Theresa Welch Fossum
Jane Cho
Curtis W. Dewey
Kei Hayashi
Janice Lynne Huntingford
Catriona M. MacPhail
Jane E. Quandt
MaryAnn G. Radlinsky
Kurt S. Schulz
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SMALL ANIMAL SURGERY

Fifth Edition



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Theresa Welch Fossum
DVM, MS, PhD, Diplomate ACVS
Vice President of Research and Strategic Initiatives
Midwestern University
Glendale, Arizona

SMALL ANIMAL SURGERY

Fifth Edition



Medical Illustrator
Laura Pardi Duprey

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1600 John F. Kennedy Blvd.
Ste 1800
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This edition is dedicated to all students of veterinary medicine and to veterinary practitioners across the globe. Special thanks and recognition go to the authors of previous editions, including Dr. Gwendolyn Carroll, Dr. Cheryl Hedlund, Dr. Caroline Horn, Dr. Donald Hulse, Dr. Ann Johnson, and Dr. Howard Seim. Their expertise and hard work were fundamental in making this textbook the fundamental resource that it has become.

T.W.F.

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ABOUT THE AUTHORS

EDITOR-IN-CHIEF



Theresa W. Fossum, DVM, MS, PhD; Diplomate ACVS

Theresa (Terry) W. Fossum is a Professor of Surgery and holds the Tom and Joan Read Chair in Veterinary Surgery at Texas A&M University (TAMU). She is a world-renowned surgeon and serves as the Director for Cardiothoracic Surgery and Biomedical Devices in the Michael E. DeBakey Institute at TAMU. She was recently appointed Director of the new Texas A&M Institute for Preclinical Studies, a $60 million venture at TAMU, which will promote public-private partnerships to speed the development of medical devices and drugs. Dr. Fossum's leadership was integral in conceptualizing and funding this Institute.

Dr. Fossum graduated from Washington State University College of Veterinary Medicine in 1982. After completing an internship at Santa Cruz Veterinary Hospital the following year, she went on to do a surgery residency and master's degree at the Ohio State University. She received board certification in Surgery (ACVS) in 1987 and joined the faculty at TAMU later that year. In 1992, she completed a PhD in veterinary microbiology. Dr. Fossum's main interests are diseases of the respiratory and cardiovascular system, including cardiopulmonary bypass procedures. Dr. Fossum has authored numerous articles on chylothorax and other respiratory and surgical diseases. She is the principal investigator of numerous research projects, including evaluation of the DeBakey Ventricular Assist Device in a calf model, evaluation of angiogenic factors for the treatment of cardiac ischemia, and vascular adaptations to hypertension. Dr. Fossum sits on the Board of Directors of the National Space Biomedical Research Institute and is the Chairman of the Board of Governors for the Foundation for Biomedical Research. Dr. Fossum is a previous recipient of the Wiley Distinguished Professor of Veterinary Medicine Award and the Carl J. Norden Distinguished Teacher Award at TAMU. In 2004, she received the Texas Society for Biomedical Research Award in recognition and appreciation for her many years of dedicated service to the science, research, and medical communities in Texas. Dr. Fossum, along with Dr. Michael E. DeBakey, founded the CARE Foundation in 2004 to provide an infrastructure for veterinary medicine to perform research using animals with spontaneous disease. She serves as president of the organization.

EDITORS



Dr. Jane Cho, DVM, DACVO

Originally from New York, Dr. Cho received her BA from the University of Pennsylvania and her DVM from Cornell University. She did her internship at the Animal Medical Center in New York City and returned to Cornell for her residency in comparative ophthalmology. After having served on the American College of Veterinary Ophthalmologists (ACVO) Exam Committee as well as on the Editorial Board for the journal *Veterinary Ophthalmology*, she was elected to the ACVO Board of Regents in 2013, currently serves as Vice President of the ACVO, and is slated to become its President in 2017. She has worked in private ophthalmology practice in New Jersey and Arkansas and is co-founder of Veterinary Eye Specialists, a private ophthalmology practice in Thornwood, NY.



Curtis W. Dewey, DVM, MS, CVA, CVCH, DACVS, DACVIM (Neurology)

Dr. Dewey was a faculty neurologist at Texas A&M University (1995–2001) and a staff neurologist at Long Island Veterinary Specialists (2001–2006) prior to returning to his alma mater, Cornell University, in 2006 as Associate Professor of Neurology/Neurosurgery. He has authored or co-authored numerous peer-reviewed journal articles and many textbook chapters. He has recently published the third edition of his own comprehensive textbook, *Practical Guide to Canine and Feline Neurology*, with his co-editor, Dr. Ronaldo C. da Costa. His current textbook project is an illustrated guide to canine acupuncture, with co-author Dr. Huisheng Xie of the Chi Institute. He is a nationally and internationally recognized speaker and has served on the editorial board of a number of veterinary journals (*Veterinary Surgery, Journal of the American Animal Hospital Association, Compendium on Continuing Education for the Practicing Veterinarian*). He has also served as an ad hoc reviewer for many other journals. He has served on the American College of Veterinary Internal Medicine (ACVIM) (Neurology) Residency Training Committee (2005–2008; committee chair 2007–2008) and the ACVIM Taskforce on Neurosurgical Training of Neurology Residents (2004–2010; committee chair 2007–2010). He consults regularly with Long Island Veterinary Specialists and Veterinary Specialists and Emergency Service of Rochester. He has been a member of the Board of Directors of the New York Veterinary Foundation since 2008. Dr. Dewey is a member of the AVMA and Veterinary Emergency and Critical Care Society. His main areas of research include seizure control and surgical management of congenital brain disorders. Dr. Dewey was the recipient of the 2014 Hills American College of Veterinary Emergency and Critical Care Jack Mara Scientific Achievement Award.

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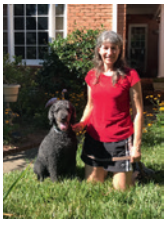
Kei Hayashi, DVM, PhD, Diplomate ACVS
Dr. Hayashi graduated from the University of Tokyo with BVMS/DVM/PhD degrees (1986–1997), and then obtained MS and PhD degrees at the University of Wisconsin (1997). He completed a small animal surgery residency at the University of Wisconsin (2003) and became a Diplomate of the ACVS. He served as an assistant/associate professor of small animal orthopedic surgery at Michigan State University (2003–2005) and University of California–Davis (2005–2012). He is currently an Associate Professor, Department of Orthopedic Surgery, at the College of Veterinary Medicine at Cornell University. Dr. Hayashi's research focus is in the pathology of ligament/tendon injury and wound healing, evaluation of total joint replacement systems, molecular profiling of osteoarthritis, and comparative orthopedics and sports medicine. His clinical interests are in arthroscopy, total joint arthroplasty, biological approach to joint surgery, minimally invasive fracture treatment, and application of novel research discoveries to clinical patients.



Janice Lynne Huntingford, DVM, DACVSMR, CVA, CVPP, CCRT, CAVCA
Dr. Huntingford is a 1984 graduate of the Ontario Veterinary College, University of Guelph, in Guelph, Ontario. She is certified in chiropractic, acupuncture, rehabilitation, and pain management. She is the owner and medical director of the Essex Animal Hospital in Essex, Ontario. In 2015 she became a Diplomate of the American College of Veterinary Sports Medicine and Rehabilitation, a consultant for the Veterinary Information Network Rehab/Sports Medicine/Chronic Pain board, and a survivor of sled dog research at the Yukon Quest in –40°C temperatures. Dr. Huntingford has lectured nationally and internationally on rehabilitation and pain management to veterinarians and veterinary technicians. She has co-authored several textbook chapters on rehabilitation and published a number of peer-reviewed manuscripts on sporting dog nutrition and exercise physiology. When not practicing she enjoys spending time on her farm/winery with her chef husband, Harold, a menagerie of dogs, cats, and horses, and even a few adult children!



Catriona M. MacPhail, DVM, PhD, Diplomate ACVS
Dr. MacPhail received her undergraduate degree from Rice University and her veterinary degree from Texas A&M University. Dr. MacPhail completed her rotating internship, surgical residency, soft tissue/surgical oncology fellowship, and PhD all at Colorado State University (CSU). She is a Diplomate of the ACVS as well as an ACVS Founding Fellow in Surgical Oncology. Dr. MacPhail has been on faculty at CSU since 2004 and became the Small Animal Chief Medical Officer for CSU in 2014. Dr. MacPhail is a frequent speaker at national and international meetings and has authored numerous journal articles and book chapters. Dr. MacPhail is the surgical laboratories coordinator for the third-year veterinary students at CSU and is an active contributor to the overall curriculum. Her primary clinical and research interests include upper and lower respiratory surgery, gastrointestinal and urinary surgery, wound reconstruction, and minimally invasive surgery. She is an active member of ACVS, Society of Veterinary Soft Tissue Surgery, and Veterinary Society of Surgical Oncology.



Jane E. Quandt, DVM, MS, DACVAA, DACVECC
Dr. Quandt graduated with a DVM from Iowa State University in 1987. Her anesthesia residency was at the University of Minnesota, and she obtained her master's degree in anesthesia in 1991. She became a diplomate of the American College of Veterinary Anesthesia and Analgesia in 1993 and was on faculty at the University of Georgia College of Veterinary Medicine for 8 years. Feeling the need to improve her ability to manage the critical and emergency patient, she completed an emergency critical care residency at the University of California–Davis and University of Minnesota. Dr. Quandt became a diplomate of the American College of Veterinary Emergency & Critical Care, small animal, in 2007, and was a faculty member at the University of Minnesota College of Veterinary Medicine for 10 years, practicing anesthesia and emergency and critical care. She rejoined the University of Georgia College of Veterinary Medicine in 2011 as a tenured associate professor in anesthesia.

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MaryAnn G. Radlinsky, DVM, MS, DACVS
Dr. Radlinsky was an academic surgeon for approximately 12 years at Kansas State University and the University of Georgia. She did her internship and residency at Texas A&M University and initially worked in private practice prior to her academic career. She published many articles in the area of minimally invasive surgery and recently became a founding fellow in the area. She has a particular interest in minimally invasive treatment of chylothorax and other diseases of the thorax. She has lectured across the United States, Europe, Asia, and South America on various topics related to soft tissue surgery. She has been an active member of the Veterinary Endoscopy Society and has been past secretary and president. She is currently a Founding Fellow, Minimally Invasive Surgery (Soft Tissue Surgery) and General Surgeon at VetMed in Phoenix, Arizona.



Kurt S. Schulz, DVM, MS, Diplomate ACVS
Dr. Schulz served on the veterinary faculty of Texas A&M University and was an associate professor at the University of California for 9 years, where he served as Chief of Small Animal Surgery from 1999 to 2003. He currently practices at Peak Veterinary Referral Center in Williston, Vermont. He has published over 50 peer-reviewed articles on veterinary surgical research and continues to teach arthroscopy and advanced orthopedic surgical techniques nationally and internationally. His other books include *Small Animal Arthroscopy* and the *Pet Lover's Guide to Canine Arthritis and Joint Problems*. He is an active member of the Veterinary Orthopedic Society, VA3, and the ACVS.



Michael D. Willard, DVM, MS, Diplomate ACVIM
Dr. Willard is an internist with a special emphasis on gastroenterology, endoscopy, pancreatology, and hepatology. Dr. Willard has received several awards for teaching excellence since 1987, among them the 1994 National Norden Award. In addition, he has numerous clinical presentations and has conducted some research in gastroenteric problems. Dr. Willard is a past secretary of the specialty of Internal Medicine and a past president of the Comparative Gastroenterology Society. He serves as a reviewer for several veterinary journals. He has contributed numerous journal articles and several monographs and book chapters. He is currently a professor in the Department of Small Animal Medicine and Surgery at the College of Veterinary Medicine at Texas A&M University.



Audrey Yu-Speight, DVM, MS, Diplomate ACVO
Dr. Yu-Speight earned her BS and MS at Stanford University and worked as a chemical engineer in the biotech industry for 4 years before attending veterinary school at Texas A&M University. She completed her internship and residency at Cornell University and served on the faculty. She has worked in private ophthalmology practice since 2003 and founded Veterinary Eye Center in Austin, Texas, in 2006. She consults on Veterinary Information Network and has worked on veterinary ophthalmology committees (Credentials, Grants, Maintenance of Certification). In 2016, she was awarded the annual Clinical Referral and Consultation Award from the Texas Academy of Veterinary Practice, an affiliate of the Texas Veterinary Medical Association.

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PREFACE

This fifth edition of *Small Animal Surgery* (*SAS*) will quickly become your daily go-to surgery reference. We hope that busy practitioners and veterinary students alike will find it to be a practical, easy-to-use, and highly valuable resource. If you have perused previous editions of *SAS*, you will note that this edition has undergone a few significant changes. While we have put a substantial effort into providing videos as an important component to *SAS*, as detailed under the Expert Consult section below, we have taken care to maintain the well-loved and highly appreciated aspects of previous editions, including (1) a limited number of contributors, (2) an excellent art program, and (3) a consistent format that varies minimally between chapters. Because of the numerous online attributes of this edition, it is available as a print book as well as on the accompanying Expert Consult website. Thus, while providing a practical, traditional textbook, we also offer a dynamic way of learning. We are extremely proud of this fifth edition and think that it is our best yet. We hope you agree.

EXPERT CONSULT

Expert Consult is the platform for the online version of the fifth edition of *Small Animal Surgery.* The website offers online access to the complete book, plus videos and some additional features such as Aftercare Instruction sheets and content on some Rarely Performed Procedures. The website contains the complete text, with full search capabilities, as well as references that link to PubMed. See the inside front cover of the book for instructions on accessing the Expert Consult site.

In the Expert Consult version of this edition, we have included numerous videos that should assist with the understanding of surgical diseases, help you with making a diagnosis, and demonstrate the specific techniques for the surgical procedures that are described. The complexity of the videos ranges from the basics of opening a peel pouch and demonstration of a gait abnormality to the more intricate manipulations required during a surgical procedure. Recognizing the busy nature of veterinary practices and the need for concise, specific information, we strived to make these videos short and succinct. Thus the disease or procedure being demonstrated typically does not have voiceover or explanatory title cards. We will continue to add new videos throughout the life of this edition and welcome approved videos from our colleagues. *To provide a video for review and possible inclusion, please contact Dr. Fossum at terry.fossum@gmail.com.*

In addition to a fully searchable text, Expert Consult offers several user-friendly tools that enhance the user experience. You will find Rarely Performed Procedures as well as Aftercare Instruction templates. To maintain a reasonably sized textbook, some of the less commonly performed procedures detailed in previous editions were removed from the print book and are presented in a fully searchable format (Rarely Performed Procedures). These procedures may, in some cases, be a historical review of procedures that are no longer used in veterinary medicine, or they may be the intervention of choice for diseases or conditions where surgical intervention is uncommon. The Aftercare Instruction templates are meant to be a time-saving tool to assist veterinarians and their staff in providing customizable, detailed discharge instructions for their clients.

UPDATES AND AUTHORS

Throughout the text, you will find that we have updated procedures with new information and, in many cases, we have added descriptions of entirely new procedures that were either rarely used or not used when the previous editions were published. It was our goal to make sure that we produced the most state-of-the-art book possible. Although it has always been our desire to provide clinically useful information rather than a monologue of research on a given topic, we have addressed the need for a review of recent research by providing up-to-date references. To make room for newer references, we have removed most references that were more than 7 years old from this edition, unless (in rare circumstances) the reference was thought to be a "classic." On Expert Consult, the references are directly linked to the original article on PubMed for easy access by the reader.

As with previous editions, the bulk of this book was written by six surgeons (Drs. Curtis Dewey, Theresa Fossum, Kei Hayashi, Catriona MacPhail, MaryAnn Radlinksy, and Kurt Schulz) and an internist (Dr. Michael Willard). However, there are also major contributions from an anesthesiologist (Dr. Jane Quandt), a sports medicine expert (Dr. Janice Huntingford), and two ophthalmologists (Drs. Audrey Yu-Speight and Jane Cho). Part One, *General Surgical Principles,* has contributions from most of the team. To provide additional information on the increasingly important subjects of anesthesia and pain management, we have divided these topics into two chapters: "Principles of Anesthesia and Anesthetics" (Dr. Quandt) and "Pain Management" (Dr. Quandt and Dr. Dewey). "Pain Management" includes a new section on acupuncture. In addition, throughout the text readers will find extensive and comprehensive tables on anesthetic management of animals with particular diseases or conditions. These tables, originally provided by Dr. Caroline Horn and updated for this edition, provide detailed information on the preoperative, intraoperative, and postoperative management of surgical cases in an easy-to-read format, complete with drug doses. Practitioners will find these tables to be a very useful, quick reference. "Fundamentals of Physical Rehabilitation" (Chapter 11) has been extensively revised by Dr. Janice Huntingford, who is a Diplomate of Canine Rehabilitation and Sports Medicine. The topic of regenerative medicine has been updated by Dr. Schulz and is now included in the chapter "Principles of Orthopedic Surgery and Regenerative Medicine" (Chapter 31). Chapter 14, updated by Drs. Willard and Schulz, includes principles of minimally invasive surgery (MIS) and a practical guide to imaging of the surgical patient. Techniques of endoscopy and MIS for specific conditions and diseases can be found throughout the text.

Drs. Catriona MacPhail and MaryAnn Radlinsky were responsible for much of Part Two, *Soft Tissue Surgery.* Dr. Kurt Schulz, and a new addition to the *SAS* team, Dr. Kei Hayashi, provided the material encompassed in Part Three, *Orthopedics.* Dr. Curtis Dewey updated the neurology chapters in Part Four, *Neurosurgery*. Drs. Audrey Yu-Speight and Jane Cho, both board-certified ophthalmologists, extensively updated Chapter 16, "Surgery of the Eye." Lastly, Dr. Mike Willard reviewed and provided his perspective on many of the chapters so that we can provide the most up-to-date information on the medical management of surgical disease.

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CONTENT

We have added a significant amount of new information to the text and have maintained the way we reorganized the coverage of minimally invasive procedures to make the text more user friendly. As with the fourth edition, we have a separate chapter on minimally invasive surgery that covers the basics of endoscopy, thoracoscopy, laparoscopy, and arthroscopy. However, we have also included the descriptions of minimally invasive procedures directly within the specific chapters where they are applicable. For example, when you read about surgical pericardiectomy, a description of thoracoscopic pericardiectomy directly follows.

As with previous editions, we believe that to be successful, surgeons must have detailed knowledge of the important issues regarding diagnosis, an awareness of potential differential diagnoses, and a thorough appreciation of preoperative concerns relative to the animal's disease or condition. These sections are each outlined in the text. In addition, anesthetic concerns, surgical anatomy, wound healing, postoperative concerns, and potential complications are detailed. The surgical technique itself is described in detail, which provides the reader with a comprehensive and thorough description of each procedure. The surgical procedure is discussed in *blue italicized type* to make it easy to distinguish from the rest of the text. While we did debate the practicality of including advanced procedures in this textbook, we decided that practitioners would benefit greatly from a better understanding of the procedures, even though they likely would choose to refer these cases to a specialist. With this in mind, we have marked some procedures as "advanced" to forewarn readers of the difficulty of the procedure. While the difficulty of any procedure lies primarily with the experience of the surgeon, the procedures denoted with the special icon AP are ones that the authors find particularly challenging and thus would recommend that they be performed by someone with advanced training or special expertise in that area.

GENERAL FORMAT

This book is composed of 44 chapters and is organized into four parts. The 14 chapters of Part One, *General Surgical Principles,* were written with veterinary medical students and practitioners in mind. The information contained in these chapters is what we teach our students in their introductory surgery courses. Found within these chapters is detailed information on the basics of sterile technique, surgical instrumentation, suturing, preoperative care, and rational antibiotic use. We have emphasized the use of scrubless and/or waterless surgeon prep solutions and new patient prep solutions in the chapters on patient and surgeon preparation. Although these solutions and techniques are not particularly new, studies suggest that they are not well known or frequently used by practicing veterinarians. Chapter 10 contains information on postoperative care, including new consideration on the nutritional management of surgical patients. Because nutrition affects many body systems and is an important adjunct to case management, we have included detailed information on techniques for hyperalimentation in this chapter. Chapter 11 details the basics of physical rehabilitation in veterinary patients. We believe that physical rehabilitation is underutilized in many veterinary practices. In addition to this basic chapter, specific recommendations for physical rehabilitation can be found throughout the orthopedic and neurologic chapters. Chapter 12 provides basic information on anesthesia techniques and drugs with important information for practitioners, and Chapter 13 details pain management. As previously noted, we have attempted to give an overview of anesthetic and analgesic techniques while providing detailed protocols for the management of specific diseases in the various chapters in Parts Two, Three, and Four. These anesthetic tables have been expanded in this edition to include preoperative and postoperative management suggestions for these cases and include recommendations for analgesia as well as suggestions for monitoring these cases.

Parts Two, Three, and Four contain information on soft tissue surgery, orthopedic surgery, and neurosurgery, respectively. These chapters are divided into a section detailing general principles and a section on specific diseases. The *General Principles and Techniques* portion begins with definitions of procedures and terms relevant to the organ system detailed. Next are sections detailing information on preoperative concerns and anesthetic considerations. This is followed by a discussion on antibiotic use (including recommendations for antibiotic prophylaxis) and a brief description of pertinent surgical anatomy. Anatomy is too often neglected in surgical textbooks or, because of formatting, is not well correlated with the techniques in a given chapter. We have circumvented this problem by including it as a separate and consistent heading under *General Principles and Techniques.* Surgical techniques that are broadly applicable to a number of diseases are also detailed in this section. However, if a surgical procedure is specific to a particular disease, the description of the technique is found instead with the specific disease description. Brief discussions on healing of the specific organ or tissue as well as suture material and special instruments follow the surgical techniques descriptions. The final headings in the *General Principles and Techniques* section are *Postoperative Care and Assessment, Complications,* and *Special Age Considerations.*

The *Specific Diseases* portion of each chapter begins with definitions and, when relevant, synonyms for the disease or techniques. Next, general considerations and clinically relevant pathophysiology are detailed. This information is meant to provide practical material for case management, rather than to serve as a supplemental text for pathophysiology. The discussions of diagnoses are detailed and include information on signalment and history, physical examination findings, diagnostic imaging, and pertinent laboratory abnormalities. Sections on differential diagnoses and medical management of affected animals are consistently provided. These are followed by a detailed description of the relevant surgical techniques. We have attempted to detail most commonly used techniques, although we may have noted our preference for a particular method. Information on positioning patients for a given procedure is provided as a separate heading. The remainder of the *Specific Diseases* section deals with postoperative care of the surgical patient, potential complications, and prognosis.

Although some of the procedures in this text are best performed only by surgeons with advanced training, we believe that practitioners referring these cases should be provided with adequate information regarding the surgery to talk with their clients in a detailed and knowledgeable fashion; thus we have elected to include some advanced procedures in this

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textbook as well as procedures more commonly performed in general practice. As with all surgical procedures, the surgeon must exercise judgment as to what their qualifications and experience are in reference to performing a particular procedure.

CHAPTER FORMAT

I. General principles and techniques
   A. Definitions
   B. Preoperative management
   C. Anesthesia
   D. Antibiotics
   E. Surgical anatomy
   F. Surgical technique
   G. Wound healing
   H. Suture materials and special instruments
   I. Postoperative care and assessment
   J. Complications
   K. Special age considerations
II. Specific diseases
   A. Definitions
   B. General considerations and clinically relevant pathophysiology
   C. Diagnosis
   D. Differential diagnosis
   E. Medical management
   F. Surgical treatment
   G. Surgical technique
   H. Suture materials and special instruments
   I. Postoperative care and assessment
   J. Complications
   K. Prognosis

ANESTHESIA PROTOCOLS

For quick reference, recommendations for anesthetizing animals with a particular disease or disorder are found in the *Specific Diseases* section of each chapter. Dr. Jane Quandt served as our anesthesia consultant for this revision. The anesthesia protocol tables include recommended drug dosages and should be extremely useful to busy practitioners. Although we recognize that many veterinarians have established protocols that they prefer and with which they are comfortable, the protocols provided in this book have proved to be a handy resource for many practitioners.

SPECIAL FEATURES

As in past editions, we have included hundreds of tables and boxes that summarize key clinical information. It has always been our intent to make this book as user friendly as possible. For this reason, we have maintained the NOTE boxes interspersed throughout most chapters, which highlight important concerns, key concepts, and precautions. To facilitate ease of access and to promote comprehension, we have created unique tables and boxes with similar types of information. These tables and boxes are color coded and are marked with distinct icons for easy content identification, as follows:

 Calculations

 General Treatment

 Analgesics/Postoperative Care/Pain Management

 Diagnosis/Differential Diagnosis

 Key Points

 Anesthetics/Sedation

 Clinical Signs

 Antibiotics

 Complications

 Causes/Etiology

 Classification of Disease

ART PROGRAM

We were extremely privileged to work with our original illustrator, Laura Pardi Duprey, on this fifth edition. In addition to being an incredibly skilled artist, she has an extremely broad-based and detailed knowledge of anatomy. You will find the illustrations in this text exceptionally clear and accurate. We have added many new images, and revised many more, in our attempt to make this book among the best illustrated textbooks in veterinary medicine.

We have added new artwork to existing procedures, and you will find that there are more color illustrations in this edition than in the last. In fact, nearly the majority of the illustrations in this edition are in full color.

INDEX

The index of *Small Animal Surgery* is thorough and exhaustive. We have avoided cross-referencing readers to separate entries in the index. Rather, we have opted to duplicate page sources each time a topic is listed because we believe that this is the most useful format for practitioners.

SUMMARY

Regardless of where you practice or when you graduated, it is the hope of the entire *Small Animal Surgery* team that you find this edition to be an extremely user-friendly, informative text that assists you in diagnosing and treating surgical conditions. We welcome your feedback and suggestions on what you appreciate about this textbook and what we might do to improve future editions. Please feel free to send your comments to terry.fossum@gmail.com.

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ACKNOWLEDGMENTS

AUTHOR ACKNOWLEDGMENTS

I would like to once again thank my past and current authors on this book. I am blessed to have been able to work with some of the best and most dedicated veterinarians in the field. This edition was in many ways the most difficult edition that we have undertaken. After the print text was submitted, we began months of working on videos. Some videos had already been in authors' possession, but many others were specifically shot and edited for this edition. The time and effort that went into creating these videos were significant, and I thank those who made providing them a priority.

PERSONAL ACKNOWLEDGMENTS

A textbook of this nature takes the input and hard work of a great number of people to ensure that it is a quality reference. Special thanks to Rae Robertson, Content Development Specialist; Jennifer Flynn-Briggs, Senior Content Strategist; and all the others at Elsevier who worked on this project. We thank them for their enthusiasm, words of encouragement, and vision, and most of all for their belief in this book. Without them, this edition would not have been possible.

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1



Principles of Surgical Asepsis

Infection remains one of the most potentially devastating and challenging complications of surgery. Infection in a veterinary surgical practice may occur during surgery or any time during hospitalization. It is incumbent upon the veterinary practitioner and staff to ensure that all possible measures are taken to reduce the risk of iatrogenic infection.

ASEPSIS VERSUS STERILITY

The terms *aseptic technique* and *sterile technique* are used interchangeably by some authors (Box 1.1), but in strict terms, *asepsis* is defined as the absence of microorganisms that cause disease, whereas *sterile* is defined as being free of all living microorganisms. Therefore techniques to maintain sterility are more appropriate for the operating room (OR) setting, but aseptic principles should be applied to the entire hospital. Outside the OR, these techniques have been referred to as *medical asepsis* and are designed to protect both the patient and hospital staff.

The complete absence of microorganisms cannot be achieved in a hospital environment, but use of aseptic techniques substantially aids in the control of pathogens and decreases the risk of infection for patients and staff. When specific procedures are discussed in this text, *aseptic technique* has been referred to as *clean technique. Sterile technique* (see Box 1.1) applies to work performed in a sterile field. The higher level of protection in a sterile field is critical because the natural defenses of the patient are breached by surgical incision, puncture, or introduction of instruments into the vascular system. Studies in human surgical practice have attempted to determine when sterile technique versus aseptic technique is necessary for certain minor procedures. For example, arthrocentesis (see p. 1136) performed under sterile technique might require the use of sterile gloves, a sterile patient preparation kit, and a small drape, whereas the same procedure performed using clean or aseptic technique would require only nonsterile gloves and an alcohol wipe. The differences in time and cost involved for sterile technique versus clean technique can be substantial.

TRANSMISSION OF MICROORGANISMS

Numerous sources of microorganisms are found in veterinary hospitals, but as in human hospitals, the hospital staff is the most likely means of transmission. Other sources of transmission include contaminated instruments and the environment. The specific means of transmission from staff to patient include airborne, droplet, and contact. Airborne and droplet types of transmission of microorganisms are common in human hospitals but less common in veterinary hospitals because of the low incidence of reverse zoonosis (transmission of disease from human to nonhuman animals). Contact, in which pathogenic microorganisms are transferred to the patient from another patient or from an environmental source, is the most common method of microorganism transmission in veterinary hospitals. During surgery, contact may also be responsible for transmission of the patient's own microorganisms from the skin or nasopharynx to the surgical wound.

Sources of Contamination

Sources of contamination may be divided into animal sources and inanimate sources.

Animal Sources

Sources of microorganisms from patients include the skin, hair, nasopharynx, and other orifices such as the vulva or anus.

Inanimate Sources

The primary sources of microorganisms from inanimate objects are fomites and air. A *fomite* is any inanimate object capable of carrying infectious organisms. Fomites may include the hospital structure (walls, floors, etc.), furniture, equipment, implants, and cleaning equipment. Protocols must be established to maintain an aseptic environment by having scheduled cleansing of potential fomites and regular monitoring for possible hospital-borne infections (see Chapter 3).

Airborne Sources

One cubic foot of air contains thousands of particles, which can increase to more than 1 million particles during a lengthy surgical procedure. This increase is due to traffic into and out of the room and other air currents that develop. Particles in air are significant sources of microorganisms and may be responsible for 80% to 90% of microbial contamination of a surgical wound. Primary sources of microorganisms found in the air include the floor, the hospital staff, and the patient.

PRINCIPLES OF HOSPITAL ASEPSIS

Minimization of infection in a surgery practice involves applying principles of aseptic technique throughout the hospital. Goals are to minimize sources of contamination and to block transmission

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ABSTRACT

Infection remains one of the most potentially devastating and challenging complications of surgery. Surgeons must understand the concepts of asepsis and sterility, how microorganisms are transmitted in the hospital, and how to prevent infection.

KEYWORDS

Asepsis
Sterility
Disinfection
Aseptic Technique
Sterile Technique
Contamination

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BOX 1.1 Glossary

Antisepsis Prevention of sepsis by exclusion, destruction, or inhibition of growth or multiplication of microorganisms from body tissues and fluids.

Antiseptics Inorganic chemical compounds that combat sepsis by inhibiting growth of microorganisms without necessarily killing them. Used primarily on skin to stop the growth of resident flora.

Asepsis Absence of microorganisms that cause disease.

Aseptic technique Method to prevent contamination by microorganisms.

Barrier A material used to reduce or inhibit the migration or transmission of microorganisms in the environment: personnel attire and gowns, furniture and patient drapes, equipment and supply packaging, and ventilating filters.

Contaminated Carrying or infected by microorganisms.

Cross-contamination Transmission of microorganisms from patient to patient or from inanimate object to patient.

Decontamination Cleaning and disinfecting or sterilizing processes carried out to make contaminated items safe to handle.

Disinfection Chemical or mechanical (friction) destruction of pathogens.

High-level disinfection Used for equipment/instruments that contact the skin or mucous membranes but do not penetrate the body (semicritical items; see Table 1.2) and where sterility is not required.

High-level sterilization Required when a surgical instrument or medical device enters beneath the skin or mucous membranes (critical items; see Table 1.2).

Irreducible minimum Microbial burden cannot get any lower; item is sterile to its highest degree.

Low-level disinfection Used for noncritical items (see Table 1.2) where the instrument or device contacts skin but does not penetrate the body or mucous membranes.

Spatial relationships Awareness of sterile, unsterile, clean, and contaminated areas, objects, and individuals and their proximity to each other.

Standard precautions Procedures followed to protect personnel from contact with the blood and body fluids of patients.

Sterile Free of living organisms.

Sterile field Area around the site of incision into tissue or the site of introduction of an instrument into a body orifice that has been prepared using sterile supplies and equipment.

Sterile technique Method by which contamination with microorganisms is prevented to maintain sterility throughout the surgical procedure.

Terminal sterilization and disinfection Procedures carried out for the destruction of pathogens at the end of the surgical procedure in the operating room after the patient has been removed.

Modified from Philips N. In: *Berry and Kohn's Operating Room Technique.* 13th ed. St. Louis: Elsevier; 2017.

of microorganisms. Some techniques that aid in reduction of the quantity of pathogenic microorganisms are listed in Box 1.2.

BOX 1.2 Techniques That Aid in Reduction of the Quantity of Pathogenic Microorganisms

- Regular hand washing by hospital staff
- Use of nonsterile or sterile gloves when handling likely sources of pathogens, including high-risk patients (e.g., patients with known infection or wounds), equipment (e.g., contaminated sponges), and hospital surfaces
- Cleaning or disposal of equipment between patients
- Containment of contaminated supplies and equipment
- Proper storage of equipment
- Regular equipment cleaning protocols
- Proper handling of soiled laundry
- Scheduled cleaning of hospital surfaces
- Proper maintenance of hospital heating, ventilation, and air conditioning systems
- Minimization of unnecessary traffic
- Isolation of patients with known pathogenic microorganisms

STERILE TECHNIQUE

All surgical procedures are ideally performed under sterile conditions. Sterile technique is designed to prevent the transmission of microorganisms into the body during surgery or other invasive procedures. General principles of aseptic technique should be familiar to all personnel working in and around the surgical environment (Table 1.1). These principles include the following: (1) use only sterile items within a sterile field; (2) sterile (scrubbed) personnel are gowned and gloved; (3) sterile personnel operate within a sterile field (sterile personnel touch only sterile items or areas, unsterile personnel touch only unsterile items or areas); (4) sterile drapes are used to create a sterile field; (5) all items used in a sterile field must be sterile; (6) all items introduced onto a sterile field should be opened, dispensed, and transferred by methods that maintain sterility and integrity; (7) a sterile field should be maintained and monitored constantly; and (8) surgical staff should be trained to recognize when they have broken technique and should know how to remedy the situation.

NOTE You must know what equipment and supplies are sterile and what are not sterile and keep the two apart. If contamination occurs, remedy the contamination immediately.

STERILIZATION AND DISINFECTION

Sterilization is the destruction of all microorganisms (bacteria, viruses, spores) on an item. It usually refers to objects (e.g., instruments, drapes, catheters, needles) that come in contact with tissue or enter the vascular system. Disinfection is the destruction of most pathogenic microorganisms on inanimate (nonliving) objects, whereas antisepsis is the destruction of most pathogenic microorganisms on animate (living) objects. Neither disinfection nor sterilization claims to kill or inactivate all microorganisms. The level of disinfection or sterilization needed depends on what the object (instrument or medical device) is being used for. Given that all invasive procedures involve contact with a patient's sterile tissue or mucous membranes, it is essential that appropriate techniques for disinfection and sterilization be utilized by veterinary surgeons. High-level sterilization is required when a surgical instrument or medical device enters beneath the skin or mucous membranes *(critical items;* Table 1.2). These instruments and implants must be handled using sterile technique. Equipment that contacts the skin or mucous membranes only for surgical purposes without penetrating the body must be cleaned and disinfected to reduce the level of microorganisms, but sterility is not required. High-level disinfection is generally used for such items (*semicritical items;* see Table 1.2); terminal sterilization is common with some of these instruments, but sterility is not maintained during the procedure. Low-level disinfection (*for noncritical items;* see Table 1.2) is needed when the instrument or device contacts skin but does not penetrate the body or mucous membranes.[1] Antiseptics are used to kill microorganisms during patient skin preparation and surgical scrubbing (see Chapters 5 and 6); however, the skin is not sterilized. Cleaning is generally restricted in meaning to the physical removal of surface contaminants, usually with detergents or soap and water, ultrasound, or other methods. Although cleaning does remove soils and bacteria, it does not kill or inactivate viruses or bacteria.

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TABLE 1.1 General Rules of Aseptic Technique

Rule	Reason
Surgical team members remain within the sterile area.	Movement out of the sterile area may encourage cross-contamination.
Talking is kept to a minimum.	Talking releases moisture droplets laden with bacteria.
Movement in the operating room (OR) by all personnel is kept to a minimum; only necessary personnel should enter the OR.	Movement in the OR may encourage turbulent airflow, resulting in cross-contamination.
Nonscrubbed personnel do not reach over sterile fields.	Dust, lint, or other vehicles of bacterial contamination may fall on the sterile field.
Scrubbed team members face each other and the sterile field at all times.	A team member's back is not considered sterile even if wearing a wraparound gown.
Equipment used during surgery must be sterilized.	Unsterile instruments may be a source of cross-contamination.
Scrubbed personnel handle only sterile items; nonscrubbed personnel handle only nonsterile items.	Nonscrubbed personnel and nonsterile items may be sources of cross-contamination.
If the sterility of an item is questioned, it is considered contaminated.	Nonsterile, contaminated equipment may be a source of cross-contamination.
Sterile tables are sterile only at table height.	Items hanging over the table edge are considered nonsterile because they are out of the surgeon's vision.
Gowns are sterile from mid-chest to waist and from gloved hand to 2 inches above the elbow.	The back of the gown is not considered sterile even if it is a wraparound gown.
Drapes covering instrument tables or the patient should be moisture proof.	Moisture carries bacteria from a nonsterile surface to a sterile surface (strike-through contamination).
If a sterile object touches the sealing edge of the pouch that holds it during opening, it is considered contaminated.	Once opened, sealed edges of pouches are not sterile.
Sterile items within a damaged or wet wrapper are considered contaminated.	Contamination can occur from perforated wrappers or from strike-through from moisture transport.
Hands may not be folded into the axillary region; rather, they are clasped in front of the body above the waist.	The axillary region of the gown is not considered sterile.
If the surgical team begins the surgery seated, they should remain seated until the surgery has been completed.	The surgical field is sterile only from table height to the chest; movement from sitting to standing during surgery may promote cross-contamination.

TABLE 1.2 Levels of Sterility and Disinfection

Level of Sterility	Definition	Requirements for Sterilization, Cleaning, and Handling	Examples
Critical	Equipment or implants entering sterile tissue of the vascular system	Requires sterilization Handle with sterile technique	Implants Catheters Surgical instruments Laparoscopes Suture material Biopsy forceps Arthroscopes[a]
Semicritical	Equipment that does not penetrate the body; they contact the skin or mucous membranes only	Requires high-level disinfection Must be cleaned before disinfection Sterility generally not maintained during procedure	Vaginoscopes Colonoscopes Flexible endoscopes[a] Some dental instruments Endotracheal tubes
Noncritical	Instruments that contact the mucous membranes or intact skin not directly associated with surgery	Terminal cleaning recommended between patients Disinfection appropriate in some cases	Laryngoscopes Ultrasound probes not used in the body Blood pressure cuffs ECG leads Pulse oximeters Stethoscopes

[a]The incidence of infection associated with endoscope use is low, but infections have occurred; thus at a minimum they should be subjected to high-level disinfection after each use.
ECG, Electrocardiogram.

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REFERENCE

1. Rutala WA, Weber DJ. Disinfection, sterilization and antisepsis: an overview. *Am J Infect Control*. 2016;44:e1–e6.

2

Care and Handling of Surgical Equipment and Supplies



Regardless of the sterilization technique used, instruments and linens (e.g., towels, gowns, drapes) must be cleaned of gross contamination. Instruments should be cleaned manually or with ultrasonic cleaning equipment and appropriate disinfectants as soon as possible after surgery, and linens should be laundered. Depending on use, surgical instruments and supplies must then be either sterilized or disinfected (see Chapter 1 and Table 1.2).

CLEANING

Cleaning should be done as soon as possible after use of the instrument to prevent drying and caking of blood and other debris on the item. It is a vital step as it removes organic material and inorganic salts that interfere with sterilization. Water with detergents or enzymatic cleaners are used, and it may be done mechanically (e.g., ultrasonic cleaner, dishwasher, utensil washer/sanitizer) or manually.

DISINFECTION

High-level disinfectants destroy all microorganisms with the exception of some bacterial spores[1] and may be accomplished via liquid immersion (see later) or pasteurization. Low-level disinfection destroys vegetative bacteria, as well as some viruses and fungi, but typically does not kill spores or mycobacteria. Disinfectants include peracetic acid–hydrogen peroxide, glutaraldehyde, hydrogen peroxide, improved hydrogen peroxide (2%), ortho-phthalaldehyde (OPA), peracetic acid, ethylene oxide (EtO), heavy metal ions, or dyes. Common disinfectants, their uses, and necessary precautions are listed in Tables 2.1 and 2.2.

STERILIZATION

The reliability of any sterilization method depends on the number, type, and inherent resistance of microorganisms on the items to be sterilized and whether other materials (e.g., soil, oil) that may shield against or inactivate the sterilizing agent are present on the items. Methods to sterilize equipment or supplies include (1) high temperature, (2) low temperature, or (3) liquid immersion. Advantages and disadvantages of commonly used sterilization technologies are summarized in Table 2.2. High-temperature (steam autoclave or dry heat) sterilization may be used for heat-tolerant critical and semicritical items; however, the high temperature and moisture of a steam autoclave make it unusable for many of today's devices. Likewise, dry-heat sterilization produces temperatures that cannot be tolerated by most devices and is seldom used. Low-temperature sterilization systems (e.g., EtO gas, hydrogen peroxide gas plasma, hydrogen peroxide and ozone, hydrogen peroxide vapor; see Table 2.2) are used for heat-sensitive critical and semicritical items (see also Table 1.2).

Liquid immersion includes chemical sterilants such as glutaraldehyde, glutaraldehyde with phenol, and various concentrations of hydrogen peroxide with peracetic acid (see Tables 2.1 and 2.2).[1] Devices cannot be wrapped when using liquid sterilants, and they typically need to be rinsed with water that often is not sterile, which limits their usefulness.

Steam Sterilization

Saturated steam under pressure is a practical and dependable agent for sterilization of heat-tolerant medical supplies and packaging. Steam rapidly destroys all known microorganisms by means of coagulation and cellular protein denaturation. To ensure the destruction of all living microorganisms, the correct relationship between temperature, pressure, and exposure time is critical. The unit used to create this high-temperature, pressurized steam is called an *autoclave*. Certain types of microorganisms have greater inherent heat resistance than do other organisms. Spores of thermophilic aerobes and anaerobes are the most resistant known forms of life to moist heat. Virus particles are more vulnerable to steam sterilization than are spores.

Types of Steam Sterilizers

Gravity Displacement Sterilization

The most commonly used steam sterilizer in veterinary practice is the gravity (or "downward") displacement sterilizer (Figs. 2.1 and 2.2). This sterilizer works on the principle that air is heavier than steam. Supplies to be sterilized are loaded into the inner chamber. A narrow, outer jacket-type chamber surrounds the inner chamber. Pressurized steam from the narrow, outer chamber enters the inner chamber and surrounds the supplies. Air in the inner chamber is pulled downward by gravity to the floor and exits through a temperature-sensitive valve. As steam accumulates and the temperature increases, the steam-release valve closes. Because the function of this sterilizer is based on the ability of air to move to the bottom of the autoclave, careful wrapping (see p. 12) and loading of supplies are critical. Table 2.3 shows the recommended sterilization times for commonly sterilized items. See Table 2.4 for minimum time and temperature standards for various types of steam sterilizers.

Prevacuum Sterilization

The prevacuum sterilizer relies on air being actively pulled out of the inner chamber, thereby creating a vacuum. Steam is injected into the chamber to replace the air. This method of sterilization provides greater steam penetration in a shorter time than the gravity displacement sterilizer (see Table 2.4).

Immediate-Use Steam Sterilization

Immediate-use, emergency, or "flash" sterilization is performed when an unwrapped, nonsterile item must be sterilized quickly.

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ABSTRACT

Proper preparation and storage of surgical equipment and supplies are critical to minimizing the risk of surgical infections and maintaining the quality of surgical equipment. A thorough understanding of the concepts of sterilization and disinfection is critical. Surgeons and staff should be familiar with differing methods of sterilization and storage of equipment.

KEYWORDS

Disinfection
Sterilization
Surgical equipment
Surgical instruments
Surgical supplies
Shelf life
Equipment packaging

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TABLE 2.1 Common Disinfectants/Sterilants Used in Veterinary Practice

Agent	Practical Use	Disinfectant Properties	Antiseptic Properties	Mechanisms of Action	Precautions
Alcohol: isopropyl alcohol (50%–70%); ethyl alcohol (70%)	Spot cleaning; injection site preparation	Good	Very good	Protein denaturation, metabolic interruption, and cell lysis	Corrosive to stainless steel; volatile; damage to shellac mountings of lensed instruments; hardens rubber and some plastic tubing after prolonged use; irritating to tissues
Chlorine compounds: hypochlorite	Cleaning floors and countertops	Good; available as liquid (sodium hypochlorite) or solid (calcium hypochlorite)	Fair	Release of free chlorine and oxygen	Inactivated by organic material; corrosive to metal; may cause skin irritation and esophageal burns if ingested; discolors fabric
Iodine compounds: iodophors (7.5%) scrub solution	Cleaning dark-colored floors and countertops	Good	Good	Iodination and oxidation of essential molecules	Stains fabric and tissue; do not use on silicone tubing
Glutaraldehyde: 2% alkaline solution	Disinfection of lenses and delicate instruments	Good; sterilizes	None	Protein and nucleic acid alkylation	Tissue reaction; odor; rinse instruments well before using; can be highly toxic
Hydrogen peroxide	Often used as an antiseptic; stabilized form used on environmental surfaces	Good	Very good, particularly against anaerobic bacteria	Formation of free hydroxyl radicals (OH), which oxidize thiol groups in enzymes and proteins	May be irritating to skin at high concentrations; irritating to eyes
Ortho-phthalaldehyde (OPA)	Chemical sterilant similar to glutaraldehyde but not irritating to eyes and nasal passages	Good; sterilizes; fast acting	None	Aldehyde binds to the outer cell wall of the organism (similar to glutaraldehyde)	Doesn't smell but wear personal protective equipment when handling; provide good ventilation; rinse equipment thoroughly
Peracetic acid	Used in automated machines to chemically sterilize instruments	Good; effective in presence of organic material; very rapid kill; no harmful residue	None	Denatures proteins, disrupts cell wall permeability, and oxidizes sulfhydryl and sulfur bonds in proteins, enzymes, and other metabolites	Unstable; quickly loses activity, particularly when diluted; corrosive; irritating to eyes, skin, and respiratory system; can corrode copper, brass, bronze, and galvanized iron

A gravity displacement sterilizer is used for this purpose. The item is placed unwrapped in a perforated metal tray and is sterilized according to the manufacturer's time and temperature recommendations (see Table 2.4). With detachable handles, sterilized items are transported to the operating room (OR) in the metal tray. It is difficult to deliver these devices aseptically; the tray is hot, wet, and unwrapped, which means it collects dust, debris, and microorganisms more readily than dry, cool trays with biobarrier protection. This type of sterilization should be used only in emergencies when no alternative is available. Immediate-use sterilization generally is not recommended for implantable medical devices or power equipment unless specifically approved by the manufacturer. If an implant must be flashed, a "rapid read" biological spore test is used and can be read in 1 hour for a flash cycle. In flash sterilization, it is important to minimize the risk of contamination during transportation. The sterilizer should be located in the restricted area of the surgical suite or treatment site. It is advised to use rigid sterilization container systems (that are validated for use in immediate-use sterilization; Fig. 2.3) and the single-wrapper technique.

Chemical (Gas) Sterilization

Ethylene Oxide

EtO is a flammable, explosive gas that kills microorganisms by altering their normal cellular metabolism and replication through alkylation of proteins, deoxyribonucleic acid (DNA), and ribonucleic acid (RNA). Advantages and disadvantages of EtO are given in Table 2.2. Flexible endoscopes typically require special EtO caps that prevent rupture of the outer plastic layer. The time required for sterilization depends on the concentration of EtO, the humidity level, the temperature, and the volume, density, and types of materials to be sterilized. Most items are sterilized at 54.4°C (130°F) for approximately 2.5 hours; heat-sensitive items are sterilized at 37.8°C (100°F) for approximately 5 hours. The manufacturer's recommendations for EtO exposure time must be followed. Compact, tabletop units (Fig. 2.4) are available that have combinations of ventilation and purge systems (e.g., Anprolene, Anderson Products). The most common veterinary units run on 12- and 24-hour cycles and operate at room temperature.

Items should be clean and dry before EtO sterilization; moisture and organic material bond with EtO and leave a toxic residue. If an item cannot be disassembled and all surfaces cleaned, it cannot be sterilized. Items are packed and loaded loosely in the sterilizer to allow gas circulation. Complex items (e.g., power equipment) are disassembled before processing (see p. 13). Items that cannot be sterilized with EtO include acrylics, some pharmaceutical items, and solutions.

The effectiveness of EtO sterilization may be changed by lumen length, lumen diameter, inorganic salts, and organic materials.

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TABLE 2.2 Methods to Sterilize or Disinfect Surgical Instruments, Implants, and Devices

Classification of Items	Level of Germicidal Action	Type of Sterilization/Disinfection	Advantages	Disadvantages
Critical (surgical instruments, implants, cardiac catheters)	Kills all microorganisms, including spores	Sterilization • Steam (autoclave)	• Nontoxic • Easy to use • Inexpensive • Rapid acting • Penetrates packing and lumens	• Deleterious for heat-labile items • Must be used carefully to avoid burns
		• Hydrogen peroxide gas plasma	• Safe to use; no toxic residues • Fast cycle time; aeration not needed • Process temp 50°C (122°F); safe for heat- and moisture-sensitive items • Simple to operate	• Cannot process cellulose (paper), linens, or liquids • Requires synthetic (polypropylene) packaging and special container trays • Cannot process large or bulky items because chamber is small
		• Ethylene oxide	• Effective • Penetrates medical packaging • Easy to monitor and control	• Potential hazard to staff • Long aeration times required
		• Vaporized hydrogen peroxide	• Safe to use; no toxic residues • Fast cycle time; aeration not needed • Safe for heat- and moisture-sensitive items	• Cannot process large or bulky items because chamber is small • Cannot use for liquid, linens, powders, or cellulose materials • Requires synthetic (polypropylene) packaging and special container trays
Semicritical (endoscopes, anesthesia equipment)	Kills all microorganisms except high numbers of bacterial spores; high-level disinfectant	• Glutaraldehyde (Glu); ≥2.0% • Ortho-phthalaldehyde; 0.55% • Hydrogen peroxide; 7.5% • Hydrogen peroxide + peracetic acid 1.0%/0.08% • Hydrogen peroxide + peracetic acid 7.5%/0.23% • Hypochlorite (free chlorine); 650–675 ppm • Accelerated hydrogen peroxide; 2% • Peracetic acid; 0.2% • Glu + isopropanol; 3.4%/26.0% • Glu + phenol/phenate; 1.21%/1.93%	• See Table 2.1	• See Table 2.1
Noncritical (walls, floors, ECG leads)	Kills vegetative bacteria, fungi, and some viruses; low-level disinfectant	• Ethyl or isopropyl alcohol; 70%–90%	• See Table 2.1	• See Table 2.1
		• Chlorine; 100 ppm (1:500 dilution)	• See Table 2.1	• See Table 2.1
		• Phenolic; use per manufacturer recommendation	• Active ingredient in Lysol, Pine Sol • Active against bacteria and some viruses • Maintain activity in presence of organic material	• Not active against nonenveloped viruses and spores • May cause tissue irritation • May be toxic to neonates
		• Iodophor; use per manufacturer recommendation	• See Table 2.1	• See Table 2.1
		• Quaternary ammonium; use per manufacturer recommendation	• Good cleaning agents but poorly effective as antiseptics • Less effective against gram-negative bacteria than gram-positive bacteria	• Not effective against nonenveloped viruses, fungi and bacterial spores

Modified from Rutala WA, Weber DJ. Disinfection and sterilization in healthcare facilities. In: The Society for Healthcare Epidemiology of America; Lautenbach E, Woeltje KF, Malani PN, eds. *Practical Healthcare Epidemiology*. 3rd ed. Chicago: University of Chicago Press; 2010:61–80.

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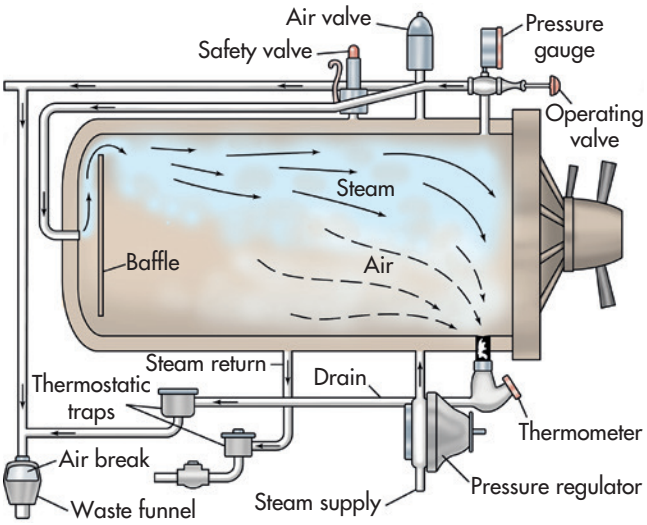


FIG. 2.1 Diagram of a gravity displacement autoclave.



FIG. 2.2 Getinge steam autoclave. (Courtesy Getinge, Gothenburg, Sweden.)

Several studies have shown failure of EtO in inactivating contaminating spores in endoscope channels or lumen test units and residual EtO levels averaging 66.2 ppm even after the standard degassing time. It is recommended that dental handpieces be steam sterilized. Environmental and safety hazards associated with EtO are numerous and severe, and the manufacturer's guidelines for equipment use should be followed carefully to prevent injury to the patient or hospital personnel. The Environmental Protection Agency has classified EtO as a Group B1, probable human carcinogen.

TABLE 2.3 Exposure Periods for Sterilization in Gravity Displacement Sterilizers

Item	Minimum Time Required (min), 121°C–123°C (250°F–254°F)
Scrub brushes (in dispensers, cans, individually wrapped)	30
Dressings (wrapped in muslin or paper)	30
Glassware (empty, inverted)	15
Instruments (wrapped in double-thickness muslin)	30
Instruments combined with suture, tubing, porous materials (wrapped in muslin or paper)	30
Metal instruments only (unwrapped)	15
Linen: maximum size 30 × 30 × 50 cm (12 × 12 × 20 in) (6 kg wrapped)	30
Needles (individually packaged in glass vials or paper, lumens moist)	30
Needles (unwrapped, lumens moist)	15
Rubber catheters, drains, tubing (wrapped in muslin or paper, lumens moist)	30
Rubber catheters, drains, tubing (unwrapped, lumens moist)	20
Utensils (wrapped in muslin or paper, on edge)	20
Utensils (unwrapped, on edge)	15
Syringes (unassembled, individually packaged in muslin or paper)	30
Syringes (unassembled, unwrapped)	15
Suture: silk, cotton, nylon (wrapped in paper or muslin)	30
Solutions:	
75–250 mL	20 (slow exhaust)
500–1000 mL	30 (slow exhaust)
1500–2000 mL	40 (slow exhaust)

Plasma Sterilization

Plasma sterilization (see Table 2.2) is a low-temperature sterilization technique that has become a method of choice for sterilizing heat-sensitive items. This process inactivates microorganisms primarily through the combined use of hydrogen peroxide gas and the generation of free radicals (hydroxyl and hydroperoxyl free radicals) during the plasma phase of the cycle. Conventional sterilization techniques (e.g., autoclaves, ovens, chemicals such as EtO) rely on irreversible metabolic inactivation or on breakdown of vital structural components of the microorganism. Plasma sterilization operates differently because it uses ultraviolet (UV) photons and free radicals. An advantage of the plasma method is the possibility of sterilizing at relatively low temperatures (50°C [122°F]), preserving the integrity of polymer-based

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TABLE 2.4 Minimum Time and Temperature Standards for Various Types of Steam Sterilizers

Item	Exposure Time at 121°C (250°F)	Exposure Time at 132°C (270°F)	Exposure Time at 134°C (273°F)	Exposure Time at 135°C (275°F)	Drying Times
Table Top Gravity-Displacement Steam Sterilization					
Wrapped Instruments	30 min				30 min
		15 min			30–45 min
			10 min		20–60 min
				10 min	15–99 min
Textiles/Porous Loads	30 min				15–99 min
Unwrapped Nonporous Items	30 min				0–1 min
(e.g., instruments)		3 min			0–30 min
		10 min	3 min		0–30 min
				3 min	0–15 min
Table Top Dynamic-Air-Removal Steam Sterilization					
Wrapped Instruments	20–30 min				6–60 min
		4 min			20–60 min
			3 min		6–99 min
				3 min	15–99 min
Textile/Porous Loads	20–30 min	4 min			9.5–99 min
			3 min		6–20 min
Unwrapped Nonporous Items	15–35 min				1–60 min
(e.g., instruments)		3 min			0–60 min
			3 min		0–30 min
				3 min	0–99 min
Immediate Use or "Flash" Sterilization					
Items should be placed in a rigid sterilization container system that is intended for the cycle parameters to be used; items should be used immediately and not stored for future use.					

Modified from Association for the Advancement of Medical Instrumentation: ANSI/AAMI ST79:2010, A1:2010, A2:2011, and A3:2012.

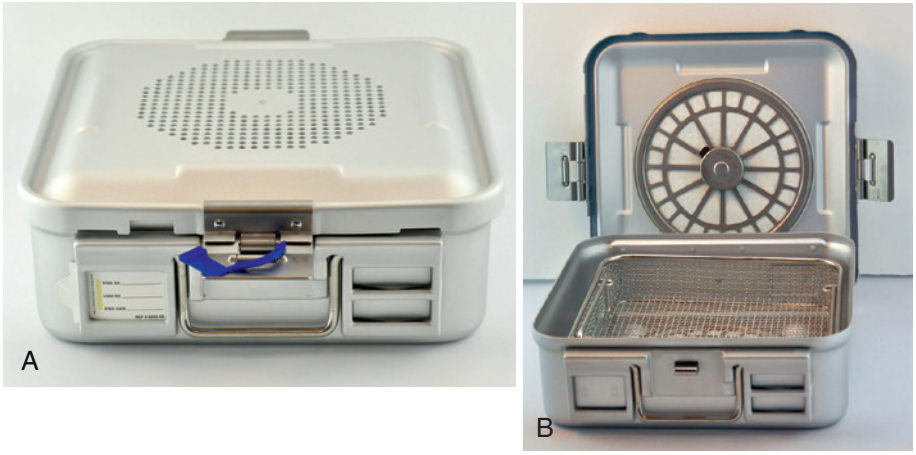


FIG. 2.3 Sterile container system. (A) Sealed container system. (B) Open container system showing inner basket and replaceable filter in the cover. (Courtesy Surgical Direct, Deland, FL.)

instruments, which cannot be subjected to autoclaves and ovens. Furthermore, plasma sterilization is safe, both for the operator and for the patient, in contrast to EtO.

Vapor phase hydrogen peroxide sterilization is a form of plasma sterilization that uses hydrogen peroxide to process instruments quickly and efficiently (Fig. 2.5). Instruments can be sterilized at low temperatures (i.e., <50°C [122°F]) and short time intervals (i.e., 45 minutes), and they are immediately available because aeration is not required. Items for sterilization must be wrapped in nonwoven polypropylene fabric or plastic (Tyvek/Mylar) pouches (Table 2.5). Items that can be sterilized through this process include stainless steel, aluminum, brass, silicone, Teflon, latex, ethyl vinyl acetate, Kraton, polycarbonate, polyethylene (high and low density), polyolefin, polyurethane, polypropylene, polyvinyl chloride, and polymethylmethacrylate. Some plastics, electrical devices, and corrosion-susceptible metal alloys can be sterilized by hydrogen peroxide gas plasma. An important shortcoming of plasma sterilization is its dependence on the actual "thickness" of the microorganisms to be inactivated because UV photons need to reach the DNA. Any material covering the microorganisms (e.g., packaging) will slow down the process. Items that cannot be sterilized safely include linen, gauze sponges, wood products (including paper), endoscopes, some plastics, liquids, items that cannot be disassembled, items that cannot be

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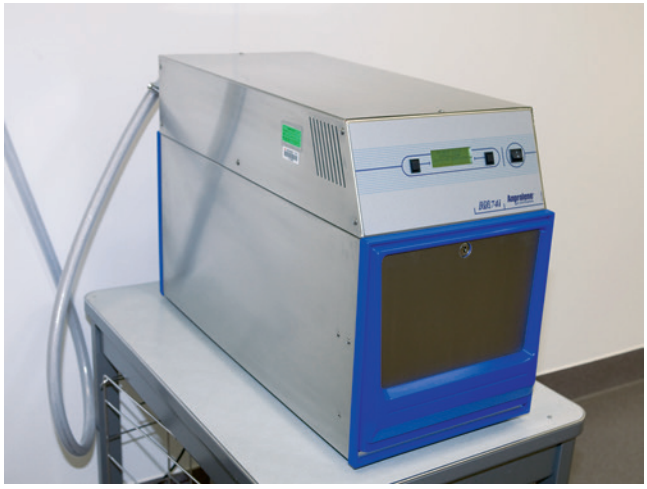


FIG. 2.4 Ethylene oxide sterilizer. (Courtesy Texas A&M Institute for Preclinical Studies.)



FIG. 2.5 Steris VHP MD140 unit that uses hydrogen peroxide in a vapor state to sterilize temperature-, radiation- and humidity-sensitive items. (Courtesy Steris, Mentor, OH.)

completely dried, items with copper or silver solder or that use bisphenol epoxy, tubes and catheters longer than 30 cm (12 inches), and tubes and catheters smaller than 1 to 3 mm in diameter. Special adapters (H_2O_2 boosters) are required for use with devices with lumens to ensure that the sterilant gains access to these areas.

TABLE 2.5 Types and Use of Sterilization Packaging Materials Based on Sterilization Method

Sterilization Method	Packaging Material Requirements	Acceptable Materials
Steam autoclave	Should allow steam to penetrate	Paper Plastic Cloth Paper peel packages Wrapped perforated cassettes Sterile container systems
Ethylene oxide	Must allow ethylene oxide to penetrate Do not use nylon, polyvinyl chloride, polyvinyl alcohol, cellophane, or aluminum foil	Cloth nonwoven fabric (polyethylene) Peel packs and pouches
Hydrogen peroxide plasma	Plasma or vapor must penetrate the wrapping Metal trays block radiofrequency waves and cannot be used Cellulose is not compatible, so paper and woven materials with cotton fibers cannot be used	Nonwoven polypropylene Tyvek

Peracetic Acid Sterilization

Peracetic acid is a highly biocidal oxidizer that maintains its efficacy in the presence of organic material. It denatures proteins, disrupts cell wall permeability, and oxidizes sulfhydryl and sulfur bonds in proteins and enzymes. The sterilant, 35% peracetic acid, and an anticorrosive agent are supplied in a single-dose container. The container is punctured at the time of use, immediately before the lid is closed and the cycle is initiated. The concentrated peracetic acid is diluted to 0.2% with filtered water (0.2 µm) at a temperature of approximately 50°C (122°F). Diluted peracetic acid is circulated within the chamber of the machine. This automated machine is used in the United States to chemically sterilize medical and surgical instruments (e.g., flexible endoscopes). Lumenated endoscopes must be connected to an appropriate channel connector to ensure that the sterilant has direct contact with the contaminated lumen. Bronchoscopy-related infections have occurred when bronchoscopes were processed using the wrong connector.[2]

Ionizing Radiation

Most equipment available prepackaged from the manufacturer has been sterilized by ionizing radiation (i.e., cobalt 60 gamma rays or electron accelerators). This low-temperature sterilization process is restricted to commercial use because of its expense. No ionizing radiation processes have been cleared by the US Food and Drug Administration (FDA) for use in health care facilities. Items commonly used in the OR that are sterilized with ionizing radiation include suture material, sponges, disposable items (e.g., gowns, drapes, table covers), powders, and petroleum goods. Resterilization by other means may not be

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possible for prepackaged sterilized items that have been opened but not used, because an alternate technique could damage the item and create a health hazard.

Cold Chemical Sterilization

Chemicals used for sterilization must be noncorrosive to the items being sterilized. Glutaraldehyde is a saturated dialdehyde that has gained wide acceptance as a high-level disinfectant and chemical sterilant. It is noncorrosive to metals, rubbers, and plastics and provides a means of sterilizing delicate lensed instruments (e.g., endoscopes, cystoscopes, bronchoscopes). The biocidal activity of glutaraldehyde is a consequence of its alkylation of sulfhydryl, hydroxyl, carboxyl, and amino groups, which alters RNA, DNA, and protein synthesis within microorganisms. Most equipment that is safe for immersion in water is safe for immersion in 2% glutaraldehyde. Glutaraldehyde products are marketed under a variety of brand names and are available in a variety of concentrations, with and without surfactants. For high-level disinfection of endoscopes, a 2% glutaraldehyde solution without surfactant is recommended.

Items for sterilization should be clean and dry; organic matter (e.g., blood, saliva) may prevent penetration into crevices or joints. Residual water causes chemical dilution. Complex instruments should be disassembled before immersion. Immersion times suggested by the manufacturer should be adhered to closely (e.g., 2% glutaraldehyde: 10 hours at 20°C–25°C [68°F–77°F] for sterilization; 10 minutes at the same temperature for disinfection). After the appropriate immersion period, instruments should be rinsed thoroughly with sterile water and dried with sterile towels to prevent damage to the patient's tissues. The major problem associated with glutaraldehyde is that it is a known respiratory and dermal irritant and sensitizer, and adverse health effects may occur in exposed workers. Failure to rinse disinfected equipment thoroughly, leaving residual glutaraldehyde on the endoscope, has led to serious conditions, including chemical colitis, pancreatitis, and mucosal damage in human patients. OPA is a new alkylating agent that contains 0.55% 1,2-benzenedicarboxaldehyde. It has shown superior mycobactericidal activity compared with glutaraldehyde with less contact time required (see Table 2.2).

STERILIZATION INDICATORS

Simply placing an item in a sterilizer and initiating the process does not ensure sterility. Failure to achieve sterility may be the result of improper cleaning (if an item cannot be disassembled and all surfaces cleaned, it cannot be sterilized), mechanical failure of the system used, improper use of equipment, improper wrapping, poor loading technique, or failure to understand the concepts of sterilization processes.

Sterilization indicators allow monitoring of the effectiveness of sterilization. Indicators may undergo a chemical or biological change in response to some combination of time and temperature. Chemical indicators, which are available for steam, gas, and plasma sterilization, generally consist of paper strips or tape impregnated with a material that changes color when a certain temperature is reached (Figs. 2.6 to 2.8). The chemical responds to conditions such as extreme heat, pressure, or humidity, but the response does not reflect the duration of exposure, which is critical to the sterilization process. Therefore it is important to remember that chemical indicators do not indicate sterility, only that certain conditions for sterility have been met. The indicators are placed in the center of each pack and on the outside of the item to be sterilized.

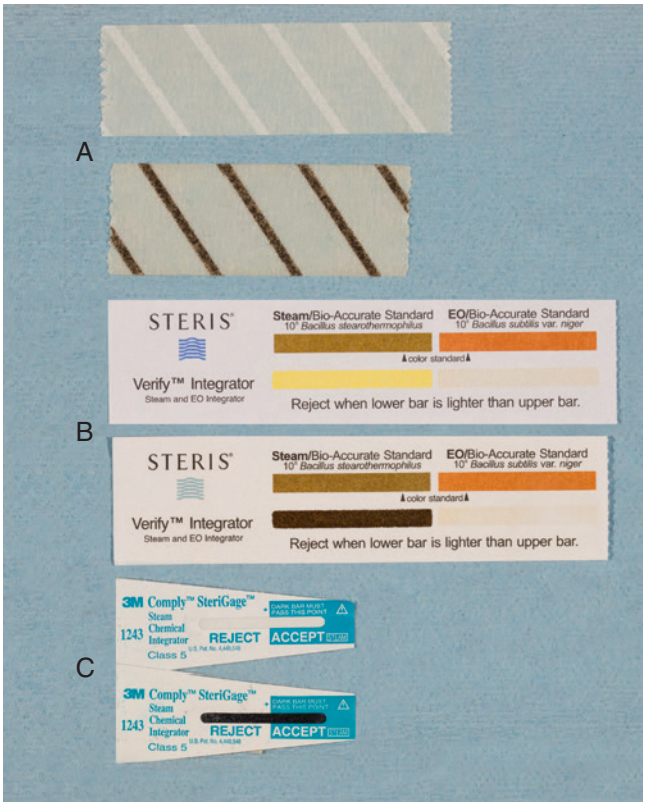


FIG. 2.6 Tape and indicator strips for steam sterilization. (A) The diagonal stripes on the tape *(top)* turn from tan to black. (B) The yellow steam strip *(top)* turns dark brown and must be darker than the line above it. (C) The clear line in the center *(top)* turns black *(bottom)*.

Some autoclaves have a temperature-time graph on the control panel. This indicator method is reliable for measuring the temperature reached and the length of time that each load is exposed to that temperature. A written record can be kept of each processed load.

Use of a biological indicator is the surest way to determine sterility. A strain of highly resistant, nonpathogenic, spore-forming bacteria (*Bacillus stearothermophilus* for steam, *Bacillus subtilis* for gas), which is contained in a glass vial or a strip of paper, is placed in the load of goods to be sterilized. After the sterilization cycle is complete, the vial or strip is recovered and cultured; growth of the organism documents inadequate sterilization. Biological indicators should be used at least weekly to test the effectiveness of the sterilization process.

Sterilization indicators should not be relied on heavily because of the problems mentioned previously. There is no substitute for close supervision of personnel, a general understanding of sterilization processes, and maintenance of high standards for preparing, packing, and loading supplies.

Pack Preparation

The procedure for wrapping items is based on enhancing the ease of sterilization and preserving the sterility of the item, not on convenience or personal preference.

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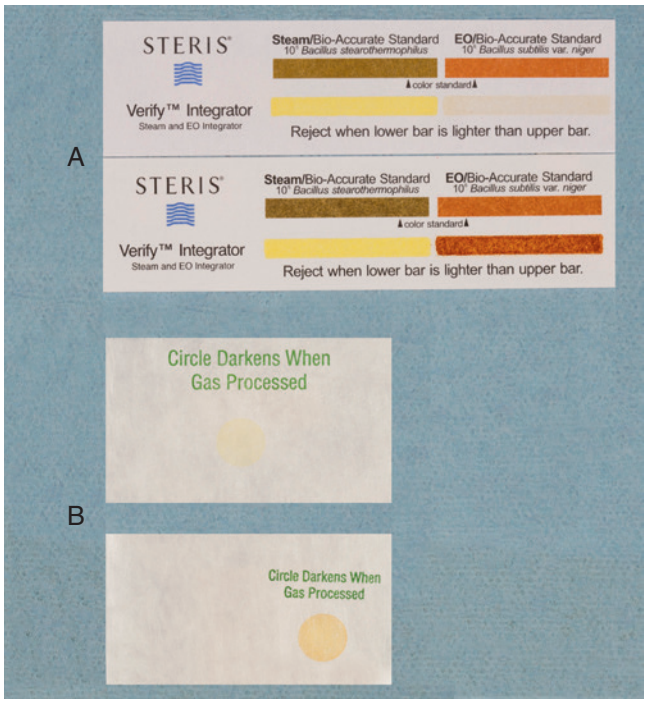


FIG. 2.7 Indicator strips and bag indicators for ethylene oxide (EtO) sterilization. (A) Gas indicator bar on strip turns from tan *(top)* to dark brown *(bottom)*. The bottom bar has to be darker than the top bar. (B) EtO bag indicators turn from a light color *(top)* to a darker shade *(bottom)*. *Note:* Color changes may vary depending on the manufacturer.

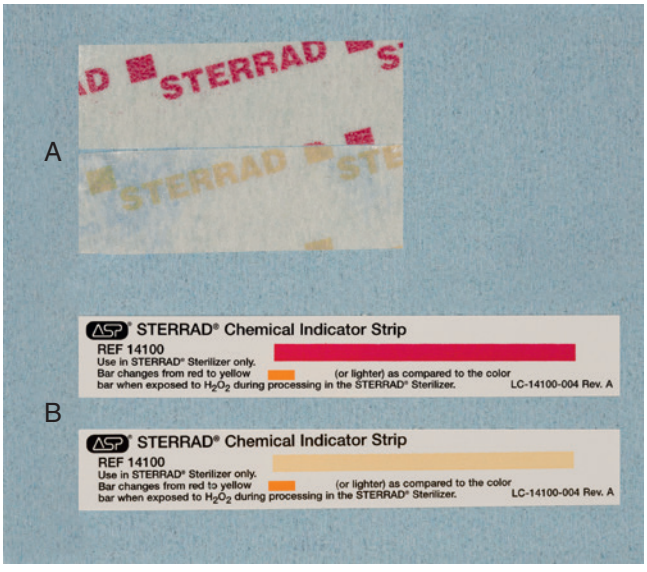


FIG. 2.8 Tape and indicator strips for plasma sterilization. (A) Red writing on tape *(top)* turns to yellow *(bottom)*. (B) Red stripe on the indicator strip *(top)* turns to yellow *(bottom)*. *Note:* Color changes may vary depending on the manufacturer.

Packaging materials (e.g., wrapped or container systems) allow penetration of the sterilization agent and maintenance of sterility after sterilization. Materials for maintaining sterility of instruments during transport and storage include wrapped, perforated instrument cassettes, peel pouches of plastic or paper, sterile container systems, and sterilization wraps (which can be woven or nonwoven). Packaging materials should be designed for the type of sterilization process being used (see Table 2.5). Items sterilized by pressurized steam or other methods (e.g., EtO, plasma) must be wrapped in a specific manner (see p. 12). Packaging materials should also be appropriate for the items being sterilized (Table 2.6). For example, nonpaper materials should be used to package sharp instruments, which can easily puncture paper packaging. Metal closures (e.g., staples, paper clips) that might puncture packaging materials should not be used.

NOTE Do not place paper-plastic pouches within wrapped sets or containment devices because they cannot be positioned to ensure adequate contact with the sterilant, air removal, or drying.[3]

Sterile container systems are typically rigid, boxlike devices made from heat-resistant and steam-sterilizable high-performance plastic or other materials in which instruments can be placed and sterilized (see Fig. 2.3). Rigid containers were first developed in Germany in the mid-1980s. The main function of these early containers was to transport sterile instruments and dressings. In that era, it was not unusual for sterile supplies to be kept in a few containers for an entire day's operating schedule. At the Association of Operating Room Nurses Congress in 1980, the concept of "rigid packaging for sterilization" was introduced in the United States. With time, sterilization containers have gained the confidence of hospital professionals. They are both durable and cost-effective, aid in pack organization, and tend to protect instruments better than wraps. Sterile container systems are the most environmentally friendly because they do not require disposable packaging or laundered cloth. Unlike cloth or paper wrapping, sterile container systems cannot be torn or easily damaged and provide superior protection for surgical equipment. They stack conveniently and can be processed much more rapidly than paper- or cloth-wrapped trays. Closed container systems require filters (in the lid only or in both the lid and the bottom of the container) and latches, seals, and/or tamper-resistant seals. Rigid containers may be a good choice if the sterilization chamber is large enough to accommodate them, and if current storage space is sufficient to accommodate the new configuration. Dozens of different container sizes and shapes are used to accommodate most commonly used instruments, including scopes, drills, and cameras.

NOTE The Association of periOperative Registered Nurses (AORN) recommends a maximum weight limit of 11 kg (25 lb) for instrument sets to avoid insufficient steam contact and a problem with drying that may result in wet packs.[3]

The original sterilization wraps were 140 thread count muslin cloth. Advantages of these cloths included that they were soft, reusable, inexpensive, absorbent, and could easily be draped over trays. However, because they were woven, bacteria could penetrate the pack. Most hospitals double-wrap packs when using cloth to reduce contamination of surgical instruments. In the 1960s, nonwoven materials were introduced that provided a more effective microbial barrier that was also water resistant. The

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TABLE 2.6 Packaging Materials Based on Device Type

Medical Device	Sterilization Method	Suggested Packaging Material
Stainless steel instrument(s) Instrument set(s)	Steam	140 count muslin SMS Woven cotton/polyester-blend fabrics Pouches Sterile container systems
Endoscopic instrument(s) Instrument set(s)	Plasma EtO	Plasma: SMS, polyester-blend fabrics, low-temp SMS pouches EtO: 140 count muslin, SMS, polyester-blend fabrics, some crepe-type papers, thermoplastic polymers (Tyvek)
Glass syringes or other medical devices made of glass	Steam	SMS pouches
	EtO Plasma	Low-temp SMS pouches Thermoplastic polymers

EtO, Ethylene oxide; *SMS,* spunbond, meltblown, spunbond.

TABLE 2.7 Advantages and Disadvantages of Wrapping Materials for Pack Preparation

Material	Advantages	Disadvantages	Sterilization Method
Cotton muslin; 140 or 270 thread count	Durable, flexible, reusable, easily handled	Requires double layer and double wrap, generates lint, not moisture resistant	Steam, EtO
Nonwoven barrier material (i.e., paper)	Inexpensive	Single use, memory, not as durable, not moisture resistant, requires double wrap	Steam, EtO
Nonwoven polypropylene fabric[a]	Flexible, durable, excellent bacterial barrier, puncture resistant, lint free	Single use, requires double wrap	Steam, EtO
Paper/plastic pouches[b] (heat sealed)	Convenient, long shelf life, water resistant	Instruments may puncture pouch	Steam, EtO
Plastic pouches[c] (heat sealed)	Convenient, long shelf life, waterproof, more puncture resistant	Instruments may puncture pouch	Plasma, EtO
Sterile container system	Convenient, long shelf life, stackable, environmental, durable, puncture proof	High initial startup cost	Steam

[a]Spunguard.
[b]Made of paper and Mylar.
[c]Made of Tyvek and Mylar.
EtO, Ethylene oxide.

material used for these wraps was derived from cellulose and was not particularly strong. Hence, sequential (double) wrapping was still necessary. The introduction of polypropylene allowed the development of wraps possessing strength, barrier, and repellent properties. Currently, the most preferred nonwoven technologies used in the medical market are spunlaced and SMS (spunbond, meltblown, spunbond). Spunlaced nonwovens are made by entangling polyester fibers with a layer of wood pulp, whereas SMS materials feature a composite of three layers—spunlace, meltblown, and spunbonded—normally using a polypropylene resin and then stacked together. These products provide excellent protection from microbial contamination. However, despite the fact that the barrier efficacy of a single sheet of wrap has improved over the years, using multiple wrap layers is common practice because of the rigors of handling packs and the consequences of bacterial contamination.

Before packing, instruments are separated and placed in order of their intended use. If steam or gas sterilization is used, the selected wrap should be penetrable by steam or gas, impermeable to microbes, durable, and flexible. Commonly used wrapping materials, the advantages and disadvantages of each, and the sterilization techniques with which each is compatible are listed in Table 2.7.

To ensure maximum penetration, specific guidelines should be followed when packs are prepared for steam and gas sterilization (see Table 2.4). Presterilization wraps for steam sterilization comprise two thicknesses of two-layer muslin or nonwoven (i.e., paper) barrier materials. The poststerilization wrap (i.e., the wrap used after sterilization and the proper cool down period) is a waterproof, heat-sealable plastic dust cover; this wrap is not necessary if the item is to be used within 24 hours of sterilization. Small items may be wrapped, sterilized, and stored in heat-sealable paper or plastic peel pouches (Video 2.1). When sterile container systems are used, the instruments are placed in the inner basket, the filter(s) are replaced, and the unit is sealed with tape or plastic locks. There are no additional storage requirements poststerilization.

Items to be gas sterilized are wrapped in heat-sealable plastic peel pouches or tubing or muslin wrap. When plasma sterilization is used, items should be wrapped in heat-sealable Tyvek-Mylar pouches or polypropylene wraps.

For steam and gas sterilization, instruments should be organized on a lint-free (huck) towel placed on the bottom of

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a perforated metal instrument tray. Instruments with boxlocks should be open when autoclaved. A 3- to 5-mm space between instruments is recommended for proper steam or gas circulation. Complex instruments should be disassembled when possible, and power equipment should be lubricated before sterilization. If the item has a lumen, a small amount of water should be flushed through it immediately before steam sterilization because water vaporizes and forces air out of the lumen; conversely, moisture left in tubing placed in a gas sterilizer may reduce the action of the gas below the lethal point. Containers (e.g., saline bowl) should be placed with the open end facing up or horizontal; containers with lids should have the lid slightly ajar. Multiple basins should be stacked with a towel between them. A standard count of radio-opaque surgical sponges should be included in each pack.

A sterilization indicator (see p. 10) (chemical indicators) should be placed in the geometric center (not on top) or in the area considered least accessible to sterilant penetration of each pack before it is wrapped.

Solutions should be steam sterilized separately from instruments, using the slow exhaust phase (see Table 2.3).

NOTE Complex instruments may require longer sterilization times to adequately penetrate the instruments or multiple tray levels (e.g., total hip replacement equipment). These extended sterilization cycle times should be described in the manufacturer's instruction for use (IFU) documentation.

Immersing instruments for long periods of time in any solution can prove damaging. Never leave instruments in any solution for longer than 20 minutes. Do not immerse instruments with tungsten carbide inserts (gold handles) in solutions containing benzyl ammonium chloride (BAC) because this chemical is known to loosen the tungsten carbide.

Linens may be steam sterilized. The maximum size and weight of linen packs that can be steam sterilized effectively are $30 \times 30 \times 50$ cm ($12 \times 12 \times 20$ in) and 6 kg, respectively. Closely woven table drapes should be packed separately. Layers of linen are alternated in their orientation to permit steam penetration. As with instruments, a sterilization indicator (see p. 10) should be placed in the center of each pack.

NOTE Manufacturers of reusable medical devices and surgical instruments for human use are required by the FDA to provide detailed and validated instructions for the disassembly, lubrication, cleaning, disinfecting, packaging, and sterilization of their products. These instructions are provided in IFU documents. IFUs are available for most veterinary surgical and medical products as well.

Wrapping Instrument Packs

Instrument packs should be wrapped so they can be easily unwrapped without breaking sterile technique (Fig. 2.9 and Video 2.2).

Folding and Wrapping Gowns

Gowns must be folded so they can be easily donned without breaking sterile technique (Fig. 2.10).

FOLDING AND WRAPPING DRAPES

Drapes should be folded so that the fenestration can be properly positioned over the surgical site without contaminating the drape (Fig. 2.11).

HANDLING AND STORAGE OF STERILIZED INSTRUMENTS AND EQUIPMENT

After removal from the autoclave, packs are allowed to cool and dry individually on racks. Placing the packs on top of each other during cooling may promote condensation of moisture, resulting in strike-through contamination. Strike-through contamination occurs when moisture carries bacteria from a nonsterile surface to a sterile surface. When wrapped sterile packs are completely dry, they should be stored in waterproof dust covers in closed cabinets (rather than uncovered on open shelves) to protect them from moisture or exposure to particulate matter (i.e., dust-borne bacteria). Sterile container systems can be stacked without additional covers. Excessive handling of sterile supplies should be avoided, especially if the items are pointed or have sharp edges. Sterile items should be handled gently and should be protected from bending, crushing, or compression forces that could break a seal or puncture the package. Sterile packs should be stored away from ventilation ducts, sprinklers, and heat-producing light. Ideal environmental conditions are characterized by low humidity, low air turbulence, and a constant, controllable room temperature.

Sterile Shelf Life

The use of published expiration dates for sterilized items in various types of wrappers is controversial. Events, not time, contaminate products. It was recently shown that if items are packaged, sterilized, and handled properly, they remain sterile unless the package is opened, gets wet, is torn, has a broken seal, or is damaged in some other way (i.e., event-related expiration). The length of time an item is considered sterile depends on a number of factors: (1) the type and configuration of the packaging materials; (2) the number of times a package is handled before use; (3) the number of personnel who may have handled the package; (4) whether the package was stored on open or closed shelves; (5) the condition of the storage area (e.g., cleanliness, temperature, humidity); and (6) the method of sealing and whether dust covers were used.[5] For an event-related expiration system to be used effectively, appropriate protocols must be adopted for sterilizing and handling items.

Handling Sterilized Items

Sterile packs should be labeled with the date on which the item was sterilized and a control lot number for tracing a nonsterile item. Heat-sealed, waterproof dust covers should be placed on items not routinely used. Items should be stored in a manner that does not compromise packaging and sterility, and they need to be rotated in such a way that the item processed first is used first.

If a sterile pack is damaged, it should not be used. Damage is defined as wraps that have moisture present; packs that have been placed in a dusty environment or stored near the source of an air current; items that have been dropped, bent, crushed, compressed, torn, or punctured; or packs that have a broken seal. Education of surgery personnel must include training in

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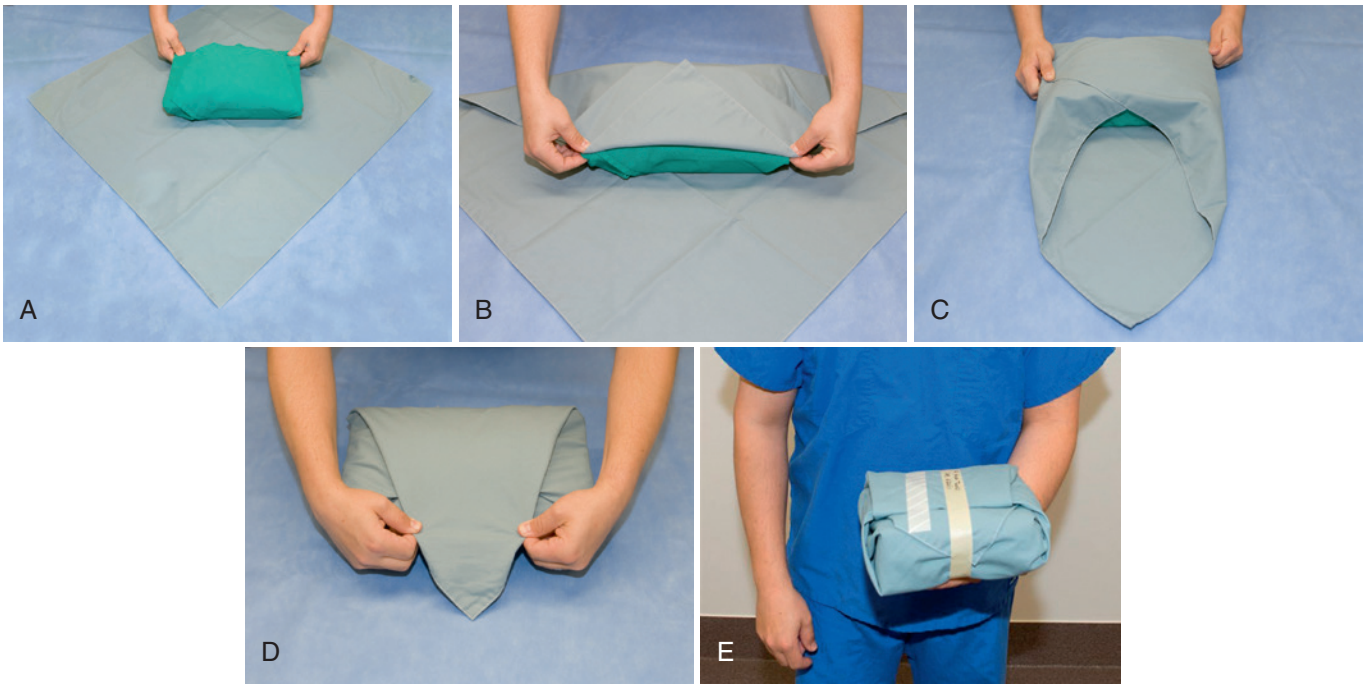


FIG. 2.9 Wrapping an instrument pack. (A) Wrap the instrument pack in a clean huck towel. Place a large, unfolded wrap in front of you and position the instrument tray in the center of the wrap so that an imaginary line drawn from one corner of the wrap to the opposite corner is perpendicular to the long axis of the instrument tray. (B) Fold the corner of the wrap that is closest to you over the instrument tray and to its far edge. Fold the tip of the wrap over so it is exposed for easy unwrapping. (C) Fold the right corner over the pack. Fold the left corner similarly. (D) Turn the pack around and fold the final corner of the wrap over the tray, tucking it tightly under the previous two folds. (E) Wrap the pack in a second layer of cloth or paper in a similar manner. Secure the last corner of the outer wrap with masking tape and a piece of heat-sensitive indicator tape.

ways to protect sterile items from events that cause loss of sterility. The integrity of sterilized items must be carefully assessed to identify damaged goods, and plastic dust covers must be removed or wiped clean before reaching the surgical area.

Wet Packs

Wet packs are sterilized containers that have interior moisture in the form of dampness, droplets, or puddles of water after the sterilization and cool down process. The American National Standards Institute and Association for the Advancement of Medical Instrumentation recommend considering such packs as contaminated as there are no scientific studies to prove otherwise.[4] Wet packs may be due to improper or excessive autoclave loading or inadequate cool down times.

Unwrapping and Opening Sterile Items

Sterile items are wrapped in a manner that allows OR personnel to unwrap the items without contaminating them. Three popular methods are used to distribute wrapped sterile items.

Unwrapping Large Sterile Linen/Paper/Polypropylene Packs That Cannot Be Held During Distribution (Video 2.3)

If the pack is too large, cumbersome, or heavy to be held during distribution, it may be opened onto a Mayo stand or back table. *Place the pack on the center of the Mayo stand or back table, and open each folded layer by pulling it toward you (this prevents your hand and arm from extending over the sterile area). Handle only the edge and underside of the wrap. Follow the same procedure for each fold. When the pack is open, have a sterile team member place it on the sterile table.*

There is disagreement over the correct way to open double-wrapped sterile packs (i.e., outer layer only or both layers), and evidence supports both techniques. The rationale for opening the outer layer only is that this technique eliminates the risk of microbial shedding from the circulating nurse's hands and arms onto the contents of the sterile package. The rationale for opening both wrappers is that when the outer surface of the inner wrapper is opened, it may become contaminated by dust particles and debris from the outer wrapper. If the circulating nurse opens this inner wrapper, the possibility of contamination is reduced. The decision of which technique to use must be based on the technical expertise of personnel and on barrier quality.

Unwrapping Sterile Linen/Paper Packages That Can Be Held During Distribution

These packs may be opened and placed on a sterile table as described in Fig. 2.12, or after opening, they may be grasped by a sterile team member.

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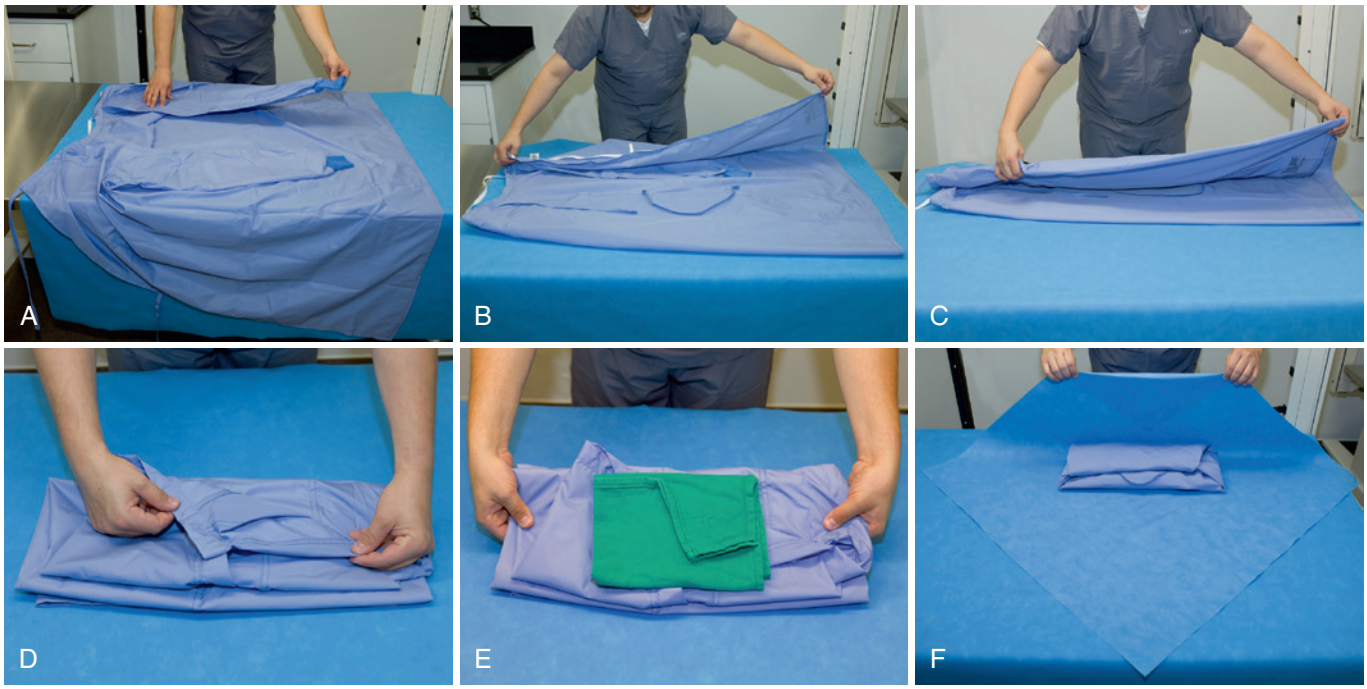


FIG. 2.10 Folding and wrapping surgical gowns. (A) Place the gown on a clean, flat surface with the front of the gown facing up. Fold the sleeves neatly toward the center of the gown with the cuffs of the sleeves facing the bottom hem. (B) Fold the sides to the center so the side seams are aligned with the sleeve seams. (C) Fold the gown in half longitudinally (the sleeves will be inside the gown). (D) Starting with the bottom hem, fanfold the gown toward the neck. (E) Fold a hand towel in half horizontally, and fanfold it into about four folds. Place it on top of the folded gown, leaving one corner turned back so that it can be easily grasped. (F) Wrap the gown and towel in two layers of paper or cloth wrap as described in Fig. 2.9.

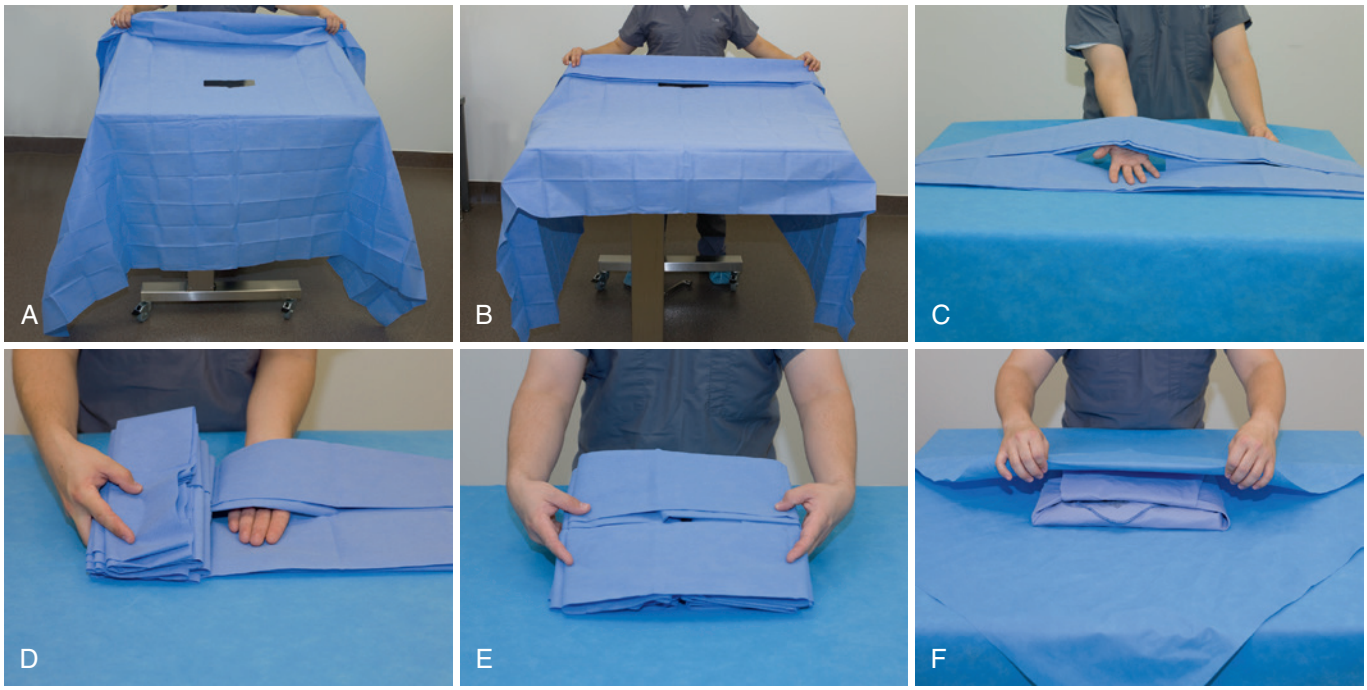


FIG. 2.11 Folding and wrapping drapes. (A) Lay the drape flat with the ends of the fenestration perpendicular to you and the sides of the fenestration parallel to you. (B) Grasp the edges of the drape nearest you and fanfold the drape to the center. The edge of the drape should be exposed (dorsal) so it can be easily grasped during unfolding. (C) Turn the drape around and fanfold the other half the same way. (D) Fanfold one end of the drape to the center (the fingers are through the fenestration); repeat with the other end. (E) If the drape has been folded properly, the fenestration is on the ventral outermost aspect. (F) Fold the drape in half, and wrap it in two layers of paper or cloth wrap as described in Fig. 2.9.

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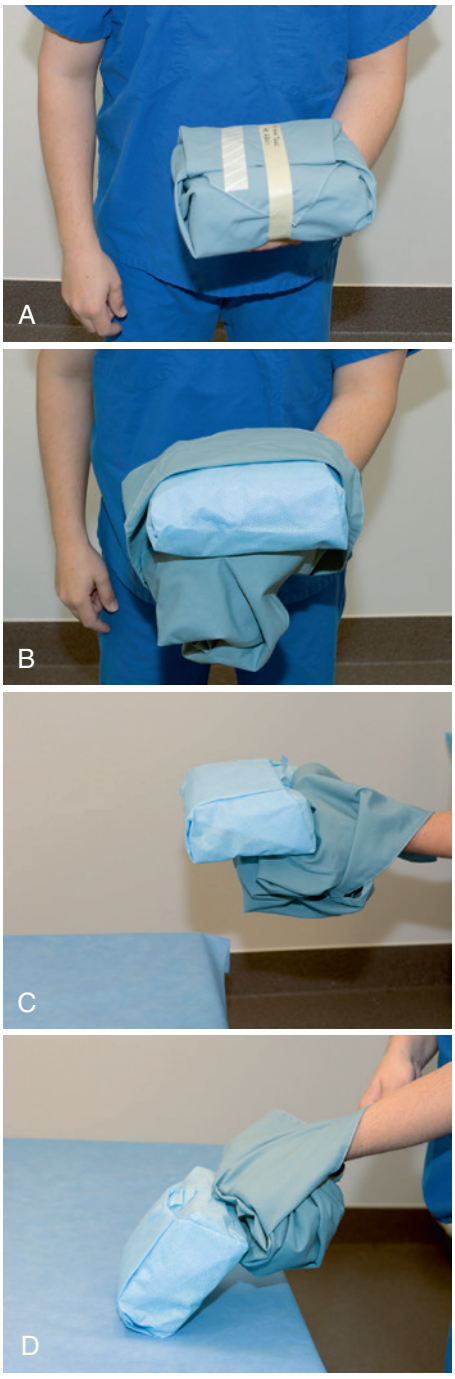


FIG. 2.12 (A) To unwrap a sterile linen pack that can be held during distribution, hold the pack in your left hand if you are right-handed (and vice versa). (B) Using your right hand, unfold one corner of the wrap at a time, being careful to secure each corner in the palm of your left hand to prevent corners from recoiling and contaminating the contents. (C) Hold the final corner with your right hand; your hand should be completely covered by the wrap. (D) When the pack is fully exposed and all corners of the wrap have been secured, gently set the pack on the sterile field, being careful not to allow your hand and arm to reach across or over the sterile field.

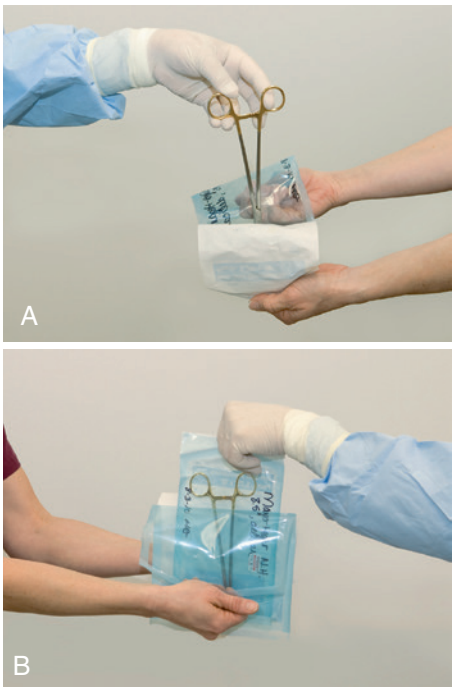


FIG. 2.13 Opening (A) single-wrapped and (B) double-wrapped peel-back pouches.

Unwrapping Sterile Items in Paper/Plastic or Plastic Peel-Back Pouches (Video 2.4)

Identify the edges of the peel-back wrapper, and carefully separate them (Fig. 2.13). Peel the edges of the wrapper back slowly and symmetrically to ensure that the sterile item does not come in contact with the torn edge of the wrapper (the torn edge of a peel-back wrapper is unsterile). If the item is small, place it on the sterile area as described earlier, being careful not to lean across the sterile table. If the item is long or cumbersome, have a sterile team member grasp it and gently pull it from the peel-back wrapper, taking care not to brush the item against the peeled edge of the wrapper. Scalpel blades and suture materials are opened in a similar manner.

Opening Sterile Container Systems (Video 2.5)

Place the container on a nonsterile surface and pop the latches by breaking the tape or plastic locks (Fig. 2.14). Remove the cover and place to the side. A sterile individual reaches in to retrieve the inner basket containing the surgical equipment.

POURING SOLUTIONS INTO BASINS

Solutions (i.e., sterile saline and antiseptics) are poured into basins. The basin should be held away from the surgical table by a sterile team member to prevent the nonsterile assistant's hand and arm from extending over the sterile area. The solution is poured without splashing, taking care to prevent it from dripping down the container onto the sterile person's hand. The solution container should not touch the sterile basin.

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FIG. 2.14 Opening a sterile container system. (A) The container is placed on a nonsterile counter. The latches are opened by breaking the tape or plastic locks, and the cover is removed. (B) A sterile individual reaches in and retrieves the inner basket containing the sterile equipment. (C) The entire basket or just its contents may be placed on the sterile instrument table. (Courtesy Surgical Direct, Deland, FA.)

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3

Surgical Facilities and Maintenance of the Surgical Environment



A variety of physical layouts are suitable for modern operating rooms (ORs) and surgical areas, but the goals of all designs should be patient safety and work efficiency. Designing an efficient workspace requires more than knowing the appropriate number of air exchanges an OR should have to reduce airborne contaminants or how many rooms are necessary to facilitate an efficient workflow. It also requires careful attention to traffic patterns, the space required for storage of supplies to be sufficient to meet the needs of the caseload, adequate space for staff and surgeons to allow them to function efficiently, and ample room for the provisions for sterile processing nearby. The surgical area should be in close proximity to the anesthesia and surgical preparation areas, critical care, radiology, and central supply. However, it should be isolated from general traffic flow (e.g., examination rooms, offices, reception area, wards). In large facilities, such as universities and surgical referral centers, anesthesia and surgical preparation areas should be a separate working unit that is isolated from general hospital traffic.

STRUCTURE AND DESIGN OF THE SURGICAL AREA

Because of the constant danger of contamination to surgical patients, the surgical area should be delineated into "restricted," "semirestricted," and "unrestricted" areas. Restricted areas include ORs, sterile supply rooms, and the scrub sink area. Scrub suits, hair coverings, and masks are required whenever anyone enters these areas (even for cleaning). The semirestricted areas include the peripheral support areas of the surgical suite, including hallways between the ORs and nurses' stations, instrument and supply processing areas, storage areas, and utility rooms. Personnel are required to wear surgical attire and cover all head and facial hair when entering these areas. Unrestricted areas include a central control point that is established to monitor the entrance of patients, personnel, and materials into the restricted areas. The anesthesia and surgical preparation rooms, dressing rooms, lounges, and offices are also in the unrestricted zone. A commonly used floor plan is one in which the surgical suites are arranged around a central workstation for OR nurses. Easy access to each OR from the workstation ensures efficient traffic flow, which reduces cross-contamination between areas. The goal is to create realistic traffic and commerce of patients, personnel, and supplies, with careful attention to restricted and semirestricted work areas.

Individuals entering a restricted area from an unrestricted area must don proper surgical attire (see Chapter 6); the ideal location for moving from an unrestricted area to a restricted one (or vice versa) is through a locker room. Surgical personnel who leave a restricted area and enter an unrestricted area must cover their clothing with a closed knee-length lab coat before they leave and discard these items when they return to the restricted area. Doors between restricted and unrestricted areas should be kept closed at all times. **Food and drink are permitted only in unrestricted areas.** Movement of clean and sterile supplies and equipment should be separated as much as possible from movement of contaminated supplies and equipment by space, time, and traffic patterns. Soiled linen and trash should be kept in an unrestricted area, and patients should be clipped and vacuumed in an unrestricted area before transport to a restricted area (e.g., the OR).

> **NOTE** To prevent contaminating the surgical suite, clip the patient and perform the initial surgical preparation in a separate (unrestricted) area. Never clip patients in the OR.

TRAFFIC PATTERNS

Because airborne contaminants can cause or aggravate infection, it is best to create traffic patterns that minimize traffic into and out of rooms. This restricts the movement of airborne contaminants, such as organisms carried and shed by people and objects. Entry into the OR and other restricted areas should be limited to essential equipment and personnel. A study presented at the 2016 American College of Surgeons National Surgical Quality Improvement Program Conference suggested that minimizing OR traffic could lower rates of surgical site infections (SSIs).[1] After review of their infection rates, a Canadian hospital determined that they had an excessively high rate of wound infections from orthopedic operations such as knee and hip joint replacements; they decided to implement practices to reduce traffic movement into the OR. Reasons for entering and exiting the OR during operations included retrieving charts, instruments, or equipment and taking a break. Over a 2-year period they were able to reduce OR traffic from between 42 and 70 door openings per case to 3.2 door openings per case. They reported that this intervention may have contributed to a decrease in orthopedic SSIs from 2.8% to 2.1%. Logically, opening the doors to the OR decreases the effectiveness of the ventilation system in effectively clearing potential contaminants from the OR outward.

Guidelines useful for limiting movement from the outside into the operating suite are provided in Box 3.1.

Most particles and bacteria found in OR air are shed from skin and hair by personnel in an OR. Although no one has established a direct relationship between the number of people in an OR and the development of postoperative infection, some studies suggest that as the number of personnel in the OR increases, so does the incidence of SSIs. Whether this has to do with the people themselves or the greater amount of traffic into and around the room is not clear.

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ABSTRACT

Patient safety and work efficiency are the most important goals of designing a surgical suite and prep area. Proper care and cleaning of the areas are critical to reduce infection.

KEYWORDS

Disenfectants
Cleaning
Maintenance
OR design
Prep room
Traffic flow
Workspace design
Restricted space
Semirestricted space
Unrestricted space

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BOX 3.1 Guidelines Useful for Limiting Movement into the Operating Room (OR)

1. Allow only those people who are essential to the procedure into the OR while operations are being performed.
2. Limit movement of personnel within the OR.
3. Keep the doors of ORs closed during surgeries/procedures.
4. Ensure that anyone entering the OR is properly trained in infection control practices and sterile technique.

DESCRIPTION AND FUNCTION OF ROOMS IN THE SURGICAL AREA

Dressing Room

The dressing room is used by surgical personnel for changing into proper surgical attire. It should have closed cabinets for storing scrub suits, shoe covers, masks, and caps and a separate area for hanging street clothes. A hamper for dirty laundry should be available to minimize carrying of contaminated linen through the hospital.

Anesthesia and Surgical Preparation Areas

The anesthesia induction and surgical preparation area should be adjacent to the surgical area yet out of major hospital traffic patterns. This room should be supplied with equipment and medications that may be necessary in an emergency (e.g., defibrillator, endotracheal tubes, suction, oxygen, crash cart). It also should have anesthetic equipment (machines and drugs), laryngoscopes, clippers (mounted on the wall or hanging from the ceiling), vacuums (large canister or central), skin preparation materials (antiseptic soaps, alcohol, sterile gauze sponges), sharps containers, needles and syringes, and monitoring equipment, all of which should be readily available to ensure efficient anesthesia and preoperative patient preparation.

NOTE Drugs and equipment necessary for an emergency can be stored in a mobile crash cart; this facilitates movement from the anesthesia preparation room to the OR and to recovery.

NOTE Clipper blades may be a source of infection between patients. Clean and disinfect them regularly by soaking them in alcohol or chlorhexidine or spraying them with an ethanol/o-phenol/phenol product.[2]

Preparation counters and surfaces should be impervious and easily cleaned and disinfected (Table 3.1). Stainless steel preparation tables with built-in sinks are ideal. Gas scavenger systems should be present at each anesthesia preparation table. General lighting is supplied by main overhead fluorescent lights, which are supplemented by a spotlight directed at each preparation table. A sink designated for cleaning anesthetic hoses, endotracheal tubes, and rebreathing bags, plus a plastic rack for draining and drying rebreathing bags and hoses, should be available. An erasable anesthesia-surgery scheduling board, easily visible to anesthesia and surgical personnel, should list the day's procedures.

Temperature in the preparation room should be kept between 17°C and 20°C (62°F and 68°F) and humidity at 50% or less to reduce microbial growth. Gurney surfaces should be padded, and circulating water and/or warm air blankets should be used to prevent hypothermia. Well-constructed gurneys (i.e., made of stainless steel or other easily cleaned materials, with relatively large wheels with bearings that can be easily lubricated, and rubber bumpers mounted on the corners to prevent damage to doors and walls) should be available for patient transportation. An adhesive microfilm dust pad should be placed at the doorway between the anesthesia preparation room and the surgical area to collect dust, hair, and other particulates on gurney rollers, shoes, and anesthetic equipment.

Anesthesia Supply Room

The anesthesia supply room should be adjacent to the anesthesia and surgical preparation room. Equipment necessary to keep anesthesia machines working properly, extra endotracheal tubes, anesthetic monitoring equipment, oxygen "E" tanks, hoses, catheters, and airway connectors are stored here. This room may also have a cabinet for storing nongaseous anesthetic agents, and it may be a convenient location for storing large oxygen tanks that supply oxygen to each anesthesia preparation table and to the OR.

Nurses' Workstation

The nurses' workstation should be centrally located in the surgical area (i.e., the restricted area). An autoclave (for flash sterilization), an incubator and/or blanket warmer (for irrigation fluids and for towels to wrap patients after surgery), a refrigerator (for medications and solutions), and formalin containers are kept in this area. A daily surgery log, OR protocols, and a telephone are also kept at the workstation. Soiled instruments may be sent to a central supply area or may be decontaminated, washed, lubricated, and wrapped or packaged here for resterilization. This area should be divided into two separate areas to prevent cross-contamination of clean supplies if it is used for decontaminating and wrapping instruments.

Sterile Instrument Room

The sterile instrument room is a restricted area that houses all sterilized and packaged instruments and supplies. It is commonly near the nurses' workstation. Surgery personnel assemble items necessary for a particular case from supplies in this room. Items should be logically arranged on shelves (e.g., alphabetical order) and routinely checked for "outdates" (i.e., time-related expiration; see p. 13) and package integrity (i.e., event-related expiration; see p. 13).

Sterile supplies should be transported into ORs in closed sterile containers. Inadequate sterilization of surgical instruments has been implicated in outbreaks of SSI. For this reason, indicators documenting adequate sterilization must be included in all sterile instrument sets (see Chapter 2).

Equipment Room

As a general rule, unless a piece of equipment is mostly or exclusively used in a given OR, it should be stored elsewhere. Large pieces of equipment, such as anesthesia machines, lasers, monitoring equipment, operating microscopes, and portable surgery lights, can be stored in an equipment room. Equipment should be kept free of dust and cleaned routinely using the protocol described for OR disinfection (see p. 21). The equipment room is a valuable area because it eliminates storage of large, expensive pieces of equipment in hallways, where they could be damaged or create a hazard.

Most equipment should be kept in an equipment storage area in the semirestricted area adjacent to the OR. For specialty rooms

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TABLE 3.1 Common Disinfectants

Product	Main Ingredient(s)	Use Instructions	Contact Time	Effective Against
Ultra Clorox	Sodium hypochlorite 6%, other ingredients 94%	Dilute at 3/4 cup/gallon of water	In kitchen or bathroom 5 minutes; for toilet bowl 10 minutes; for mildew 15 minutes	Parvovirus, gram-positive and gram-negative bacteria
Roccal D Plus	Didecyl dimethyl ammonium chloride, alkyl dimethyl benzyl ammonium chloride, alkyl dimethyl benzyl ammonium chloride, bis-*n*-tributyltin oxide, inert ingredients	Hard surfaces, 3.75 g/L (1/2 oz/gallon) of water; boot bath, 7.5 g/L (1 oz/gallon) of water; badly soiled areas, 11.23 g/L (1.5 oz/gallon) of water	10 minutes	Gram-positive and gram-negative bacteria, *Aspergillus fumigatus, Trichophyton mentagrophytes, Candida albicans,* parvovirus, parainfluenza
Clorox Clean-Up	Sodium hypochlorite 1.84%, other ingredients 98.16%	Mop floors with 34 g/L (1 cup/gallon) of water	5 minutes	*Escherichia coli, Salmonella choleraesuis, Staphylococcus aureus, Streptococcus pyogenes*
Nolvasan Solution	Chlorhexidine diacetate 2%, other ingredients 98%	7.5 g/L (1 oz/gallon) of water; for rabies, 3 oz/gallon of water	10 minutes	Gram-positive and gram-negative bacteria, canine distemper virus, feline rhinotracheitis virus, canine coronavirus
Snap 'n' Go #6 (Neutral Disinfectant, purple)	Octyl decyl ammonium chloride 6.51%, dioctyl dimethyl ammonium chloride 3.25%, alkyl benzyl ammonium chloride 8.68%, inert ingredients	3.75 g/L (1/2 oz/gallon) water or full strength	10 minutes	Gram-positive and gram-negative bacteria; use this dilution for feline leukemia virus, canine distemper virus
Snap 'n' Go #14 (Veterinary Disinfectant, green)	*n*-Alkyl dimethyl ethylbenzyl ammonium chloride 4.5%, dimethyl ethylbenzyl ammonium chloride, inert ingredients	7.5 g/L (1 oz/gallon) water or 30 g/L (4 oz/gallon) water	10 minutes	Gram-positive and gram-negative bacteria, canine distemper virus, feline calicivirus; use this dilution for canine parvovirus, adenovirus, coronavirus, and parainfluenza viruses and for feline panleukopenia, coronavirus, and picornavirus
Virkon *S*	Potassium peroxymonosulfate 20.4%, sodium chloride 1.5%, other ingredients 78.1%	For 1% solution, add one (1) tablet to 120 g (16 fl oz) of water; solution stable for 7 days	10 minutes	Gram-positive and gram-negative bacteria; *Aspergillus fumigatus, Candida albicans, Microsporum canis;* canine parvovirus and distemper; feline parvovirus, herpes, and calicivirus; and *Leptospira canicola*

(cardiology, neurology), an adjacent space in the restricted area near the OR should be designated as storage. The tendency to favor additional OR space, at the expense of storage space, when designing an OR suite results in cluttered corridors and ORs, which can become safety hazards.

Other storage spaces are designated for clean supplies and packaged reusable items and should be in a separate space from the soiled workroom. Carts and gurneys must not be stored where they obstruct corridors.

Housekeeping Supply Room

Supplies used to decontaminate and clean surgical suites may be stored in the supply room or closet. Cleaning equipment and supplies stored here must be restricted to use in the OR to prevent cross-contamination from other hospital areas.

Scrub Sink Area

Scrub sink areas should be centrally located for the OR suites. Antiseptic soap in an appropriate dispenser (i.e., foot activated or motion sensitive), scrub brushes (i.e., sterilized reusable brushes or a disposable polyurethane brush-sponge combination, unless brushless scrub solutions are used [see Chapter 6]), and fingernail cleaners should be kept within easy reach at each scrubbing station. Deep stainless steel sinks equipped with knee-, elbow-, or foot-operated or motion-sensitive water activators are ideal. Electronic sensory activated scrub sinks as shown in Fig. 3.1 allow "no touch" activation. With reusable brushes, the dispensing container and clean brushes must be detached and autoclaved regularly. The scrub sink area must be located away from wrapped sterile supplies because of possible contamination by water droplets and spray from the sinks. Scrub sinks should never be used to clean equipment or instruments or to dispose of body fluids.



FIG. 3.1 Motion-sensitive water-activated scrub sink. (Courtesy Texas A&M Institute for Preclinical Studies.)

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Gowning and Gloving Area

Gowning and gloving can be done outside or inside the OR. Controversy continues over which location results in the least cross-contamination, but no evidence supports one location over another.

NOTE If the OR is small, or if several people are scrubbing in, gowning and gloving in a separate area may help prevent contamination of personnel, sterile supplies, or the prepped surgical site.

Operating Room

ORs are individual rooms where surgeries are performed. The room should be large enough to allow personnel to move around sterile equipment without contaminating it and to accommodate the large pieces of equipment necessary for some procedures.

Guidelines from the American Institute of Architects recommend that newly constructed general ORs should have a minimum clear floor area of 37 m_2 (400 square feet) exclusive of fixed or wall-mounted cabinets and built-in shelves, with a minimum of 6 m (20 feet) clear dimension between fixed cabinets and built-in shelves.[3] These guidelines also state that rooms for cardiovascular, neurologic, orthopedic, and other special procedures that require additional personnel or large equipment should have a minimum clear floor area of 55.7 m_2 (600 square feet) and a minimum of 6 m (20 feet) clear dimension exclusive of fixed or wall-mounted cabinets and built-in shelves.

For renovations, the minimum clear floor area for general purpose rooms and orthopedic rooms should be 33.4 m_2 (360 square feet). Cardiovascular, neurologic, and other special purpose rooms should be 37 m_2 (400 square feet). Windows should have coved or sloped edges and should be without ledges that can collect dust. They should be well sealed and should not be allowed to be opened. Studies have shown that natural light enhances staff morale.

Some suppliers that offer lights and tables (e.g., Skytron, Berchtold Corp.) also supply voice and touch screen activation systems, video monitors, cameras, power booms, and more for an integrated OR that is efficient and multifunctional. Integrated OR suites can be custom-configured to be minimal or extravagant and may be built from scratch or retrofitted.

The OR should be uncluttered and simple so that no areas trap dust or are difficult to clean. Floors, ceilings, walls, and other surfaces should be smooth, nonporous, and constructed of fireproof materials. Smooth surfaces can be thoroughly cleaned and disinfected (see Table 3.1), which helps prevent trapping of biological material that could cross-contaminate. Surface materials should be able to withstand frequent washing and cleaning with strong disinfectants.

Uniform ceilings are recommended for ORs. Most ceilings are made of plaster; however, moisture-resistant gypsum wallboard systems with special epoxy-based coatings may be less costly. Ceiling-mounted lights or fixtures should be sealed so that dust and contaminants cannot enter through openings and so that there is no compromise to the ventilation system. In semirestricted and unrestricted areas (e.g., recovery and holding areas), lay-in ceilings with clips that ensure that dust and other contaminants do not enter the room may be used[4]; however, they are not permitted in ORs.

Proper air handling is the single most important environmental factor in the prevention of SSI. Operating suites are ideally maintained at positive pressure so that air flows from the cleanest areas to the least clean areas. Air should flow from the OR toward corridors and adjacent areas. Current requirements for airflow conditions in hospital ORs are regulated by the international technical society organized to advance the arts and sciences of heating, ventilation, air-conditioning and refrigeration (ASHRAE Standard 170-2008). Although this standard contains airflow requirements such as the number of exchanges and the temperature and humidity of the air, it does not address airborne contaminant levels. At least 15 air changes per minute should be maintained, and at least 3 air changes per minute should be made up of fresh air. ORs are designed to introduce air at the ceiling with exhaust near the floor (plenum system). Rooms may be engineered for horizontal laminar flow, in which particle-free air is moved over the operative field at a uniform velocity, picking up particles in its path and passing them through a high-efficiency particulate air (HEPA) filter. Although the benefit of laminar flow systems is questionable, HEPA filters do appear to provide the best environment for reducing microbial contaminants in the air of the OR. Regularly scheduled surveillance and maintenance of the air handling system is crucial. This involves checking for moisture in walls, ceilings, and other potentially porous materials; ensuring the integrity of air ducts; and checking fan settings and filters. Scavenging systems that pull anesthetic gases out of the OR air should be installed in each room.

General lighting in the OR is supplied by main overhead fluorescent lights, which are supplemented by one or preferably two halogen spotlights. Surgery lights are designed to emit a soft white light that is of high intensity, low heat, and true color and that reduces shadows without glare. Options include single, double, and triple light-head configurations in wall, ceiling, and stand mounts. Surgical lights are generally ceiling mounted. Track lighting should be avoided because dust and bacteria may become trapped in the tracks. Rotation capabilities are important; most lights rotate 360 degrees at the light-head axis, but some are even more flexible. Many lights can incorporate cameras. Fiberoptic headlamps worn by many surgeons are now available in comfortable, lightweight models that virtually eliminate shadowing of the surgical site (Fig. 3.2).

Stainless steel operating tables should be fully adjustable for height (hydraulic mechanism) and degree of tilt (Fig. 3.3). Tabletops should include a flat, one-piece surface or should have V-trough capabilities. Tables vary not only in terms of the types of procedures that can be performed on them, but also in imaging



FIG. 3.2 Fiberoptic headlamp.

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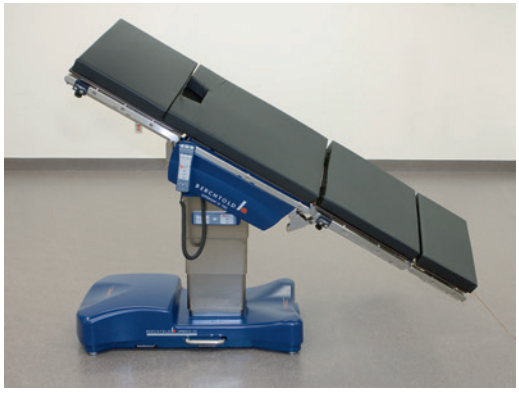


FIG. 3.3 Operating room table with remote control to allow tipping of the table in any direction during the surgical procedure. (Courtesy Texas A&M Institute for Preclinical Studies.)

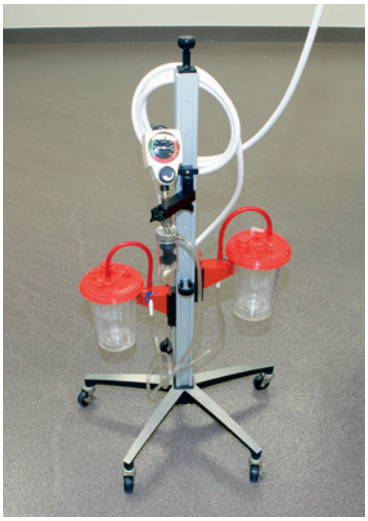


FIG. 3.4 Portable suction unit. (Courtesy Texas A&M Institute for Preclinical Studies.)

capabilities, mobility, and availability of accessory options. Tables that provide full-function tilt, Trendelenburg, reverse Trendelenburg, and other positioning capabilities are particularly useful. Tables with removable leg sections are convenient for orthopedic procedures. Specialized tables are available for intraoperative imaging when 100% of the tabletop is radiolucent. Portable V-troughs and insulating table pads should be available. Special tabletop attachments that allow the anesthesiologist to see the patient's head should be provided so the patient can be monitored without contamination of the surgical field.

An instrument table (e.g., Mayo stand) or a back table should be available. The table should be large enough to accommodate all instrumentation required for the surgical procedure. Instrument tables should be made of stainless steel and be adjustable in height. A kick bucket is used by surgical team members to discard soiled sponges during surgery. The bucket frame should have wheels so it can be moved easily (i.e., kicked) about the OR. Plastic bag liners for kick bucket containers facilitate cleanup.

Suction (portable or piped in; Fig. 3.4) should be available in each OR. Suction units with disposable containers are reliable, easily cleaned, and cost-efficient. Suction hoses should not be reused unless they are sterilized because they are a common source of surgical wound contamination. Other accessory equipment, such as physiologic monitors, the anesthesia supply cart, intravenous stands, and sitting stools, should be available. Each OR should be provided with a method to view radiographs (radiographic view box preferably flush-mounted to facilitate cleaning, or a digital screen). Portable imaging devices are optimal for evaluating placement of orthopedic implants. The OR should have a wall clock to determine elapsed time, particularly when vascular occlusion is necessary. Supply cabinets with tight-fitting doors (to minimize dust accumulation) may be located in each OR for storing suture material, dressings, sponges, scalpel blades, and frequently used instruments. Doors to the OR should be kept closed to reduce the mixing of OR air with corridor air.

Patient body temperature must be maintained during surgery, especially if the animal weighs less than 10 kg, or if the surgical procedure is prolonged. Hypothermia has numerous deleterious effects on the patient, including decreased cerebral blood flow, vasodilation, increased cardiac irritability, and decreased cardiac output.[5] Importantly, heat loss is significant after the animal has been premedicated; thus attempts should be made to prevent the deleterious effects of hypothermia as early as possible, but certainly as soon after anesthetic induction as is feasible. In a 2015 study, prewarming dogs weighing less than 10 kg in an incubator prior to inhalation anesthesia did not improve or maintain their body temperature during inhalation anesthesia with isoflurane in oxygen.[6] Warming inhaled gases (e.g., Darvall's anesthesia breathing circuits) might be more effective than warming blankets or devices, including providing warmed intravenous fluids. Different types of patient warming devices are available in veterinary medicine, including warm-air circulating devices (e.g., Bair Hugger, Hot Dog Patient Warming System, Darvall Vet Cocoon), water circulating systems, and electric warming pads. The ChillBuster Vet Surgical System (ThermoGear) consists of a surgical warming blanket, warming cage mat, and disposable covers to keep the surgical and recovery areas free of contamination. Some systems now come with a HEPA filter that is built into the unit so that dirt and debris are not recirculated (e.g., SurgiVet Equator Convective Warming System, Smiths Medical). These units may also have a thermostat that is located at the blanket itself, rather than at the heating unit, which may prevent overheating and burning of the patient. Electric-style pads are increasing in popularity because the temperature is fixed (38.9°C [102°F]), they can be cleaned and sanitized, and unlike water circulating pads, they cannot be punctured. Triple lumen fluid warmers (e.g., SurgiVet Hotline fluid warmer [Smiths Medical]) that deliver fluids and blood products to the patient at a consistent temperature may be of some value during surgery and postoperative care of surgical patients.

Room "Turnover"

Standard practice is to routinely clean rooms after each operation to provide a clean environment for the next one. Centers for Disease Control and Prevention guidelines specify use of a one-step process and a hospital detergent registered with the Environmental Protection Agency (EPA) and designed for general housekeeping purposes when blood or body fluid contamination of surfaces may occur. Likewise, the Occupational Safety and Health Administration requires that any equipment or surfaces contaminated with blood or potentially infectious agents should be cleaned and decontaminated. If single-use, disposable mops and cloths are not utilized, mop heads and cloths should be

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cleaned after each use and allowed to dry before reuse. After the last surgical procedure of the day or night, OR floors should be wet mopped with a single-use mop and an EPA-registered hospital disinfectant (see Table 3.1). Tacky surfaces and mats should not be used at the entrances of ORs. Ultraviolet lights also should not be used. Despite the practice of closing ORs or following special procedures to clean rooms after contaminated or dirty operations, no data support such practices.[4]

Postoperative Recovery Area

The postoperative recovery area should be adjacent to the surgical area, yet separate from other hospitalized patients. Patients should be placed in individual, heated cages and carefully monitored until recovery is complete. Patients that require intensive care should be taken directly to the critical care facility. Temperature in the recovery room should be warmer than in the OR (i.e., 21°C–25°C [70°F–77°F]). Warming cabinets with a supply of warm fluids and blankets should be available. Analgesic medications and any equipment or medications that may be necessary in an emergency (e.g., defibrillator, laryngoscopes, endotracheal tubes, suction, oxygen, the crash cart) should be available.

Minor Procedures Surgery Room

A separate room adjacent to the anesthesia preparation area should be designated for minor contaminated surgical procedures (e.g., lacerations, biopsies, wound management, dental procedures, endoscopy). The room should be equipped with an operating table, spotlight, gas and suction lines for anesthesia equipment, suture material, antiseptic preparation materials, and instrument packs for minor surgery. Because of the nature of surgical procedures performed in this room, it should be properly cleaned and disinfected after each surgical procedure and at the end of each surgery day.

PERSONNEL

Responsibilities and functions of every member of the surgical team should be clearly defined in writing. This is done to clarify the job description and establish the accountability of each employee. These policies must be carefully followed and strictly enforced to ensure safe and efficient operation of the surgical area. All staff members should be evaluated periodically. Provision should be made for training programs, educational self-improvement, and information dissemination, and current books, periodicals, and audiovisual tapes of new procedures and techniques should be available.

The surgeon's role is to guide the flow and scope of what happens in the OR during surgery. Surgical assistance is often provided by a veterinary technician or nurse. Surgical assistants carry out functions that assist the surgeon in performing a safe operation, including developing a working knowledge of the procedure being performed, providing retraction and hemostasis, and manipulating instrumentation and tissues into proper position to complete the surgical task. A knowledgeable surgical assistant is invaluable. Both veterinary technicians and assistants must practice the hospital policy and procedures, involving a unified approach to aseptic technique.

An anesthesiologist is responsible for meticulous monitoring and adjustment of the patient's physiologic status during surgery. Anesthesiologists are trained to render immediate care in a physiologic crisis. Occasionally, the surgeon and the anesthesiologist must work together to carefully time surgical maneuvers, as in cardiothoracic surgery. A properly trained anesthesiologist allows the surgeon to concentrate on the surgical procedure.

Operating Room Supervisor and Surgical Technician

In large facilities, the OR supervisor oversees technicians working in the surgical area. This individual is responsible for organizing work schedules, training new staff, setting policies for the surgical area, implementing and enforcing policies, and developing educational programs and seminars. The OR supervisor also participates in the day-to-day technical aspects of running a surgical area (i.e., circulates, opens surgical packs, and retrieves special instruments).

In a small facility (i.e., one that has only one or two ORs), the OR supervisor assumes all the tasks of the surgical technician already mentioned. The supervisor may also have other technical tasks to perform as a veterinary technician, such as administering anesthetics, providing restraint, and serving as receptionist. Qualifications of a well-educated technician include graduation from an approved veterinary technician/nurse program and 1 to 2 years of basic training in a veterinary practice or veterinary teaching hospital.

REPAIRS AND RENOVATIONS

In human hospitals, nosocomial infections ranging from *Aspergillus* and mold in ceilings to *Legionella* in wet areas prompted support of mandatory proactive infection control risk assessments (ICRAs) by the Joint Commission for the Accreditation of Hospitals, the American Society of Hospital Epidemiologists, and the American Institute of Architects. ICRAs should be initiated in the earliest stages of the design or development phase of a project to identify potential infectious risks to the patient population, any risks associated with the mechanical systems of the building, and areas that will be influenced by the project. Goals include defining necessary containment measures (e.g., traffic diversion, waste removal, and work disruption) during construction and anticipating any safety hazards that can result from the construction.[4]

Care and Maintenance of the Surgical Environment

Surgery puts patients at risk for nosocomial (hospital-acquired) infection unless strict environmental, equipment care, and maintenance standards are established and followed. Because most surgical infections develop from bacteria entering the incision site during surgery, proper preparation of the surgical environment is crucial in reducing the likelihood of infection.

The OR is considered a restricted area, and appropriate attire must be worn by all personnel entering or leaving it (see Chapter 6). To keep the surgical environment as free of microorganisms as possible, routine cleaning and disinfection should be performed. The term **cleaning** refers to removal of soil (i.e., blood, serum, urine, or pus); the term **disinfection** refers to treatment of surfaces, materials, and equipment with chemicals to reduce bacterial numbers (see also Chapter 2). Cleaning and disinfection are usually performed simultaneously, except when large quantities of organic material or other body fluids are present.

DAILY CLEANING ROUTINES

Operating Room

At the beginning of each surgery day, all horizontal surfaces, lights, OR equipment, and furniture should be damp dusted

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BOX 3.2 Daily Care and Maintenance of the Operating Room

At the Beginning of Each Day

- Wipe flat surfaces of furnishings and lights with a cloth dampened with a disinfectant solution.

After Each Surgical Procedure

- Collect used instruments and place them in a cool water and detergent or enzymatic solution.
- Collect waste materials and soiled linens and place them in the proper containers.
- Wipe instrument and surgical tables, stands, kick buckets, and heating pads with a disinfectant.
- If necessary, clean the floor (move the surgical table and clean under it if body fluids have collected there).

After the Last Surgical Procedure of the Day

- Clean and disinfect kick buckets.
- Check ceilings, walls, cabinet doors, counter surfaces, and all furniture, and clean as necessary.
- Clean and care for individual items (e.g., monitoring devices, anesthesia equipment, surgical lights) according to the manufacturer's instructions.
- Wipe counter surfaces and cabinet doors with a disinfectant solution.
- Wipe instrument and surgical tables, stands, heating pads, and light fixtures with a disinfectant solution. Disassemble the surgical table if necessary to clean it thoroughly.
- Check supplies and restock as necessary.
- Roll wheeled equipment (e.g., surgical table, monitoring devices) through a small amount of disinfectant solution placed on the floor.
- Wet vacuum or damp mop the floor.

with a lint-free cloth and hospital-grade disinfectant (Box 3.2). After each surgical procedure, areas contaminated by organic debris (e.g., floors, doors, counters, equipment, operating table) should be cleaned and disinfected. If biohazards (i.e., infectious diseases or chemotherapeutic agents) are encountered during surgery, special precautions (i.e., specific disinfectant, cleaning time, and disinfectant contact time) should be taken during cleaning and disinfection.

At the close of each day, operating tables, counters, lights, equipment, floors, windows, cabinets, and doors should be cleaned and disinfected in preparation for the next day's activities. Linen and waste bags should be collected, the linen laundered, and waste disposed of properly. Kick buckets should be disinfected and lined with new plastic bags. Surgical lights and monitoring and anesthetic equipment are cleaned and disinfected according to manufacturers' specifications. Wheels and coasters of all movable equipment and gurneys are cleaned and disinfected. The OR should be restocked with commonly used instruments, suture material, gauze sponges, needles, and syringes, and the floor should be wet vacuumed or damp mopped. Wet vacuuming is preferred because wet mops support heavy bacterial growth and can be a major source of contamination and infection. If mops are used, mop heads should be laundered and dried daily. They should be rinsed between uses and soaked in disinfectant.

Scrub Sinks

Scrub sink areas need special attention during the day because water is frequently splashed on floors and walls, and blood and other organic debris can be tracked from the scrub sink area to the surgical suite (Box 3.3). Water and soap splashed on the floor should be routinely removed to avoid slips, falls, and other accidents by surgical personnel. This area should be cleaned as needed throughout the day, with used scrub brushes and fingernail cleaners removed, soap dispensers cleaned, and sinks and walls washed. This area should be disinfected at the end of the day.

BOX 3.3 Daily Care and Maintenance of the Scrub Room and Sinks

Between Scrubbing Sessions

- Dispose of wrappings from packs.
- Dispose of debris in sinks.

After the Last Surgical Procedure of the Day

- Remove waste and clean waste receptacles with a disinfectant. Line waste receptacles with a plastic bag.
- Check supplies and restock.
- Clean and refill soap dispensers.
- Wipe counter surfaces, cabinet doors, walls adjacent to sink, and switch plates.
- Scrub and disinfect sinks.
- Wet vacuum or damp mop the floor.

BOX 3.4 Daily Care and Maintenance of the Patient Preparation Area

Between Patient Preparations

- Discard waste material (e.g., feces).
- Properly dispose of urine and clean the sink.
- Remove hair from clipper blades and lubricate according to manufacturer's instructions.
- Check walls, counters, and cabinet doors and clean with a disinfectant if necessary.
- Vacuum and clean the floor as necessary to remove hair clippings.

At the End of the Day

- Remove waste and clean waste receptacles with a disinfectant. Line waste receptacles with a plastic bag.
- Wipe light fixtures and supply lines with a disinfectant.
- Clean clippers according to manufacturer's instructions.
- Vacuum the floor to remove hair clippings. Change the filter in the vacuum. Wipe the outside of the vacuum, the hose, and the nozzle with a disinfectant.
- Check the walls and ceiling, and clean as necessary.
- Check supplies and restock.
- Wipe counter surfaces, cabinet doors, walls adjacent to sink, and switch plates with a disinfectant.
- Scrub and disinfect sinks.
- Wet vacuum or damp mop the floor.

Anesthesia and Patient Preparation Room

Sinks, vacuum canisters, trash buckets, gurneys, and anesthesia preparation tables should be kept clean of organic debris and disinfected as necessary throughout the day (Box 3.4). Hair removed during patient preparation should be vacuumed from surgical tables and floors. Blood, urine, feces, tissue, serum, and purulent material should be contained and discarded. Needles and other sharp instruments should be disposed of in appropriate containers. Discarded biohazard materials should be disposed of in color-coded bags or should be clearly marked as a biohazard.

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Plumbing fixtures, floors, cabinets, anesthetic equipment, utility rooms, furniture, and other equipment should be cleaned and disinfected daily. At the end of the day, the sink in the preparation area should be disinfected and a cup of disinfectant solution poured down the drain. The inner surface of garbage containers should be disinfected. Bags and filters of portable vacuums should be removed and replaced as necessary; the outside surfaces of the vacuum (including the hose and nozzle) should be wiped clean and disinfected. Clippers should be cleaned according to manufacturer's instructions. Floors should be wet vacuumed or damp mopped and supplies restocked. If a mop is used, the mop bucket should be emptied and cleaned, and all cleaning equipment and supplies should be returned to a designated storage closet.

Recovery Room

Cages, sinks, trash buckets, and gurneys should be cleaned of organic debris and disinfected as needed throughout the day. Plumbing fixtures, floors, cabinets, anesthetic equipment, utility rooms, furniture, and other equipment should be cleaned and disinfected daily as described in the previous section.

After a surgical patient has vacated a recovery room cage, it must be carefully disinfected before it is used by the next patient. Before the cage is disinfected, padding, paper, and organic matter should be removed. Disinfectant should be sprayed on all surfaces of the cage, including the door. Dry organic matter should be scrubbed with a brush until it is released. Lastly, the area in front of the cage should be cleaned and disinfected. Linen (e.g., pads, blankets, and heating blanket covers) should be laundered before reuse. Plastic circulating-water heating blankets should be cleaned and disinfected. This protocol helps maintain a consistently low level of microbes in the surgical recovery area, which reduces the incidence of nosocomial infection. However, some infectious diseases (e.g., parvovirus) require special precautions.

WEEKLY AND MONTHLY CLEANING ROUTINES

Surgical suites should be emptied of movable equipment and thoroughly cleaned once a week. Shelves of supply cabinets, walls, windows, windowsills, ceilings, light fixtures, surgical tables, utility and supply carts (and castors), utility rooms, equipment storage areas, and infrequently used equipment also should be cleaned and disinfected. OR floors should be wet vacuumed at least weekly, and the grills of ventilation ducts vacuumed. Walls, floors, and ceilings should be mopped once a month, and wheels and other movable parts of equipment and gurneys should be lubricated.

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4

Preoperative and Intraoperative Care of the Surgical Patient



The selection and preparation of surgical patients require attention to a number of details. The patient should always receive a complete physical examination, followed by the appropriate laboratory workup. A thorough history helps determine the extent of the physical and laboratory examinations. Obtaining preoperative information also allows comparison of the animal's status before and after surgery (e.g., ability to micturate before and after spinal surgery). General assessment and stabilization of the surgical patient are discussed in this chapter; preoperative considerations for specific diseases are provided throughout the text.

HISTORY TAKING

A thorough history from the owner or caregiver aids in evaluation of the underlying disease process and identification of other abnormalities that might affect the surgical outcome. Although in emergencies an abbreviated history is often necessary, eventually a thorough history should always be obtained. The history should include the signalment, diet, exercise, environment, past medical problems, recent treatments (especially antiinflammatory, antimicrobial, and potentially nephrotoxic or hepatotoxic therapy), and any evidence of infection. Before embarking upon a detailed chronology of endless detail, the presenting complaint should be defined, specifically: (1) When did the current problem start? (2) What did the problem look like when it first began? and (3) Has the problem gotten better or worse, and if so, how? After the chief complaint has been defined, then one has a framework in which the details will make more sense.

Questions should be framed to avoid vague responses and to obtain specific information. For example, "When was your dog last vaccinated?" is a better question than "Is your dog current on his vaccinations?" Vomiting, diarrhea, altered appetite, exposure to toxins or foreign bodies, coughing, exercise intolerance, and other abnormalities should be noted. Animals with a history of previous drug reactions or seizures must be identified so that certain drugs can be avoided.

PHYSICAL EXAMINATION

The animal should be systematically evaluated during a physical examination, in which all body systems are evaluated. The animal's general condition (body condition, attitude, and mental status) should be noted. If indicated, animals should have a neurologic examination (see Chapter 38) and an orthopedic examination (see Chapter 31) in addition to evaluation of the respiratory, gastrointestinal, cardiovascular, and urinary systems. Emergencies may allow only a cursory examination until the animal has been stabilized. Evaluation of the preanesthetic physical status (Table 4.1) is a good determinant of the likelihood of cardiopulmonary emergencies during or after surgery; the more deteriorated the physical status, the higher the risk of anesthetic and surgical complications.

LABORATORY DATA

The animal's physical status and the procedure to be performed dictate the extensiveness of the laboratory workup. Determination of the hematocrit, total protein (TP), blood urea nitrogen or preferably serum creatinine concentration, and urine specific gravity may suffice for young, healthy animals undergoing elective procedures (e.g., ovariohysterectomy and declawing) and in healthy animals with localized disease (e.g., patellar luxation). If the animal is older than 5 to 7 years, even with a physical status of I or II (see Table 4.1), or has systemic signs (e.g., dyspnea, heart murmur, anemia, ruptured bladder, gastric dilation-volvulus, shock, and hemorrhage), and/or if the anticipated surgery time is longer than 1 to 2 hours, a complete blood count, serum biochemistry profile, and urinalysis should be done. Venous blood gas analysis could also be considered to determine the animal's acid-base status.

The necessity for additional laboratory data is dictated by the animal's presenting signs and underlying disease (Table 4.2). Identification of associated or underlying disease influences preoperative management, the surgical procedure performed, the prognosis, and postoperative care. Animals with neoplasia should be evaluated for metastasis (e.g., thoracic radiographs, abdominal ultrasound, and/or lymph node aspiration). Those with cardiac disease should have thoracic radiographs, cardiac ultrasound scans, and/or electrocardiograms (see Chapter 27). In endemic areas, the patient's heartworm status should be checked before surgery. Traumatized animals should have thoracic radiographs so that the diaphragm, pleural space, and lungs can be evaluated for conditions such as pulmonary contusion, pneumothorax, pleural effusion, or diaphragmatic hernia. Full-body sedated computed tomography (CT) exams can also be considered for traumatized animals to assess for unidentified injuries. Although economic considerations are important, a thorough preoperative examination is cost-effective because it often prevents or predicts costly complications.

NOTE Remember that there are age-related differences in hematologic and serum biochemical values in dogs. Growth and maturation of puppies influences some of these values such that they diverge greatly from those for adults (e.g., white blood cell count, red blood cell count, hematocrit, alkaline phosphatase activity, hemoglobin, calcium, phosphorus, protein, and globulin concentrations).

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ABSTRACT

The surgical patient requires a thorough assessment before sedation, anesthesia, or surgical intervention in order to rate the physical status of the animal and determine surgical risk. The patient's history, clinical signs, physical examination findings, laboratory test results, and cursory diagnostic imaging findings are all critical for complete evaluation. If indicated and if possible, the patient should receive stabilization measures before surgery, including but not limited to oxygen therapy, fluid therapy, and correction of acid-base and electrolyte abnormalities.

KEYWORDS

patient assessment
stabilization
oxygen therapy
fluid therapy
checklists

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TABLE 4.1 Rating of Physical Status in Surgical Patients

Physical Status	Animal's Condition	Examples
I	Healthy with no discernible disease	Patient is undergoing an elective procedure (e.g., ovariohysterectomy, declaw, castration)
II	Healthy with localized disease or mild systemic disease	Patellar luxation, skin tumor, cleft palate without aspiration pneumonia
III	Severe systemic disease	Pneumonia, fever, dehydration, heart murmur, anemia
IV	Severe systemic disease that is life threatening	Heart failure, renal failure, hepatic failure, severe hypovolemia, severe hemorrhage
V	Moribund; patient not expected to live longer than 24 hours with or without surgery	Endotoxic shock, multiorgan failure, severe trauma

DETERMINATION OF SURGICAL RISK

Once the history, physical examination, and laboratory tests have been completed, the surgical risk can be estimated and a prognosis given (Table 4.3). An *excellent prognosis* should be given if the potential for complications is minimal and there is a high probability that the patient will return to normal after surgery. If there is a high probability of a good outcome but some potential for complications, a *good prognosis* is warranted. If serious complications are possible but uncommon, if recovery may be prolonged, or if the animal may not return to its presurgical function, a *fair prognosis* is warranted. If the underlying disease or the surgical procedure is associated with many or severe complications (or both), if recovery is expected to be prolonged, if the likelihood of death during or after the procedure is high, or if the animal is unlikely to return to its presurgical function, a *poor prognosis* should be given. A *guarded prognosis* is often given when the outcome is highly variable or unknown.

Occasionally the risk of the surgical procedure may outweigh its potential benefits. For example, removal of an apparently benign skin mass may not be warranted in an animal with hepatic or renal dysfunction. Likewise, patients with thoracic metastases may not benefit from removal of the primary tumor. Quality of life must be considered for veterinary patients; those with severe, debilitating, untreatable disease may not benefit from surgery. However, for some patients surgery may improve the quality of life even if length of life is limited.

CLIENT COMMUNICATION

Communication with the client is extremely important to ensure the owner's satisfaction after surgery. Owners should be informed before surgery of the diagnosis, surgical or nonsurgical options, potential complications, postoperative care, prognosis, and cost. Although cost cannot always be predicted because of unanticipated complications, owners should be kept apprised of the animal's status and of procedures that may affect the initial cost estimate. If the disease is hereditary, neutering should be recommended. A waiver signed by the owner, authorizing surgery and accepting anesthetic and surgical risks, is mandatory and should be part of the medical record.

PATIENT STABILIZATION

Patients should be stabilized as thoroughly as possible before surgery. Occasionally, stabilization is impossible, and surgical intervention must be done rapidly; however, replacing fluid deficits and correcting acid-base and electrolyte abnormalities before induction of anesthesia usually are justified. Intravenous (IV) fluids are indicated for all animals undergoing general anesthesia and surgery, including healthy animals having elective procedures. The need for perioperative antibiotics is dictated by the animal's disease and the procedure being performed. Recommendations for antibiotic prophylaxis and therapy are given with discussions of specific diseases throughout this text. Perioperative antibiotic use is discussed in Chapter 9.

The patient history, clinical signs, physical examination findings, electrolytes, and total carbon dioxide (CO_2) are helpful in identifying significant acid-base abnormalities. The blood pH, arterial oxygen partial pressure (PaO_2), arterial carbon dioxide partial pressure ($PaCO_2$), and bicarbonate concentration may be measured to determine the extent of such abnormalities. If the animal is notably acidemic (arterial pH <7.2), efforts to optimize ventilation and capillary perfusion should be instituted. As a result of production and retention of CO_2 in the tissues, correcting base deficits with sodium bicarbonate without concurrent ventilatory and hemodynamic support may be detrimental; most acidotic patients do not require bicarbonate administration. The amount of bicarbonate necessary to give for a given base deficit can be calculated using the formula in Box 4.1.

The patient's nutritional state is often critical in chronically diseased animals. Preoperative parenteral or enteral hyperalimentation (see Chapter 10) is sometimes recommended to

BOX 4.1 Calculation of Volumes Needed for Blood Transfusion or Bicarbonate Therapy

Blood Transfusion

Blood needed (mL) = Recipient's weight (kg) × Desired PCV – Recipient PCV Donor's PCV × 70 (cat) or 90 (dog)[a]

Note: A rough estimate is 2.2 mL of blood/kg of body weight increases the recipient's PCV by 1%.

Bicarbonate Therapy

Bicarbonate needed (mEq) = 0.3 × Base deficit[b] (mEq) × Body weight (kg)

Give one half intravenously (IV) over 10 to 15 minutes and reevaluate; give remainder over 4 to 6 hours if necessary *or* give 1–2 mEq/kg IV; repeat only if indicated based on assessment of acid-base balance and potassium concentration.

Note: Because carbon dioxide is an end product of bicarbonate administration, ensure adequate ventilation.

[a]Total blood volume is estimated at 90 mL/kg for dogs and 70 mL/kg for cats.
[b]Some calculate the base deficit as the difference between the desired bicarbonate and the actual bicarbonate (rather than the normal bicarbonate and the actual bicarbonate). Animals that are acidotic enough to require bicarbonate therapy need continual monitoring.
PCV, Packed cell volume.

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TABLE 4.2 Brief Considerations for Selected Clinical Pathologic Findings

Laboratory Abnormality	Comments	Major Differential Diagnoses
High BUN	Obtain urine specific gravity before initiating fluid therapy; measure serum creatinine concentration	Prerenal azotemia, primary renal disease, postrenal azotemia
Low BUN		Hepatic insufficiency (e.g., portosystemic shunt, cirrhosis), severe polyuria-polydipsia, low-protein diet
High ALT	ALT may be normal in some animals with severe hepatic disease	Hepatic disease: the magnitude of the increase in ALT is neither diagnostic for any particular disease nor prognostic; severe muscle disease can cause increases in ALT
Low albumin	Substantial inconsistencies between laboratories, methodology used to measure albumin in people can severely underestimate canine albumin concentration	Hepatic disease, loss from kidneys or gastrointestinal tract, severe exudative cutaneous lesion (e.g., burn); lack of nutrition is not the sole cause for a serum albumin <2.0 g/dL
High SAP	Commonly elevated in young growing animals or caused by steroids or anticonvulsants; falsely elevated with severe lipemia or severe bilirubinemia (>8 g/dL)	Hepatic disease, steroid therapy, extrahepatic biliary obstruction, some neoplasms; many dogs with increased SAP as the only biochemical abnormality do not have clinically significant hepatobiliary tract disease
High bilirubin	Exposure to fluorescent light may degrade bilirubin	Hepatocellular disease, extrahepatic biliary obstruction, intrahepatic cholestasis, hemolytic anemia, severe sepsis
High calcium	It is probably best to measure ionized serum calcium because serum albumin concentrations can have a substantial effect on total serum calcium concentrations, masking hypercalcemia	Paraneoplastic syndrome (lymphosarcoma, anal sac adenocarcinoma), primary hyperparathyroidism, calciferol-containing rodenticides, hypervitaminosis D, hypoadrenocorticism, granulomatous disease, chronic renal failure
Low calcium	Artificially low in animals with low albumin	Renal disease (especially acute), pregnancy (eclampsia), hypovitaminosis D, hypoparathyroidism
High phosphorus	Normal in young, growing dogs	Renal failure (acute and more severe chronic)
Low phosphorus		Refeeding syndrome, excessive insulin—especially in ketoacidotic cats (can cause hemolysis)
High creatinine	Emaciated animals have a falsely reduced serum creatinine	Renal disease, prerenal azotemia, uroabdomen, muscle trauma (very minor elevations)
High glucose	Stress may increase glucose to 200–400 mg/dL in cats	Diabetes mellitus
Low glucose	Delayed separation of RBCs falsely lowers glucose	Hepatic disease, insulinoma, hypoadrenocorticism, extrahepatic neoplasms, septicemia or toxemia, starvation of neonates
High sodium	Primarily caused by loss of free water	Vomiting, diarrhea, renal failure, diabetes insipidus, inappropriate fluid therapy, adipsia for any reason, free water loss for any reason
High potassium	Thrombocytosis may falsely elevate potassium; hemolysis increases potassium in selected breeds	Hypoadrenocorticism, severe renal failure, uroabdomen, selected drugs
Low potassium		Vomiting, diarrhea, diuretic therapy, chronic renal failure (especially in cats), inappropriate fluid therapy, refeeding syndrome
High total CO_2		Usually means metabolic alkalosis due to vomiting gastric contents, excessive diuretic administration, sodium bicarbonate administration, inappropriate fluid therapy. Can also mean a compensated respiratory acidosis (exceedingly rare)
Low total CO_2		Usually means metabolic acidosis from any of a number of causes, rarely means compensated respiratory alkalosis
High eosinophils		Parasitism (heartworm), eosinophilic diseases, mast cell tumor, hypersensitivities
High basophils		Parasitism (heartworm), mast cell tumors
High lymphocytes	Can be found in young animals	± Lymphosarcoma, ± feline leukemia virus, chronic lymphocytic leukemia, some dogs with ehrlichiosis
Low lymphocytes		Severe stress, lymphangiectasia, chylothorax, acute viral diseases
High RBCs	Some breeds (e.g., greyhounds) normally have higher packed cell volume (e.g., 55%) than other breeds	Dehydration, polycythemia, hypoxia (right to left shunts)

ALT, Alanine aminotransferase; *BUN,* blood urea nitrogen; *RBC,* red blood cells; *SAP,* serum alkaline phosphatase.

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improve nutritional status before surgery. For example, in patients with cleft palate, cleaning particulate matter from the nasal cavity, administering the appropriate antibiotics, and providing enteral hyperalimentation for several weeks before surgery may reduce infection and improve wound healing.

Traumatized patients must be evaluated swiftly to detect life-threatening abnormalities. The cardiovascular and respiratory systems should be assessed by evaluating the pulse quality and rate, respiratory rate and effort, mucous membrane color, and capillary refill time (Videos 4.1 and 4.2). The heart should be

TABLE 4.3 Guidelines for Determining Surgical Prognosis

Prognosis	Criteria
Excellent	Potential for complications is minimal High probability that patient will return to normal after surgery
Good	Some potential for complications High probability of a good outcome
Fair	Serious complications are possible, but uncommon Recovery may be prolonged Animal may not return to its presurgical function
Poor	Underlying disease or surgical procedure is associated with many or severe complications Recovery is expected to be prolonged Likelihood of death during or after the procedure is high The animal is unlikely to return to its presurgical function
Guarded	Outcome is unknown or uncertain

BOX 4.2 Abdominal Focused Assessment With Sonography for Trauma (AFAST) Protocol to Detect Free Abdominal Fluid in Dogs (see Fig. 4.1)

The AFAST examination consists of two ultrasonographic views (transverse and longitudinal) in each of the following four areas:
1. Diaphragmaticohepatic (DH) view: probe placed subxiphoid
2. Splenorenal (SR) view: probe placed at left flank
3. Hepatorenal (HR) view: probe placed at right flank
4. Cystocolic (CC) view: probe placed at midline over urinary bladder

At each area, the ultrasound probe (5- or 7.5-MHz curvilinear probe) is fanned through an angle of 45 degrees from the long- and short-axis positions and is moved 2 to 3 cm (1 in) in each of four directions (i.e., cranial, caudal, left, and right) from the starting point until target organs are located.

auscultated for evidence of murmurs or an arrhythmia, and the lungs should be evaluated for crackles or wheezes. Diminished heart or lung sounds suggesting the presence of space-occupying pleural disease or a diaphragmatic hernia should be noted. Oxygen therapy should be given to animals that appear to be in respiratory distress or have other signs of oxygen deprivation. Thoracic radiographs or thoracic CT should be considered in traumatized patients regardless of the lack of obvious respiratory distress.

The use of ultrasound as a point-of-care test for animals presenting with emergent conditions has been gaining wide acceptance.[1] Abdominal and thoracic focused assessment with sonography for trauma (AFAST and TFAST) allow for detection of peritoneal, pleural, and pericardial fluid, in addition to rapidly identifying pneumothorax. The AFAST examination is a four-quadrant protocol (Box 4.2; Fig. 4.1) that is performed with the animal in right or left recumbency. An abdominal fluid score is recorded, ranging from 0 (no fluid in any quadrant) to 4 (peritoneal fluid in all quadrants). Serial AFAST examinations performed every 2 to 4 hours allow detection of delayed accumulation of peritoneal fluid. The TFAST examination is a five-point scan that evaluates the animal in both right and left lateral recumbencies (Box 4.3; Fig. 4.2).

OXYGEN THERAPY

Clinical signs of hypoxia include dyspnea, cyanosis, tachycardia, tachypnea, postural changes, anxiety, and/or central nervous system depression. If clinical signs, arterial blood gases, pulse oximetry, or the patient's disease suggest hypoxia, supplemental oxygen may be administered via mask, flow-by, or nasal catheter, or the animal may be placed in an oxygen cage or tent.

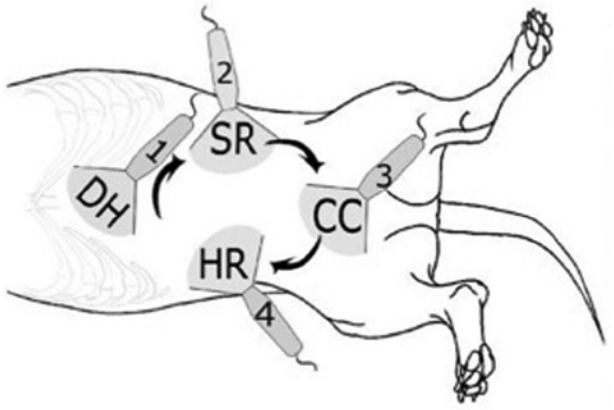


FIG. 4.1 Four views of the abdominal focused assessment with sonography for trauma (AFAST) examination: (1) diaphragmaticohepatic *(DH)*, (2) splenorenal *(SR)*, (3) cystocolic *(CC)*, and (4) hepatorenal *(HR)*. (From Boysen SR, Lisciandro GR. The use of ultrasound for dogs and cats in the emergency room. *Vet Clin North Am Small Anim Pract.* 2013;43[4]:773–797.)

BOX 4.3 Thoracic Focused Assessment With Sonography for Trauma (TFAST) Protocol to Detect Pleural Fluid, Pericardial Fluid, and Pneumothorax in Dogs (see Fig. 4.2)

The TFAST examination consists of examination of five areas:
1. Left and right chest tube site (CTS): probe placed at the seventh to ninth dorsolateral intercostal space
2. Left and right pericardial sac (PCS) view: probe placed at the fifth to sixth ventrolateral intercostal space
3. Diaphragmaticohepatic (DH) view: probe placed subxiphoid

NOTE Remember that patients can be hypoxic without showing signs of cyanosis because greater than 5 g/dL of deoxygenated hemoglobin must be present in the circulation before cyanosis can be detected. The patient must have a PCV of approximately 15% to have 5 g hemoglobin/dL.

Flow-by oxygen may be the easiest way to provide supplemental oxygen in an emergency situation (Fig. 4.3; Table 4.4). The oxygen line is placed within 1 to 3 cm of the patient's nose and mouth, which creates a small area where the fraction of inspired oxygen (F_iO_2) is increased. However, because it requires a care provider to be present to hold the oxygen line and to ensure that the patient does not move away from it, and because a high oxygen flow rate is required, it is not always practical or the best choice. Furthermore, it is not nearly as effective as the other methods described below.

Face mask delivery of oxygen is a useful short-term method to provide supplemental oxygen. With an oxygen flow rate of 6

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TABLE 4.4 Methods for Supplementing Oxygen

Mode of Oxygen Delivery	Indication	Oxygen Flow Rate	Fraction of Inspired Oxygen
Face mask	Short-term emergency stabilization	6–10 L/min (be sure face mask fits well)	35%–55%
Flow-by	Short-term emergency stabilization; face mask not tolerated	6–8 L/min	25%–45%
Tent or Elizabethan collar canopy	Nasal catheter not tolerated; oxygen cage not available	0.75–1 L/min	30%–40%
Nasal catheter	Postoperative oxygen delivery; prolonged delivery	1–6 L/min; 50–100 mL/kg/min	30%–50%
Nasotracheal tube	Upper airway obstruction; nasal catheter not tolerated	50 mL/kg/min	40%–60%
Oxygen cage	Prolonged delivery of oxygen; limited access to patient		40%–50%

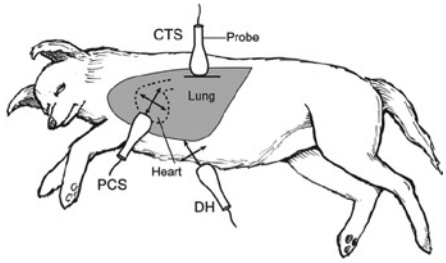


FIG. 4.2 Five views of the thoracic focused assessment with sonography for trauma (TFAST) examination: (1) right and left chest tube site *(CTS)*, (2) right and left pericardial sac *(PCS)*, (3) midline diaphragmaticohepatic *(DH)*. (From Boysen SR, Lisciandro GR. The use of ultrasound for dogs and cats in the emergency room. *Vet Clin North Am Small Anim Pract.* 2013;43[4]: 773–797.)



FIG. 4.3 A 12-year-old large mixed-breed dog receiving flow-by oxygen supplementation.



FIG. 4.4 Makeshift oxygen tent.

to 10 L/min and a well-fitted mask, an F_iO_2 of 0.35 to 0.55 may be achieved (see Table 4.4). Be aware that face masks may not be tolerated (especially in severely dyspneic animals) and are often difficult to fit well to the faces of cats and brachycephalic dogs. An alternative is to use an Elizabethan collar covered with plastic wrap to create an oxygen-enriched environment. The end of the oxygen tube should be fed up through the collar and secured (Fig. 4.4). To allow elimination of CO_2, make a small hole in the plastic wrap.

Nasal catheters may be used when more prolonged oxygen delivery is desired than can be achieved with flow-by or face mask techniques (Box 4.4). Other advantages of nasal catheter delivery of oxygen are that it permits access to the patient without loss of the oxygen-rich environment (versus an oxygen cage), and it is well tolerated in most patients. When necessary, bilateral catheters can be placed. The appropriate oxygen flow rate is based upon assessment of the degree of respiratory distress, the patient's respiratory rate and pattern, and the patient's size. The recommended initial therapeutic dose for unilateral nasal oxygen supplementation is approximately 50 to 100 mL/kg per minute. These flow rates can achieve a tracheal F_iO_2 of approximately 50%. Although high gas flow rates can be administered through a single nasal catheter, these high flow rates are associated with patient discomfort, nasal turbinate desiccation, and airway constriction. In such cases, administering oxygen through bilateral nasal catheters can improve patient comfort. Nasotracheal tubes

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BOX 4.4 Nasal Oxygen Insufflation

1. Select a small, red rubber feeding tube (3.5–5 Fr for cats, 5–8 Fr for dogs) to serve as a catheter and lubricate the tip with lidocaine gel.
2. Place one or two drops of local anesthetic (e.g., 2% lidocaine or proparacaine) in the nostril.
3. Premeasure the catheter to the medial canthus of the eye or the caudal ramus of the mandible.
4. Elevate the dorsal aspect of the nose and feed the lubricated catheter into the nostril the predetermined distance.
5. Suture or glue (e.g., VetBond) the catheter to the external naris and muzzle, and over the frontal sinus or along the jaw. With cats, do not allow the tube to touch the whiskers.
6. Place an Elizabethan collar on the animal.
7. Attach the tube to an oxygen source and administer humidified oxygen to maintain oxygen saturation at greater than 90%; typically start at 50 mL/kg/min and adjust as necessary.[a]

[a]Gastric distention may occur if the flow rate is too high.



FIG. 4.5 A 5-year-old English Bulldog with nasotracheal intubation to facilitate postoperative recovery.

can be used in animals, particularly brachycephalic breeds, which will not tolerate an intranasal catheter (Fig. 4.5; see Table 4.4).

An oxygen cage provides a sealed environment in which the F_iO_2, ambient temperature, and humidity can be controlled. An ambient temperature of 70°F and a relative humidity of 40% to 50% is desired. The major disadvantage of an oxygen cage is that it isolates the patient from the clinician because each time the oxygen cage door is opened there is a loss of the oxygen-rich environment.

FLUID THERAPY

Fluid therapy should be initiated if hemorrhage or shock is suspected (Table 4.5). The normal blood volume of dogs is approximately 90 mL/kg and that of cats is approximately 60 to 70 mL/kg. Treatment of acute hypovolemia is intended to establish a circulating blood volume that allows adequate tissue perfusion. Generally, hypovolemic patients can be given polyionic isotonic fluid intravenously in the first hour (60–90 mL/kg in dogs, 45–60 mL/kg in cats) without adverse effects; however, patients with pulmonary, cardiovascular, or severe renal disease may be less tolerant of rapid fluid administration. Commonly, one-quarter to one-half the calculated shock dose is administered over 15 to 30 minutes, and the patient is carefully reassessed for changes in vital signs. If hemodilution is not a concern, balanced electrolyte solutions (e.g., lactated Ringer's solution, Normosol-R) may be given. The duration of action of infused crystalloids is short, with only approximately 10% of the solution remaining in the intravascular space at 1 hour.

Limited-volume fluid resuscitation (LVFR) is an alternate means for animals presenting with hemorrhagic shock. The smallest fluid volume required to restore intravascular volume is used, thereby minimizing extravasation of fluid into the interstitium and decreasing the risk of blood clot disruption. LVFR protocols typically use hypertonic saline or colloids instead of crystalloid solutions.

Hypertonic saline solutions are beneficial for reducing total fluid requirements, limiting edema, and increasing cardiac output. Adding a colloid (e.g., hetastarch, plasma) to hypertonic saline prolongs the effect of the volume expansion. However, animals with protein-losing nephropathy or enteropathy quickly lose the albumin they receive from plasma, making this an expensive and poorly effective therapy. Synthetic colloids are a better option in these patients. Colloids could also be considered for animals that are hypoproteinemic (i.e., total solids less than 4.5 g/dL). Fresh-frozen plasma (FFP) is beneficial for patients in need of coagulation factors because of consumption or dilution (e.g., when large doses of synthetic colloids have been given). The major preoperative concern in anemic patients is maintaining oxygen-carrying capacity; therefore transfusions (i.e., whole blood or packed red cells) may be necessary in anemic patients. Animals with a preoperative packed cell volume (PCV) less than or equal to 20% usually benefit from transfusions. The amount of donor blood necessary can be estimated by the formula presented in Box 4.1.

Crystalloid Solutions

Crystalloids are solutions containing electrolyte and nonelectrolyte solutes capable of entering all body fluid compartments. Advantages of crystalloid fluid therapy include replacement of interstitial and intravascular fluid losses, minimal impairment of coagulation, and no risk for allergic reaction, as well as low cost and wide availability. The main disadvantage is the limited duration of IV volume expansion when using crystalloid therapy alone. Large volume of crystalloid administration can contribute to decreased colloid oncotic pressure, resulting in tissue edema and other serious consequences.

Examples of crystalloid solutions include **Normosol-R, lactated Ringer's solution**, **5% dextrose**, **Plasma-lyte A**, and **normal saline (0.9%)**. Supplementation of these fluids with potassium chloride (KCl) is indicated if the patient is hypokalemic or likely to become so (e.g., from vomiting). A sliding scale for potassium addition to parenterally administered fluids is provided in Table 4.6. Thorough mixing of the crystalloid solution following addition of KCl should be performed to avoid potentially life-threatening infusion of potassium.

The choice of fluid to administer depends on the nature of the disease process and the composition of the body fluid losses. Patients vomiting gastric contents may become hypokalemic or hypochloremic. They can develop metabolic alkalosis (see p. 398), in which case 0.9% NaCl with 20 to 30 mEq KCl per liter is a reasonable choice, or they can become acidotic. The acid-base status cannot be accurately predicted. If the vomitus is not primarily stomach contents, lactated Ringer's may be used initially while awaiting laboratory results.

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TABLE 4.5 Blood and Fluid Products: Indications for Use and Doses

Product	Indications	Infusion Rate
Isotonic crystalloid solutions[a]	Shock Dehydration Maintenance	*Dogs:* Up to 90 mL/kg (to effect) *Cats:* Up to 60 mL/kg (to effect) Maintenance rate is approximately 66 mL/kg/day for a 10-kg dog; larger dogs need less (e.g., 44 mL/kg/day for a 40-kg dog) and smaller dogs need more (e.g., 81 mL/kg/day for a 5-kg dog)
Hetastarch	Shock Hypoalbuminemia	*Dogs:* 10–20 mL/kg/h (shock) *Cats:* 10–15 mL/kg/h over 10–15 min (shock) 5–10 mL/kg (can repeat) *or* constant-rate infusion (1–2 mL/kg/h) up to 20 mL/kg/day (hypoalbuminemia)
Pentastarch	Shock Hypoalbuminemia	*Dogs:* 10–20 mL/kg/h (shock) *Cats:* 10–15 mL/kg/h (shock) 5–10 mL/kg (can repeat) *or* constant-rate infusion (1–2 mL/kg/h) up to 20 mL/kg/day (hypoalbuminemia)
25% Human serum albumin	Shock Hypoalbuminemia	5–25 mL/kg; maximal volume 2–4 mL/kg (bolus or slow push) 0.1–1.7 mL/kg/h as a constant-rate infusion[b]
7% Hypertonic saline[c]	Shock Hypoalbuminemia	4 mL/kg over 5 min, then isotonic crystalloids (10–20 mL/kg/h) to effect
Fresh whole blood	Anemia Hemorrhage Coagulopathy Shock	10–22 mL/kg (see also Box 4.1); in general, 2 mL/kg will raise the PCV by 1% For shock: 22 mL/kg/h maximum
Stored whole blood	Anemia Hemorrhage	10–22 mL/kg (see also Box 4.1); in general, 2 mL/kg will raise the PCV by 1%
Packed red blood cells	Anemia Hemorrhage	6–10 mL/kg and then reassess PCV to determine if more is necessary; in general, 1 mL/kg will raise the PCV by 1%
Platelet-rich plasma	Thrombocytopenia Coagulopathy	1 unit/3–10 kg
Fresh-frozen plasma	Coagulopathy Hypoproteinemia DIC	10–20 mL/kg; then reassess serum albumin or AT III concentration to determine if more is necessary
Cryoprecipitate	Von Willebrand disease Hemophilia	1 unit/5–15 kg
Oxyglobin	Anemia Shock	*Dogs:* 15–30 mL/kg at a maximum rate of 10 mL/kg/h *Cats:* 5–10 mL/kg at a maximum rate of 5 mL/kg/h

[a]Monitor central venous pressure to prevent fluid overload.
[b]From Mathews KA, Barry M. The use of 25% human serum albumin: outcome and efficacy in raising serum albumin and systemic blood pressure in critically ill dogs and cats. *J Vet Emerg Crit Care.* 2005;15:110.
[c]To prolong the effect of hypertonic saline, hetastarch or other colloid may be administered simultaneously. Do not exceed the maximum rate for either fluid.
AT III, Antithrombin III; *DIC,* disseminated intravascular coagulation; *PCV,* packed cell volume.

TABLE 4.6 Sliding Scale for Potassium Supplementation

Serum Potassium (mEq/L)	mEq KCl to Add to 250 mL Fluid	Maximal Fluid Infusion Rate[a] (mL/kg/h)
<2.0	20	6
2.1–2.5	15	8
2.6–3.0	10	12
3.1–3.5	7	16

[a]Do not exceed 0.5 mEq/kg/h.

Hypertonic saline (7.2%) can be used for restoration of intravascular volume in patients with severe hypovolemic shock or head trauma. Its use requires preexisting normal hydration, and thus hypertonic saline is primarily useful in dogs or cats with sudden development of hypovolemia rather than hypovolemia from untreated dehydration. If it is given as a rapid bolus, hypotension may occur and can be fatal. Lower doses should be used in patients with cardiac disease, and central venous pressure should be monitored during administration. Hypertonic saline should be avoided in patients with severe dehydration and hyperosmolar conditions. It is given as an IV infusion (1 mL/kg per minute) at a dose of 4 to 6 mL/kg, or can be used for low-volume fluid resuscitation.

Colloid Solutions

Colloids are large-molecular-weight substances (e.g., plasma, dextrans, and hetastarch) that are restricted to the plasma compartment because of their size. These solutions are often used in animals in shock or that are severely hypoalbuminemic (i.e., serum albumin <1.5 g/dL); however, colloids should be used with caution due to the potential for adverse outcomes (see Note on next page). Factors that influence the duration and volume of intravascular expansion associated with artificial colloids are the species of animal, dose, specific colloid

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formulation, preinfusion intravascular volume status, and microvascular permeability. After administration, albumin rapidly disappears from the intravascular space; however, synthetic colloids contain molecules that vary in molecular weight. Smaller molecules are rapidly excreted, whereas the larger molecules remain in the circulation and are gradually hydrolyzed or removed by the reticuloendothelial system. The benefits of colloid therapy include rapid volume expansion with low volume administration as compared with crystalloids. The calculated volume of required crystalloid therapy can be reduced by 40% to 60% when administered concurrently with synthetic or natural colloid to achieve the same anticipated outcomes. Disadvantages of colloid solutions include possible allergic reactions, possible renal impairment, interference with coagulation, and increased expense.

The artificial colloids used most frequently in the United States are **hetastarch** and **dextran 70**. The recommended dosage for both is 20 mL/kg per day (see Table 4.5). All the commonly used artificial colloids can cause abnormal coagulation when large doses are given, when they are given repeatedly, or when there is reduced intravascular degradation. These coagulopathies may be associated with a reduction in factor VIII and von Willebrand factor. The low-molecular-weight dextrans (e.g., dextran 40) may be associated with acute renal failure and should not be used.

NOTE Use colloids judiciously as studies have suggested that their use may be associated with increased risk of acute kidney injury or death.[2]

Albumin has a molecular weight of approximately 69,000 Daltons and is most commonly given to small animal patients as stored plasma or FFP, stored whole blood, or fresh whole blood. Because it equilibrates with the interstitial space rapidly, relatively large volumes must be given to achieve a sustained rise in plasma colloid osmotic pressure (COP). Plasma can be administered at a rate of 4 to 6 mL/min in an acute hypovolemic crisis, whereas in normovolemic patients, plasma can be administered at 6 to 22 mL/kg over a 24-hour period.

Human serum albumin (HSA) can be used for volume expansion but is more often utilized for treatment of severe hypoalbuminemia. Hypoalbuminemia is often a consequence of critical illness and is associated with increases in morbidity and mortality in both humans and dogs (Box 4.5). HSA has been shown to effectively increase serum albumin, total solids, and COP in critically ill dogs. However, administration of HSA is controversial as it has (rarely) been associated with serious adverse reactions, including death. There are variable data in the veterinary literature regarding the incidence of complications with HSA use, with a low number of mild adverse reactions reported in some studies and several serious complications potentially attributable to HSA administration in others.[3] In addition, HSA has been shown to be highly antigenic in both ill and normal dogs, and presents a significant risk if administered more than once. Goals for HSA administration in dogs are to increase serum albumin to 2.0 to 2.5 g/dL and COP to 14 to 20 mmHg (Box 4.6).

Blood Products

Whole blood is the blood product most commonly transfused into dogs and cats. It contains donor blood plus anticoagulant. Although no standards have been established for the volume of blood that constitutes 1 unit, a human collection system generally contains approximately 450 mL of blood and 63 mL of anticoagulant and is designated as 1 unit. Whole blood contains red blood cells, clotting factors, proteins, and platelets. The initial dose is 10 to 22 mL/kg (see Table 4.5).

Packed red cells are the red cells and a small amount of plasma that remains after most of the plasma has been removed. Approximately 200 mL of packed red cells is obtained from 450 mL of whole blood. Because packed red cells do not contain clotting factors or platelets, they are typically used to treat anemia. The initial dose is 6 to 10 mL/kg (see Table 4.5). **FFP** is the plasma obtained from a unit of whole blood plus the anticoagulant. If frozen at −30°C (−22°F), clotting factors contained in FFP remain viable for approximately 1 year, whereas albumin is preserved for 5 years. The dose of plasma that is required to increase the albumin concentration of blood by 1 g/dL is approximately 45 mL/kg, which makes its use in most hypoproteinemic animals cost-prohibitive. It is typically used to treat coagulopathies that arise from a congenital deficiency of clotting factors (e.g., von Willebrand disease or hemophilia). The dose is 10 to 20 mL/kg. **Platelet-rich plasma** and platelet concentrates are prepared from fresh whole blood by slow centrifugation. The platelets are then suspended in plasma for transfusion. Transfused platelets may be rapidly destroyed in patients with immune-mediated thrombocytopenia. **Cryoprecipitate** is a concentrated source of von Willebrand factor, factors XIII and VIII, and fibrinogen. It is primarily used to treat the corresponding clotting disorders (i.e., von Willebrand disease and hemophilia).

Whole blood transfusions are readily available in many practices. Whole blood should be serotyped to avoid allergic reactions in dogs and cats. Typing sera are available for six blood types in dogs (dog erythrocyte antigen [DEA] 1.1, 1.2, 3, 4, 5, 7) and three in cats (A, B, AB). The most antigenic canine blood type appears to be DEA 1.1. A DEA 1.1-negative dog that has been previously sensitized with DEA 1.1-positive blood will

BOX 4.5 Consequences of Severe Hypoalbuminemia

Systemic organ dysfunction
Pulmonary edema
Poor and delayed wound healing
Gastrointestinal ileus
Interstitial edema
Hypercoagulability
Increased morbidity and mortality

BOX 4.6 Calculation to Correct Hypoalbuminemia

Fresh-Frozen Plasma

Plasma (mL) = Desired recipient TS (g/dL) − Recipient TS (g/dL) × 50/Donor TS (g/dL)

25% Human Serum Albumin (Diluted to 5%–10%)

Albumin deficit (g) = 10 × (Serum albumin desired − Serum albumin of patient) × Body weight (kg) × 0.3

TS, Total solids.

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develop an acute hemolytic transfusion reaction after repeated transfusions of type DEA 1.1-positive blood, thus blood typing of blood donor and patient before the first transfusion is recommended, or at least before a second transfusion if only DEA 1.1-negative blood donors are used.

There is wide breed and geographic variation in the proportion of type B cats. It is thought that more than 99% of domestic cats in the United States are type A, but this number may be decreasing. The frequency of type B cats is markedly higher in Australia, Asia, and Europe. Cats develop naturally occurring antibodies against foreign blood types that result in premature destruction of transfused red cells, clinically severe transfusion reactions, and neonatal isoerythrolysis. Type B cats that are given type A blood typically develop a rapid, potentially fatal transfusion reaction following even a single transfusion of a small volume of blood. Type A cats given type B blood may develop a mild transfusion reaction that is often not clinically apparent; however, the PCV typically falls to pretransfusion levels within a few days of the transfusion. Type A and type AB kittens born to a type B queen receiving anti-A alloantibodies through the colostrum are at risk for developing isoerythrolysis during the first few days of life. Type AB is extremely rare in domestic cats, and blood from a type A donor is adequate. Cross-matching should be performed to detect antibodies in the plasma of the recipient or donor.

In a massive hemorrhage, blood may be given as rapidly as possible. In stable patients, starting with a low dose (i.e., 0.25 mL/kg) to ascertain whether a transfusion reaction is likely is wise (see Table 4.5). If no adverse reaction is noted, the rate can be increased. A dosage of 4 mL/kg per hour has been recommended as the upper limit in patients with heart disease. The volume of whole blood or packed red blood cells necessary to elevate the patient's PCV to the desired level (generally 25%–30% in dogs and 20% in cats) can be calculated using the formula in Box 4.1. Transfusion reactions may be acute or chronic. The incidence of transfusion reactions in dogs and cats has been reported as high as 15%, and this risk increases in untyped or unmatched recipient animals. Dogs with immune-mediated disease appear to be at increased risk. Transfusion reactions are classified as immunologic or nonimmunologic, and acute or delayed. Acute immunologic reactions include immune-mediated hemolysis, febrile nonhemolytic reactions, and hypersensitivity reactions. Nonimmunologic acute reactions include nonimmune-mediated hemolysis, volume overload, hypocalcemia, air embolism, and bacterial contamination leading to sepsis. Delayed transfusion reaction is typically manifested as immune-mediated hemolysis 3 to 5 days posttransfusion. Premedication prior to blood transfusion with antihistamines or corticosteroids does not appear to reduce the risk of transfusion reactions.

If an acute reaction is suspected, the transfusion should immediately be discontinued. For mild reactions (fever, urticaria), the transfusion can often be continued at a slower rate. Short-acting glucocorticoids (methylprednisolone succinate, 30 mg/kg IV once; or dexamethasone sodium phosphate, 0.5–1 mg/kg IV once) and antihistamines (diphenhydramine, 2 mg/kg IV) can also be administered. Antihistamines are also often given prior to administration of blood or plasma products to help prevent mild reactions. More severe reactions should receive IV fluid therapy and be closely monitored and treated for signs of shock, disseminated intravascular coagulation, renal failure, and sepsis.

NOTE Hemoglobin-based, oxygen-carrying fluid or blood substitutes have been approved for use in dogs; however, the availability of such products has been inconsistent and unreliable for several years.

Intraoperative Fluid Therapy

Intraoperative fluid therapy must take into consideration the effects of anesthesia and surgery on fluid hemodynamics. A dosage rate of 10 to 15 mL/kg per hour of crystalloid fluids during surgery is generally recommended to offset hypotension and maintain perfusion during anesthesia. Lower rates (5 mL/kg per hour) may be adequate for healthy patients undergoing elective procedures. Prewarming fluids, especially for young or small patients, is recommended.

If blood losses exceed 10% of the blood volume in an animal with a normal PCV and TP, blood replacement during surgery is indicated. Although an awake animal may tolerate acute blood loss of up to 25% of their total blood volume, anesthetized animals are less tolerant of rapid blood loss. Blood loss during surgery should be calculated by counting blood-saturated sponges and cotton-tipped applicators and monitoring blood suctioned from the field. As a general rule of thumb, a blood-soaked sponge (4 × 4) will contain 5 to 10 mL of blood, whereas a premoistened (with sterile saline) blood-soaked laparotomy sponge will contain up to 50 mL of blood. To estimate the amount of blood lost in aspirated fluids, the PCV of the aspirated fluid can be multiplied by the volume of fluid aspirated and this figure divided by the PCV of the patient.

SURGICAL SAFETY CHECKLISTS

In human medicine, a significant reduction in postoperative complications and mortality has been associated with the use of surgical safety checklists. Implementation of the use of checklists should be considered for veterinary hospitals as well, as one recent study found a significant decrease in the frequency and severity of postoperative complications. Fig. 4.6 is an example of a surgical checklist used at an academic veterinary teaching hospital.

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Veterinary Hospital: Surgical Safety Checklist

Date:_____

Clinician:_____

Patient location: Anesthesia ____

Surgical prep site is confirmed with margins identified
- **Graft sites included?**

Patient positioning is confirmed _____

Imaging displayed/set-up in the O.R.? (circle all that apply)

RADS/CT/MRI **C-ARM** **Tower/RIS**

Patient concerns: Infectious, allergies, ETC.

_____

Antibiotic prophylaxis with patient if needed? **Yes** **N/A**

Pre-operative (before incision) ____

All team members introduce themselves and role
team confirms patient:

- Identity
- Procedure _____
- Site _____

R **L** **N/A**

Have antibiotics been initiated?
Yes **N/A**

Confirm client notification
Surgeons: Inform team of anticipated complications
Anesthesia: Inform team how patient is doing anesthetically
- Crash cart, blood typed, ETC.

Nurse: Address concerns within the O.R.

Patient label

Pre-operative continued

Post-op analgesia

Can patient have NSAIDS?

Yes _____ **No**

Confirm patient's post-op destination
(circle all that apply)

CCU **ICW** **Radiology**

Post-operative (BEG. closing) ____

Nurse confirms with team:

Any equipment concerns?
(Lights, power equip., scopes, ETC.)

No
Yes _____

Purse string removal reminder

Specimen(s) correctly:

Yes: **-Labeled** **N/A**

-Archived

-Inked

Charges complete

FIG. 4.6 Example of a surgical safety checklist.

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5

Preparation of the Operative Site



Endogenous microbial flora (especially *Staphylococcus pseudintermedius)* are the most common source of **surgical site infection** (SSI) in dogs.[1] The term *SSI* is more appropriate than "surgical wound contaminant" because it includes infection that directly results from surgical procedures that involve other areas of the body, such as organs or internal spaces that are manipulated during the operation (see Box 9.1). **Antisepsis** is the prevention of sepsis by preventing or inhibiting the growth of resident and transient microbes.

Normal or resident organisms live in the skin's superficial cornified layers and outer hair follicles. *S. pseudintermedius* can be cultured from the skin of most normal dogs and is the principal bacterial skin pathogen in this species. Staphylococci can cause a wide range of infections when the immune system becomes compromised or select comorbidities are present (i.e., renal failure, congestive heart failure, diabetes). Other transient pathogens in canine skin include *Enterococcus* spp., *Klebsiella* spp., *Pasteurella* spp., and *Streptococcus* spp. Reducing or eliminating exposure to this flora is extremely important during surgery. Although it is impossible to sterilize skin without impairing its natural protective function and interfering with wound healing, appropriate preoperative preparation reduces the number of bacteria and the likelihood of infection.

The prevalence of SSI is a major concern because of the associated increased incidence of morbidity and mortality, length of hospitalization, and cost of care for postoperative patients. Although similar data are not available for veterinary hospitals, in 2007 it was estimated that direct medical costs to US hospitals of all hospital-acquired infections among hospital patients was between $28.4 and $45 billion.[2] Preventive preoperative measures that can reduce the risk of SSI include administration of antimicrobial prophylaxis (see Chapter 9) and proper utilization of skin antiseptic agents for the surgical team (see Chapter 6) and the patient. Among human patients with SSI who die in the postoperative period, death was directly related to SSI in over 75% of cases[3]; thus it is imperative that veterinarians and their staff be proactive in preventing infection during surgery by using proper techniques and solutions to prepare the skin for surgery.

DIETARY RESTRICTIONS

In adult animals, food intake is generally restricted 8 to 12 hours before induction of anesthesia to avoid intraoperative or postoperative emesis and aspiration pneumonia. Shorter periods may be recommended in some small breed dogs to prevent hypoglycemia. Access to water generally is not generally curtailed. Operations of the large intestine (see Chapter 18) often require specialized preparations (e.g., dietary restriction for 48 hours) and/or enteric antibiotics (e.g., oral kanamycin, neomycin, penicillin G, metronidazole). Food should not be withheld from young animals for longer than 4 to 6 hours because hypoglycemia may occur.

EXCRETIONS

The animal should be allowed to defecate and urinate shortly before induction of anesthesia. Colonic surgery may require enemas (see Chapter 18). An empty urinary bladder often facilitates abdominal procedures. If urine is not evacuated naturally, the bladder may be manually expressed with the animal under general anesthesia, or a sterile urethral catheter may be passed into the bladder.

TREATMENT OF HAIR

Before a patient is prepared for surgery, the patient's identity, surgical procedure to be performed, and surgical site should be verified (see preoperative checklist; Fig. 4.6). In some cases, bathing the animal the day before surgery to remove loose hair, debris, and external parasites may be warranted. Whenever possible, infection remote to the surgical site should be identified and treated before an elective operation is performed. Elective procedures should be postponed until the infection has resolved.

Hair should be removed as close to the time of surgery as possible, and hair removal should always occur outside the room where the surgical procedure will be performed (e.g., in the prep room). **Removal of hair the night before surgery is associated with a significantly higher superficial skin infection rate than removing the hair immediately before surgery.** The surgical site should be identified, and hair should be liberally clipped around the proposed incision site so that the incision can be extended within a sterile field (Fig. 5.1). The prepared area should be large enough to accommodate extension of the incision, additional incisions (if needed), and all possible drain sites. It must also be large enough that inadvertent wound contamination is avoided if the drapes move during the procedure. A general guideline is to clip at least 20 cm on each side of the incision. The hair can be removed most effectively with an electric clipper and a No. 40 clipper blade. Patients with dense hair coats may be clipped first with a coarser blade (No. 10); the higher the blade number, the shorter the remaining hair. Clippers should be held in a "pencil grip," and initial clipping should be done with the hair growth pattern. Subsequent clipping should be against the pattern of hair growth to obtain a closer clip. Depilatory creams are less traumatic than other hair removal methods, but they induce a mild dermal lymphocytic reaction. They are most useful in irregular areas where adequate hair removal is difficult. Razors are occasionally used for hair removal (e.g., around the eye) but can cause microlacerations in skin that may

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ABSTRACT

Proper preparation of the surgical patient is vital to reduce the incidence of surgical site infections. One-step alcohol-based solutions may be more cost effective than using surgical sponges once all costs, including pack preparation, are considered. Chlorhexidine solutions appear to be more effective than povidone-iodine solutions when used for surgical skin preparation.

KEYWORDS

surgical site infection
antisepsis
chlorhexidine gluconate
povidone-iodine
one-step alcohol-based solutions
dietary restrictions
draping

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FIG. 5.1 Liberally clip hair around the proposed incision site so the incision can be extended within a sterile field. In male dogs, be sure to clip the prepuce.



FIG. 5.2 For limb procedures that do not require exposure of the paw, exclude the paw from the surgical area by placing a latex glove over the distal extremity and securing it to the limb with tape. Wrap the glove with tape or Vetrap.

increase irritation and promote infection. After hair removal is complete, loose hair is removed with a vacuum.

For limb procedures in which exposure of the paw is unnecessary, the paw can be excluded from the surgical area by placing a latex glove over the distal extremity and securing it to the limb with tape (Fig. 5.2). The glove should be covered with tape or self-adherent bandaging tape (e.g., Vetrap, COHERE, Coban). The foot is then "draped out" of the sterile field (see later). To enhance manipulation of limbs during surgery, a hanging-leg preparation may be done. The limb is circumferentially clipped and then is hung from an intravenous (IV) pole during prepping to allow all sides to be scrubbed.



FIG. 5.3 Flush the prepuce of male dogs with antiseptic solution before performing the sterile preparation.

Before the animal is transported to the surgical suite, the incision site is given a general cleansing scrub, and ophthalmic antibiotic ointments or lubricants are placed on the cornea and conjunctiva. Studies suggest that using *clean,* rather than sterile, supplies for this initial cleansing prep does not influence the infection rate if the skin is intact. Lesions, eruptions, abrasions, irritations, rashes, dermatitis, burns, denuded or traumatized areas, or other similar medical conditions that could provide a portal of entry for a pathogen should not be present. In male dogs undergoing abdominal procedures, the prepuce should be flushed with an antiseptic solution (Fig. 5.3). The skin is scrubbed with germicidal soaps to remove debris and reduce bacterial populations. The area is lathered well until all dirt and oils have been removed. This is a generous scrub that often encompasses the hair surrounding the operation site to remove unattached hairs and dander that may be disturbed during draping.

Commonly used scrubbing solutions include iodophors, chlorhexidine gluconate (CHG), alcohols, hexachlorophene, and quaternary ammonium salts. Alcohol is not effective against spores but produces a fast kill of bacteria and acts as a defatting agent. Using alcohol by itself is not recommended, but it is commonly used in conjunction with CHG or povidone-iodine (PVI; see later). Hexachlorophene and quaternary ammonium salts are less effective than other available agents and are no longer recommended for preoperative skin preparation. It is important to avoid abrading the skin by excessive scrubbing with gauze sponges.

POSITIONING

Before sterile application of the epidermal germicide, the animal is moved to the operating room, positioned so the operative site is accessible to the surgeon, and secured with ropes, sandbags, troughs, tape, or vacuum-activated positioning devices. When these restraints are applied, interference with respiratory function and peripheral circulation, as well as the musculature and its innervation, must be avoided. Monitoring devices should be connected or the connections rechecked after the patient is positioned. Warm-air circulating devices (e.g., Bair Hugger, Hot Dog Patient Warming System, Darvall Vet Cocoon), water circulating systems, or electric warming pads (see Chapter 3) are placed adjacent to or over the patient, depending on the site of the surgical incision. If electrocautery is to be used, the ground plate should be positioned under the patient. If a hanging-leg preparation is to be done, the limb should be carefully suspended with tape from an IV pole.

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STERILE SKIN PREPARATION

The purposes of the preoperative skin preparation are to (1) remove soil and transient microorganisms from the skin, (2) reduce the resident microbial count to subpathogenic levels in a short time and with the least amount of tissue irritation, and (3) inhibit rapid rebound growth of microorganisms. "Sterile" preparation begins after positioning of the animal has been completed. For some solutions, gauze sponges are sterilized in a pack, along with bowls into which the germicides can be poured. Newer solutions that are prepackaged with an applicator do not require sponges and may be more economical (see later). *Handle sponges with sterile sponge forceps or a gloved hand using aseptic technique. Use your dominant hand to perform the sterile preparation and your less dominant hand to retrieve sponges from the preparation bowl.* Transferring sterile sponges to the dominant hand before scrubbing the animal helps ensure that the hand picking up the sponges is not contaminated during the procedure. *Begin scrubbing at the incision site, usually near the center of the clipped area. Use a linear or circular scrubbing motion, moving from the center to the periphery. Do not return sponges from the periphery to the center because bacteria could be transferred onto the incision site; discard sponges after reaching the periphery.*

NOTE Using concentric circles, rather than a horizontal or linear preparation, has not been shown to reduce surgical site infection.[4]

Characteristics of an ideal preoperative antiseptic are provided in Box 5.1. Alcohol-based solutions that require shorter prep times than aqueous-based solutions (e.g., PVI, CHG) are now commonly used in human medicine, and their use is increasing in veterinary medicine. These newer solutions, which typically combine alcohol with PVI, CHG, or zinc pyrithione, appear to have better antimicrobial activity than PVI, CHG, or alcohol alone and provide a more persistent reduction in the baseline number of bacteria (Table 5.1). **In humans, evidence suggests that CHG is a more effective skin preparation agent than PVI.**[4–6] A meta-analysis conducted to verify whether recent evidence supports the hypothesis that CHG is more efficient than other antiseptics in reducing SSI rates was published in 2016.[6] The authors conducted a systematic review from 2000–14 in all languages. The primary end point was SSI incidence and secondary skin bacterial colonization. The authors concluded that there is moderate-quality evidence supporting the use of CHG for preoperative skin antisepsis and high-quality evidence that the use of CHG is associated with fewer positive skin cultures. CHG-alcohol is also recommended by the Centers for Disease Control and Prevention as the antiseptic of choice to reduce vascular catheter–associated bloodstream infection. If CHG (see Table 5.1) is the preparation solution, it remains in contact with the skin at the end of the preparation procedure or may be rinsed with saline. Because CHG binds to keratin, contact time is less critical than with PVI. Two 30-second applications have been advocated as adequate for antimicrobial activity.

When PVI (see Table 5.1) and alcohol are used, the site is typically scrubbed alternately with each solution three times to allow 5 minutes of contact time; however, using alcohol (isopropyl alcohol) between PVI scrubs reduces the contact time of PVI with skin and may diminish its efficacy; thus this is not recommended. Excess solution on the table or in body "pockets" should be blotted with a sterile towel or sponges. When the final PVI scrub is finished, a 10% PVI solution should be sprayed or painted on the operative site (Fig. 5.4).

One-Step Alcohol-Based Solutions

Using one-step alcohol-based solution (e.g., ACTIPREP, ChloraPrep, DuraPrep), apply the solution with a sterile applicator, working

BOX 5.1 Characteristics of an Ideal Preoperative Antiseptic

- Kill all bacteria, fungi, viruses, protozoa, tubercle bacilli, and spores
- Be hypoallergenic
- Be nontoxic
- Have residual activity
- Not be absorbed
- Be nontoxic and be able to be used repeatedly safely
- Be safe to use on all parts of the body and in all body systems

TABLE 5.1 Properties of Antiseptics Used for Preoperative Skin Preparation

Antiseptic	Mode of Action	Activity	Examples
Iodine/iodophors (PVI)	Penetration of the cell wall and oxidation and replacement of intracellular molecules with free iodine. *Iodophors* are solutions of iodine with a surfactant or stabilizing agent that liberates free iodine	Wide range of bacteria, tubercle bacilli, and some spores (e.g., *Clostridia*); activity is greatly reduced by the presence of organic material (pus and exudates)	10% PVI: Betadine
Alcohol (IPA)	Rapidly denatures bacterial cell wall proteins and biomolecules (DNA, RNA, lipids)	Wide range of bacteria, tubercle bacilli, and many fungi and viruses	70% isopropyl alcohol
CHG	Disrupts cell membrane and precipitates cellular contents	Wide range of bacteria; more effective against gram-positive bacteria than gram-negative bacteria; minimal activity against tubercle bacteria, *Mycobacterium* spp., and fungi	4% CHG: Hibiclens, Betasept 2% CHG: Nolvasan
Alcohol-based solutions	Combination of previously listed modes of action	Broad spectrum of activity due to the combination of several antiseptics with different modes of action	2% CHG + 70% IPA: ChloraPrep 83% ethanol + zinc pyrithione: Actiprep PVI (0.7% available iodine) + 74% IPA: DuraPrep

CHG, Chlorhexidine gluconate; *IPA,* isopropyl alcohol; *PVI,* povidone-iodine.

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FIG. 5.4 If using povidone-iodine, spray or paint a 10% solution on the operative site after preparation has been completed.

from the center outward (Fig. 5.5). Once a uniform application has been applied, allow the surgical site to dry for approximately 2 to 3 minutes before draping or using an electrosurgical unit.

NOTE One-step alcohol-based solutions such as ChloraPrep are more effective than PVI, CHG, or alcohol alone. They may be cheaper to use when all costs (e.g., prep pack sterilization) are taken into consideration.

NOTE Prevent pooling of alcohol or alcohol-based solutions, particularly if electrocautery is being used, as these solutions are flammable. Do not drape until the solution is thoroughly dried.

DRAPING

Once the patient has been positioned and the skin prepared, the animal is ready to be draped. If electrocautery is to be used, sufficient time should elapse between skin preparation and the application of drapes to permit complete evaporation of flammable substances (e.g., alcohol, defatting agents) from the skin. If an abdominal incision extends to the pubis in male dogs, the prepuce should be clamped to one side with a sterile towel clamp (Fig. 5.6). The purpose of drapes is to create and maintain a sterile field around the operative site. Draping is performed by a gowned and gloved surgical team member and begins with placement of field drapes (quarter drapes) to isolate the unprepared portion of the animal. These towels should be placed one at a time at the periphery of the prepared area. Field drapes may be huck towels or disposable, nonabsorbent towels. Drapes should not be flipped, fanned, or shaken because rapid movement of drapes creates air currents on which dust, lint, and droplet nuclei can migrate. Drapes, supplies, and equipment that extend over or drop below table level should be considered nonsterile because they are not within the visual field of the surgeon and their sterility cannot be verified.

Once the towels have been placed, they should not be readjusted toward the incision site because this carries bacteria onto the prepared skin. Towels are secured at the corners with sterile Backhaus towel clamps (Fig. 5.7). The tips of the towel clamps, once placed through the skin, are considered nonsterile and should be handled appropriately. Generally, field towels do not

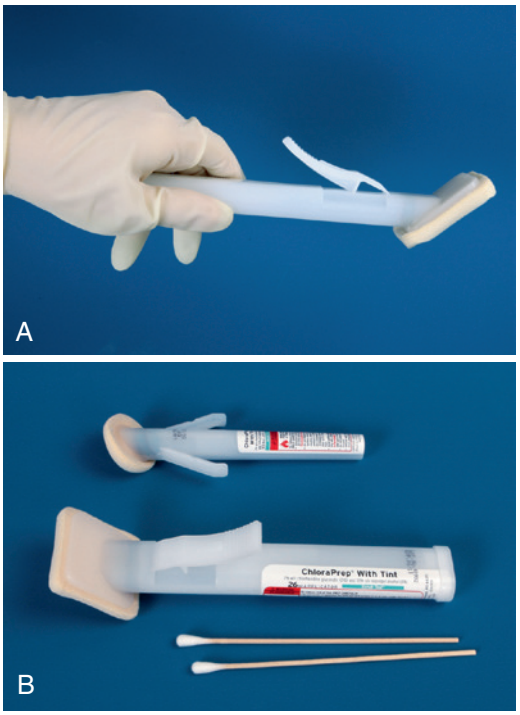


FIG. 5.5 (A) A one-step alcohol-based solution such as ChloraPrep is applied with a sterile applicator, working from the center outward. Once a uniform application has been applied, the surgical site is allowed to dry for approximately 2 to 3 minutes before draping or use of an electrosurgical unit. Rinsing and repeated applications are not necessary. (B) The solution comes in 10.5 and 26 mL applicators for skin preparation.



FIG. 5.6 If an abdominal incision extends to the pubis in male dogs, clamp the prepuce to one side with a sterile towel clamp.

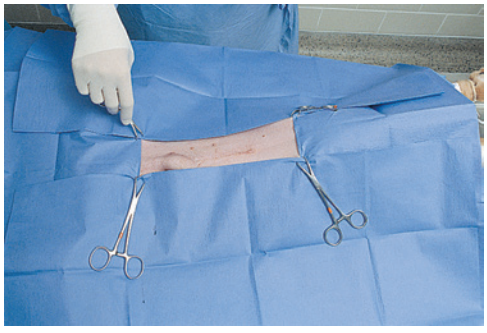


FIG. 5.7 Secure field drapes at the corners with sterile Backhaus towel clamps. The tips of the towel clamps are considered nonsterile once they have been placed through the skin and should be handled appropriately.

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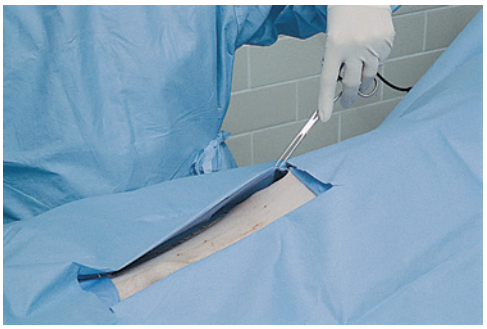


FIG. 5.8 If the drape does not have a fenestration, cut one to the appropriate size. The edges of the drape can be secured to the field drapes with Allis tissue forceps (not towel clamps). Do not put holes through the outer drape.



FIG. 5.9 When performing a hanging-leg preparation, place field drapes around the limb and secure them with towel clamps.



FIG. 5.10 The limb is placed through a fenestration of a lap or fanfold drape, and the drape is secured. A plastic adhesive drape has been applied to the skin and surrounding drapes.

cover the edges of the table, and care should be taken not to brush a sterile gown against this nonsterile field. Once the animal and the incision site are protected by field drapes, final draping can be performed (Fig. 5.8). A large drape is placed over the animal and the entire surgical table to provide a continuous sterile field. Cloth drapes should have an opening of appropriate size and position that can be placed over the incision site while the drape covers the remaining surfaces.

To drape a limb, field drapes should be placed and secured as described previously to isolate the surgical site or the proximal aspect of the limb if the leg is hung (Fig. 5.9 and Video 5.1). The unprepared area of the limb is held by a nonsterile member of the surgical team, and the tape holding the elevated limb is cut. The limb is presented to the sterile surgical member, so it may be taken with a hand in a sterile stockinette or towel. The limb should not be turned loose until it is securely held by the sterile surgical team member. If a stockinette is used, it should be carefully unrolled down the limb and secured with towel clamps. If a sterile towel is used, the limb should be carefully wrapped with the towel before it is secured to the skin with a towel clamp. Water-impermeable (disposable) towels (plus the towel clamp) should then be covered with sterile Kling. If a cloth towel is used, it (and the towel clamp) should be covered with sterile Vetrap. The limb is now ready to be placed through a fenestration of a lap or fanfold drape and the drape secured (Fig. 5.10). The end of the stockinette is wrapped with a self-adherent cohesive bandage material (e.g., Vetrap, COHERE, Coban).

The World Health Organization (WHO) Global Guidelines for the Prevention of Surgical Site Infection recently suggested that antimicrobial sealants, such as plastic adhesive drapes with or without antimicrobial properties, *not* be recommended for the purpose of preventing SSI.[5] To reduce skin exposure and subsequent contamination during surgery, additional skin draping, or "toweling-in," is sometimes performed after the skin incision has been made, but its value is debatable. Wound protector devices consist of a semirigid plastic ring placed into the abdomen through the laparotomy wound to which an impervious drape is circumferentially attached. This plastic drape comes up and out of the wound on to the skin surface, thus protecting the cut edges of the wound. Wound protection devices may be of benefit in clean-contaminated, contaminated, and dirty abdominal surgical procedures for the purpose of reducing the rate of SSI, but their value has not been established and is controversial.[5,7]

After the animal and nearby nonsterile surfaces have been covered with sterile drapes, the instrument tray can be arranged, and surgery can begin.

OTHER CONSIDERATIONS TO REDUCE THE INCIDENCE OF SURGICAL SITE INFECTION

Additional recommendations made in the latest report of the WHO guidelines for the prevention of SSI include (1) providing enhanced nutritional support (oral or enteral multiple nutrient–enhanced nutritional formulas) in underweight patients who

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undergo major surgical operations (see Chapter 10), (2) perioperative oxygenation (see later and Chapter 4), (3) maintenance of normal body temperature (see Chapter 3), (4) use of protocols for intensive perioperative blood glucose control in both diabetic and nondiabetic patients, and (5) maintenance of adequate circulating volume control/normovolemia (see Chapter 12).[5] They noted that a moderate quality of evidence shows that providing a high fraction of inspired oxygen (FiO_2; 80%) is beneficial in patients undergoing procedures under general anesthesia with endotracheal intubation and results in a significant decrease of the risk of SSI compared with 30% to 35% FiO_2. As a result, the panel unanimously agreed to recommend that patients undergoing surgical procedures under general anesthesia should receive 80% FiO_2 intraoperatively and in the immediate postoperative period for 2 to 6 hours, if feasible. The panel unanimously agreed that warming devices should be used to avoid patient hypothermia in the operating room and during the surgical procedure to reduce the risk of SSI and, more importantly, other complications associated with surgery such as a decrease in myocardial events, blood loss, and transfusion requirements. The panel suggested that immunosuppressive agents *not* be discontinued perioperatively for the purpose of reducing SSI.

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6

Preparation of the Surgical Team



Although most surgical site infections (SSIs) are caused by the patient's endogenous flora, operating room (OR) personnel are also an important source of bacterial contamination.[1] Although careful preparation of the surgical team and nonsterile personnel is important to reduce the number of bacteria in the surgical suite, it does not eliminate them. It is estimated that individuals shed approximately 1 million microorganisms from their bodies each day,[2] and a correlation has been noted between the number of people, their movements, and the number of airborne bacteria in a surgical suite. Therefore surgical room personnel should be reduced to only those essential for anesthesia or surgical support. To minimize contamination during surgery, strict guidelines should be followed regarding surgical attire for all surgical room personnel, including observers, and guidelines for performing surgical scrubs and preps (see Chapter 5) should be closely followed.

SURGICAL ATTIRE

Although there have been drastic improvements in the arena of SSI prevention over the last century, evidence supporting many current wardrobe and aseptic environment practices in the OR continues to be limited. Nonetheless, current recommendations suggest that all those entering the OR suite should be appropriately clothed, regardless of whether surgery is in progress or not. Surgical attire includes scrub suits, hair coverings, masks, footwear and shoe covers, gowns, and gloves.

Scrub Suits, Jackets, and Laboratory Coats

To minimize microbial contamination from OR personnel, scrub suits rather than street clothes should be worn in the operating suite. With two-piece pant suits, loose-fitting tops should be tucked into the trousers. Tunic tops that fit close to the body may be worn outside the trousers. The sleeves of the top should be short enough to allow the hands and arms to be scrubbed. Pants should have an elastic waist or drawstring closure. Scrub clothes should be laundered between wearings and changed if they become visibly soiled or wet to prevent transfer of microorganisms to the surgical environment. Wearing scrub clothes outside the surgical environment increases microbial contamination. In human hospitals, evidence does not support wearing cover apparel (e.g., laboratory coats) to protect scrub attire from contamination; in fact, there is evidence that in these settings laboratory coats worn as cover apparel can be contaminated with large numbers of pathogenic microorganisms, leading to contamination of scrub suits.[3] However, the large amount of loose hair present in veterinary hospitals makes cover attire more likely to be beneficial rather than detrimental, as long as it is laundered daily.

Nonscrubbed personnel should wear long-sleeved jackets over their scrub clothes because opening sterile supplies onto the sterile field without wearing a long-sleeved jacket may allow skin squames from the perioperative team member's bare arms to drop onto the sterile field and increase the risk for an SSI.[3] Jackets should be worn buttoned or snapped closed to minimize risk of the edges inadvertently contaminating sterile surfaces.

Hair Coverings

Hair is a significant carrier of bacteria and, when left uncovered, may act as a filter and collect bacteria. Although the evidence for wearing a head covering is weak, complete coverage of the hair is recommended. Caps should completely cover all hair, and sideburns and beards require hoods for complete coverage (Fig. 6.1). Skullcaps that do not cover the side hair above the ears and the hair at the nape of the neck are inappropriate.

Footwear and Shoe Covers

Any footwear that is comfortable can be worn in the surgery area. Using dedicated shoes in the operating theatre is a sensible practice that may reduce spreading hair and other contaminants from the prep room to the OR. If dedicated shoes are not used, shoe covers should be provided. Shoe covers are generally made of disposable materials that are water repellent and tear resistant. Although the value of shoe covers is debatable in human ORs, most veterinary facilities recommend that they be donned when first entering the surgical area and worn when leaving it to keep shoes clean. **New shoe covers should be donned upon returning to the surgical area.** Because of the abundance of animal hair in veterinary hospitals, changing shoe covers immediately before entry into the surgical theater may decrease the amount of hair tracked into the OR by surgical room personnel.

Masks

Masks constructed from lint-free material containing a hydrophilic filter web sandwiched between two outer layers should be worn whenever entering a sterile area. Their major function is to filter and contain droplets of microorganisms expelled from the mouth and nasopharynx during talking, sneezing, and coughing. Masks must be fitted over the mouth and nose and must be secured in a manner that prevents venting. The dorsal aspect of the mask is secured by shaping the reinforcing top edge tightly around the nose.

Surgical Gowns

Factors to consider when purchasing surgical gowns and drape products are that they must provide a barrier to contamination and should be resistant to tears, punctures, and abrasions. The barrier properties of the critical zone of the gown or drape are

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ABSTRACT

The most important advances in surgical hand asepsis in the past decade include no longer using scrub brushes, the introduction of alchohol rubs, and a reduction in the duration of the prep. These advances have been slow to be adopted by veterinary practitioners, but doing so will save both time and money and should reduce the number of surgical site infections seen in veterinary patients.

KEYWORDS

surgical hand asepsis
gowning
gloving
assisted gloving
open gloving
closed gloving
alchohol rubs

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FIG. 6.1 Facial hair and sideburns should be covered with a hood.

labeled by the manufacturer as level 1, 2, 3, or 4, with 4 being the most protective. The critical zone is the area where direct contact with blood, body fluids, or other potentially infectious materials is most likely to occur. The type of procedure (entering a body cavity vs. a superficial mass removal), duration of the procedure, potential for splash or soaking of the gown, and how likely contamination is to occur based on the individual's role in the surgery (surgeon, anesthetist, etc.) should also be considered when choosing gowns and drapes.

Surgical gowns may be reusable and made of woven materials (usually cotton), or they may be disposable. Which to use is largely due to individual choice because there is little consensus on which is better. Primary considerations include environmental concerns and cost.[4] One study found that the number of microorganisms isolated from the surgical environment was lower when disposable, nonwoven materials were used, resulting in lower rates of contamination in the OR.[5] These authors recommended that disposable paper gowns be used for all surgical cases, especially those involving implants, because of the heightened risk of infection with reusable gowns.

Reusable gowns are made by a number of companies and, depending on the manufacturer, may be of variable thickness and may have extra paneling added to the abdominal and arm regions for added protection. These different gowns have different levels of performance (level 1 to 4; see earlier) depending on the type of surgery for which they will be used. The more expensive gowns often can be laundered more frequently than those that are less expensive, without impairing the barrier function.

Disposable gowns are meant to be used during one surgery and then thrown away. They are nonwoven and are made by a nonwoven technique from fibers such as polypropylene and polyester.[6] Most manufacturers of disposable gowns and drapes (e.g., Medline Industries, 3M, Precision Fabrics Group, Kimberly Clark) use a technique known as SMS, which stands for spunbond, meltblown, spunbond.[7–9] In some cases the SMS process can include more than one meltblown layer between the outer spunbond layers. The purpose of the spunbond layer closest to the skin is primarily to keep the surgeon cool and maintain a highly breathable environment. The outer layer of spunbonded material is to give the gown tensile strength to prevent ripping or tearing.[6]

Surgical Gloves

Relying on gloves alone (without a surgical scrub) to prevent microbial contamination is not recommended because many surgical gloves have holes at the completion of surgery, and punctured gloves may increase the risk of SSIs, particularly when prophylactic antibiotics have not been administered (see Chapter 9). In human surgeries it has been noted that approximately 20% of gloves have tiny punctures after surgery, the majority of which go unnoticed by the surgeon. Although few studies have been done in veterinary medicine, one prospective study in small animal surgical procedures found that of 78 gloves sampled at the end of surgery from 39 procedures, bacterial contamination was present in 21% of gloves from 31% of the surgeries.[10] This study found no difference related to the left- or right-hand glove, surgical time, dominant hand, surgery type (orthopedic, soft tissue, neurologic), or surgical wound classification on the incidence of contamination. A prospective, randomized controlled trial designed to determine whether use of colored indicator gloves affects perforation detection rate and to identify risk factors for glove perforation during veterinary orthopedic surgery was recently reported.[11] In this study of 574 double pairs of gloves worn during 300 orthopedic surgical procedures (2296 gloves), perforations occurred during 43% of surgeries, with a mean of 2.3 holes per surgery. Inner gloves were intact in 63% of glove pairs where an outer perforation occurred. Intraoperative perforation detection was improved when colored indicator gloves were worn (83% sensitivity) versus standard gloves (34% sensitivity). Independent risk factors for perforation were placement of plates and/or screws, placement of an external skeletal fixator, use of orthopedic wire, and primary surgeon being board certified. However, the cost benefit of wearing an indicator glove versus double gloving is questionable.

NOTE Given the high rate of glove defects, you may wish to double glove and periodically change the outer glove or remove it during key points such as handling of an implant (e.g., in total hip replacement surgeries) or during laparotomies in animals at high risk of infection (i.e., immunocompromised, diabetic, or cushingoid animals).

SURGICAL SCRUB

All sterile surgical team members should perform a hand and arm scrub before entering the surgical suite. In 1840, a Hungarian obstetrician, Ignaz Philipp Semmelweis, discovered that antiseptic hand washing could reduce patient deaths from pathogens transmitted by hands. He reduced the maternal mortality rate from puerperal fever by approximately 90% over a 3-year period by initiating hand washing before touching hospital patients. Hand antisepsis was further advocated by Joseph Lister, Professor of Surgery at the Glasgow Royal Infirmary, in 1860 to promote asepsis and reduce SSI. Lister initiated the first known disinfection program in which the clinical environment and all equipment were cleaned with carbolic acid. Additionally, the surgical team was asked to wash their hands in a solution of 5% carbolic acid before undertaking surgery.

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The objectives of a surgical scrub include mechanical removal of dirt and oil, reduction of the transient bacterial population (i.e., bacteria deposited from the environment), and residual depression of the skin's resident bacterial population (i.e., bacteria persistently isolated from the skin) during the procedure. Rapid multiplication of skin bacteria occurs under surgical gloves if hands are washed with a nonantimicrobial soap, whereas it occurs more slowly following preoperative scrubbing with a medicated soap.

Scrub Technique

Three main developments in surgical hand antisepsis have occurred in the past decade. These include (1) no longer using scrub brushes (rubbing vs. scrubbing) (Fig. 6.2), (2) the introduction of alcohol rubs (Box 6.1), and (3) a reduction in the duration of the prep. In veterinary schools, instructions for surgical hand antisepsis have, until recently, included scrubbing the hands and arms with a brush. This practice is no longer recommended because studies have determined that scrubbing with a brush results in skin damage and increased bacterial counts. Scrubbing with a brush has been demonstrated to remove epidermal layers, increase shedding of bacterial squamous cells, increase (not decrease) microbial counts, and change microbial flora. T**hus most experts agree that the time-honored convention of vigorously scrubbing off the uppermost layers of skin with a brush is not only unnecessary, but also unwise.** A recent review of eight peer-reviewed, randomized controlled trials and two nonrandomized controlled trials from 1990 to 2015 compared traditional hand scrubbing with chlorhexidine gluconate or povidone-iodine against alcohol-based hand rubbing, scrubbing with a brush versus without a brush, and detergent-based antiseptics alone versus antiseptics incorporating alcohol solutions.[12] Evidence showed that hand rubbing techniques are as effective as traditional scrubbing and seem to be better tolerated. Hand rubbing appears to cause less skin damage than traditional scrub protocols, and scrub personnel tolerated brushless techniques better than scrubbing with a brush. Despite such evidence, veterinarians have been slow to adopt these practices.[13]

Before scrubbing, all items of jewelry (including watches) should be removed from the hands and forearms because they are reservoirs for bacteria. Fingernails should be kept short, clean, natural, and healthy. Studies have shown no increase in microbial growth related to wearing freshly applied nail polish; however, nail polish that is obviously chipped or has been worn longer than 4 days is associated with the presence of greater numbers of bacteria and has been associated with infection. Artificial nails (e.g., bondings, tips, wrappings, tapes) should not be worn. A higher number of gram-negative microorganisms have been cultured from the fingertips of personnel wearing artificial nails than from personnel with natural nails, both before and after hand washing. Fungal growth between artificial nails and the natural nail has also been reported and can contaminate the surgical wound. Hands and forearms should be free of open lesions and breaks in skin integrity because skin infection may contaminate surgical wounds.

Once the scrub has been started, nonsterile items cannot be handled. If the hands or arms are inadvertently touched by a nonsterile object (including surgical personnel), the scrub should be repeated. During and after scrubbing procedures, the hands should be kept higher than the elbows. This allows water and soap to flow from the cleanest area (hands) to a less clean area (elbows).

When the scrub has been completed, the hands and arms should be dried with a sterile towel. *Pick up the sterile towel from the table, taking care not to drip water on the gown beneath it, and step back from the sterile table. Hold the towel lengthwise and, using a blotting motion, dry one hand and arm, working from hand to elbow with one end of the towel (Fig. 6.3). Bend over at the waist when drying the arms so that the end of the towel does not brush against your scrub suit. Once the hand and arm are dry, bring the dry hand to the opposite end of the towel. Dry the other hand and arm in a similar manner. Drop the towel into the proper receptacle or onto the floor if a receptacle has not been provided. Do not lower your hands below waist level.*

Scrub Solutions

Antimicrobial soaps and detergents used for scrubbing should be rapid acting, broad spectrum, and nonirritating and should inhibit rapid rebound microbial growth. Some hand scrubs can bind with the stratum corneum, resulting in residual activity. Because bacteria proliferate under gloves, particularly if the gloves are damaged during surgery, persistent chemical activity is desirable. Aqueous scrubs and alcohol rubs are the two most common forms of hand antisepsis. The former are water-based solutions that contain antiseptic agents such as chlorhexidine or povidone-iodine. Scrubbing involves wetting the hands and arms and then using either a brush or a sponge following a specific protocol (i.e., either timed or anatomic; see later). The most commonly used surgical scrub solutions in veterinary medicine are alcohol, chlorhexidine gluconate (Hibiclens, Betasept), povidone-iodine (Betadine), and hexachlorophene (Phisohex) (Table 6.1). Alcohol solutions, on the other hand, contain active ingredients (typically alcohol plus chlorhexidine, povidone-iodine, or other antiseptic agents) and are rubbed on the hands and forearms without using a brush or sponge (Video 6.1).

Alcohol rubs, which do not require rinsing, were first introduced around the late 1990s as an alternative to traditional aqueous-based antiseptic solutions. Alcohol-based solutions may be categorized as *alcohol-based waterless* (ABWL) or *alcohol-based water-aided* (ABWA). ABWL are often preferred over other products because they are easy to use and have few side effects. The new brushless solutions generally have a very rapid killing effect and require a shortened contact time compared with traditional povidone-iodine or chlorhexidine solutions (see later).

BOX 6.1 Surgical Hand Antisepsis/ Hand Rub With an Alcohol-Based Surgical Hand Rub[a]

A standardized protocol for alcohol-based surgical hand rubs should follow the manufacturer's written instructions and should include, but may not be limited to, the following:

1. Wash hands and forearms with soap and water if they are visibly soiled or contaminated with blood or saliva. Dry thoroughly.
2. Dispense the manufacturer-recommended amount of the surgical hand rub product. Apply the product to the hands and forearms, following the manufacturer's written instructions. Some manufacturers may require the use of water as part of the process.
3. Rub thoroughly until dry if using a waterless solution.
4. Repeat the product application process if indicated in the manufacturer's written instructions.

[a]Data from Recommended practices for surgical hand antisepsis/hand scrubs. In: *Standards, Recommended Practices, and Guidelines.* Denver: AORN; 2009.

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FIG. 6.2 Use of a brushless surgical scrub solution. (A) If desired, clean under your fingernails with a nail pick. (*Note:* Nail picks have not been found to decrease bacterial numbers.) (B) Wet your hands and arms. (C) Dispense the appropriate amount of solution (e.g., Triseptin, 1/4 oz) into the palm of your hand by depressing the foot pump. (D) Insert and twist the fingertips of your opposite hand into the solution for several seconds. Transfer the solution to your opposite hand and repeat this step with the fingers of your other hand. (E) Rub your hands together, moving up the forearms to slightly past the elbows. (F) Add water throughout the wash to create additional lather. (G) Rinse thoroughly and repeat steps B through F. Stop below the elbows on the second application. Total scrub time: 2 to 7 minutes.

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TABLE 6.1 Common Antimicrobial Soaps Available for Surgical Scrubs

Antimicrobial Soap	Mechanism of Action	Properties
Chlorhexidine gluconate	Disruption of cell wall and precipitation of cell proteins	• Broad spectrum (more effective against gram-positive than gram-negative bacteria or fungi) • Good virucide • Residual activity because it binds to keratin • Not inactivated by organic material • May be less irritating to skin than iodophors
Hexachlorophene	Disruption of cell wall and precipitation of cell proteins	• Bacteriostatic for gram-positive cocci • Minimal activity against gram-negative bacteria, fungi, or viruses • Not inactivated by organic material • Cumulative (nullified by alcohol) • May be neurotoxic
Iodophors (e.g., povidone-iodine)	Cell wall penetration, oxidation; replaces microbial contents with free iodine	• Broad spectrum (gram-negative and gram-positive bacteria, fungi, and viruses) • Some activity against spores • Inactivated by organic material • Requires minimum of 2 minutes of skin contact
Parachlorometaxylenol	Disruption of cell wall and enzyme inactivation	• Broad spectrum (more effective against gram-positive than gram-negative bacteria, fungi, or viruses) • Slow onset of action
Triclosan	Disruption of cell wall	• Broad spectrum (ineffective against many *Pseudomonas* spp.) • Minimally affected by organic material
Alcohol-based solutions	Combination of above	• Broad spectrum (gram-negative and gram-positive bacteria, fungi, and viruses)



FIG. 6.3 When drying your hands and arms, use one end of the towel to dry one hand and arm (work from hand to elbow). Then bring the dry hand to the opposite end of the towel and dry the other hand and arm in a similar manner.

Examples of brushless solutions on the market include Triseptin (Healthpoint Ltd.), Avagard (3M), Sterillium Rub (Bode Chemic), Purell Waterless Surgical Scrub (GOJO Healthcare), and Endure 450 (Ecolab Inc.).

NOTE A simple hand wash with nonmedicated soap and water before entering the operating theatre area is highly recommended to eliminate any risk of colonization with bacterial spores. Between surgeries, repeating hand rubbing, **without prior hand wash or scrub,** is recommended.

A recent meta-analysis of 14 studies compared a range of methods for performing surgical hand antisepsis.[14] Outcomes were the number of cases of SSIs in patients and the number of viable bacteria or fungal cells (colony-forming units [CFUs]) on the hand of the person operating, before and after surgery. Most of the studies were determined to be too small or sufficiently flawed to determine whether the method of hand antisepsis influenced the risks of SSI. There was some evidence that hand antisepsis with chlorhexidine may reduce the number of bacteria on the hands of health professionals compared with povidone-iodine. There was also some evidence that alcohol rubs with additional antiseptic agents may reduce CFUs compared with aqueous scrubs.

Scrub Duration

The third development within hand antisepsis has been the move toward shorter scrub duration. Over the past 3 decades, scrubs have gradually been reduced from 10 minutes to 2 minutes. In the aforementioned meta-analysis,[14] evidence for the effectiveness of different scrub durations varied. Four studies compared the effect of different durations of scrubs and rubs on the number of CFUs on hands. There was evidence that a 3-minute scrub reduced the number of CFUs compared with a 2-minute scrub; however, the evidence was considered low quality and was downgraded for imprecision and indirectness of outcome. Contact time between the antimicrobial soap or detergent and the skin should be based on documentation of a product's efficacy in the scientific literature and the manufacturer's guidelines. A 5- to 7-minute scrub for the first case of the day, followed by a 2- to 3-minute scrub between subsequent operations, generally is adequate. If the hands and arms are grossly soiled, scrub time should be extended or brush counts increased; however, skin irritation or abrasion should be avoided because this causes bacteria that reside in deeper tissues (e.g., around the base of hair follicles) to become more superficial, increasing the number of potentially infective organisms on the skin surface.

GOWNING (VIDEO 6.2)

Gowns serve as a barrier between the skin of the surgical team member and the patient. They should be constructed of a material that eliminates the passage of microorganisms between sterile

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FIG. 6.4 (A) Have an assistant pull the gown up and over your shoulders and secure it by closing the neck fasteners and tying the inside waist tie. (B) If a sterile-back gown is used, do not secure the front tie until you have donned sterile gloves.

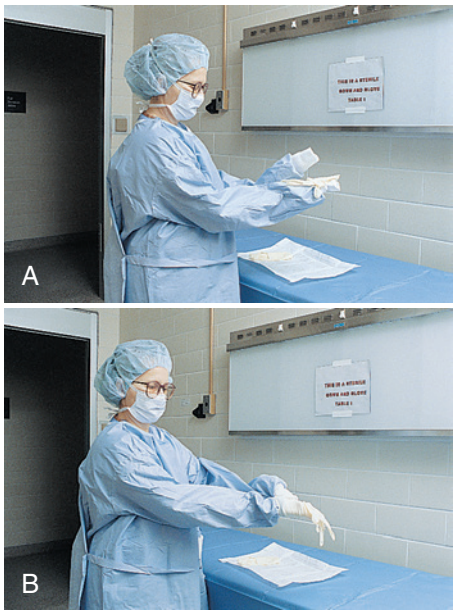


FIG. 6.5 Closed gloving. (A) Working through the gown sleeve, pick up one glove from the wrapper. Lay the glove palm down over the cuff of the gown with the thumb and fingers of the glove facing toward your elbow. (B) Grasp the cuff of the glove with your index finger and thumb. With the index finger and thumb of the other hand (within the cuff), take hold of the opposite side of the edge of the glove. Lift the cuff on the glove up and over the gown cuff and hand. Turn loose and come to the palm side of the glove and take hold of the gown and glove, pulling them toward the elbow while pushing the hand through the cuff and into the glove. Proceed with the opposite hand using the same technique.

and nonsterile areas and should be resistant to fluid, linting, stretch, pressure, and friction (especially in the forearm, elbow, and abdominal areas; see earlier). They should also be comfortable, economical, and fire resistant.

The technique of gowning is described here and is illustrated in Fig. 6.4. Gowning and gloving should be done from a surface separate from other sterile supplies (the surgical table) and from the surgical patient to avoid dripping water onto the sterile field and contaminating it. Gowns are folded so that the inside of the gown faces outward. *Grasp the gown firmly and gently lift it away from the table. Step back from the sterile table to allow room for gowning. Hold the gown at the shoulders and allow it to gently unfold. Do not shake the gown because this increases the risk of contamination. Once the gown is open, identify the armholes and guide each arm through the sleeves. Keep your hands within the cuffs of the gown. Have an assistant pull the gown up over your shoulders and secure it by closing the neck fasteners and tying the inside waist tie (see Fig. 6.4A). If a sterile-back gown is used, do not secure the front tie until you have donned sterile gloves (see Fig. 6.4B).*

GLOVING

Latex rubber gloves serve as barriers between the surgical team member and the patient; however, they are not a substitute for proper scrubbing methods. Lubricating agents for latex gloves such as magnesium silicate (talcum) or low cross-linked cornstarch allow gloves to slide more easily onto the hand. However, these agents can cause considerable irritation to various tissues, even if gloves are vigorously rinsed in sterile saline before surgery. Therefore the surgeon should use gloves in which the inner surfaces have been lubricated with an adherent coating of hydrogel. Potentially pathogenic gram-positive bacteria may be cultured from water droplets collected from surgeons after scrubbing. It is thought that these organisms are derived from scrub room tap water. It has also been demonstrated that gram-positive bacteria strike through the glove wrappings within 2 minutes of wetting the paper. **Optimally, gloves should not be dropped onto the opened gown pack before scrubbing and should not be placed onto a sterile field until the surgeon's arms have been dried.**

Gloving can be performed by three separate methods: (1) gloving yourself using a closed method; (2) gloving yourself using an open method; and (3) assisted gloving.

Closed Gloving

The closed method of gloving ensures that the hand never comes in contact with the outside of the gown or glove (Fig. 6.5 and Video 6.3).

Open Gloving

The open method of gloving is used when only the hands need to be covered (as for urinary catheterization, bone marrow biopsy, or sterile patient preparation) and during surgery when one glove becomes contaminated and must be changed. This method should not be used routinely for gowning and gloving (Fig. 6.6). The procedure used when both gloves are donned is shown in Fig. 6.7 and Video 6.4.

Assisted Gloving

The steps involved in assisted gloving are shown in Fig. 6.8 and Video 6.5. Both open and closed gloving are frequently associated with gown contamination, especially at the cuff. Scrub staff–assisted gloving, although uncommonly performed

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FIG. 6.6 Open gloving when one hand is sterile. (A) Open the glove wrapper and pick up the correct glove at the folded edge with your sterile hand. Gently put your hand into the glove until your fingers are in the fingers of the glove. Place your thumb inside or near the thumb of the glove and hook the cuff of the glove over your thumb. Let go of the glove. (B) Place the finger of your sterile hand under the cuff at the palm of the glove. (C) Bend the wrist of the hand being gloved 90 degrees. (D) Gently walk your fingers around the cuff until they are at the front of the cuff and, at the same time, pull the cuff up and over your gown.

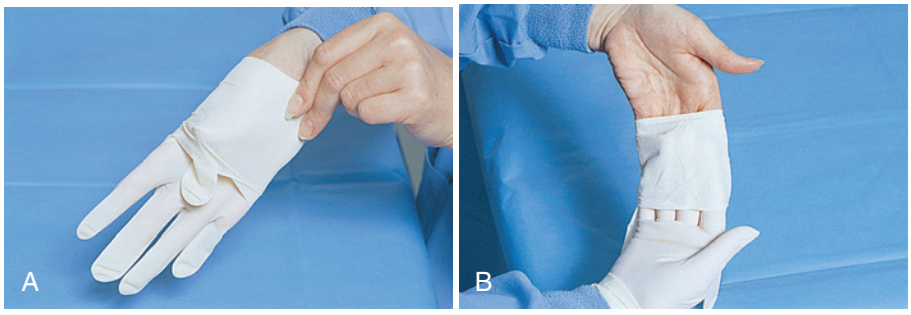


FIG. 6.7 Open gloving when neither hand is sterile. (A) Pick up one glove by its inner cuff with the opposite hand. Slide the glove onto the opposite hand; leave the cuff down. (B) Using the partially gloved hand, slide your fingers into the outer side of the opposite glove cuff. Slide your hand into the glove and unfold the cuff; do not touch your bare arm as the cuff is unfolded. With the gloved hand, slide your fingers under the outside edge of the opposite cuff and unfold it.

in veterinary medicine, may minimize the possibility of gown contamination.

Removing Gloves Aseptically

The procedure for removing gloves in an aseptic manner is described in Fig. 6.9.

MAINTAINING STERILITY DURING SURGERY

The techniques described in this chapter for gowning and gloving minimize the risk that the operative team may contaminate the surgical field. However, vigilance is necessary to prevent contamination of gowns or gloves. Once gowned, surgical team

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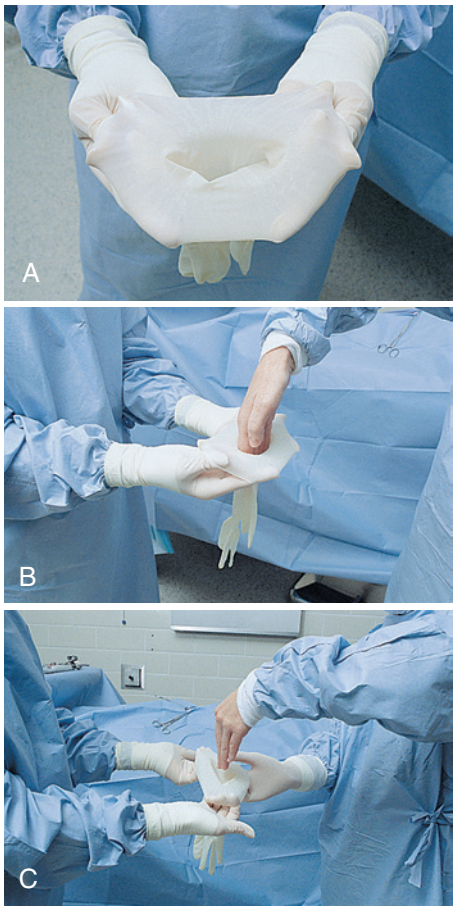


FIG. 6.8 Assisted gloving. (A) Have the assistant pick up one glove and place his or her fingers and thumb under the cuff of the glove. (B) With the thumb of the glove facing you, slip your hand into the glove; then have the assistant bring the cuff of the glove up and over the cuff of your gown and gently let it go. (C) Have the assistant pick up the other glove. Assist by holding the cuff of the glove open with the fingers of your sterile hand while putting your ungloved hand into the open glove. The assistant's thumbs are kept under the cuff while you thrust your hand into it.

members should always face the sterile field and should not touch or lean over a nonsterile area. The arms and hands should remain above waist level and below shoulder level. The arms should not be folded; they should be clasped in front of the body, above the waist. Scrubbed personnel should avoid changing position levels; they should be seated only if the entire surgical procedure will be performed at this level.

The front of the gown should be considered sterile from the chest to the level of the sterile field; the back of the gown is not considered sterile (even if a sterile-back or wraparound gown is used) because it cannot be seen by the scrubbed individual. The sleeves should be considered sterile from 2 inches above the elbow to the stockinette cuff. Because the stockinette cuff collects moisture (making it an ineffective microbial barrier), it is considered nonsterile and should be covered by sterile gloves at



FIG. 6.9 To remove gloves aseptically, the nonsterile assistant grasps the glove near the cuff (being careful not to touch the gown) and pulls it gently from the fingertips. Regloving should be performed using the assisted gloving technique described in Fig. 6.8.

all times. The neckline, the shoulders, and the area under the arms should be considered nonsterile because they may be contaminated by perspiration or by rubbing together of collar and shoulder surfaces during head and neck movement.

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7

Surgical Instrumentation



INSTRUMENT CATEGORIES

Each type of surgical instrument is designed for a particular use and should be used only for that purpose. Using instruments for procedures for which they are not designed (e.g., using Metzenbaum scissors to cut suture or tissue forceps to hold bone) may dull or break them. Specialized instruments for ophthalmologic surgery are discussed in Chapter 16.

Scalpels

Scalpels are the primary cutting instruments used to incise tissue (Fig. 7.1). Reusable scalpel handles (No. 3 and 4) with detachable blades are most commonly used in veterinary medicine; however, disposable handles and blades are available. Disposable scalpels with a locking retractable shield are designed to minimize the risk of surgical blade injuries while passing blades between procedural steps and during disposal. Blades are available in various sizes and shapes, depending on the intended task. A No. 10 blade is most commonly used in small animal surgery for incision and excision of tissues. A No. 15 blade is a smaller version of a No. 10 and is used for precise incisions in smaller tissues. A No. 11 blade is ideal for stab incisions into fluid-filled structures or organs. The curved angle of the No. 12 blade limits its applicability, but it is most often used in cats for elective dissection onychectomy (declawing).

Scalpels are usually used in a slide-cutting fashion, which means that the direction of pressure applied to the knife blade is at a right angle to the direction of scalpel pressure. When incising skin, the scalpel blade should be kept perpendicular to the skin surface. Scalpels can be held with a pencil grip, a fingertip grip, or a palmed grip. The pencil grip allows shorter, finer, and more precise incisions than the other grips because the scalpel is at a 30- to 40-degree greater angle to the tissue (Fig. 7.2). However, this angle reduces cutting edge contact, making this grip less useful for long incisions. The fingertip grip offers the best accuracy and stability for long incisions. The palmed grip is the strongest hold on the scalpel and allows exertion of great pressure on the tissue, but this is often unnecessary in surgical situations.

Scissors

Scissors come in a variety of shapes, sizes, and weights and generally are classified according to the type of point (e.g., blunt-blunt, sharp-sharp, sharp-blunt), blade shape (e.g., straight, curved), or cutting edge (e.g., plain, serrated) (Fig. 7.3). Curved scissors offer greater maneuverability and visibility, whereas straight scissors provide the greatest mechanical advantage when cutting tough or thick tissue. Metzenbaum (also called Metz, Nelson, delicate, or tissue scissors) or Mayo scissors are most commonly used in small animal surgery. Metzenbaum scissors are more delicate than Mayo scissors and are designed for sharp and blunt dissection or incision of finer tissues; Mayo scissors are used for cutting dense, heavy tissue, such as fascia. Heavy scissors are used to cut sutures, but these are separate instruments from those used to cut tissue. Suture scissors used in the operating room are different from suture removal scissors. The latter have a concavity on one blade to gently hook the suture away from the skin and facilitate easy removal. Delicate scissors (e.g., tenotomy scissors, iris scissors) are often used in ophthalmic procedures and other meticulous surgeries, such as perineal urethrostomy, that require fine, precise cuts. Bandage scissors have a blunt tip, which reduces the risk of cutting skin when the scissors are introduced under the bandage. Scissors should be utilized only for their specific purpose and should be regularly maintained to keep them sharp.

Scissors may be used for sharp cutting or blunt dissection. They are held with the tips of the thumb and ring finger through the finger rings and with the index finger resting on the shanks near the fulcrum. The ring finger or thumb should not be allowed to "fall through" the handle; the rings should be kept near the distal finger joint. This is referred to as a *wide-based tripod grip.* Most scissors are designed for use with a right-handed grip, so the natural pushing of the thumb and pulling of the fingers in a gripping motion apply maximal shear and torque to the blades. When used in the left hand, loss of shear and torque forces results in less precision and increased tissue trauma. Therefore left-handed surgeons should learn to cut with scissors with their right hand or should invest in specifically designed left-handed scissors.

Direction, control, and accuracy in cutting depend on the stability of the tissue between the blades of the scissors and the stability of the scissors in the operator's grip. The larger the angle between the blades when cutting, the less the scissors stabilize the tissue and the less accurate the cut. Using the end of the blade stabilizes tissue more securely and allows a more precise cut. Scissors should not be completely closed if the incision is to be continued because the result is a ragged incision; scissors should be nearly closed, advanced, and nearly closed again. Blunt dissection (i.e., separation of tissue by inserting the points and opening the handle) may be used to separate loosely bound tissues, such as muscle or fat, or to undermine skin edges for wound closure. Blunt dissection should not be used in tougher tissue or where precise cuts are possible.

Needle Holders

Needle holders grasp and manipulate curved needles (Fig. 7.4). Size and type of needle holder are determined by characteristics of the needle to be held and location of tissue to be sutured. Larger needles require wider, heavier jawed needle holders. If needle holders are used to hold suture, the jaws should be finely

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ABSTRACT

General surgical instrumentation is designed to cut, grasp, manipulate, or retract tissue. The choice of instrument for a particular surgical task should be based on minimizing the amount of tissue trauma, which also relies on using that instrument properly. Tissue trauma can also be limited by appropriate care and maintenance of surgical instrumentation.

KEYWORDS

scalpel
scissors
forceps
retractors
hemostasis
tissue trauma
sterilization

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FIG. 7.1 Scalpel handles (*left,* No. 3; *right,* No. 4) and blades *(top to bottom)*: No. 10, 11, 12, 15, and 20.



FIG. 7.2 Scalpels are generally held with a pencil grip because this allows short, fine, precise incisions.

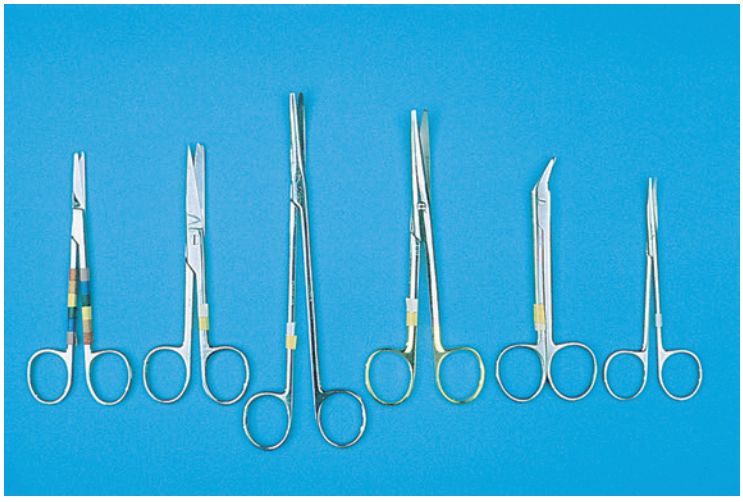


FIG. 7.3 Scissors. *Left to right:* stitch (suture removal), sharp-blunt, Metzenbaum, Mayo, wire, and tenotomy.

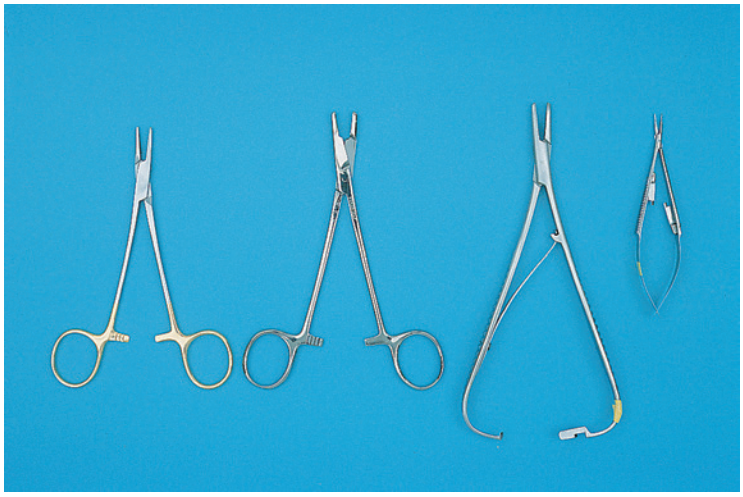


FIG. 7.4 Needle holders. *Left to right:* Mayo-Hegar, Olsen-Hegar, Mathieu, and Castroviejo.

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serrated or smooth to prevent damaging the suture by fraying or cutting it. Long needle holders facilitate working in deep wounds. High-quality needle holders are made of noncorrosive, high-strength alloy and have a glare-free finish. The tips are hardened by coating them with a diamond surface or by fusing tungsten carbide to the face. Tungsten carbide inserts may be replaced when they become damaged or fail to hold suture adequately.

Most needle holders (e.g., Mayo-Hegar, Olsen-Hegar types) have a ratchet lock just distal to the thumb, but some (e.g., Castroviejo type) have a spring and latch mechanism for locking. Mayo-Hegar needle holders are commonly used in veterinary medicine for manipulating medium to coarse needles. Olsen-Hegar needle holders are used similarly but have scissor blades that allow suture to be tied and cut with the same instrument. The disadvantage of Olsen-Hegar needle holders is that expertise is required to prevent cutting the suture during knot tying. Mathieu needle holders have a ratchet lock at the proximal end of the handles of the needle holder, which permits locking and unlocking simply by progressively squeezing the handles together.

Needles generally should be placed perpendicular to the needle holder, which allows greatest maneuverability. When needles are placed at an angle, the handles must move through a wide arc during suturing. A needle is generally grasped near its center to allow it to be advanced through tissue with greater force and less risk of breakage. When the needle is grasped near the eye or swage, maximum needle length is available for suturing and risk of needle slippage is reduced; however, the needle is more likely to bend or break unless delicate tissue is being sutured. Conversely, holding the needle near the pointed end allows the greatest driving force when suturing tough tissue, but extracting the needle is difficult.

Needle holders may be held using a *palmed grip* (no fingers are placed in the rings, and the upper ring rests against the ball of the thumb [Fig. 7.5]), a *thenar grip* (the upper ring rests on the ball of the thumb, and the ring finger is inserted through the lower ring [Fig. 7.6]), a *thumb-ring finger grip* (thumb is placed through the upper ring and the ring finger through the lower ring [Fig. 7.7]), or a *pencil grip* (index finger and thumb rest on the shafts of the needle holder [Fig. 7.8]), which is used with Castroviejo needle holders. The palmed grip is most advantageous for suturing tough tissue that requires a strong needle-driving force; however, the needle cannot be easily released and regrasped after a stitch without changing to another grip, making suturing less precise.

NOTE Left-handed surgeons cannot palm right-handed instruments because the boxlock closes rather than opens with pressure.



FIG. 7.5 A palmed grip provides a strong driving force but less precision.

The thenar grip allows the needle to be released and regrasped for extraction without changing grips. Although it allows mobility, releasing the needle holder by exerting pressure on the upper ring with the ball of the thumb causes the needle holder handles to "pop" apart, and some needle movement occurs during this process. The greatest advantage of a thumb-ring finger grip is that it allows precision when releasing a needle. Although slower than the palmed or thenar grip, it is preferred when tissue is delicate or when precise suturing is required.

Tissue Forceps

Tissue (thumb) forceps are tweezer-like, nonlocking instruments used to grasp tissue (Fig. 7.9). The proximal ends are bonded together to allow the grasping ends to spring open or be squeezed shut. They are available in various shapes and sizes; tips (grasping ends) may be pointed, flat, round, smooth, or serrated with small or large teeth. Tissue forceps with large teeth should not

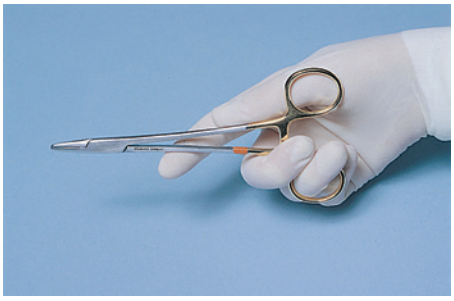


FIG. 7.6 A thenar grip provides good mobility, but releasing the needle holder by applying pressure with the ball of the thumb to the upper ring causes the handles to "pop" apart. This causes some movement of the needle in the tissue being sutured.



FIG. 7.7 The thumb-ring finger grip allows for the best precision of all grips and is preferable when suturing delicate tissue.

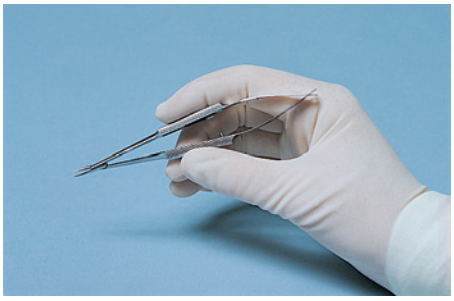


FIG. 7.8 A pencil grip is used with Castroviejo needle holders.

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FIG. 7.9 Tissue forceps. *Left to right:* Bishop-Harmon (smooth tip), Bishop-Harmon (toothed), Brown-Adson, 132 tissue, serrated, and DeBakey.

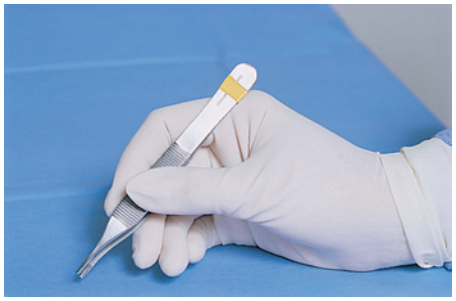


FIG. 7.10 Holding tissue forceps with a pencil grip provides greater maneuverability than is attained with other grips.

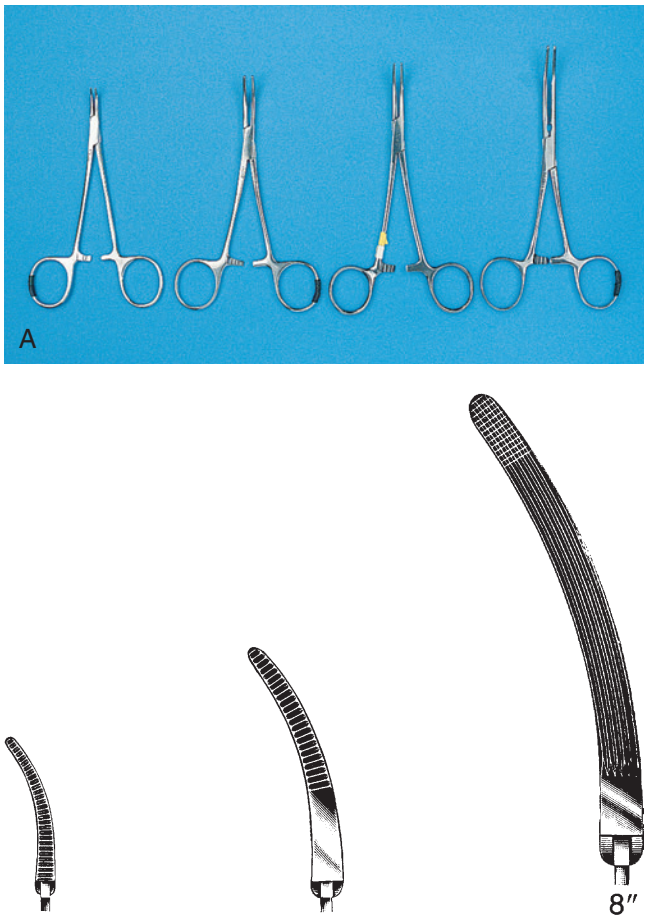


FIG. 7.11 (A) Hemostat forceps. *Left to right*: mosquito, Kelly, Crile, and Rochester-Carmalt. (B) Jaw detail of hemostatic forceps. *Left to right:* mosquito, Kelly, and Rochester-Carmalt.

be used to handle tissue that is easily traumatized. Tissue forceps with smooth tips, such as DeBakey forceps, are recommended for manipulation of delicate tissue, such as viscera or blood vessels. The most commonly used tissue forceps (i.e., Brown-Adson forceps) have small serrations on the tips that minimize trauma but facilitate holding tissue securely.

Tissue forceps are generally used in the nondominant hand. They should be held so that one blade functions as an extension of the thumb and the other blade functions as an extension of the opposing fingers (i.e., pencil position [Fig. 7.10]). Holding the shanks in the palm greatly limits maneuverability. When forceps are not in use, they can be palmed and held with the ring and little fingers, leaving the index and middle fingers free.

Tissue forceps are used to stabilize tissue and/or expose tissue layers during suturing. During suturing, tissue forceps are used on the far side of the wound to grasp the layer above the one being sutured. This layer is retracted upward and outward with the forceps, exposing the layer to be sutured. The needle point can then be placed at the desired level. Before the needle is driven completely through the tissue, the forceps should be moved from the superficial layer to grasp the layer being sutured. This layer can then be lifted to expose the needle's exit as it is passed through the tissue. The tissue layer on the near side being sutured is grasped and lifted to expose the desired needle entrance site. After the needle point has been placed at the desired site, the tissue forceps are moved and are used to retract the more superficial layer, thereby exposing the exit site. When needles are grasped with forceps during suturing, they should be grasped perpendicular to the shaft.

Hemostat Forceps

Hemostat forceps are crushing instruments used to clamp blood vessels (Fig. 7.11). They are available with straight or curved tips and vary in size from smaller (7.6 cm [3 in]) Mosquito hemostats with transverse jaw serrations to larger (22.9 cm [9 in]) angiotribes. Serrations on the jaws of larger hemostat forceps may be transverse, longitudinal, diagonal, or a combination of these. Longitudinal serrations are generally gentler to tissue than cross-serrations. Serrations usually extend from the tips of the jaws to the boxlocks, but in Kelly forceps, transverse (i.e., horizontal) serrations extend only over the distal portion of the jaws. Similarly sized Crile forceps have transverse serrations that extend the entire length of the jaw. Kelly and Crile forceps are used on larger vessels. Rochester-Carmalt forceps are larger crushing forceps often used to control large tissue bundles, such as during an ovariohysterectomy. They have longitudinal grooves with cross-grooves at the tip ends to prevent tissue slippage. Specialized

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cardiovascular forceps (e.g., Satinsky forceps) allow occlusion of only a portion of the vessel. Serrations of cardiovascular clamps provide tissue compression without cutting delicate vessel walls. Large teeth at the tip ends of some forceps (i.e., Ochsner) help prevent tissue slippage within forceps.

Curved hemostats should be placed on tissue with the curve facing up. As little tissue as possible should be grasped to minimize trauma, and the smallest hemostat forceps that can accomplish the job should be used. To prevent having fingers momentarily trapped in the rings of hemostats, fingertips should be placed on the finger rings, or fingers should be inserted into the rings only as far as the first joint.

Tissue Forceps

Tissue forceps are used to grasp or clamp tissue, varying the degree of tissue trauma that is created. Allis tissue forceps have interlocking sharp teeth; this instrument is used to firmly grasp tissue that is going to be removed from the body. Babcock tissue forceps have broad, flared, and blunt grasping tips that are more delicate than Allis tissue forceps and can be used carefully on tissue remaining in the body. Doyen intestinal forceps are noncrushing, occluding forceps with shallow longitudinal striations that are used to temporarily occlude the lumen of the bowel.

Retractors

Handheld retractors (Fig. 7.12) and self-retaining retractors (Fig. 7.13) are used to retract tissue and improve visualization. The ends of handheld retractors may be hooked, curved, spatula-shaped (i.e., Hohmann), or toothed. Some handheld retractors (e.g., malleable or ribbon retractors) may be bent by the surgeon to conform to the structure or area of the body being retracted. Senn (rake) retractors are small, double-ended retractors with three small, fingerlike projections on one end and a flat, curved blade on the other. Army-Navy retractors are larger with blunt, broad blades on each end for retraction of large amounts of tissue. Self-retaining retractors (e.g., Gelpi, Weitlaner) maintain tension on tissue and are held open with a boxlock or another device (e.g., a set-screw, such as in Balfour and Finochietto retractors [Fig. 7.14]). Balfour retractors generally are used to retract the abdominal wall, and Finochietto retractors are commonly used during thoracotomies.

Miscellaneous Instruments

Instruments are available to suction fluid (Fig. 7.15), clamp drapes or other inanimate objects (Fig. 7.16), cut and remove pieces of bone (rongeurs [Figs. 7.17 and 7.18]), hold bones during fracture repair (Fig. 7.19), scrape surfaces of dense tissue (curettes), remove periosteum (periosteal elevators [Fig. 7.20]), cut or shape bone and cartilage (osteotomes and chisels [Fig. 7.21]), and bore holes in bone (trephines). Magnifying loupes are useful when precise cutting or suturing of tissue is necessary (e.g., cardiovascular or

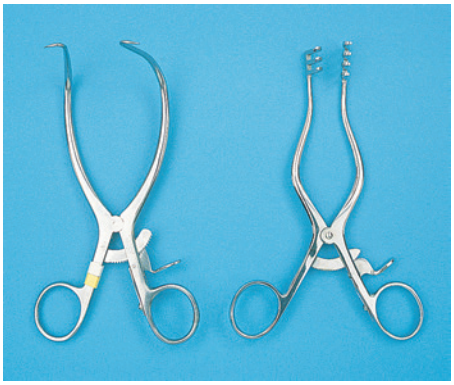


FIG. 7.13 Self-retaining retractors. *Left,* Gelpi; *right,* Weitlaner.

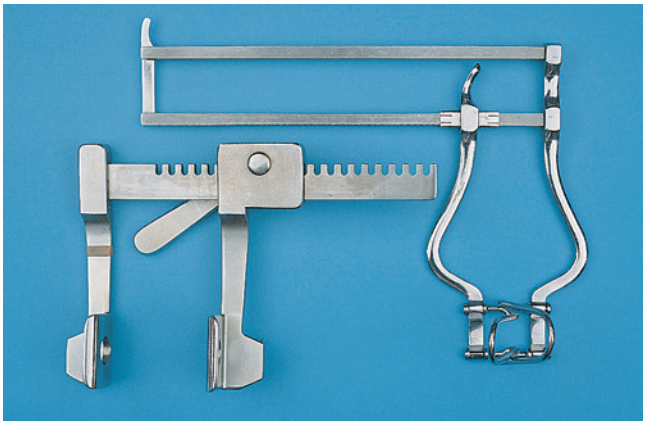


FIG. 7.14 Self-retaining retractors. *Left,* Finochietto; *right,* Balfour.

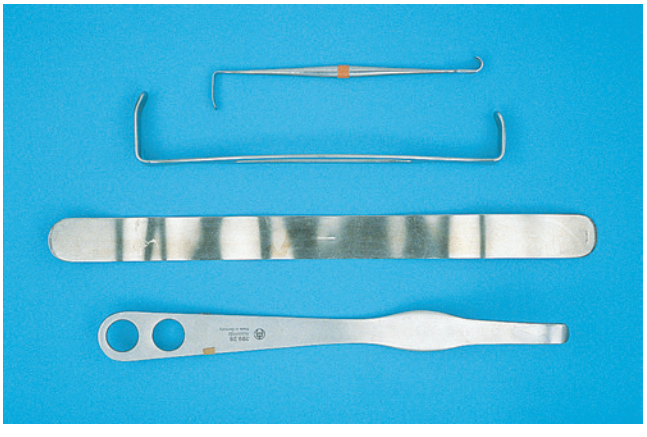


FIG. 7.12 Handheld retractors. *Top to bottom:* Senn, Army-Navy, malleable, and Hohmann.

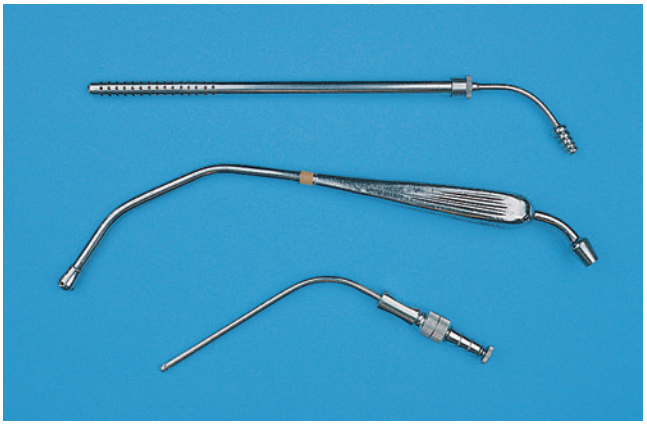


FIG. 7.15 Suction tips. *Top to bottom:* Poole, Yankauer, and Frazier.

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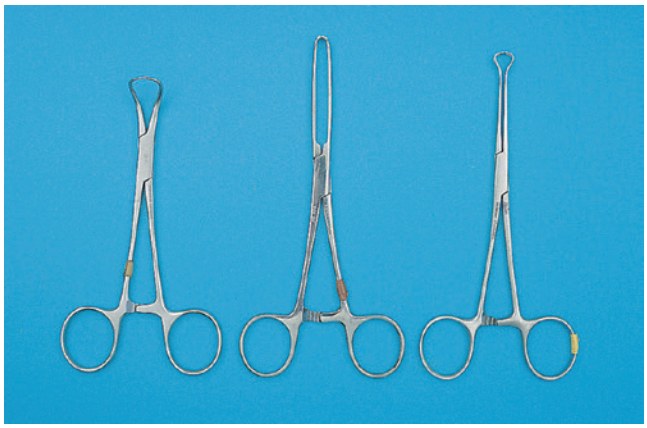


FIG. 7.16 Clamps and forceps. *Left to right:* Backhaus towel clamp, Allis tissue forceps, and Babcock forceps.

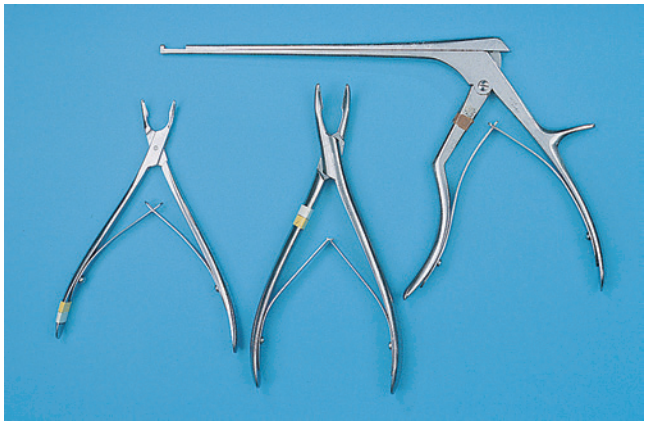


FIG. 7.17 Rongeurs. *Left to right:* Lempert, Ruskin, and Kerrison.

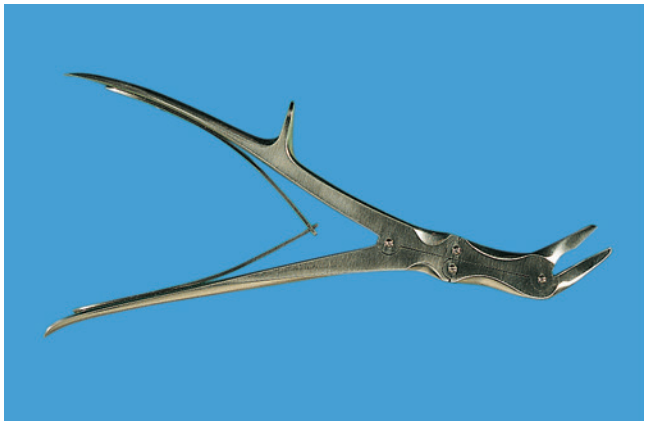


FIG. 7.18 Duck-bill double-action rongeurs.

neurologic surgery), and when relatively small tissues are handled (e.g., ureteral anastomoses). Numerous other specialized instruments have been developed to facilitate specific surgical procedures, particularly for minimally invasive surgery. Instrumentation for laparoscopy and thoracoscopy are described in Chapter 14. Some instruments used in orthopedic and neurologic procedures are shown in Figs. 7.22 to 7.24. Other orthopedic instruments are described in Chapter 32.

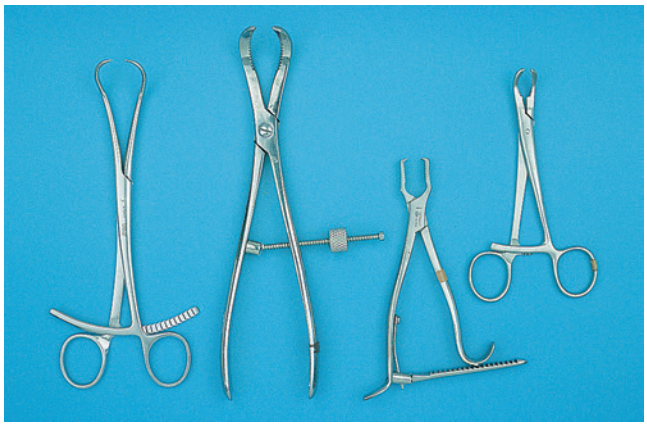


FIG. 7.19 Bone-holding forceps. *Left to right:* AO reduction forceps, large speed-lock reduction forceps, Lane bone-holding forceps, and small clamshell reduction forceps.



FIG. 7.20 Periosteal elevators. *Left,* AO round edge; *right,* AO curved blade and straight edge.

INSTRUMENT CARE AND MAINTENANCE

Good surgical instruments are valuable investments. They must be used properly and must receive routine care and maintenance to prevent corrosion, pitting, and discoloration and to prolong the life of the instruments (Table 7.1). Instruments should be rinsed in warm water immediately after the surgical procedure to prevent blood, tissue, saline, or other foreign matter from drying on them. If instruments cannot be immediately cleaned, they should be kept moist under a wet towel. Key takeaways from the Association of periOperative Registered Nurses (AORN) *Guideline for Cleaning and Care of Surgical Instruments* are listed in Box 7.1.[1]

Many manufacturers recommend that instruments be rinsed, cleaned, and sterilized in distilled or deionized water because tap water contains varying degrees of minerals that may discolor

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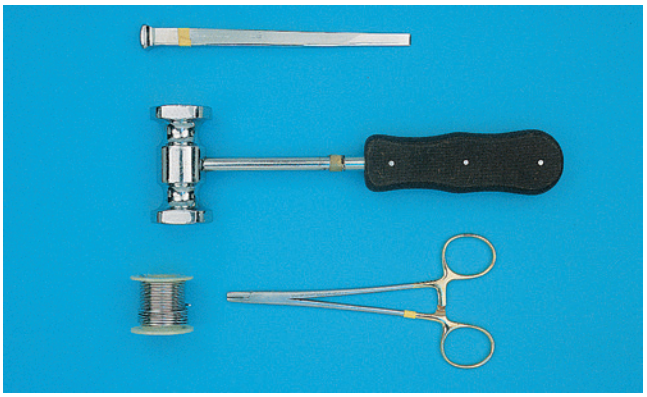


FIG. 7.21 Orthopedic equipment. *Top to bottom:* chisel, mallet, orthopedic wire, and wire twisters.

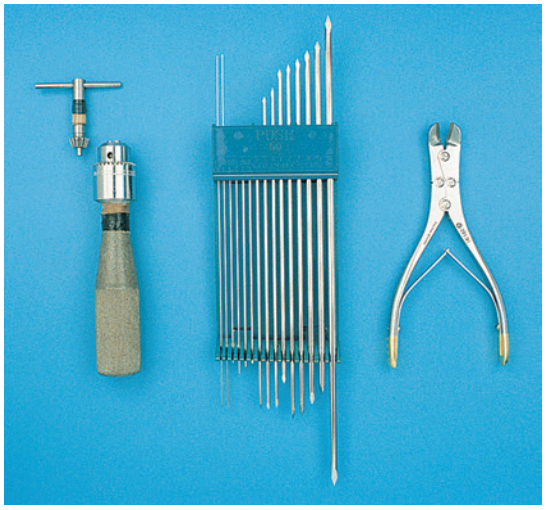


FIG. 7.22 Orthopedic equipment. *Left to right:* Jacobs chuck and key, Steinmann pins and Kirschner wires (pin caddy), and bone cutter.

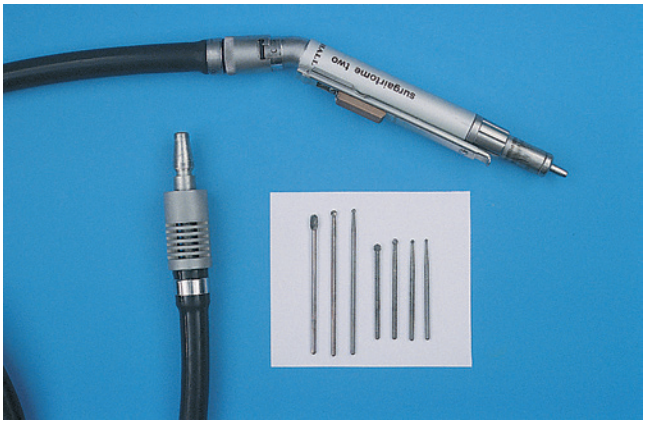


FIG. 7.23 Hall air drill and assorted bits.

and stain the instruments. If tap water is used for rinsing, instruments should be dried thoroughly to prevent staining and spotting. Instruments with several components should be disassembled before cleaning. Delicate instruments should be cleaned and sterilized separately.

BOX 7.1 Key Takeaways From 2014 AORN Guidelines for Cleaning and Care of Surgical Instruments[a]

- Clean and decontaminate instruments as soon as possible after use because this helps prevent biofilm formation. To reduce biofilm formation:
  - Remove gross soil at the point of use as dried blood and other organic material can be corrosive to instrument surfaces.
  - Do not allow blood or other bioburden to dry as this could make it more difficult to remove them and could compromise the effectiveness of the subsequent disinfection or sterilization.
  - Periodically irrigate the instruments with lumens to remove gross soil and reduce the risk of biofilm formation.
  - Keep the instruments moist by placing a towel moistened with sterile water over the instruments. DO NOT USE SALINE, which may cause pitting of the instruments.
- Laryngoscope blades may be a source of contamination to other patients because they come in contact with mucous membranes. To prevent this:
  - Clean blades and high-level disinfect or sterilize them after each use.
  - Handles are classified as a noncritical device; thus they require only cleaning and low-level disinfection. However, the rough surface of the laryngoscope handle can accumulate bioburden and occasional sterilization is warranted.
- Ophthalmic instruments have been associated with a syndrome of acute inflammation of the anterior segment of the eye in humans. To prevent this:
  - Ensure instruments are rinsed well and that no detergent residues remain.
  - Ensure that dried debris and residues of ophthalmic viscoelastic material have been cleaned from the instruments.
  - Rinse lumens and expel fluid from the lumen into the drain to prevent recontamination of the rinse water with debris from inside the lumen.
  - Use sterile, deionized water to rinse the instruments because untreated water may contain endotoxins or may cause stains, deposits, or corrosion of the instrument surface.
  - Dry lumens thoroughly with compressed air to eliminate moisture that could foster microbial growth.
  - Use cleaning products recommended by the manufacturer.

[a]Modified from Cowperthwaite L, Holm RL. Guideline implementation: surgical instrument cleaning. *AORN J.* 2015;101(5):542–549.
AORN, Association of periOperative Registered Nurses.

Cleaning

For instrument cleaning, use detergents with neutral or near-neutral pH (between 7 and 8); low-pH detergents corrode the protective surface of stainless steel if not fully rinsed off, whereas high-pH detergents corrode or cause "browning" of instruments and may impair function. Stains should be distinguished from rust (Table 7.2). Stains can be removed, whereas rust causes permanent damage. Enzymatic solutions containing proteases, lipases, and amylases are often added to neutral pH detergents to effectively and efficiently remove organic material from general surgical instruments and endoscopic equipment; these solutions must be thoroughly rinsed from instrumentation to avoid adverse reactions. Studies differ with regard to effectiveness of enzymatic versus nonenzymatic cleaners.

NOTE Do not use Betadine solution, dish soap, laundry soap, or hand scrubbing solutions to clean instruments because spotting and corrosion will occur.

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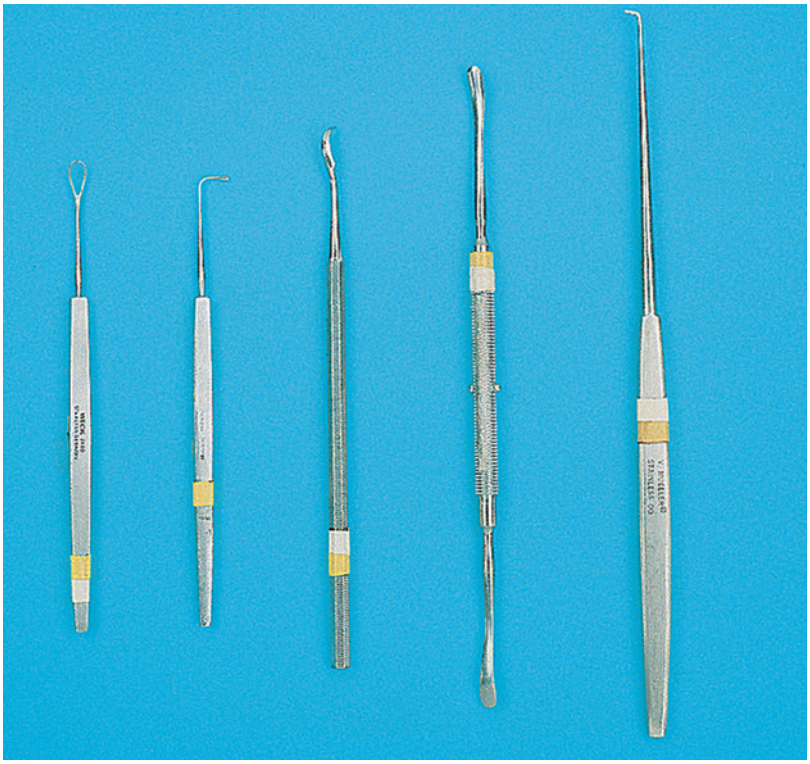


FIG. 7.24 Neurosurgery equipment. *Left to right:* lens loop, small nerve root retractor, tartar scraper, Freer dissector, and large right-angle nerve root retractor.

TABLE 7.1 Causes of Instrument Corrosion, Pitting, or Discoloration

Type and Cause of Damage	Solution
Corrosion	
• Excessive moisture left on the surface of the instrument or in the instrument pack	• Preheat the autoclave; allow instruments to cool slowly; check autoclave valves for leaks
• Rinsing with tap water; deposition of alkali earth on walls of autoclave, which deposits on instruments	• Use distilled or deionized water during sterilization; periodically clean autoclave with acetic acid
• Prolonged exposure to enzymatic cleaning solutions	• Do not expose carbon steel instruments to enzymatic cleaners for longer than 5 minutes
Pitting	
• Exposing instruments to saline or foreign materials	• Rinse instruments with distilled water immediately after the procedure
• Detergent residue on instruments during autoclaving	• Avoid detergents with a chloride base, which form hydrochloric acid when combined with steam
• Use of alkaline detergents that remove the chromium oxide coat	• Use detergents that have a pH near 7
• Simultaneous cleaning of metals of dissimilar composition in an ultrasound cleaner	• During cleaning, separate instruments made from dissimilar metals
Rust Deposition	
• Deposition of iron on instrument from tap water	• Use distilled or deionized water during cleaning, rinsing, and sterilization
• Deposition and oxidation of carbon particles on stainless steel instruments when they are sterilized with chrome-plated instruments that have exposed metal	• Separate the two types of steel during sterilization; replace plated instruments that are peeling or imperfect
Spotting	
• Condensation and slow evaporation of water droplets containing sodium, calcium, and/or magnesium on instruments	• Follow instructions for autoclave use; open door after steam has been exhausted; check valves or gaskets; use distilled or deionized water

Ultrasonic cleaning can be used to follow manual cleaning of surgical instruments. This method removes small or microscopic organic material by cavitation and implosion in which waves of acoustic energy are propagated in aqueous solutions to disrupt the bonds that hold particulate matter to surfaces. Dissimilar metals (e.g., chrome and stainless steel) should not be mixed in the same ultrasonic cycle. All instruments should be placed in the ultrasonic cleaner with ratchets and boxlocks open and should not be piled on top of each other because delicate instruments could be damaged. Instruments should be

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TABLE 7.2 Troubleshooting Stain Guide for Surgical Instruments[a]

Stain Color	Cause
Brown/orange	High-pH detergents, chlorhexidine, or improper soaking of instruments. May also be caused by soaking in tap water.
Dark brown	Low-pH instrument solutions. The brownish-colored film may also be caused by a malfunctioning sterilizer. Similar localized stain spots may be a result of baked-on blood.
Bluish black	Reverse plating, when instruments of different metals (e.g., chrome, stainless steel) are ultrasonically processed together. Additionally, exposure to saline, blood, or potassium chloride will cause this.
Multicolor	Excessive heat by a localized hot spot in the sterilizer. The rainbow-colored stain can be removed.
Light- and dark-colored spots	Water droplets drying on the instruments. Slow evaporation leaves sodium, calcium, and magnesium deposits.
Bluish gray	Liquid (cold) sterilization solutions are being used beyond manufacturer's recommendations.
Black	Contact was made with ammonia or a solution containing ammonia.
Gray	A liquid rust remover is being used in excess of manufacturer's recommendations.
Rust	Dried blood that has become baked on the serrated or hinged areas of surgical instruments. This organic material, once baked on, may appear dark in color. Rust also can be caused by soaking in tap water.

[a]Modified from Spectrum surgical instruments, repairs, instrument accessories; Spectrum Surgical Instruments Corp. http://www.spectrumsurgical.com.

removed from the cleaner and rinsed and dried at completion of the cycle.

If ultrasonic cleaning is unavailable, instruments should be cleaned as thoroughly as possible. Use an instrument cleaning brush to remove debris from the jaw serrations, teeth, and hinged areas. Various specialized instrument brushes are available (e.g., Frazier suction tube brushes, laparoscopic brushes, bone reamer brushes, endoscopic brushes), along with general instrument cleaning brushes. A soft nylon brush or toothbrush may also be used; rasps and serrated areas may require a wire brush. Dry surgical instruments on a clean paper towel. Place the fine tips of surgical hemostats face up on towels to prevent damage.

Lubricating and Autoclaving

Instruments with boxlocks, hinges, and power equipment should be lubricated before autoclaving. Only surgical lubricants should be used because they are steam penetrable; industrial oils interfere with steam sterilization and should not be used. Lubricant sprays are preferred over submersion in lubricant milk due to the potential for bacterial contamination from previously dipped instruments.

Surgical instruments are generally grouped into packs or kits according to their use (Tables 7.3 and 7.4). Before autoclaving, instruments should be wrapped in cloth or placed on a cloth inside a fenestrated pan to absorb moisture. Instruments should be sterilized with boxlocks or hinges open.

NOTE Never lock an instrument during autoclaving; this prevents steam from reaching and sterilizing overlapping metal surfaces. Hinge areas of forceps and hemostats can expand and crack when exposed to heat during autoclaving if they are locked.

The chamber should not be overloaded, and stacking of instruments should be avoided to prevent damage to delicate instruments. Kits should be double-wrapped (see p. 11) and sealed with autoclave tape. A sterilization monitor (e.g., OK sterilization indicators [Propper Manufacturing Company], Sterrad chemical indicator strips [Johnson & Johnson]) should be added before autoclaving (see p. 10). Rapid cooling of instruments should be avoided to prevent condensation. Additional information on autoclaving and other methods of sterilization can be found in Chapter 2.

TABLE 7.3 Suggestions for a Basic Soft Tissue Pack[a]

Instrument	Quantity
Halsted-mosquito hemostats, curved, 5 inch (12.5 cm)	2
Halsted-mosquito hemostats, straight, 5 inch (12.5 cm)	2
Kelly hemostats, curved, 5.5 inch (14 cm)	2
Crile forceps, straight, 5.5 inch (14 cm)	2
Rochester-Carmalt hemostats, curved, 7.25 inch (18.5 cm)	4
Mayo-Hegar or Olsen-Hegar needle holders, 7 inch (18 cm)	1
Brown-Adson tissue forceps	1
Allis tissue forceps, 5 × 6 teeth, 6 inch	4
Backhaus towel clamps, 5.25 inch (13 cm)	4
Metzenbaum scissors, curved, 8 inch (20 cm)	1
Mayo scissors, curved, 8 inch (20 cm)	1
Suture scissors, sharp-blunt, straight, 5 inch (12.5 cm)	1
Instrument tray	1
Senn retractors	2
Blade handle, No. 3	1
Ovariohysterectomy "spay" hook	1
Saline bowl	1
Radio-opaque sponges (4 × 4 inches; 10.2 × 10.2 cm))	20

[a]For spaying, laparotomy, or wound repair.

NOTE To determine whether a brown or orange discoloration is a stain or rust, use the eraser test. Rub a pencil eraser over the discoloration. If the eraser removes discoloration and the metal underneath is smooth and clean, this is a stain. If a pit mark appears under the discoloration, this is corrosion or rust.

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TABLE 7.4 Suggestions for a Basic Orthopedic Pack[a]

Instrument	Quantity
Jacobs chuck and key	1
Hohmann retractor	2
Army-Navy retractor	2
Periosteal elevator	1
Wire twister	1
Medium pin cutter	1
Kern or Lane bone-holding forceps	2
Reduction forceps	1
Orthopedic wire (18, 20, and 22 gauge)	1 each size
Kirschner wires	2 each size
Intramedullary pins	2 each size

[a]Augmented with a general pack (see Table 7.3).

Cold Sterilization

Cold sterilization is used for some instruments but does not guarantee sterility. Instruments that cannot be autoclaved are best sterilized using alternate means (e.g., ethylene oxide or plasma sterilization; see pp. 5–8). Solutions that contain benzyl ammonium chloride (BAC) should not be used with instruments that have tungsten carbide inserts because BAC dissolves tungsten.

DRAPING AND ORGANIZING THE INSTRUMENT TABLE

Instrument tables should be height adjustable to allow them to be positioned within reach of surgical personnel. The instrument table should not be opened until the animal has been positioned on the surgical table and draped. Large, water-impermeable table drapes should cover the entire instrument table. To open these drapes, the drape and outer wrap are positioned on the instrument table and the exposed underneath surface of the drape is gently grasped. The ends and then the sides are unfolded. Once the drape has been opened, nonsterile personnel should not reach over it. Mayo stands often are used in procedures that require additional instruments, such as bone plating; specially designed stand covers are available for these tables. After the instrument pack has been opened (see p. 14), instruments should be positioned so that they can be readily retrieved. The layout is generally determined by the surgeon's preference, but grouping of similar instruments (e.g., scissors, retractors) facilitates their use. Whenever a body cavity is opened, sponges should be counted at the beginning of the procedure (before the incision is made) and again before closure to ensure that none have been inadvertently left in the cavity. Contaminated instruments and soiled sponges should not be placed back on the instrument table.

REFERENCE

1. Guideline for cleaning and care of surgical instruments. In: *Guideline for Perioperative Practice.* Denver: AORN; pp. 615-650, 2015.

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8

Biomaterials, Suturing, and Hemostasis



SUTURES AND SUTURE SELECTION

Suture plays an important role in wound repair by providing hemostasis and support for healing tissue. Tissues have different requirements for suture support, depending on the type of tissue and anticipated duration of healing. Some tissues need support for only a few days (e.g., muscle, subcutaneous tissue, skin), whereas others require weeks (fascia) or months (tendon) to heal. Individual patient variation further affects suture choice. Healing of wounds may be delayed by infection, obesity, malnutrition, neoplasia, drugs (e.g., steroids), and collagen disorders. In rapidly healing tissue, a suture that will lose its tensile strength at a similar rate that the tissue gains strength, and that will be absorbed by the tissue so that no foreign material remains in the wound, is ideal. Minimally invasive surgical techniques (see Chapter 14) put additional demands on the performance of surgical sutures. Not only must good knot security be maintained, but also the surface lubricant must ensure ease of manipulation, minimal tissue drag, and good biocompatibility with minimal inflammatory responses. Subjective preferences, such as familiarity with the material and availability, need also be taken into consideration when choosing a suture material.

Suture Characteristics

The ideal suture is easy to handle, reacts minimally in tissue, inhibits bacterial growth, holds securely when knotted, resists shrinking in tissue, absorbs with minimal reaction after the tissue has healed, and is noncapillary, nonallergenic, noncarcinogenic, and nonferromagnetic; however, such a material does not exist. Therefore surgeons must choose a suture that most closely approximates the ideal for a given procedure and tissue to be sutured. A wide variety of suture and needle combinations are available.

Suture Size

The smallest diameter suture that will adequately secure wounded tissue should be used to minimize trauma as the suture is passed through the tissue, and to reduce the amount of foreign material left in the wound. There is no advantage to using a suture that is stronger than the tissue to be sutured. The most commonly used standard for suture size is the United States Pharmacopeia (USP), which denotes dimensions from fine to coarse (with diameters in inches) according to a numeric scale, with 12-0 being the smallest and 7 the largest. The USP uses different standards for surgical gut and other materials (Table 8.1). The smaller the suture size, the less tensile strength it has. Stainless steel wire is sized according to the metric or USP scale or by the Brown and Sharpe (B and S) wire gauge (see Table 8.1).

Flexibility

The flexibility of a suture is determined by its torsional stiffness and diameter, which influence its handling and use. Flexible sutures are indicated for ligating vessels or performing continuous suture patterns. Less flexible sutures (e.g., wire) cannot be used to ligate small bleeders. Nylon and surgical gut are relatively stiff compared with silk suture; braided polyester sutures have intermediate stiffness.

Surface Characteristics and Coating

The surface characteristics of a suture influence the ease with which it is pulled through tissue (i.e., the amount of friction or "drag") and the amount of trauma caused. Rough sutures cause more injury than smooth sutures. Smooth surfaces are particularly important in delicate tissues, such as the eye. However, sutures with smooth surfaces also require greater tension to ensure good apposition of tissues, and have less knot security. Braided materials have more drag than monofilament sutures. Braided materials are often coated to reduce capillarity (see later discussion), but this also provides a smooth surface. Teflon, silicone, wax, paraffin-wax, and calcium stearate are used for coating sutures.

Capillarity

Capillarity is the process by which fluid and bacteria are carried into the interstices of multifilament fibers. Because neutrophils and macrophages are too large to enter the interstices of the fiber, infection may persist, particularly in nonabsorbable sutures. All braided materials (e.g., polyglycolic acid, silk) have degrees of capillarity, whereas monofilament sutures are considered noncapillary. Coating reduces the capillarity of some sutures, but regardless, capillary suture materials should not be used in contaminated or infected sites.

Knot Tensile Strength

Knot tensile strength is measured by the force in pounds that the suture strand can withstand before it breaks when knotted (Box 8.1). Sutures should be as strong as the normal tissue through which they are being placed; however, the tensile strength of the suture should not greatly exceed the tensile strength of the tissue.

Relative Knot Security

Relative knot security is the holding capacity of a suture expressed as a percentage of its tensile strength. The knot-holding capacity of a suture material is the strength required to untie or break a defined knot by loading the part of the suture that forms the loop; the suture material's tensile strength is the strength required to break an untied fiber with a force applied in the direction of its length (see Box 8.1).

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ABSTRACT

When choosing a suture material, the surgeon must be knowledgeable about the healing properties of the particular tissue, the wound healing capability of the patient, and the characteristics of suitable suture materials (particularly tissue reactivity, tensile strength, knot security, and rate of absorption). The type of suture to be used depends on the procedure being performed and the tissue in which it will be placed. The suture material chosen must retain its strength until the wound heals sufficiently. Other methods for hemostasis and tissue apposition include, but are not limited to, energy-based devices and tissue adhesives (e.g., cyanoacrylates).

KEYWORDS

knot security
tensile strength
absorbable
nonabsorbable
monofilament
multifilament
interrupted
continuous
cyanoacrylate
energy-based hemostasis

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TABLE 8.1 Suture Sizes

Synthetic Suture Materials (USP)	Surgical Gut (USP)	Brown and Sharpe Wire Gauge	Metric Gauge	Actual Size (mm)
10-0			0.2	0.02
9-0			0.3	0.03
8-0			0.4	0.04
7-0	8-0	41	0.5	0.05
6-0	7-0	38–40	0.7	0.07
5-0	6-0	35	1	0.1
4-0	5-0	32–34	1.5	0.15
3-0	4-0	30	2	0.2
2-0	3-0	28	3	0.3
0	2-0	26	3.5	0.35
1	0	25	4	0.4
2	1	24	5	0.5
3,4	2	22	6	0.6
5	3	20	7	0.7
6	4	19	8	0.8
7		18	9	0.9

USP, United States Pharmacopeia.

BOX 8.1 Terminology Used to Describe Suture Characteristics

- *Absorbability.* Progressive loss of mass and/or volume of suture material; does not correlate with initial tensile strength.
- *Breaking strength.* Limit of tensile strength at which suture failure occurs.
- *Capillarity.* Extent to which absorbed fluid is transferred along the suture.
- *Elasticity.* Measure of the ability of the material to regain its original form and length after deformation.
- *Fluid absorption.* Ability to take up fluid after immersion.
- *Knot-pull tensile strength.* Breaking strength of knotted suture material (10%–40% weaker after deformation by knot placement).
- *Knot strength.* Amount of force necessary to cause a knot to slip (related to the coefficient of static friction and plasticity of a given material).
- *Memory.* Inherent capability of suture to return to or maintain its original gross shape (related to elasticity, plasticity, and diameter).
- *Plasticity.* Measure of the ability to deform without breaking and to maintain a new form after relief of the deforming force.
- *Pliability.* Ease of handling of suture material; ability to adjust knot tension and to secure knots (related to suture material, filament type, and diameter).
- *Straight-pull tensile strength.* Linear breaking strength of suture material.
- *Suture pullout value.* The application of force to a loop of suture located where tissue failure occurs, which measures the strength of a particular tissue; variable depending on anatomic site and histologic composition (fat, 0.2 kg; muscle, 1.27 kg; skin, 1.82 kg; fascia, 3.77 kg).
- *Tensile strength.* Measure of the ability of a material or tissue to resist deformation and breakage.
- *Wound breaking strength.* Limit of tensile strength of a healing wound at which separation of the wound edges occurs.

From Lai SY, Becker DG. Sutures and needles, e-medicine. *Topic.* 2004;38.

Specific Suturing Materials

Suture materials may be classified according to their behavior in tissue (absorbable or nonabsorbable), their structure (monofilament or multifilament), or their origin (synthetic, organic, or metallic) (Fig. 8.1 and Table 8.2). Two major mechanisms of absorption result in the degradation of absorbable sutures. Sutures of organic origin, such as surgical gut, are gradually digested by tissue enzymes and phagocytized, whereas sutures manufactured from synthetic polymers are principally broken down by hydrolysis. Nonabsorbable sutures are ultimately encapsulated or walled off by fibrous tissue.

Monofilament sutures are made of a single strand of material and therefore have less tissue drag than multifilament sutures and do not have interstices that may harbor bacteria or fluid. Care should be used in handling monofilament sutures because nicking or damaging the material with forceps or needle holders may weaken the suture and predispose it to breakage. Multifilament sutures consist of several strands of suture that are twisted or braided together. Multifilament sutures generally are more pliable and flexible than monofilament sutures. They may be coated to reduce tissue drag and enhance handling characteristics (see previous discussion).

Absorbable Suture Materials

Absorbable suture materials lose most of their tensile strength within 60 days and eventually disappear from the tissue implantation site because they have been phagocytized or hydrolyzed (see Fig. 8.1 and Table 8.2). The time to loss of strength and for complete absorption varies among suture materials.

Organic absorbable materials. Catgut (surgical gut) is the most common nonsynthetic absorbable suture material. The word *catgut* is derived from the term *kitgut* or *kitstring* (the string used on a kit, or fiddle). Misinterpretation of the word *kit* as referring to a young cat led to the use of the term catgut. Surgical gut is made from the submucosa of sheep intestine or the serosa of bovine intestine, and is approximately 90% collagen. It is broken down by phagocytosis and, in contrast with other suture materials, elicits a notable inflammatory reaction. Plain surgical gut loses strength rapidly after tissue implantation. "Tanning" (cross-linking of collagen fibers), which occurs by exposure to chrome or aldehyde, slows absorption. Surgical gut is available as plain, medium chromic, or chromic; increased tanning generally implies prolonged strength and reduced tissue reaction. Surgical gut is rapidly removed from infected sites or areas where it is exposed to digestive enzymes, and is quickly degraded in catabolic patients. The knots may loosen when wet.

Synthetic absorbable materials. Synthetic absorbable materials (see Table 8.2) are generally broken down by hydrolysis and cause minimal tissue reaction. The time to loss of strength and to absorption is fairly constant in different tissue. Infection or exposure to digestive enzymes does not significantly influence the rate of absorption of most synthetic absorbable sutures. Polyglactin 910 and polyglycolic acid are more rapidly hydrolyzed in alkaline environments but are relatively stable in contaminated wounds. Polyglycolic acid, polyglactin 910, and poliglecaprone 25 may be rapidly degraded in infected urine; polydioxanone, polyglyconate, and glycomer 631 are acceptable for use in sterile bladders and those infected with *E. coli*. However, use of any suture that is degraded via hydrolysis may be at risk for accelerated degradation when the bladder is infected with *Proteus* spp. (see also p. 696) as all common monofilament absorbable sutures have been shown to degrade within 7 days in *P. mirabilis*-inoculated urine.

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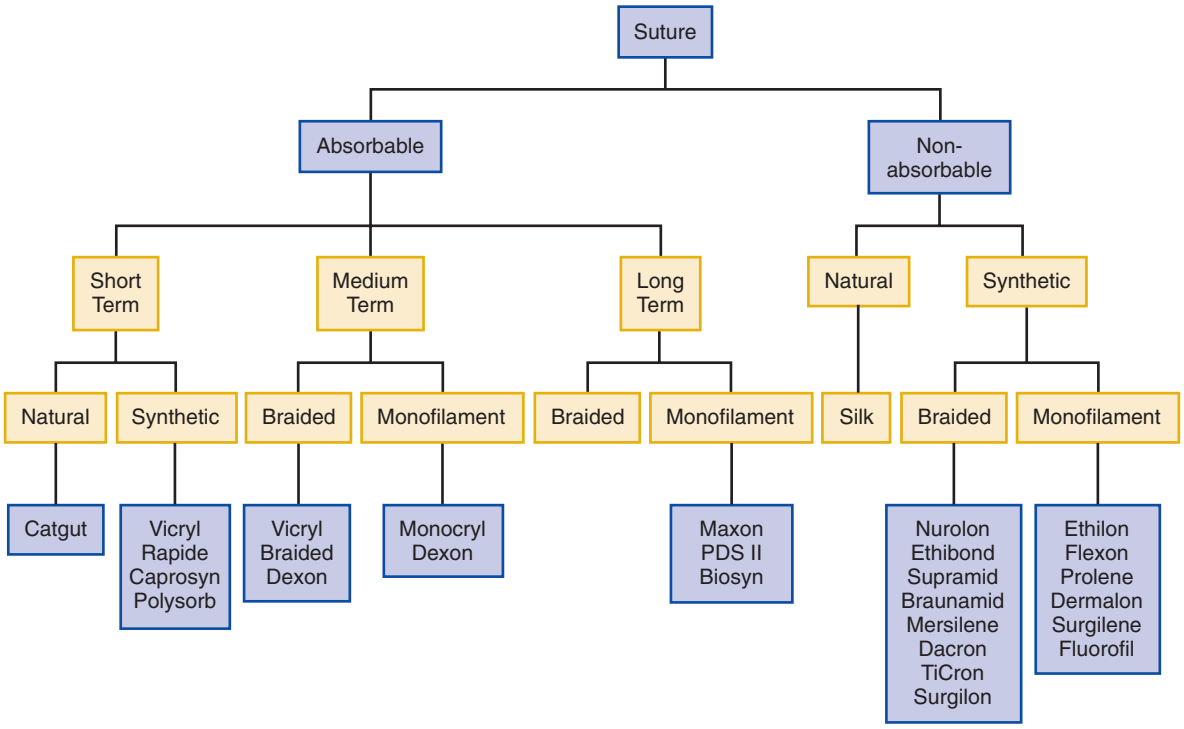


FIG. 8.1 Characteristics of sutures used in veterinary medicine.

Monofilament absorbable materials. Polydioxanone (PDS II) and polyglyconate (Maxon) are classic monofilament sutures that retain their tensile strength longer than multifilament sutures, with complete absorption occurring in 6 months. Poliglecaprone 25 (Monocryl) and Glycomer 631 (Biosyn) are monofilament rapidly absorbable synthetic materials that are pliable, lack stiffness, and have good handling characteristics. These sutures have good initial tensile strength that deteriorates in 2 to 3 weeks following implantation, and are completely absorbed by 120 days.

Polyglytone 6211 (Caprosyn) is a relatively new, rapidly absorbable monofilament suture. It is a synthetic polyester of glycolide, caprolactone, trimethylene carbonate, and lactide. Absorption of this material is essentially complete in 56 days, which is thought to lead to less wound complications and tissue reactions. This suture retains up to 30% knot strength at 10 days postimplantation and has excellent handling characteristics; however, its rapid absorption also limits its application.

Multifilament absorbable materials. Polyglycolic acid (Dexon) is braided from filaments extracted from glycolic acid and is available in both coated and uncoated forms. Polyglactin 910 (Vicryl) is a multifilament suture made of a copolymer of lactide and glycolide with polyglactin 370. It is coated with calcium stearate, and its rate of loss of tensile strength is similar to that of polyglycolic acid. Vicryl Rapide is a rapidly absorbed, synthetic braided suture that has an initial strength that is comparable to nylon and gut. However, the tensile strength declines to 50% in 5 to 6 days, and it is completely absorbed in 42 days. This suture is indicated for superficial closure of mucosa, gingival closure, and periocular skin closure.

Polysorb sutures are composed of Lactomer glycolide/lactide copolymer, which is a synthetic polyester composed of glycolide and lactide (derived from glycolic and lactic acids). Polysorb maintains 80% of its tensile strength at 2 weeks and 30% at 3 weeks, with absorption ranging from 56 to 70 days. Velosorb Fast sutures are composed of absorbable synthetic polyester comprised of glycolide and lactide. This suture is indicated for use in soft tissue approximation of the skin and mucosa, when only 7 to 10 days of wound support is required, as it retains the majority of its tensile strength until approximately 5 days. Complete absorption of Velosorb takes 40 to 50 days.

Nonabsorbable Suture Materials

Organic nonabsorbable materials. Silk is the most common organic nonabsorbable suture material used. It is a braided multifilament suture made by a special type of silkworm, and is marketed as uncoated or coated. Silk has excellent handling characteristics and is often used in cardiovascular procedures; however, it does not maintain significant tensile strength after 6 months and is therefore contraindicated for use in vascular grafts. It should also be avoided in contaminated sites; one silk suture may reduce the number of bacteria required to induce infection in a wound from 10^6 to 10^3.

Synthetic nonabsorbable materials. Synthetic nonabsorbable suture materials (see Table 8.2) are marketed as braided multifilament threads (e.g., polyester or coated caprolactam) or monofilament threads (e.g., polypropylene, polyamide, or polybutester). These sutures are typically strong and induce minimal tissue reaction. Nonabsorbable suture materials with an inner core and an outer sheath (e.g., Supramid) should not be buried in tissue because it may predispose to infection and fistulation. The outer sheath frequently is broken, which allows bacteria to reside underneath it.

NOTE Nylon cable ties are not recommended to be implanted in the body (e.g., used to ligate ovarian pedicles), as toxic substances are released during degradation, and may result in abscess or tumor formation.

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TABLE 8.2 Characteristics of Suture Materials Commonly Used in Veterinary Medicine

Generic Name	Trade Name	Manufacturer	Suture Characteristics	Reduction in Tensile Strength[a]	Complete Absorption (Days)	Relative Knot Security[b]	Tissue Reaction[c]
Chromic surgical gut (catgut)	—	—	Absorbable Multifilament	33% at 7 days	60	— (wet)	+++
Polyglactin 910	Vicryl and Vicryl Plus	Ethicon	Absorbable Multifilament	25% at 14 days 50% at 21 days	56–70	++ ++	+ +
	Vicryl Rapide	Ethicon	Absorbable Multifilament	50% at 5 days 100% at 14 days	42	++	+
Polyglycolic acid	Dexon "S" (uncoated) Dexon II (coated)	Medtronic	Absorbable Multifilament	35% at 14 days 65% at 21 days	60–90	++	+
Glycolide/ lactide polymer	Polysorb	Medtronic	Absorbable Multifilament	20% at 14 days 70% at 21 days	60	+++	—
Polydioxanone	PDS II	Ethicon	Absorbable Monofilament	14% at 14 days 31% at 42 days	180	++	+
Polyglyconate	Maxon	Medtronic	Absorbable Monofilament	30% at 14 days 45% at 21 days	180	++	+
Poliglecaprone 25	Monocryl	Ethicon	Absorbable Monofilament	40%–50% at 7 days 70%–80% at 14 days	90–120	++	+
Glycomer 631	Biosyn	Medtronic	Absorbable Monofilament	25% at 14 days 60% at 21 days	90–110	++	+
Polyglytone 6211	Caprosyn	Medtronic	Absorbable Monofilament	40%–50% at 5 days 70%–80% at 10 days	56	+++	+
Silk	Perma-Hand	Ethicon Medtronic	Nonabsorbable Multifilament	30% at 14 days 50% at 1 year	> 2 years	—	+++
Polyester	Mersilene (uncoated) Ethibond (coated) Dacron (uncoated) Ticron (coated)	Ethicon Ethicon Medtronic Medtronic	Nonabsorbable Multifilament			—	++
Polyamide (Nylon)	Ethilon (monofilament) Nurolon (multifilament) Dermalon (monofilament) Surgilon (multifilament)	Ethicon Ethicon Medtronic Medtronic	Nonabsorbable Monofilament or multifilament	30% at 2 years (monofilament) 75% at 180 days (multifilament)		+	—
Polypropylene	Prolene Surgilene Fluorofil	Ethicon Medtronic Mallinckrodt Veterinary	Nonabsorbable Monofilament			+++	—
Polybutester	Novafil	Medtronic	Nonabsorbable Monofilament			++	—
Polymerized caprolactam	Supramid Braunamid Vetcassette II	S. Jackson B. Braun Melsungen Ag Mallinckrodt Veterinary	Nonabsorbable Multifilament			++	++ (if coating breaks)
Stainless steel wire	Flexon (multifilament)	Medtronic Ethicon	Nonabsorbable Monofilament or Multifilament			+++	—

[a]Values given are approximate. Actual loss of tensile strength may vary depending on suture and tissue.
[b](–), Poor (<60%); (+), fair (60%–70%); (++), good (70%–85%); (+++), excellent (>85%).
[c](–), Minimal to none; (+), mild; (++), moderate; (+++), severe.

Metallic sutures. Stainless steel is the metallic suture most commonly used and is available as a monofilament or multifilament twisted wire. Surgical steel is strong and inert with minimal tissue reaction, but knot ends evoke an inflammatory reaction. Stainless steel has a tendency to cut tissue and may fragment and migrate. It is stable in contaminated wounds and is the standard for judging knot security and tissue reaction to suture materials.

Antimicrobial-coated sutures. Triclosan-impregnated suture is available in certain monofilament (PDS Plus, Monocryl Plus) and multifilament (Vicryl Plus) absorbable sutures. These sutures create an in vitro zone of inhibition against *Staphylococcus aureus*,

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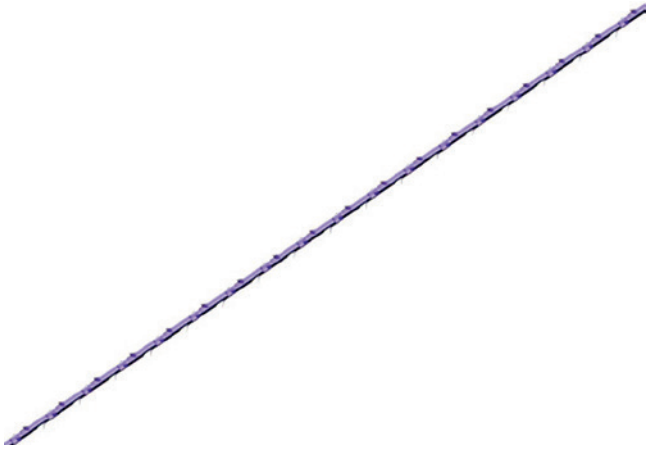


FIG. 8.2 Barbed suture material with unidirectional barbs notched into the strand.

Staphylococcus epidermidis, and *Escherichia coli* and are used to decrease rates of surgical site infection. Systematic reviews and meta-analyses in the human literature have failed to find evidence demonstrating the protective effect of triclosan-impregnated sutures on the occurrence of surgical site infections.[1,2] In the veterinary literature, a retrospective cohort study did not find additional benefit for using triclosan-impregnated suture for wound closure following tibial plateau leveling osteotomy in dogs.[3] However, in vitro comparison of impregnated versus nonimpregnated suture for adherence of methicillin-resistant *Staphylococcus pseudointermedius* found PDS Plus to have significantly less adherence than PDS II, Monocryl, Vicryl, and Stratafix Spiral PDO.[4]

Knotless barbed suture. Barbed suture material (e.g., V-Loc, Stratafix, Quill) has unidirectional barbs placed in a helical fashion around the suture strand (Fig. 8.2). These barbs allow the suture to become lodged in the tissue, creating multiple anchor points to distribute tension along the suture line. These sutures are also knotless as there is a preconstructed loop on the end to allow initial anchor of the suture line. Due to the unidirectional movement and anchoring of the suture through the tissue, there is no need for an end knot. Bidirectional barbs with double-swaged needles are also available on certain knotless suture materials (i.e., Quill, Statafix). Multiple experimental biomechanical studies have demonstrated similar tensile strength to standard monofilament suture material in a variety of tissues and procedures.[5–7] However, for tendon repair, smooth monofilament sutures were consistently stronger and were associated with less gap formation than knotless barbed suture.[8,9] Barbed monofilament suture (Stratofix Spiral PDO) has also been shown to have a comparable bacterial adherence profile of methicillin-resistant *S. pseudintermedius* to nonbarbed monofilament suture.[4]

Surgical Needles

A variety of needle shapes and sizes are available; selection of a needle depends on the type of tissue to be sutured (e.g., penetrability, density, elasticity, and thickness), topography of the wound (e.g., deep or narrow), and characteristics of the needle (i.e., type of eye, length, and diameter). Needle strength, ductility, and sharpness are important factors in determining the handling characteristics and use of a needle. The amount of angular deformation a needle can withstand before becoming permanently deformed is called *surgical yield*. Ductility is the needle's resistance to breaking under a specified amount of bending. The sharpness of a needle is related to the angle of the point (see p. 65) and the taper ratio of the needle. The sharpest needles have a long, thin, tapered point with smooth cutting edges. Most surgical needles are made from stainless steel wire because it is strong, corrosion free, and does not harbor bacteria.

The three basic components of a needle are the attachment end (i.e., swaged or eyed end), the body, and the point (Fig. 8.3A). Eyed needles must be threaded, and because a double strand of suture is pulled through the tissue, a larger hole is created than when swaged suture material is used. Eyed needles may be closed (i.e., round, oblong, or square) or French (i.e., with a slit from the inside of the eye to the end of the needle for ease of threading) (see Fig. 8.3B). Eyed needles are threaded from the inside curvature and are uncommonly used in surgical practice. With swaged sutures, the needle and suture are a continuous unit, which minimizes tissue trauma and increases ease of use.

The needle body comes in a variety of shapes (see Fig. 8.3C); the tissue type and depth and the size of the wound determine the appropriate needle shape. Straight (Keith) needles are generally used in accessible places where the needle can be manipulated directly with the fingers (e.g., placement of purse-string sutures in the anus). Curved needles are manipulated with needle holders. The depth and diameter of a wound are important when selecting the most appropriate curved needle. One-fourth circle needles are primarily used in ophthalmic procedures. Three-eighths and one-half circle needles are the most commonly used surgical needles in veterinary medicine. Three-eighths circle needles are more easily manipulated than one-half circle needles because they require less pronation and supination of the wrist. However, because of the larger arc of manipulation required, they are awkward to use in deep or inaccessible locations. A one-half circle or five-eighths circle needle, despite requiring more pronation and supination of the wrist, is easier to use in confined locations.

The needle point (i.e., cutting, taper, reverse cutting, or side cutting) (see Fig. 8.3D) affects the sharpness of a needle and the type of tissue in which the needle can be used. Cutting needles generally have two or three opposing cutting edges and are designed for use in tissues that are difficult to penetrate, such as skin. With conventional *cutting needles*, the third cutting edge is on the inside (i.e., concave) curvature of the needle. The location of the inside cutting edge may promote more "cut out" of tissue because it cuts toward the edges of the wound or incision. Reverse cutting needles have a third cutting edge on the outer (i.e., convex) curvature of the needle; this makes them stronger than similarly sized conventional cutting needles and reduces the amount of tissue cut out. Side cutting needles (i.e., spatula needles) are flat on the top and bottom and are generally used for ophthalmic procedures. Taper needles (i.e., round needles) have a sharp tip that pierces and spreads tissues without cutting them. They are generally used in easily penetrated tissues, such as the intestine, subcutaneous tissue, or fascia. Tapercut needles, which are a combination of a reverse cutting edge tip and a taperpoint body, are generally used for suturing dense, tough fibrous tissue, such as a tendon, and for some cardiovascular procedures, such as vascular grafts. Bluntpoint needles have a rounded, blunt point that can dissect through friable tissue without cutting. They are occasionally used for suturing soft, parenchymal organs, such as the liver or kidney.

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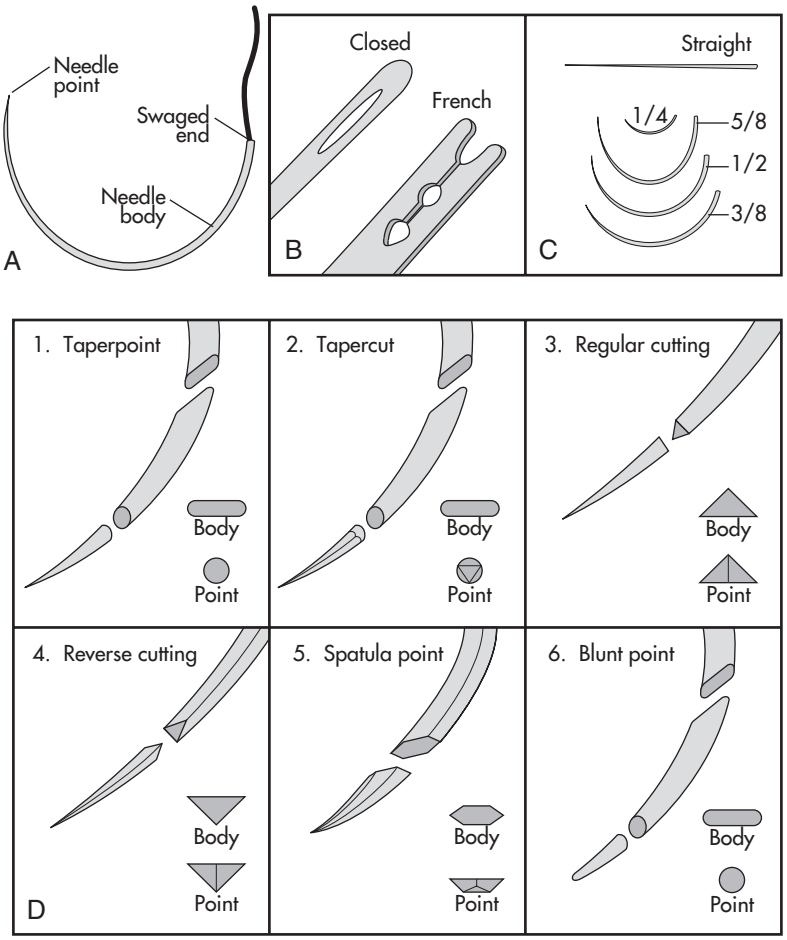


FIG. 8.3 (A) Basic components of a needle. (B) Types of eyed needles. (C) Shapes and sizes of needle bodies. (D) Detail of needle points.

Suture Selection for Different Tissue Types

Considerations for suture selection include the length of time the suture will be required to help strengthen the wound or tissue, the risk of infection, the effect of the suture material on wound healing, and the dimension and strength of the suture required.

Skin

Monofilament sutures should be used in skin to prevent wicking or capillary transport of bacteria to deeper tissue. Synthetic monofilament nonabsorbable sutures (e.g., nylon, polypropylene) generally have good relative knot security and are relatively noncapillary. Polymerized caprolactam (Supramid, Vetafil) has good handling characteristics, but it is braided and should not be buried in deeper tissue. Absorbable sutures (e.g., polydioxanone, polyconate) may be used in skin, but they should ultimately be removed because absorption requires contact with body fluids. Subcutaneous sutures are used to obliterate dead space and reduce tension on skin edges; multifilament or monofilament absorbable suture material is preferred.

Abdominal Closure

The rectus fascia may be closed with either an interrupted or a continuous suture pattern; however, most surgeons routinely close the rectus fascia with a simple continuous suture pattern. When using an interrupted pattern, numerous suture materials are adequate; however, suture that is rapidly removed (e.g., surgical gut) should be avoided in catabolic (i.e., hypoalbuminemic and malnourished) patients. When a continuous suture pattern is used, a standard absorbable or nonabsorbable monofilament suture with good knot security should be used (e.g., polypropylene, polybutester, polydioxanone, polyglyconate). One size larger suture than would normally be used is preferred for a continuous suture pattern. The knots should be tied carefully, and three or four square knots (six or eight throws) should be placed. Standard absorbable suture (e.g., polydioxanone or polyglyconate) may be preferable to prevent large amounts of foreign material from remaining permanently in the incision.

Muscle and Tendon

Muscle has poor holding power and is difficult to suture. Absorbable or nonabsorbable suture material may be used. Sutures placed parallel to the muscle fibers are likely to pull out, so consideration should be given to the type of suture pattern chosen (see p. 68). Suture material used for tendon repair should be strong, nonabsorbable, and minimally reactive. Suturing with a taper or taper-cut needle is generally less traumatic to these tissues. The largest suture that will pass without trauma through the tendon should be used.

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Parenchymal Organs

Parenchymal organs, such as the liver, spleen, and kidneys, are generally sutured with absorbable monofilament sutures, as multifilament sutures tend to cut through this type of tissue because of the increased drag.

Hollow Viscus Organs

Absorbable monofilament sutures are generally recommended in hollow viscus organs, such as the trachea, gastrointestinal tract, or bladder, to prevent tissue retention of foreign material once the wound is healed. In addition, nonabsorbable suture may be calculogenic when placed in the urinary bladder or gallbladder and may be extruded into the lumen when implanted in intestine. Polyglycolic acid suture rapidly dissolves when incubated in sterile urine (6 days) or infected urine (3 days).

Infected or Contaminated Wounds

If possible, sutures should be avoided in highly contaminated or infected wounds because even the least-reactive nonabsorbable sutures elicit some degree of infection in tissue contaminated with either *Escherichia coli* or *Staphylococcus aureus*. Multifilament nonabsorbable sutures (e.g., silk or polyester) should not be used in infected tissue because they potentiate infection and may fistulate. Absorbable suture material is preferred; however, surgical gut should be avoided because its absorption in infected tissue is unpredictable. Synthetic monofilament nylon and polypropylene sutures may elicit less infection in contaminated tissue than metallic sutures.

Vessels and Vascular Anastomoses

Vessels should be ligated with absorbable suture material. Vascular anastomoses are typically performed with monofilament nonabsorbable suture material such as polypropylene. Nonabsorbable suture should also be used for vascular grafts. Arterial anastomoses may be performed in an end-end (Fig. 8.4A) or end-side fashion (see Fig. 8.4B). Arteriotomies may be closed using a vertical (Fig. 8.5A) or transverse (see Fig. 8.5B) method.

Reduction of blood loss from a vascular anastomosis (e.g., when a polytetrafluoroethylene graft is used) can be affected by suture choice, even when a technically perfect anastomosis has been performed, as bleeding may occur from the needle holes. Sutures having a needle-to-suture ratio of 2:1 or 3:1 are associated with more bleeding than when the needle-to-suture diameter ratio is 1:1, which allows the suture to completely fill the graft needle hole.

OTHER BIOMATERIALS

Tissue Adhesives

Cyanoacrylates (e.g., N-butyl and isobutyl-2-cyanoacrylate; Vetbond, LiquiVet, GLUture) are commonly used for tissue

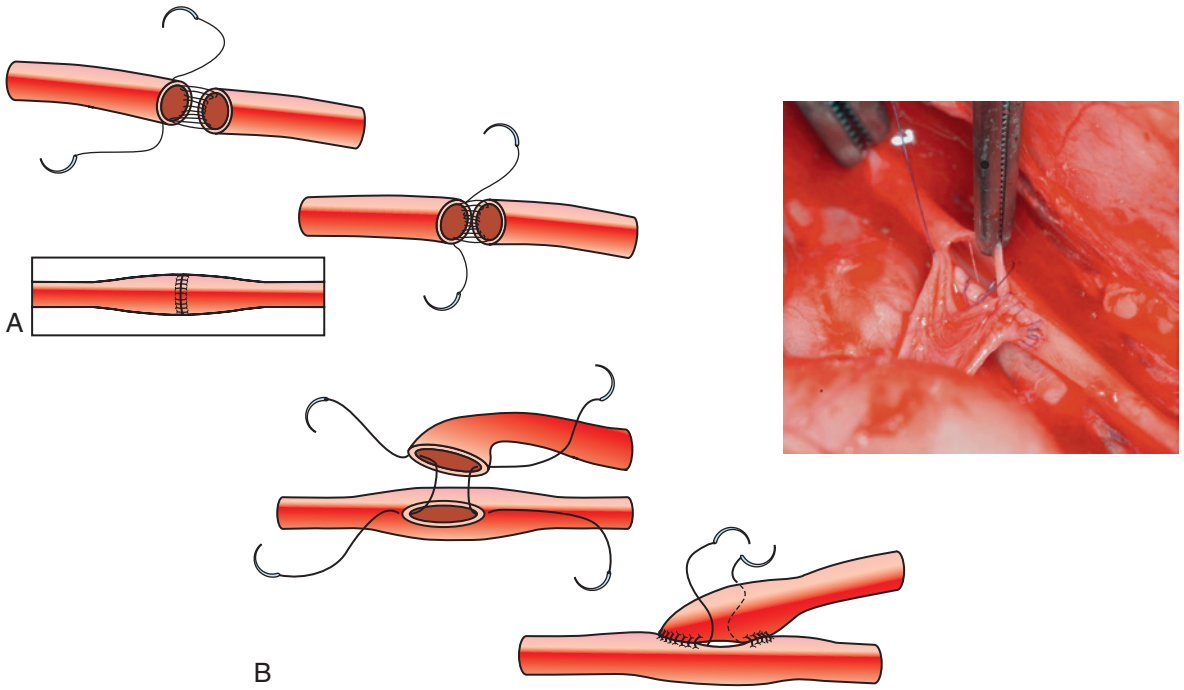


FIG. 8.4 (A) To perform an end-end arterial anastomosis, approximate the vessel ends and place two stay sutures at equidistant points (usually at the corners) between the ends. Use these sutures to hold the vessel steady and rotate the vessel if required while the anastomosis is being performed. Place continuous sutures at 2-mm intervals, 2 mm from the edge of the vessel beginning at the posterior wall (away from the surgeon) and continuing to the anterior wall. If stenosis is a concern, spatulate the ends. (B) For an end-side anastomosis, place sutures initially at the cranial (head) and caudal aspect (toe) of the two ends. Perform the anastomosis circumferentially, beginning with the posterior wall first and progressing to the anterior wall. *Inset:* End-side anastomosis of external jugular vein to carotid artery in a pig to create an experimental aneurysm.

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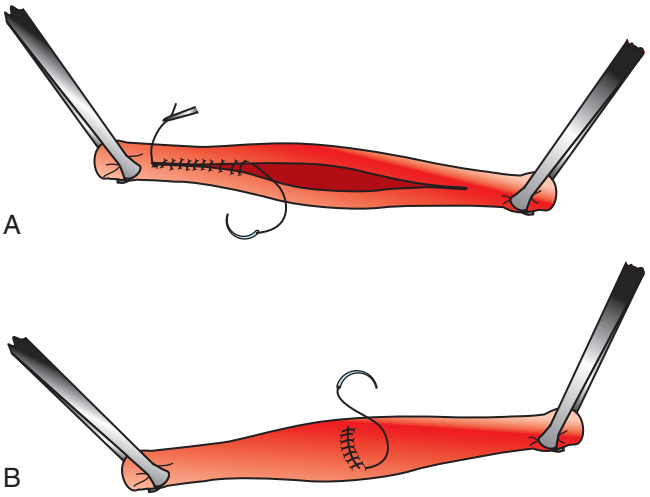


FIG. 8.5 An arteriotomy may be closed (A) vertically or (B) transversely.

adhesion in certain procedures, such as declawing, tail docking, and small (<5 cm) skin lacerations. These adhesives rapidly polymerize in the presence of moisture and produce a strong, flexible bond. Adhesion of the contact tissue generally takes less than a minute, but may be prolonged with excessive hemorrhage. Persistence of the glue in the dermis may result in granuloma formation or dehiscence, and placement in an infected site may be associated with fistulation. Heat generated during the procedure may cause tissue burns. Cyanoacrylate adhesives also cause an intense inflammatory reaction within subcutaneous tissue and should never be applied within deep wounds.

Dermabond (2-octylcyanoacrylate) is an adhesive to which special plasticizers have been added to provide flexibility. It reaches maximum bonding strength within 2.5 minutes and is equivalent in strength to healed tissue at 7 days after repair. It is marketed for humans as a replacement for sutures that are 4-0 to 5-0 or smaller in diameter for incisional or laceration repair.

Adhesives should not be used on bite wounds, severely contaminated wounds, ulcers, puncture wounds, mucous membranes, near the eye, or in areas of high moisture content. Adhesives are most useful when used on wounds that may close spontaneously, have clean or sharp edges, and are located on clean, nonmobile areas. Wounds in which the edges are separated more than 5 mm by the underlying skin tension are unlikely to stay closed with tissue adhesives alone and should be supported with subcutaneous sutures. Reliable closure of lacerations longer than 5 cm is also uncertain when tissue adhesives alone are used.

To apply tissue adhesives, clean the wound and control bleeding. Hold the wound together manually or with tissue forceps, with the edges slightly everted. Apply the adhesive by lightly wiping the applicator tip over the area in the direction of the long axis of the wound, at least 5 mm beyond the skin edges. Apply three to four thin layers successively; avoid a droplet or a single thick layer. Hold the wound edges together for 30 to 60 seconds after the last adhesive application to ensure that the adhesive has time to set fully. Remove adhesive applied to unwanted areas by using petroleum jelly or acetone. Do not cover with ointment, bandage, or a dressing. After 24 hours, gently wash the area with plain water, but do not scrub, soak, or expose it to moisture for any length of time. The adhesive will spontaneously slough off in 5 to 10 days.

Fibrin glue (e.g., Tisseel) is a relatively new generation of tissue adhesive. The product is composed of two separate solutions of concentrated human fibrinogen and bovine thrombin that, when combined, replicate the final stage of the coagulation cascade. A fibrin clot is generated in 10 to 60 seconds. Autologous fibrin glue (e.g., Vivostat) is made from the patient's blood before surgery, combining a biologic adhesive made of fibrinogen, factor XIII, fibronectin, thrombin, aprotinin, and calcium chloride. In humans, uses of the glue include fixation of skin grafts (i.e., without sutures) and stabilization of gastrointestinal and nerve anastomoses. It has also been used as a preclot material on vascular grafts and as a seal for sutured vascular anastomoses. However, there is limited experience with the use of fibrin glue in small animals.

Bioglue is a relatively new two-component surgical adhesive comprised of purified bovine serum albumin and glutaraldehyde. The two components are dispensed from a double-chamber syringe at the time of application. The amine proteins within the bovine albumin are bridged to the extracellular protein matrix within the tissue by the glutaraldehyde, creating a strong covalent bond. Complete polymerization takes 2 to 3 minutes. Bioglue is most commonly utilized in cardiovascular procedures because the glue also adheres to synthetic graft materials.

Ligating Clips

Clips (e.g., hemoclips or ligaclips) may be used for vessel ligation and are particularly useful when the vessel is difficult to reach or when multiple small vessels must be ligated. However, ligating clips are not recommended for use on vessels larger than 11 mm in diameter. The vessel should be dissected free of surrounding tissue before the clip is applied, and 2 to 3 mm of vessel should extend beyond the clip to prevent slippage. The vessel should be one-third to two-thirds the size of the clip.

Skin Staples

Rapid and precise wound closure can be achieved through the use of skin staplers. Numerous models are available (e.g., Precise Vista, Proximate Plus, Appose ULC), but most consist of a disposable hand-held unit that dispenses a single rectangular staple. Although there are slight variations between manufacturers, a standard skin staple is made of 316 L stainless steel and has a diameter of 0.5 mm, a span of 6 to 10 mm, and a leg length of 3.5 mm. Several staplers come with wide staples that span from 7 to 13 mm and have a leg length of 4.0 mm. Most staples penetrate the skin first and are then formed to the final configuration. This allows for good skin closure and a degree of tissue eversion that is favorable for wound healing. Other beneficial characteristics of the skin stapler include an angled head to allow visualization of staple deployment, a rotating head to facilitate precise staple placement, and an ergonomic, lightweight design to minimize hand and arm fatigue. A special staple remover facilitates clip removal after healing.

When placing staples, hold the wound edges together with tissue forceps. Place the stapling device gently (not firmly or with pressure) against the skin surface and slowly squeeze the trigger.

Absorbable skin staples (e.g., Insorb) have been developed to provide a rapid and cosmetic skin closure and eliminate the

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need for staple removal. These staples are placed in the subcutaneous tissue to securely approximate wound edges. Absorbable subcuticular staples are made of synthetic polymers composed of polyglycolic and polylactic acids and are gradually absorbed by the body over a period of months.

Synthetic Surgical Mesh

Surgical mesh may be used to repair hernias (e.g., perineal hernias) or reinforce traumatized or devitalized tissue (abdominal hernias). It is also used to reconstruct large tissue defects following resection of devitalized, traumatized, or neoplastic tissue (see p. 913). Surgical mesh is available in nonabsorbable forms (e.g., polyester [Mersilene] fiber mesh and polypropylene [Prolene] mesh) or absorbable forms (e.g., polyglactin 910 [Vicryl] and polyglycolic acid [Dexon]) and in woven and knitted forms. Surgical mesh has been developed that has a thin, bioresorbable fabric layer (Proceed Mesh) that effectively separates the strong, supportive mesh from underlying tissue; it is being advocated for use in the abdominal cavity.

Although surgical mesh is generally elastic, it does not stretch significantly as the patient grows and thus should be used cautiously in immature patients. Fibrous tissue grows through the mesh interstices. Nonabsorbable mesh placed in contaminated wounds may extrude or fistulate and should be removed when the tissue has healed and the mesh is no longer required for support. Polypropylene mesh has been shown to facilitate the reconstruction of large tissue defects in small animals without serious complications.

Biologic Tissue Grafts

Animal tissue biomaterials are becoming more commonly considered as soft tissue replacements for hernia repair or reconstruction of tissue defects. Autologous free fascia lata grafts have been described for various orthopedic procedures and isolated clinical case reports for soft tissue reconstruction, as well as described experimentally for repair of diaphragmatic hernias, urethral defects, cranial cruciate ligament injury, and Achilles tendon rupture. If there is concern regarding donor site morbidity, canine allogenic fascia lata grafts are available.

Porcine small intestinal submucosa (Vet BioSISt) can be used as a surgical patch for soft tissue reconstruction or reinforcement. It is an absorbable, biocompatible material that acts as scaffold for tissue ingrowth. It is best used for enhancement of wound healing but should be used with caution in areas of tension, as degree of mechanical support is limited. In those situations, a multilaminate four-layer product is available that may provide adequate strength in low-strain areas.

COMMON SUTURE TECHNIQUES

Suture Patterns

Suture patterns can be classified as interrupted or continuous by the way they appose tissue (e.g., appositional, everting, or inverting) or by which tissues they primarily appose (e.g., subcutaneous or subcuticular). Appositional sutures (e.g., simple interrupted sutures) bring the tissue in close approximation; everting sutures (e.g., continuous mattress sutures) turn the tissue edges outward, away from the patient and toward the surgeon. Inverting sutures (e.g., Lembert, Connell, and Cushing sutures) turn tissue away from the surgeon, or toward the lumen of a hollow viscus organ.

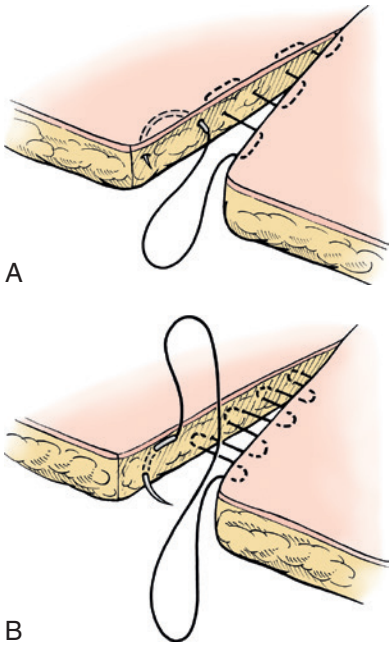


FIG. 8.6 Suture patterns. (A) Subcutaneous. (B) Subcuticular.

Subcutaneous and Subcuticular Patterns (Video 8.1)

Subcutaneous sutures are placed to eliminate dead space and provide some apposition of skin so that less tension is placed on skin sutures (Fig. 8.6A). Subcutaneous sutures are generally placed in a simple continuous manner; however, in some instances, such as when drainage might be necessary, simple interrupted sutures are preferable. Subcuticular or intradermal closures may be used in place of skin sutures to reduce scarring or eliminate the need for suture removal (e.g., fractious patient). The suture line is begun by burying the knot in the dermis (see p. 74). The suture is advanced in the dermal tissue, but, in contrast to a continuous subcutaneous line, the bites are parallel to the long axis of the incision (see Fig. 8.6B). The suture line is completed with a buried knot. Absorbable suture materials with a cutting needle are preferred for this suture pattern.

Interrupted Suture Patterns

Simple Interrupted Pattern

A simple interrupted suture is made by inserting the needle through tissue on one side of an incision or wound, passing it to the opposite side, and tying (Fig. 8.7A). The knot is offset so that it does not rest on top of the incision, and the ends of the suture are cut (for skin sutures, the ends are left long enough to allow them to be grasped during removal). The sutures should be placed approximately 2 to 3 mm away from the skin edge. Right-handed surgeons place sutures from right to left in a horizontal fashion; left-handed surgeons perform the opposite.

Simple interrupted sutures are easy and quick to place. They are appositional unless excessive tension is applied, then inversion may occur. Inversion of skin results in poor healing; therefore care should be taken to ensure that skin sutures are loose and the edges are apposed. The primary advantage of simple interrupted sutures is that disruption of a single suture does not cause the entire suture line to fail. However, simple interrupted

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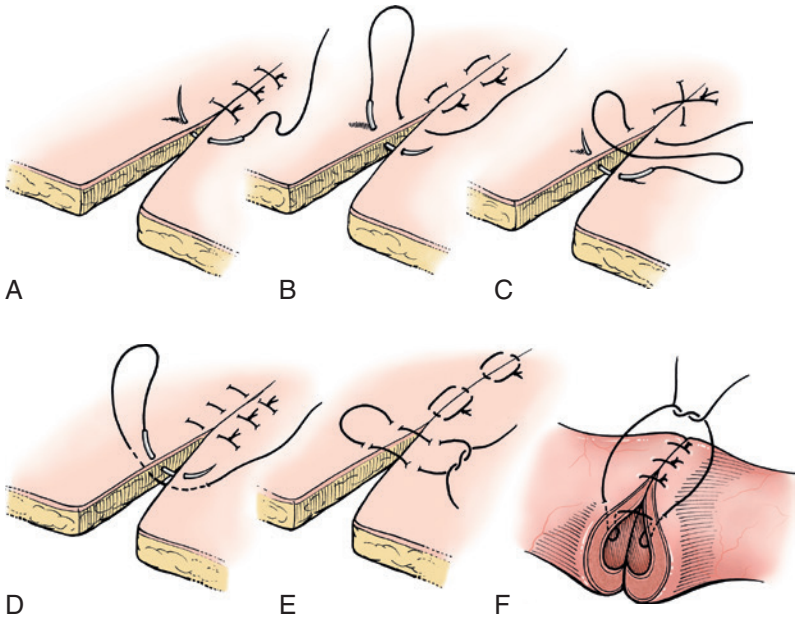


FIG. 8.7 Interrupted suture patterns. (A) Simple interrupted. (B) Horizontal mattress. (C) Cruciate. (D) Vertical mattress. (E) Halsted. (F) Gambee.

sutures take more time than continuous patterns and result in more foreign material (knots) in the wound.

Cruciate Pattern

When two simple interrupted sutures are placed parallel to each other and then tied across the incision to create an "X," a cruciate pattern is formed (see Fig. 8.7C). Cruciate sutures are appositional and can relieve low to moderate tension across an incision. Less suture material is used to close a skin incision with cruciate sutures than simple interrupted, yet cruciate sutures still afford the security of an interrupted pattern.

Horizontal Mattress Pattern

Horizontal mattress sutures are placed by inserting the needle on the far side of the incision, passing it across the incision, and exiting it on the near side, as described for a simple interrupted suture (see Fig. 8.7B). The needle is then advanced 6 to 8 mm along the incision and reintroduced through the skin on the near side. It then crosses the incision, exiting from the skin on the far side, and the knot is tied. Horizontal mattress sutures are generally separated by 4 to 5 mm. They are used primarily in areas of tension and can be placed rapidly; however, they often cause tissue eversion. Care should be exercised to appose, rather than evert, tissue margins, and the suture should be angled through the tissue so that it passes just below the dermis.

Vertical Mattress Pattern

To place a vertical mattress suture, the needle is introduced approximately 8 to 10 mm from the incision edge on one side, passed across the incision line, and exited at an equal distance on the opposite side (see Fig. 8.7D). The needle is reversed and inserted through skin on the same side and exited on the far side, each bite approximately 4 mm from the skin edge, and the knot is tied. Vertical mattress sutures are stronger than horizontal mattress sutures when used in areas of tension, and are preferred when addressing tension in skin closure, as there is less disruption to the blood supply of the wound edges. Placement of vertical mattress sutures is relatively time-consuming, but eversion of the skin margins is less of a problem than with horizontal mattress sutures. Both horizontal and vertical mattress pattern can be bolstered using rubber stents and buttons.

Halstead Pattern

A Halstead suture is another interrupted mattress pattern that is a modification of a continuous Lembert pattern (p. 70). It is an infrequent technique used in veterinary medicine, but does provide exact skin approximation. The needle enters and exits perpendicular to and on the same side of the incision. The needle then crosses over the wound and is passed in and out of the skin in a similar fashion. The needle is advanced down the incision, and this pattern is repeated in reverse, back across the incision, and the knot is tied (see Fig. 8.7E).

Gambee Pattern

A Gambee suture is an interrupted pattern used in intestinal surgery to reduce mucosal eversion. The suture is introduced the same as a simple interrupted suture from the serosa through the muscularis and mucosa to the lumen (Fig. 8.7F). The needle is then returned from the lumen through the mucosa to the muscularis before it crosses the incision. After crossing the incision, it is introduced in the muscularis and is continued through the mucosa to the lumen. The needle is then reintroduced through the mucosa and muscularis to exit from the serosal surface, and the suture is tied. Gambee sutures reduce mucosal inversion and may reduce wicking of material from the intestinal lumen to the exterior.

Continuous Suture Patterns

Simple Continuous Pattern

A simple continuous suture consists of a series of simple interrupted sutures with a knot on either end; the suture is continuous

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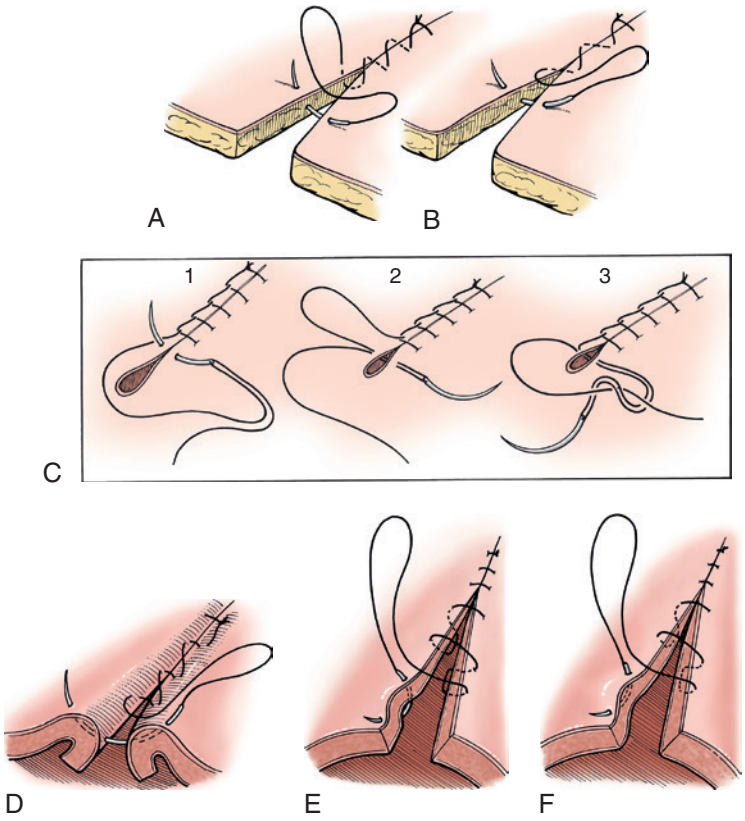


FIG. 8.8 Continuous suture patterns. (A) Simple continuous. (B) Running. (C) Ford interlocking (*C2* and *C3* illustrate how to end the suture line). (D) Lembert. (E) Connell. (F) Cushing.

between the knots (Fig. 8.8A). To begin a simple continuous suture line, a simple interrupted suture is placed and knotted, but only the end that is not attached to the needle is cut. The needle is then passed through the tissue from one side to the other, perpendicular to the incision. The suture is advanced above the incision line at a diagonal. A running suture is created if the suture is advanced above and below the incision line, but this is not as secure as less tissue is purchased (see Fig. 8.8B). To end a continuous suture, the needle end of the suture is tied to the last loop of suture that is exterior to the tissue.

Simple continuous suture lines provide maximum tissue apposition and are relatively air and fluid tight compared with a series of simple interrupted sutures. Simple continuous suture lines are frequently used to close the linea alba and subcutaneous tissue. Care should be taken when placing continuous suture lines in areas where tightening of the suture may result in a purse-stringlike effect, such as with an intestinal anastomosis.

Ford Interlocking Pattern

This pattern is a modification of a simple continuous pattern in which each passage through the tissue is partly locked (see Fig. 8.8C1). Each pass through the tissue is linked to the previous passage as the suture exits the tissue through a created loop of material. To terminate this suture pattern, the needle is reversed and introduced in the opposite direction from that used previously. The loop of the suture formed on the opposite side is tied to the single end (see Fig. 8.8C2 and C3). Locked suture patterns may be placed quickly and may appose tissue better than a simple interrupted pattern. This pattern also provides greater stability than a simple continuous pattern in the event of a partial break along the line. However, a larger amount of suture material is used, and sutures may be more difficult to remove.

Lembert Pattern

A Lembert pattern is a variation of a vertical mattress pattern applied in a continuous fashion. It is an inverting pattern that is often used to close hollow viscera. The needle penetrates the serosa and muscularis approximately 8 to 10 mm from the incision edge and exits near the wound margin on the same side. After passing over the incision, the needle penetrates approximately 3 to 4 mm from the wound margin and exits 8 to 10 mm away from the incision. This pattern is repeated along the length of the incision (see Fig. 8.8D).

Connell and Cushing Patterns

These are inverting patterns that are used to close hollow organs. A watertight seal is created by the inversion; however, appositional patterns are advocated for visceral closure in small animals, as this facilitates rapid intestinal healing. The Connell and Cushing patterns are similar, except that a Connell pattern enters the lumen, whereas a Cushing pattern extends only to the submucosal layer (see Fig. 8.8E and F). It was previously thought that a Cushing pattern would be preferable to a Connell for cystotomy closure because suture material in the lumen might be calculogenic; however, the use of rapidly absorbed monofilament sutures negate this concern.

The suture line is begun with a simple interrupted or vertical mattress suture. The needle is advanced parallel to the incision and introduced into the serosa, passing through the muscular and submucosal surfaces. From the deep surface (the lumen with a Connell suture), the needle is advanced parallel along the incision and returned through the tissue to the serosal surface.

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Once outside the viscera, the needle and suture are passed across the incision and introduced at a point that corresponds to the exit point on the contralateral side. The suture is then repeated. The suture should cross the incision perpendicularly. When the suture is tightened, the incision inverts.

A Parker-Kerr suture is a modification of the Cushing and Lembert patterns that has been described for closing the stump of hollow viscera. It is seldom used because it causes excessive tissue inversion.

Tendon Sutures

Specific suture configurations are used to approximate severed ends of a tendon or to secure one end of a tendon to bone or muscle; these include Kessler locking-loop, Bunnel-Mayer, Krackow, and three-loop pulley suture patterns, among others. Select pattern descriptions are listed and more information can be found on p. 1283.

Three-Loop Pulley Suture

The three-loop pulley pattern is made with three loops oriented approximately 120 degrees to each other. The initial loop is placed perpendicular to the long axis of the tendon ends in a near-far fashion (Fig. 8.9). The second loop is placed in a plane 120 degrees from the first, at a point midway between the near and far positions. The final loop is placed in a far-near pattern 120 degrees from the first two sutures. This pattern is considered to be biomechanically superior to other tendon patterns, as it is more resistant to gap formation during tensile loading.

Bunnell Suture

A modified Bunnell suture pattern may be used to appose severed tendons. The needle is passed from one side of the proximal end of the severed tendon and crossed diagonally across the tendon to the opposite side, where it exits (see Fig. 8.9). The suture is reintroduced approximately 1 mm distal to the exit site and crossed diagonally to the other side of the tendon, where it exits from the severed end. It is introduced into the distal portion of the severed tendon from the cut end, and two cruciate sutures are placed. The suture exits at the severed end of the distal portion of the tendon and is reintroduced into the proximal tendon. The pattern is repeated in this portion of the tendon, with the suture exiting near the original entrance site. The tendon ends are apposed and the suture tightened.

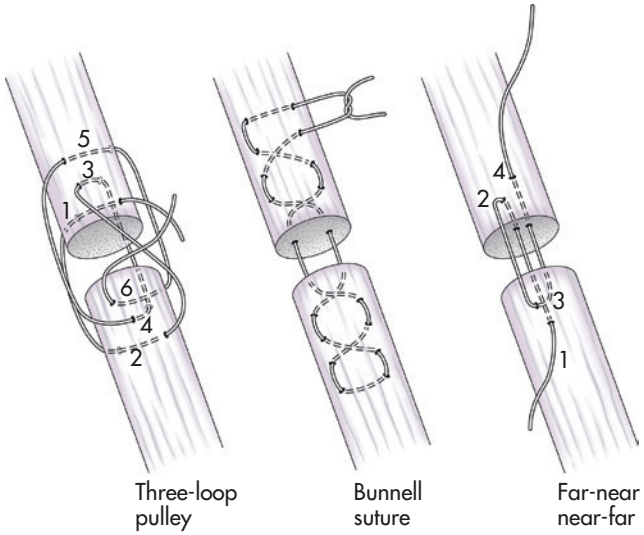


FIG. 8.9 Tendon sutures.

This pattern is not commonly used because it is difficult to place and may damage the tendon's microcirculation. Ischemia resulting from the suture may cause the suture to pull out or may result in death of the tendon ends. The resultant gap must then be filled with fibrous tissue.

Far-Near-Near-Far Suture

A far-near-near-far suture may be used in flat tendons. The needle is passed through the tendon perpendicular to and 5 mm from the severed tendon end (see Fig. 8.9). The needle then enters the distal section of the severed tendon in the same vertical plane 2 mm from the tendon edge. It is looped back to the proximal section of tendon, where it enters 2 mm from the severed edge. The suture is again looped back to the distal section of the tendon to enter 5 mm from the severed tendon edge. The suture ends are pulled taut and tied with a surgeon's knot. This pattern minimally disrupts blood flow and provides good resistance to tension because all suture passes are in the same vertical plane.

Knot Tying

The knot is the weakest point of a suture. A knot consists of at least two throws laid on top of each other and tightened. The throws can be joined parallel, as in a square knot, or crosswise, as in a granny knot (Fig. 8.10). Correct knot-tying technique is important because incorrectly tied knots (e.g., tumbled knots, half-hitches, or granny knots) may lead to dehiscence. Factors that influence knot security are the type of material, the length of the cut ends, the structural configuration of the knot, the number of throws used in knot construction, and surgeon experience.[10]

The most reliable configuration for a knot is superimposition of squared knots. A surgeon's (friction) knot involves passage of suture material twice on the first throw (see Fig. 8.10). Due to the extra suture material, this knot cannot be easily tightened and can withstand only a slight strain on the suture loop. Although it is often used in areas of tension, it is generally not recommended for use with coated or monofilament materials, and should be avoided unless tissue tension is such that use of the standard square knot would result in poor tissue apposition. A surgeon's knot is not recommended for vessel ligation. A surgeon's throw

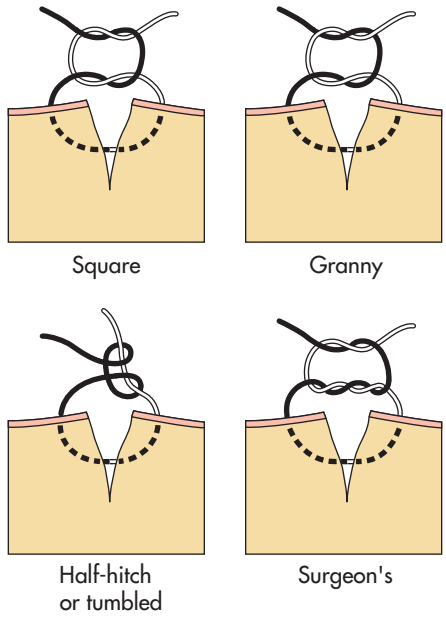


FIG. 8.10 Types of knots.

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was recently shown to be less reliable in a vascular pedicle ligation model when compared to five other friction knots.[11] The Miller's knot, constrictor knot, and strangle knot were considered to be the most reliable when used as a first throw for vessel ligation. All three of these configurations have an overriding turn, which is a section of suture that passes on top of another section of suture, often perpendicular or at an angle to the underlying section (Fig. 8.11). Tension is maintained by providing compression on the underlying turns of suture.

Multifilament sutures generally have better knot-holding properties than monofilament materials; however, coating the suture to decrease tissue drag reduces knot security. To prevent strangulating tissue, excessive tension should be avoided when tying knots (except when ligatures are applied for hemostasis). Excessively tight skin sutures cause the patient discomfort and increase the likelihood that the animal will remove the sutures prematurely.

Instrument Ties

In veterinary medicine, instrument ties (Fig. 8.12) are more commonly used than hand ties because it is thought there is less waste of the suture. The first loop is made as shown in Fig. 8.12, after which the suture should not be lifted or have uneven pressure applied to either end, or the throw will loosen. If one end is pulled with greater tension than the other, a half-hitch will form (see Fig. 8.10). Opposing suture ends should be pulled perpendicular to the long axis of the incision. Lifting one hand causes the suture to tumble, forming a sliding two half-hitch knot. Failure to correctly cross the hands results in a granny knot.

Hand Ties

Hand ties are particularly useful in confined or hard to reach areas or when sutures have been preplaced, as in a thoracotomy closure. Hand ties generally require that suture ends be left longer than for an instrument tie, but can be placed much faster and more securely than instrument ties. A one-handed or two-handed technique may be used. The two-handed technique generally allows better control and accuracy; however, the one-handed technique is more useful in confined areas. Techniques for tying one-handed and two-handed knots are shown in Figs. 8.13 and 8.14.



FIG. 8.11 Strangle knot.

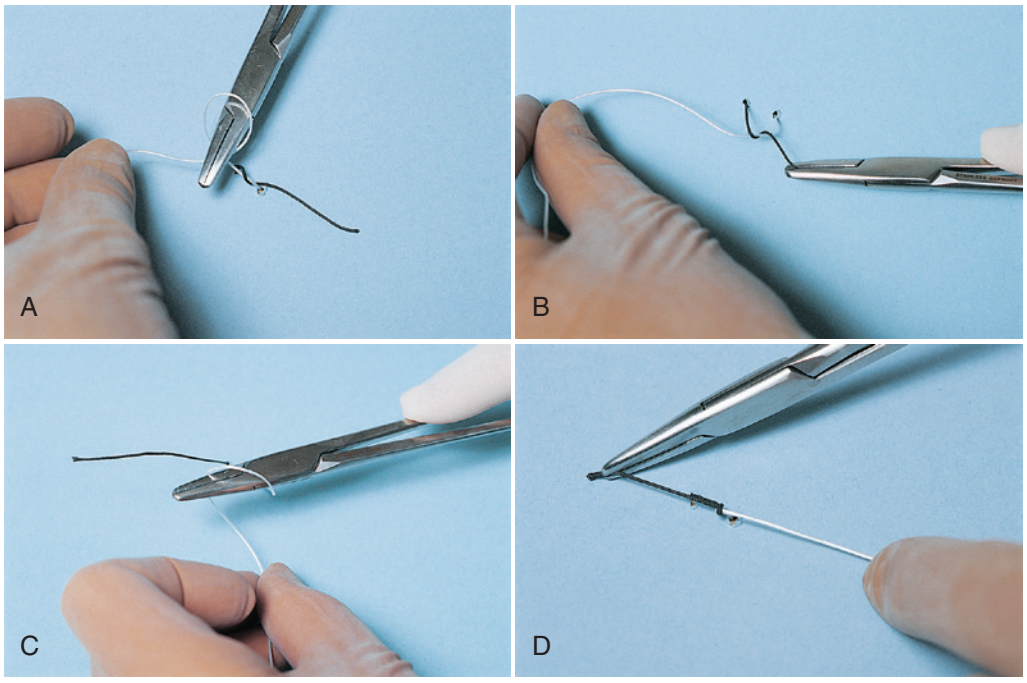


FIG. 8.12 Instrument tie. (A) Place the tips of the needle holders between the two strands of suture. Wrap the strand nearest you (*white*, or long end) around the needle holders to form a loop and grasp the end of the far piece of suture (*black,* or short end) in your needle holders. (B) Bring the short end toward you (through the loop) by reversing your hands, and tighten the suture gently. (C) For the second throw, wrap the strand farthest from you (*white,* or long end) over the needle holders to form a loop, grasp the end of the suture nearest you (*black,* or short end), and (D) pull it through the loop, snugly laying the knot down to prevent tightening the suture excessively. Keep your hands low and parallel when tightening the suture to prevent causing the knot to tumble.

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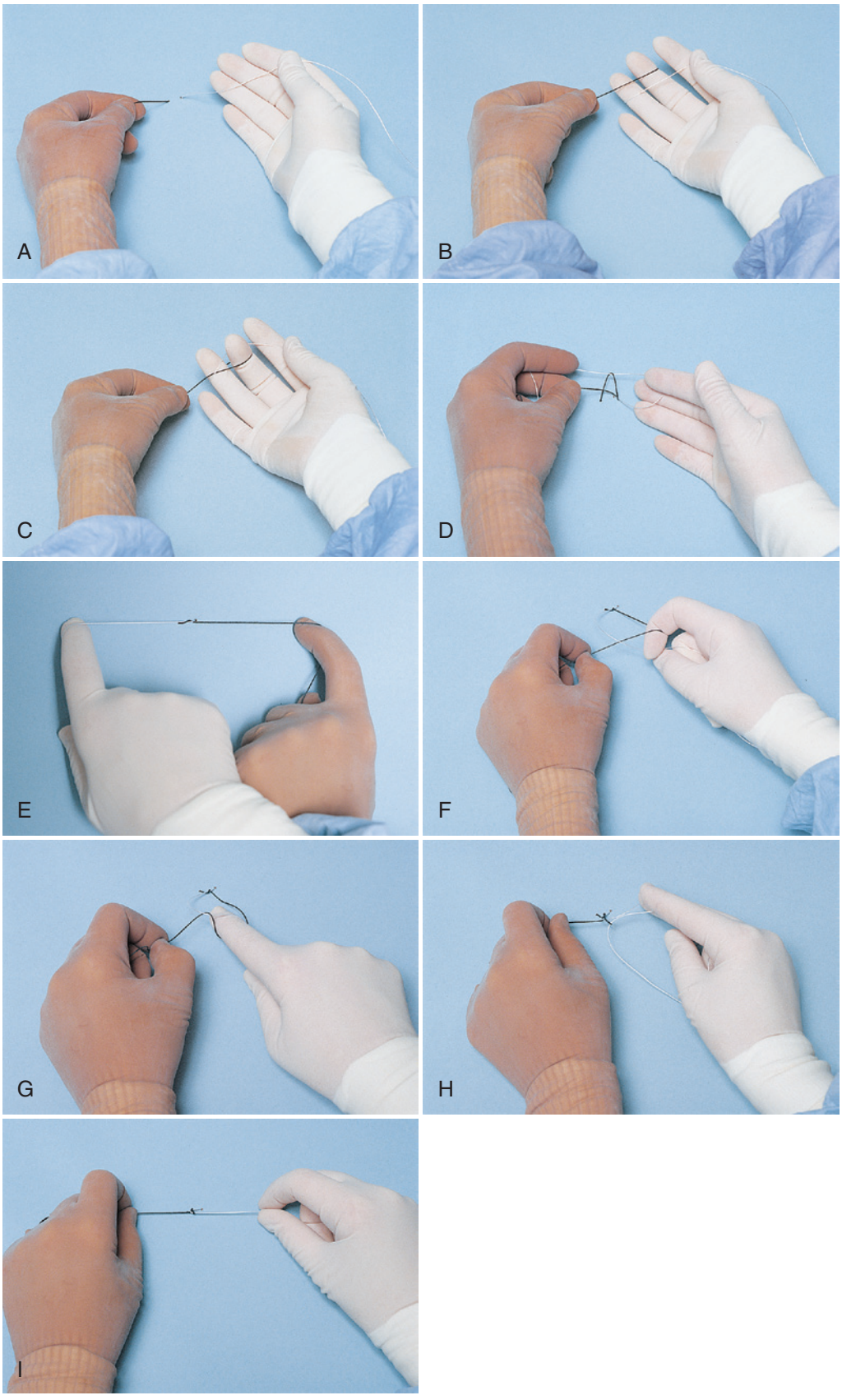


FIG. 8.13 One-handed square knot (right-handed). (A) Reflect the right suture *(white)* between the three fingers of your right hand *(white glove)* and hold it between your index finger and thumb. (B) Hold the left suture *(black)* in your left hand *(dark glove)* and pass it between the index finger and second finger of your right hand. (C) Flex the distal phalanx of the second finger of your right hand and draw the left strand to the right of the right strand. Extend the tip of the second finger so that the white strand is drawn with it through the loop. (D) Pull the right strand through the loop by the tips of the second and third fingers of your right hand. (E) Cross your hands and apply even tension to the two strands. (F) Place the index finger of your right hand between the right *(black)* and left *(white)* strands so that the left-hand strand forms a loop with the right. Flex the distal phalanx of your right index finger. One-handed square knot (right-handed). (G) Extend the distal phalanx of your right index finger to draw the right-hand strand through the loop. (H) Pull the right strand through the loop and (I) apply even tension to complete the square knot. (Modified from Knecht CD, Allen A, Williams DJ, et al. *Fundamental Techniques in Veterinary Surgery.* 3rd ed. Philadelphia: WB Saunders; 1981.)

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Burying the Knot (Video 8.2)

The knots of subcutaneous and subcuticular suture patterns are often buried to reduce irritation caused by the knots rubbing against more superficial tissue. Fig. 8.15 presents a description of this procedure using buried square knots. Alternatively, a self-locking knot (e.g., Aberdeen knot) can be used at the beginning and end of continuous closure of subcutaneous and intradermal suture lines. Aberdeen knots are as secure as square knots but have less suture volume.[12]

Suture Removal

Skin sutures should generally be removed once healing is sufficient to prevent dehiscence, usually within 10 to 14 days after surgery. However, prolonged healing, such as in extremely debilitated animals, may require that the sutures be left in place longer. In addition, if fibrosis is desired (e.g., aural hematoma repair), delaying suture removal may be considered.

HEMOSTATIC TECHNIQUES AND MATERIALS

Hemostasis is a complex process that involves platelet activation and circulating clotting factors. Numerous diseases or conditions may interfere with clotting in surgical patients. The reader is referred to other veterinary medicine texts for an in-depth discussion of normal clotting and alterations of clotting caused by disease. In surgery, obtaining hemostasis allows appropriate visualization of tissue during the procedure and prevents life-threatening hemorrhage. Low pressure hemorrhage from small vessels can be controlled by applying pressure to the bleeding points with gauze sponges. Once a thrombus has formed, the sponge should be gently removed to prevent disrupting clots. Soaking the sponge with saline before removal may also help prevent clot disruption. Small hemostatic forceps (e.g., mosquito forceps) may also be used to control bleeding from small vessels. The vessel is grasped and clamped with the forceps for several minutes until coagulation occurs.

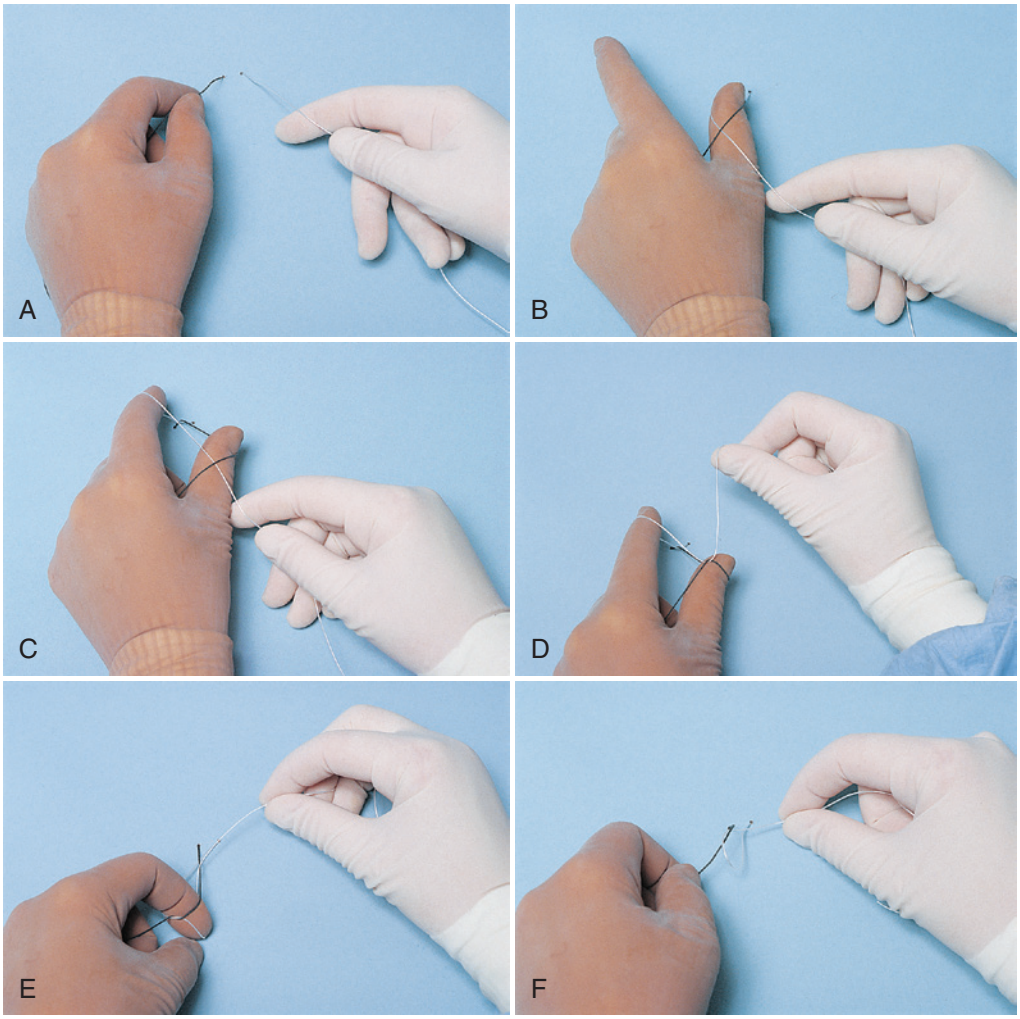


FIG. 8.14 Two-handed square knot (right-handed). (A) Extend the index finger of your right hand *(white glove)* as a bridge and place the right *(white)* strand over it. Hold the left *(black)* strand in the palm of your left hand *(dark glove)*. (B) Pass your left thumb below and around the right strand and then to the left of the left strand. (C) Introduce your left index finger between the crossed strands (with your left thumb). (D) Carry the right strand to your left index finger and thumb and, (E) using your left index finger and thumb, carry it through the loop. (F) Return the suture to your right hand. Two-handed square knot (right-handed).

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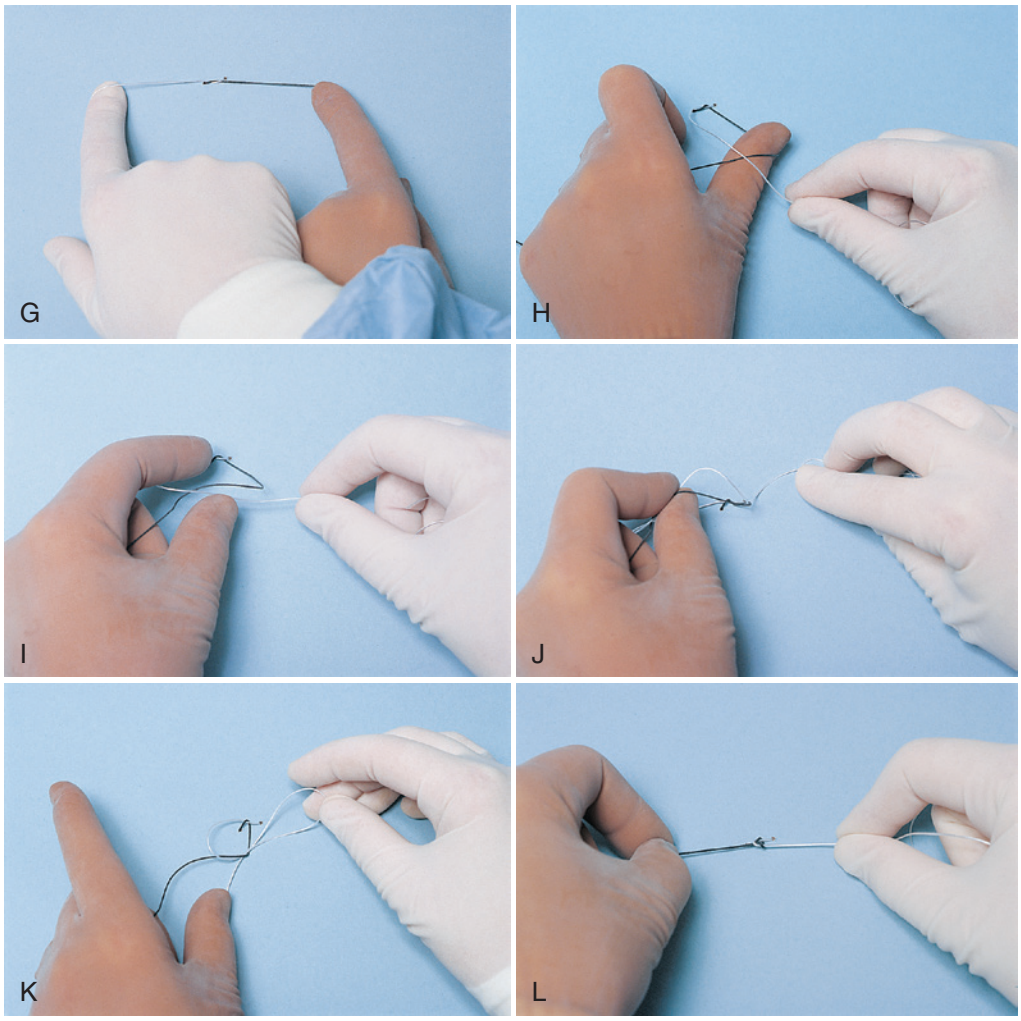


FIG. 8.14, cont'd (G) Cross your hands and apply even tension to the suture ends. (H) Place your left thumb between the two strands and make a loop with your right hand. (I) Place your left index finger through the loop and use it and your left thumb to grasp the left *(white)* strand and (J) pull or push it through the loop. (K) Pass the left strand from your left hand to your right thumb and index finger after passing it through the loop and (L) apply even tension to the suture strands to tighten the square knot. (Modified from Knecht CD, Allen A, Williams DJ, et al. *Fundamental Techniques in Veterinary Surgery*. 2nd ed. Philadelphia: WB Saunders; 1981.)

Alternatively, a ligature may be placed to ensure the bleeding is stopped.

Larger vessels must be ligated. Double ligatures are recommended for larger vessels, particularly arteries. Transfixation ligatures (Fig. 8.16) may be indicated for larger arteries to prevent the ligature from slipping off the vessel end. Using the smallest suture possible for vessel ligation improves knot security. A surgeon's throw should not be used for vessel ligation (see p. 72), although other friction knots would be appropriate (e.g., Miller's knot, constrictor knot, strangle knot).

Topical Hemostatic Agents

Several topical substances are available to control hemorrhage during surgery. Bone wax is a sterile mixture of beeswax, paraffin, and isopropyl palmitate. It is pressed into bleeding channels of bone to control hemorrhage through a tamponade effect. Bone wax should be used sparingly and with caution as it is nonabsorbable and may interfere with bacterial clearance, act as a foreign body, and become a nidus for infection.

Gelfoam is an absorbable gelatin sponge that can be used to control hemorrhage. This material provides a physical matrix that initiates clotting through contact activation. In addition, when applied to an area of hemorrhage, Gelfoam swells and exerts pressure on the wound as it absorbs many times its weight in blood. Absorption occurs in 4 to 6 weeks but should not be left in infected sites and areas with a high risk of infection or in confined places as it may exert unnecessary pressure on neighboring vital structures.

Other gelatin-based hemostatic products include SurgiFlow Hemostatic Matrix and Vetspon. Gelatin products should not be used in closure of skin incisions as they may interfere with healing.

Surgicel is made of oxidized regenerated cellulose. When saturated with blood, it becomes a gelatinous mass that provides

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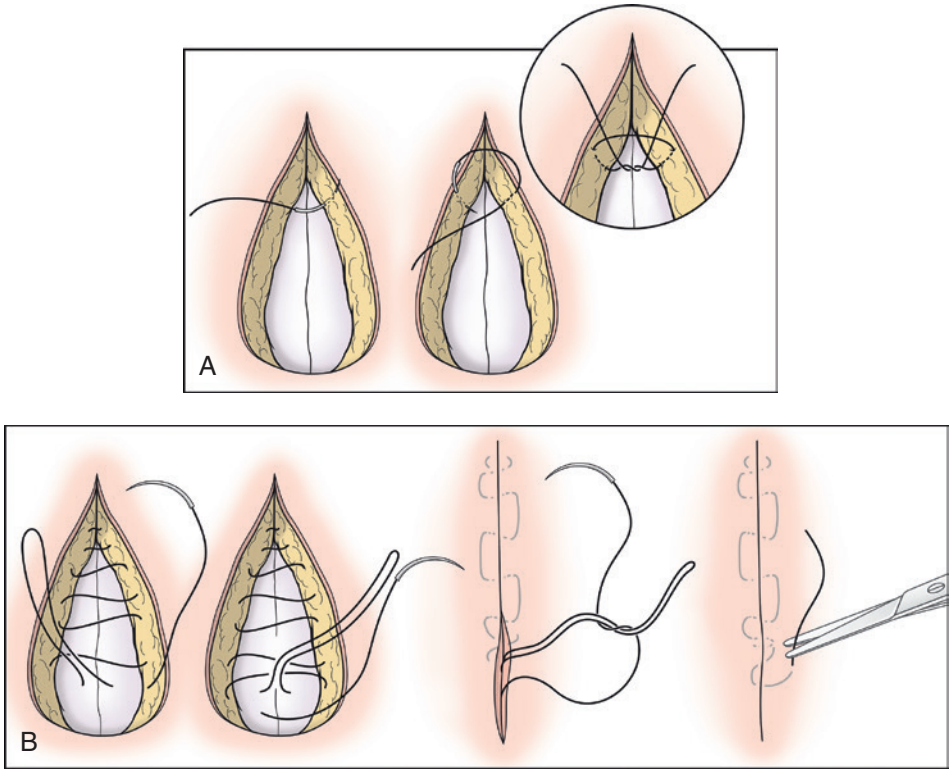


FIG. 8.15 (A) To bury a simple interrupted suture, introduce the needle deep in the far subcutaneous tissue and pass it toward the dermis. Then pass it across the incision line and reintroduce it in the near subcutaneous tissue at the dermis, exiting deep in the incision line. (B) To bury a knot at the end of a continuous suture line, lift a loop of suture from the incision line, introduce the needle from deep to superficial on one side, pass it across the incision, and insert it from superficial to deep in the tissue near the loop. Alternately, after the continuous pattern is completed, advance the needle 2 to 3 mm to the opposite side. Place a vertical bite from the mid-dermis down to subcutaneous tissue. Then insert the needle on the opposite side, vertically aiming up from the subcutaneous tissue, exiting at the mid-dermis within 2 to 3 mm of the commissure. Create a 2-cm loop of suture between the two vertical bites. Take a third vertical bite parallel to the first, initiating in the mid-dermis, but exiting deeper in the subcutaneous layer. Bring the needle up between the exposed loop and final suture crossing the incision. Apply tension to the exposed loop to tighten the horizontal sutures and appose the wound margins, then tie the free suture end to the exposed loop with four to five throws to complete the knot and close the wound. Trim the loop 2 to 3 mm above the knot. Insert the needle close to the knot, aiming to exit the dermis at least 1 cm lateral to the incision. As tension is applied to the suture, the knot is pulled deeper into the tissue, below the dermis. Finally, under tension, trim the free end of suture flush with the skin.

a substrate for clot formation. It can be cut to the desired size and placed on an area of hemorrhage. Surgicel is absorbed by the body, but removal is recommended because it may inhibit callus formation and promote infection. It is not activated by tissue fluids other than blood and therefore should be used only at sites of hemorrhage.

Both gelatin- and cellulose-based materials can be used in conjunction with thrombin to convert fibrin from fibrinogen and promote platelet aggregation (e.g., Floseal and GelFoam Plus).

Avitene is microfibrillar collagen derived from bovine skin that comes in a light flour form or nonwoven web form. It binds tightly to blood surfaces causing contact activation, but also directly activates platelets. It is absorbed from the body in two to three months.

Energy-Based Hemostasis–Electrosurgery

The language regarding the use of electrical current for hemostasis is confusing. The terms *electrocautery* and *electrosurgery* are often used interchangeably when the words actually mean distinctly different things. *Electrocautery* coagulates small vessels or cuts tissue by using heat generated by direct electric current into a metal wire or probe. Electrical current does not enter the patient's body. *Electrosurgery* refers to generating heat inside the tissue using an alternating electric current that passes through the tissue creating a circuit. Electrosurgery is widely used for hemostasis for vessels less than 1.5 to 2 mm in diameter; larger vessels should be addressed by other means.

Standard electrical current alternates at a frequency of 60 cycles per second (Hz). Because nerve and muscle stimulation

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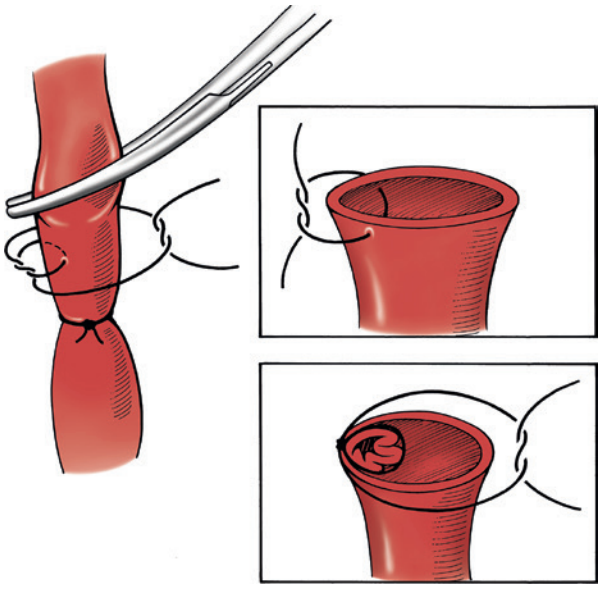


FIG. 8.16 To place a transfixation ligature on a vessel, introduce the needle through the previously ligated tissue. Place a single throw in the suture on the near side, then tie the suture (two square knots) on the opposite side of the vessel.

cease at 100,000 cycles/second (100 kHz), an electrosurgical generator takes 60 cycle current and increases the frequency to more than 200,000 cycles per second. This affords minimal neuromuscular stimulation without risk of electrocution. Electrosurgical generators are able to produce a variety of waveforms that have different effects on tissue. A constant waveform occurs on the "cut" setting; it produces heat quickly to vaporize or cut tissue. The "coagulation" setting uses an intermittent waveform, which produces less heat and creates a coagulum rather than vaporizing tissue. Modern, high-quality electrosurgery units (e.g., Force FX Electrosurgical Generator C; Medtronic Inc.) have a blend setting, which is an intermittent waveform at a higher duty cycle, so that it produces more heat. More coagulation occurs than with a pure cut setting, and more tissue cutting occurs than with a pure coagulation setting.

NOTE Research studies have confirmed that the smoke plume given off during electrocautery can contain toxic gases and vapors, such as benzene, hydrogen cyanide, formaldehyde, bioaerosols, dead and live cellular material (including blood fragments), and viruses. Thus the Occupational Safety and Health Administration recommends that smoke evacuation systems be used to reduce potential acute and chronic health risks to patients and personnel.

Electrosurgery may be performed with monopolar or bipolar devices, in a direct or indirect fashion.

Monopolar Electrosurgery

Monopolar electrosurgery is the most commonly used method of electrosurgery. It involves the flow of current from an active electrode (handpiece) through the patient to a ground plate. The small surface area of the handpiece concentrates the current density, increasing the temperature of the contact tissue and causing coagulation or tissue vaporization. The larger surface area of the ground plate reduces the current density so that minimal tissue heating occurs as the circuit is completed. If an adequate low-impedance ground pad is not present, alternate paths to ground will be inadvertently used by the circuit, resulting in burns to the patient. Most modern electrosurgery units have circuitry to eliminate this danger, but safe practices are still important. The amount of current multiplied by the amount of time the current is applied, and divided by the area of the return path, is proportional to the probability of a burn. Therefore a small ground plate or an alternate path to ground (i.e., through an ECG pad or lead) can easily produce a severe burn. *To reduce the risk of a burn, use a large pad placed in good contact on well-vascularized area of tissue that is close to the operative site.* In addition, keeping electrodes clean and free of eschar will enhance performance by maintaining lower resistance within the circuit. Towels dampened with water or conduction gel can be placed on metal ground plates to improve contact. Adhesive hydrogel patient return electrodes are available with some electrosurgery systems. These electrodes conform to the surface of the patient and provide improved contact over metal plates. Certain models (REM Polyhesive Patient Return Electrode) monitor patient impedance levels and will deactivate the generator if a fault (poor contact) at the electrode is detected.

With monopolar coagulation, the field must be relatively dry and the electrode kept clean and free of debris. Direct contact of the electrode with the tissue produces lower heat sufficient to coagulate. The use of the arc between the electrode and tissue produces higher heat and consequently a cutting or vaporization action. The standard flat-tipped electrode is designed to create an eschar or coagulum directly on tissue. When there is direct contact of the tip with the tissue, the cutting cycle will use far less voltage to accomplish the same coagulation as the coagulation cycle produces. Indirect contact involves touching the electrode to an instrument, usually hemostatic or tissue forceps that has been applied to the bleeding vessel. This technique allows more precise application of energy and effective coagulation.

NOTE Do not activate the generator while the active electrode is touching or in close proximity to another metal object. The instrument (particularly in body cavities) when energized can seek its own pathway to the return electrode, resulting in patient injury. Gloves can occasionally act in a capacitance function and cause burns to the surgeon.

Bipolar Electrosurgery

Bipolar electrosurgery involves the use of a forceps-like handpiece. Instead of passing through the patient's body, the current passes from one tip of the forceps to the opposite tip through the tissue being held between the tips. The tips must be held approximately 1 mm apart for a current to be generated. A ground plate or pad is not necessary for bipolar electrosurgery. Bipolar coagulation is used when precise coagulation is necessary and to prevent damage to adjacent structures, such as in spinal surgery, thyroidectomy, or ophthalmic procedures.

Radiosurgery generators and handpieces (e.g., Surgitron Dual Frequency RF) are similar to standard electrosurgery units, except

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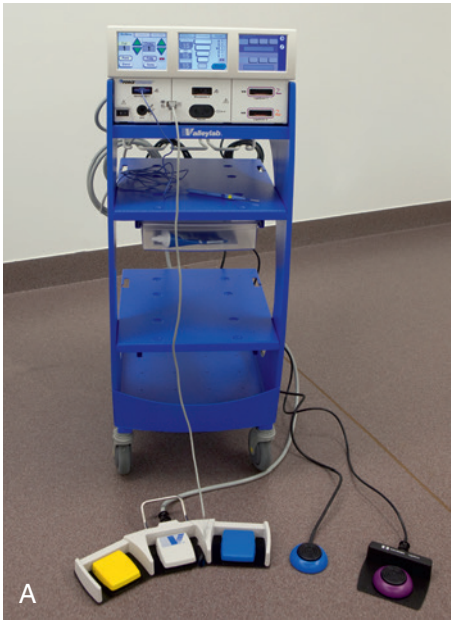




FIG. 8.17 (A and B) Force Triad Energy Platform with monopoly electrosurgical cutting and coagulation, bipolar functionality, and vessel sealing system.

that the energy is generated by low-temperature, high-frequency current (4.0 MHz). The radio waves pass from an active electrode in the handpiece to a passive electrode beneath or near the patient. Tissue resistance to the radio waves causes an ionic agitation in the cells at the tip of the active electrode. This results in molecular friction within the tissues and generation of heat. With this technology, there is precise tissue dissection with excellent incisional hemostasis, but with minimal thermal damage to the surrounding tissues.

Carbon Dioxide Laser

Although there are numerous types of surgical lasers available, the most widely used laser in small animal surgery is the carbon dioxide (CO_2) laser. The laser generates a wavelength of 10,600 nm, which is highly and selectively absorbed by water. Tissue vaporization occurs as this light energy is absorbed by water into the soft tissues. Penetration into tissue is shallow and precise with little heat dissipated to surrounding tissues. Proposed advantages of the CO_2 laser include less bleeding, less pain, less tissue swelling, and decreased risk of infection.

Vessel Sealing

Electrothermal, feedback-controlled, bipolar vessel sealing systems (Ligasure, Enseal) (Fig. 8.17) can permanently seal blood vessels up to 7 mm in diameter. The device uses pressure and pulsed, low voltage energy to fuse collagen and elastin of the vessel wall and achieve hemostasis. The amount and duration of energy delivered automatically adjusts to the degree of tissue impedance. A 2014 study showed that a single seal created with a single activation cycle is adequate for sealing canine carotid arteries.[13] This technology is utilized for hemostasis in laparoscopic (e.g., ovariectomy, cryptorchidectomy) and thoracoscopic (e.g., partial pericardectomy, lung lobectomy) surgery, as well as open abdominal procedures (e.g., splenectomy, liver lobectomy, partial pancreatectomy) and upper airway surgery (e.g., soft palate resection, tonsillectomy).

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9 Surgical Infections and Antibiotic Selection

Modern antibiotic therapy began with the discovery and mass production of penicillin in 1941. Since then, many otherwise fatal infections have been resolved or prevented through their use. Unfortunately, their great success lead to rampant misuse. Widespread use of prophylactic antibiotics in surgical patients has resulted in de-emphasis of surgical asepsis and development of antibiotic-resistant bacteria. Nosocomial infections with resistant bacteria have been promulgated by prolonged and extensive surgical procedures, invasive supportive therapy, lengthy hospital stays, inappropriate use of antibiotics, increased survival of geriatric and debilitated patients, and use of immunosuppressive drugs.

Antibiotic selection is often based on personal bias versus actual/expected bacterial flora. Antibiotic therapy may be either *prophylactic* or *therapeutic* in nature. Prophylactic antibiotic therapy should primarily be used when there is clearly an increased likelihood of infection or when infection would be catastrophic. Selection of prophylactic antibiotics should be based on expected bacterial flora in the targeted tissue and their anticipated resistance patterns as determined by recent laboratory data. Choice of antibiotics for an established infection (therapeutic antibiotics) is ideally based on culture and susceptibility results; however, antibiotic therapy often needs to be initiated before this information is available. Therefore initial selection should be based on expected flora with a subsequent modification based on culture and sensitivity results.

NOTE Inappropriate administration of antibiotics may not only harm the patient being treated but also promote development of resistant microbes that can harm future patients.

Bacterial survival in a host depends on bacterial virulence and numbers, host immunocompetence, and wound factors that antagonize host defenses (e.g., blood clots, ischemic tissue, fluid pockets, foreign material). Successful antibiotic therapy requires reducing bacterial numbers to the point that host defenses are effective. With competent host defenses, bacteriostatic agents that slow protein synthesis or prevent bacterial replication are often adequate (see later discussion). If host defenses have been significantly compromised, then bactericidal concentrations of antibiotics are generally preferred. In addition to using appropriate antibiotics, wound factors may need to be corrected through wound debridement, drainage, and/or removal of foreign material to achieve a successful outcome.

NOTE Some antibiotics are bacteriostatic at low concentrations and bactericidal at higher concentrations. If susceptibility testing has been done, use an antibiotic to which the bacteria are susceptible regardless of whether it is bacteriostatic or bactericidal.

MECHANISMS OF ANTIBIOTIC ACTION

When antibiotics only inhibit bacterial growth, they are noted as being *bacteriostatic*; when they kill bacteria, they are noted as being *bactericidal*. The distinction between bactericidal and bacteriostatic classifications of antibiotics is relative and depends on the ratio between the minimum bactericidal concentration (MBC) and the minimum inhibitory concentration (MIC). The MIC, generally expressed in micrograms per milliliter (μg/mL), is the lowest concentration of a drug that inhibits visible bacterial growth after overnight incubation. The MBC is the lowest concentration of a drug that kills 99.9% of bacteria after overnight incubation. Antibiotics with a MBC to MIC ratio of 4 or less are classified as bactericidal because plasma and tissue levels that kill 99.9% of the bacteria are typically achieved. Conversely, drugs with a large MBC to MIC ratio may make it difficult to achieve plasma or tissue levels that kill bacteria; such drugs are considered bacteriostatic. However, nonuniform distribution of antibiotics in body tissues can cause culture and susceptibility results to be misleading. An antibiotic may be present in higher than anticipated concentrations in some tissues, making a "bacteriostatic" drug effectively bactericidal for certain bacteria in that tissue. A common example is treatment of urinary tract infections (UTIs). A UTI characterized as "resistant" to a particular antibiotic based on susceptibility testing might be successfully treated if the antibiotic is concentrated in urine. Conversely, a bacteria considered "sensitive" will probably not respond if the infection involves the central nervous system (CNS) and the antibiotic does not penetrate the blood-brain barrier.

Finally, the antibiotic must kill bacteria without harming the host. When the dose required to kill bacteria is greater than can be tolerated by the host (or achieved in the target tissue), then the bacteria are considered "resistant" to that drug.

Antibiotics typically are classified according to their mechanism of action. Broadly, they may destroy or alter the bacterial cell wall or inhibit its synthesis, or inhibit protein or deoxyribonucleic acid (DNA) synthesis.

Destruction of Bacterial Cell Walls

Antibiotics that inhibit synthesis or promote destruction of bacterial cell walls include the β-lactam ring antibiotics (e.g., penicillins, cephalosporins, carbapenems, and monobactams); vancomycin; bacitracin; polymyxin; the antifungal drugs nystatin and amphotericin B; and the imidazoles. β-Lactams bind to proteins in the cell wall (e.g., penicillin-binding proteins [PBPs]), thereby impairing cell wall synthesis, reducing its strength and rigidity, and ultimately causing increased permeability and cell lysis. β-Lactam antibiotics tend to be time dependent, bactericidal, and are typically eliminated via the kidneys. Being time dependent,

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ABSTRACT

Surgical infections remain a serious risk in small animal surgery. A thorough understanding of the variety of bacteria and methods of diagnosis and treatment is critical for surgeons and support staff. In addition, the increasing issues with bacterial resistance and methods to combat resistant bacteria should be thoroughly understood.

KEYWORDS

antibiotic
bacterial resistance
penicillin
amoxicillin
ampicillin
piperacillin
sulbactam
tazobactam
cephalosporin
clindamycin
tetracycline
doxycycline
minocycline
chloramphenicol
azithromycin
metronidazole
enrofloxacin
potentiated sulfa drug
drugs of last resort
difloxacin
orbifloxacin
marbofloxacin
aminoglycoside
amikacin

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it is important to administer the drugs frequently enough so that MBCs are maintained for 80% of the treatment interval. Most β-lactams are relatively safe in dogs and cats.

There are over 15 different types of penicillins and over 20 different cephalosporins; therefore only representative examples are considered in this discussion. **Aminopenicillins** (e.g., amoxicillin, ampicillin) are effective against many gram-positive aerobes and some gram-positive and gram-negative anaerobes. The carboxypenicillins (e.g., piperacillin/tazobactam) have better gram-negative and anaerobe spectrums than the aminopenicillins, whereas the ureidopenicillins (e.g., mezlocillin) have the best gram-negative spectrums of all the penicillins. Typical indications include urinary tract, respiratory, soft tissue, and cutaneous infections. Many gram-negative bacteria have resistance factors that inhibit penicillin's activity. Resistance to penicillins is mediated by bacterial penicillinases (a type of β-lactamase), decreased permeability of the cell wall to penicillins caused by altered porin size, and altered PBP structure, which prevents penicillins from binding (e.g., methicillin-resistant staphylococci). Penicillinase inhibitors (e.g., clavulanic acid, sulbactam) may be combined with penicillins (e.g., amoxicillin, ampicillin, piperacillin/tazobactam) to enhance their activity, but these inhibitors do not counter all mechanisms of resistance.

The **cephalosporins** (Table 9.1) are typically more effective than penicillin against gram-negative rods (e.g., Enterobacteriaceae). Resistance to cephalosporins is mediated by the same mechanisms that cause resistance to penicillins. First-generation cephalosporins (e.g., cefazolin) are usually effective against most gram-positive and some gram-negative organisms, but not anaerobes. Second-generation cephalosporins (e.g., cefoxitin) have greater activity against gram-negative bacteria and anaerobes but no additional efficacy against gram-positive organisms. Third-generation cephalosporins (e.g., cefotaxime) are highly effective against more than 90% of gram-negative bacteria, but they often are less active against gram-positive organisms than first-generation cephalosporins. Some third-generation cephalosporins have specific gram-negative spectra, and just because one third-generation cephalosporin is effective in a patient does not mean that another third-generation cephalosporin will also be effective. Cefpodoxime proxetil (Simplicef) is a prodrug whose metabolite is cefpodoxime, a third-generation cephalosporin with good activity against most streptococci and staphylococci and Enterobacteriaceae. It is designed to be given once daily, usually for skin and ear infections, as well as upper respiratory and urinary tract infections.

Imipenem and meropenem are carbapenems (a type of β-lactam antibiotic) that have broad gram-negative and gram-positive spectra and are highly resistant to most β-lactamases, except for the New Delhi metallo-β-lactamase or the *Klebsiella pneumoniae* carbapenemase. Unlike aminoglycosides, they are not nephrotoxic. Imipenem and meropenem are usually effective against most clinically relevant bacterial species, including gram-negative and gram-positive anaerobes and aerobes, but not methicillin-resistant staphylococci or resistant strains of *Enterococcus faecium*. Imipenem should be used only for severely ill patients with sensitivity results documenting that the bacteria is resistant

TABLE 9.1 Select Cephalosporin Drugs

Drug Name	Brand Name	Indications	Dosage
First-Generation Cephalosporins			
Cephalexin	Keflex	Wide spectrum of activity against gram-positive organisms; variable against gram-negative organisms; poor activity against anaerobic infections	22–30 mg/kg PO bid-qid
Cefazolin	Kefzol	As above	20–35 mg/kg IV, IM, SC; tid
Cefadroxil	Cefa-tabs	As above	22–35 mg/kg PO bid
Second- and Third-Generation Cephalosporins			
Cefepime	Maxipime	Fourth-generation cephalosporin with better gram-positive coverage than third-generation drugs	40 mg/kg IV, IM; qid
Cefotetan	Cefotan	Anaerobes and gram-negative bacilli (e.g., septic peritonitis, *Escherichia coli*)	30 mg/kg IV, SC; tid
Cefoxitin	Mefoxin	Anaerobes and gram-negative bacilli (e.g., septic peritonitis)	30 mg/kg IV, IM; tid-qid 22 mg/kg IV (presurgical)
Cefotaxime	Claforan	Wide spectrum of activity against both gram-negative and gram-positive organisms; most active in this group against staphylococci; reaches good concentrations in spinal fluid	*Dogs:* 50 mg/kg IV, IM, SC; bid *Cats:* 20–80 mg/kg IV, IM; qid
Cefovecin	Convenia	For skin and soft tissue infections where first-line drugs fail	8 mg/kg SC every 14 days
Ceftazidime	Fortaz	Effective against *Pseudomonas aeruginosa*	25–30 mg/kg; IM, SC (tid), or IV (qid)
Cefixime	Suprax	Limited activity against most gram-positive organisms; can be given orally; use lower dose for urinary tract infections and higher dose for other infections	10 mg/kg PO bid
Cefpodoxime	Proxetil	Better activity against *Staphylococcus* spp. than other oral third-generation cephalosporins	5–10 mg/kg PO qd
Cefoperazone	Cefobid	Effective against Enterobacteriaceae	22 mg/kg IV, IM; bid-qid
Cefquinome	Cobactan	A fourth-generation cephalosporin with good activity against Enterobacteriaceae	2 mg/kg IV, IM, SC; bid
Ceftriaxone	Rocephin	Often used in CNS infections and borreliosis	25–50 mg/kg IV, IM; qd-bid
Ceftiofur	Naxcel	Activity against gram-positive organisms; can be given once a day for urinary tract infections; not recommended for soft tissue infections in dogs or cats; not effective against enterococci	2.2–4.4 mg/kg SC qd-bid
Cefuroxime	Biociclin	Gram-positive except Enterococcus and MRSP	15–30 mg/kg IV, PO; bid-tid

bid, Twice a day; CNS, central nervous system; *IM,* intramuscular; *IV,* intravenous; *MRSP,* methicillin-resistant *Staphylococcus pseudintermedius; PO,* oral; *qd,* once a day; *qid,* four times a day; *SC,* subcutaneous; *tid,* three times a day.

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to all other antibiotics. Unfortunately, excessive use has already produced resistant bacteria, especially Enterobacteriaceae (i.e., *Klebsiella* and *E. coli*).

Aztreonam is a monobactam (another β-lactam) that is resistant to many β-lactamases. It is highly effective against many gram-negative aerobes but has little activity against anaerobes. It has no activity against gram-positive bacteria and must be used in combination with other drugs to achieve broad-spectrum activity.

Inhibition of Protein Synthesis

Chloramphenicol, tetracycline, erythromycin, and clindamycin bind to bacterial ribosomes, causing reversible inhibition of protein synthesis. Chloramphenicol (Table 9.2) has broad-spectrum activity against most anaerobic and aerobic bacteria, as well as *Ehrlichia* spp. and *Rickettsia* spp., but poor activity against *Pseudomonas* spp. It is highly lipophilic and penetrates most organs (e.g., CNS, eye, prostate). Although considered bacteriostatic, it often achieves tissue levels high enough to make it bactericidal. It is eliminated by hepatic metabolism and urinary excretion. The drug may cause idiosyncratic fatal anemia in people, but dogs and cats experience only a mild, transient anemia, if that. It inhibits hepatic P450 cytochromes and can thus affect hepatic metabolism of certain other drugs (e.g., propofol, opiates, barbiturates).

Tetracyclines are effective against many gram-positive and gram-negative bacteria, including *Chlamydia* spp., rickettsia, spirochetes, *Mycoplasma* spp., bacterial L-forms, and some protozoa. They are usually ineffective against staphylococci, enterococci, *Pseudomonas* spp., and Enterobacteriaceae. Doxycycline (see Table 9.2) is usually the preferred tetracycline in small animal practice because it has fewer side effects, penetrates cells better, and has less resistance develop to it compared with other tetracyclines. It is well distributed to most tissues and achieves good intracellular concentrations, although not to the CNS. Products containing calcium chelate and tetracyclines interfere with oral absorption. Binding of the drugs to calcium can be a problem in young or pregnant animals, and tooth discoloration and inhibited bone growth can occur. Doxycycline is primarily eliminated through the intestines in the dog. Typical indications include tick-borne diseases as well as respiratory and prostatic infections. Doxycycline causes hepatotoxicity in some patients, but its main adverse side effect is related to causing esophagitis after oral administration.

NOTE Tetracyclines are caustic, and it is critical that the patient drink water immediately after swallowing a tablet or capsule (in the case of doxycycline, the patient may eat nondairy food). Otherwise the tablet may lodge in the esophagus and cause esophagitis and even a benign stricture (especially in cats).

Erythromycin is a macrolide that is readily absorbed from the upper gastrointestinal system and diffuses well throughout most tissues. All macrolides tend to accumulate in phagocytic cells and are usually eliminated in the bile. Erythromycin has a narrow spectrum of activity and may be associated with nausea and

TABLE 9.2 Dosages of Selected Antibiotics

Generic and Trade Names	Dosage
Amoxicillin plus clavulanate (Clavamox)	*Dogs*: 12.5–25 mg/kg PO bid *Cats*: 62.5 mg/cat PO bid (tid for gram-negative infections)
Azithromycin (Zithromax)	*Dogs*: 10 mg/kg PO qd for 7 days, followed by every other day *Cats*: 5–10 mg/kg PO qd for 7 days, then give every other day; for upper respiratory infections, give 15 mg/kg qd PO for 3 d, then q72h
Clindamycin (Antirobe)	*Dogs:* 11–33 mg/kg PO bid *or* 11 mg/kg IV, bid; dilute and give slowing when giving IV *Cats*: 11–33 mg/kg PO qd *or* 10 mg/kg IV, IM bid
Amikacin (Amiglyde-V)	*Dogs:* 15–22 mg/kg IV, SC, IM; qd (preferably in the morning)
Piperacillin/tazobactam (Zosyn)	50 mg/kg IV, IM; qid
Ampicillin plus sulbactam (Unisyn)	10–20 mg/kg IV, IM; tid
Chloramphenicol (Chloromycetin)	*Dogs*: 50 mg/kg PO, or 10–15 mg/kg IV tid *Cats*: 12.5–20 mg/cat IV, IM bid
Doxycycline (Vibramycin)	5 mg/kg PO bid *or* 10 mg/kg PO qd
Minocycline (Minocin)	*Dogs*: 5–10 mg/kg PO bid *Cats*: 8.8 mg/kg PO qd
Enrofloxacin (Baytril)[a]	*Urinary tract infection:* 2.5 mg/kg IV, IM, SC, PO bid *Deep tissue infection:* 7–20 mg/kg IV, IM, SC, PO qd
Difloxacin (Dicural)	5–10 mg/kg PO qd (use higher dose for organisms with higher MIC)
Orbifloxacin (Orbax)	2.5–7.5 mg/kg PO qd (use higher dose for organisms with higher MIC)
Marbofloxacin (Zeniqun)	2.75–5.5 mg/kg PO qd
Pradofloxacin (Veraflox)	*Dogs*: 3–5 mg/kg PO qd *Cats*: 3–5 mg/kg PO qd for tablets; 5–7.5 mg/kg PO qd for suspension
Trimethoprim-Sulfadiazine (Tribrissen)	15 mg/kg PO bid *or* 30 mg/kg PO qd–bid
Metronidazole (Flagyl)	7.5–10 mg/kg PO, IV bid–tid (dilute and give slowly over 20 min)

[a]When given IV, enrofloxacin is typically diluted and given over 10 to 20 minutes. Recent reports suggest that enrofloxacin may be associated with blindness in cats when doses >5 mg/kg are used. It can be given as a single IV injection daily.

bid, Twice a day; *IM,* intramuscular; *IV,* intravenous; *MIC,* minimum inhibitory concentration; *PO,* oral; *qd,* once a day; *qid,* four times a day; *qod,* every other day; *SC,* subcutaneous; *tid,* three times a day.

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vomiting because of its prokinetic activity. New derivatives include clarithromycin and azithromycin. Azithromycin (see Table 9.2) is active against aerobic bacteria, such as staphylococci and streptococci, and anaerobes. It also has good activity against *Mycoplasma* spp. and intracellular organisms, such as *Bartonella* spp., *Toxoplasma* spp., and atypical mycobacteria. Absorption of azithromycin after oral administration is high, and it is well tolerated. The drug achieves extremely high tissue concentrations and only needs to be given once daily.

Clindamycin (see Table 9.2) is a lincosamide that is primarily eliminated by biliary excretion and achieves excellent concentrations in the skin and bones, as well as high concentrations in white blood cells. It is effective against most anaerobic bacteria, plus it is active against many gram-positive pathogens. Typical indications include intra-abdominal infections, osteomyelitis, discospondylitis, and oral/dental disease. Clindamycin can be used in combination therapy directed at *Pseudomonas aeruginosa*; it inhibits bacterial adherence to epithelial cells, making it more susceptible to other antibiotics. Clindamycin is effective against toxoplasmosis and *Neospora*, and it can cause esophagitis if the pill stays in the esophagus (especially in cats).

The aminoglycosides (e.g., amikacin, gentamicin, kanamycin, neomycin, netilmicin, and tobramycin) disrupt protein synthesis by binding irreversibly to bacterial ribosomes, making them bactericidal. They are effective against gram-negative and gram-positive bacteria, including Enterobacteriaceae and pseudomonads, but not anaerobes. Their activity is reduced in necrotic tissue because of free nucleic acid material. Aminoglycosides are polar (i.e., lipid insoluble) and have limited distribution in the extracellular and cerebrospinal fluids. However, distribution into the pleural fluid, bone, joints, and peritoneal cavity is good. Aminoglycosides are poorly absorbed orally. They are concentration dependent rather than time dependent, meaning that they can be given at higher doses at longer intervals (e.g., once daily), which maintains effectiveness but reduces renal toxicity. Amikacin (see Table 9.2) is typically used. Dehydration, electrolyte loss, preexisting renal disease, and concurrent use of other nephrotoxic drugs (e.g., nonsteroidal antiinflammatory drugs [NSAIDs]) increase the nephrotoxicity of aminoglycosides. Ototoxicosis and neuromuscular blockade are other possible adverse effects. Using a combination of a β-lactam and an aminoglycoside is often synergistic, plus it helps prevent bacteria from becoming resistant to these drugs.

NOTE A β-lactam drug should never be mixed in the same syringe, vial, or intravenous (IV) line with an aminoglycoside; mixing the two can result in a chemical reaction that inactivates one or both of the drugs.

Inhibition of DNA Synthesis

Fluoroquinolones (e.g., enrofloxacin, difloxacin, ciprofloxacin, ofloxacin, marbofloxacin, pradofloxacin) (see Table 9.2) and potentiated sulfas (e.g., trimethoprim-sulfa) inhibit DNA synthesis. Fluoroquinolones inhibit DNA synthesis by inactivating DNA gyrase. They are bactericidal, concentration-dependent drugs with a prolonged postantibiotic effect. Fluoroquinolones tend to accumulate in phagocytic white blood cells. Enrofloxacin is primarily excreted in the urine; however, approximately 25% is metabolized to ciprofloxacin. It has a principally gram-negative spectrum but is also effective against *Rickettsia rickettsii*, Mycobacteria, and possibly L-form bacteria. Enrofloxacin is poorly effective against most gram-positive cocci and anaerobic bacteria, but the newer fluoroquinolones (e.g., pradofloxacin) have enhanced anaerobic activity. Enrofloxacin has "flexible" dosing depending upon the target bacteria and target tissue (see Table 9.2). Resistance to fluoroquinolones has become common. Side effects include blindness in cats, CNS effects, cartilage lesions in rapidly growing large breed dogs, and vomiting. Rapid IV injection of undiluted enrofloxacin can be fatal.

NOTE When given intravenously, enrofloxacin must be given slowly, in a diluted solution, or the patient may experience morbidity or even death.

Oral ciprofloxacin is less expensive than enrofloxacin but is also less bioavailable in dogs (approximately 30%–40%) than in people (approximately 70%–80%). Therefore it is commonly underdosed when administered to dogs. Marbofloxacin has a broad spectrum of activity against the major pathogens encountered in surgical infections. It is safe in dogs, and a single IV injection of 2 to 4 mg/kg maintains plasma concentrations above the MIC for Enterobacteriaceae and staphylococci for 12 to 24 hours.

Trimethoprim-sulfonamide combinations inhibit sequential steps in folate synthesis, making them time-dependent, bactericidal drugs to which bacteria have difficulty developing resistance. Penetration into tissues depends on the specific sulfonamide used; sulfadiazine penetrates most body tissues well, whereas sulfamethoxazole is limited to the interstitial fluid compartment. Sulfas are renally excreted, but some are metabolized by the liver. In general, they are effective for the treatment of osteomyelitis, prostatitis, pneumonia, tracheobronchitis, pyoderma, and UTI. Trimethoprim-sulfonamide combinations have a broad spectrum of activity against gram-positive and gram-negative bacteria, as well as *Nocardia* spp. and anaerobic bacteria. They are usually not effective against pseudomonads. In vitro and in vivo susceptibilities do not always correlate. Sulfas tend to have poor efficacy in the presence of pus or necrotic tissue. Possible side effects include keratoconjunctivitis sicca, thrombocytopenia, anemia, neutropenia, bone marrow suppression, fever, vomiting, hypersensitivity (i.e., vasculitis or arthritis), and hepatic disease. Some breeds (e.g., miniature Schnauzers, Samoyeds, Doberman pinschers) and some families of dogs seem more likely to suffer side effects, probably because of altered hepatic metabolism.

Metronidazole is very effective against most anaerobic bacteria. It penetrates most body tissues well. Dose-dependent CNS toxicity is common if excessive doses are administered.

DRUGS OF LAST RESORT

The number of antibiotics that are effective against multidrug resistant (MDR) infections (e.g., methicillin-resistant *S. aureus* [MRSA], vancomycin-resistant enterococci, MDR gram-negative rods) is ever diminishing due to transmission of resistance between bacteria, especially in hospital settings. Currently, there are some MDR bacteria for which only one or two drugs are effective.

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The antibiotics reserved for such infections are sometimes referred to as *drugs of last resort*. They include vancomycin, the carbapenems (i.e., imipenem, meropenem), the oxazolidinone linezolid (Zyvox), the streptogramin combination of dalfopristin and quinupristin (Synercid), the cyclic lipopeptide daptomycin (Cubicin), the glycopeptide telavancin (Vibativ), the tetracycline tigecycline (Tygacil), the oxazolidinone telithromycin (Ketek), and the newest cephalosporins (i.e., ceftaroline and ceftobiprole). Although tempting to use these antibiotics when faced with a patient seriously ill from a bacterial infection (especially when the clinician wants to impress the client with the fact that their pet is receiving the very newest and most powerful antibiotic available), it is a moral and ethical imperative that veterinarians only use such drugs when the bacteria has been isolated and determined to only respond to one of these antibiotics. The more these drugs are used, the sooner resistant strains will develop. Most dogs with extraintestinal infection by MDR *E. coli* and *Enterobacter* have had prior antibiotic treatment, prolonged hospitalization, or prior surgery (the urinary tract appears to be the most common site of infection with these bacteria).

CAUSES OF ANTIBIOTIC FAILURE AND MECHANISMS OF ANTIBIOTIC RESISTANCE

Successful antibiotic therapy requires administering the appropriate dose of an effective antibiotic that can reach the site of infection so that the bacteria there are killed or suppressed sufficiently to allow the patient's immune system to control the infection. Factors contributing to therapeutic failure include inappropriate dose, frequency, or route of administration; inadequate duration of treatment; inappropriate antibiotic selection; presence of foreign material (i.e., foreign body or implant); inability of sufficient antibiotic to reach the target tissue (e.g., cross the blood-brain barrier); bacterial resistance (see later discussion); depressed host immunity (e.g., concurrent debilitating illness, immunosuppressive drug therapy); pharmacokinetics of the drug; drug reactions; antibiotic antagonism; and incorrect diagnoses (i.e., viral diseases or foreign bodies misdiagnosed as primary bacterial infections).

Unfortunately, even appropriate administration of antibiotics is eventually associated with development of resistance, usually within 1 to 2 decades of introducing a particular drug, although in some cases it occurs within 3 to 4 years. There are two main ways that bacteria develop resistance; they can alter the site that the antibiotic targets (i.e., receptors in/on the bacteria), or they can alter the concentration of the antibiotic in the bacteria. Intracellular concentrations may be altered by enzymatic destruction of the antibiotic (e.g., β-lactamases), decreasing bacterial permeability to the antibiotic (e.g., altering porin size such that antibiotics can no longer access intracellular targets), or developing efflux proteins (i.e., these are closely associated with porins, and they pump specific chemicals out of the bacteria). Alteration of the structural target for the antibiotic includes alteration of PBPs in the cell wall and binding sites on ribosomes.

SURGICAL INFECTIONS

Definitions of Infection

There are numerous means of defining the existence of bacterial infection. Bacterial infection has been defined quantitatively as a wound having more than 10^5 bacteria per gram of tissue. This definition has limited clinical applicability when attempting to determine when to treat an infection or for broader screening and reporting purposes. Alternatively, the Centers for Disease Control and Prevention (CDC) more specifically defines infection based on specific body systems (e.g., bone and joint, CNS, cardiovascular) with subcategories (osteomyelitis, discospondylitis) that require fulfillment of criteria that may or may not include the actual culture of bacteria. *This is significant as bacterial infection can frequently be present in spite of the inability to demonstrate it with a positive culture.* This is particularly true of canine and human joint infections. An additional CDC category of infections is the surgical site infection (SSI). There are no agreed-upon categorizations of SSI in veterinary medicine. Adaptation of the CDC categories are likely appropriate in veterinary patients and include superficial incisional, deep incisional, and organ/space (Fig. 9.1). The criteria for diagnosing an infection in each of these three locations are listed in Box 9.1.

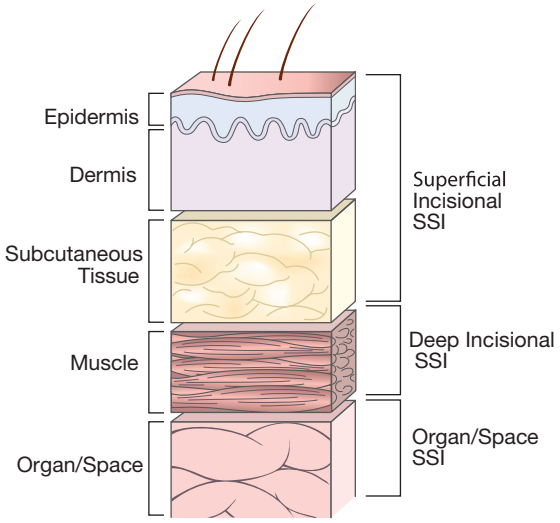


FIG. 9.1 Categorization of surgical site infections (SSIs) by tissue layer. Superficial incisional SSIs are the most commonly reported surgical infections, accounting for more SSIs than deep incisional SSIs and organ space SSIs combined.

Sources of Surgical Site Infections

Sources of surgical infections are separated into endogenous and exogenous. Endogenous sources include skin flora, mucous membranes, gastrointestinal tract, and preexisting infections at the surgical site (abscesses, open wounds). Additionally, endogenous sources of SSI include distant sites of infection such as dental disease, UTI, or endocarditis. Exogenous sources of SSI include surgical personnel, the operating room (OR) environment and ventilation, and surgical tools, equipment, and materials.

Reported Rates of Small Animal Surgical Site Infections

Veterinary studies have estimated the rates of SSI to be between 3% and 10%; however, data from some studies are difficult to interpret based on the variability in their definitions of infection, retrospective nature, small sample size, and use of medical record

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BOX 9.1 Surgical Site Infection (CDC Definition)

Superficial Incisional Surgical Site Infection (SSI)

Infection within 30 days after the operation and only involves skin and subcutaneous tissue of the incision and at least one of the following:

1. Purulent drainage, with or without laboratory confirmation, from the superficial incision.
2. Organisms isolated from an aseptically obtained culture of fluid or tissue from the superficial incision.
3. At least one of the following signs or symptoms of infection: pain or tenderness, localized swelling, redness, or heat and superficial incision is deliberately opened by surgeon, unless incision is culture negative.
4. Diagnosis of superficial incisional SSI made by a surgeon or attending physician.

Deep Incisional Surgical Site Infection

Infection occurs within 30 days after the operation if no implant is left in place or within 1 year if implant is in place and the infection appears to be related to the operation and infection involves deep soft tissue (e.g., fascia, muscle) of the incision and at least one of the following:

1. Purulent drainage from the deep incision but not from the organ/space component of the surgical site.
2. A deep incision spontaneously dehisces or is deliberately opened by a surgeon when the patient has at least one of the following signs or symptoms: fever (>38°C [100°F]), localized pain or tenderness, unless incision is culture negative.
3. An abscess or other evidence of infection involving the deep incision is found on direct examination, during reoperation, or by histopathologic or radiologic examination.
4. Diagnosis of deep incisional SSI made by a surgeon or attending physician.

Organ/Space Surgical Site Infection

Infection occurs within 30 days after the operation if no implant is left in place or within 1 year if implant is in place and the infection appears to be related to the operation and infection involves any part of the anatomy (e.g., organs and spaces) other than the incision that was opened or manipulated during an operation and at least one of the following:

1. Purulent drainage from a drain that is placed through a stab wound into the organ/space.
2. Organisms isolated from an aseptically obtained culture of fluid or tissue in the organ/space.
3. An abscess or other evidence of infection involving the organ/space that is found on direct examination, during reoperation, or by histopathologic or radiologic examination.
4. Diagnosis of organ/space SSI made by a surgeon or attending physician.

CDC, Centers for Disease Control and Prevention.
Modified from Mangram AJ, Horan TC, Pearson ML, et al. Guideline for prevention of surgical site infection 1999. Hospital Infection Control Practices Advisory Committee. *Infec Control Hosp Epidemiol.* 1999;20(4):250–278.

review to identify infection. A 2015 prospective cohort study of 846 dogs examined the incidence of SSI in dogs using the CDC definitions and classifications of SSI.[1] The incidence of SSI in this study was 3% overall with 42% classified as superficial, 50% classified as deep, and 8% as organ/space. The most prominent bacterial species cultured in this study was methicillin-resistant *Staphylococcus pseudintermedius* (MRSP) at 47%. The majority of SSIs are recognized within 30 days of surgery, and the CDC has reduced the screening period for SSIs from 1 year postoperatively to 90 days postoperatively.

Factors Affecting Surgical Site Infections

Numerous factors determine the likelihood of development of an SSI. The most commonly identified of these include the classification of the wound, patient-related factors, and procedure-related factors.

Classification of Wounds

Wounds, including surgical wounds, are classified by degree of contamination to help predict the likelihood that infection will develop. The classification scheme was developed by the National Research Council (Table 9.3) to provide a basis for comparison between types of wounds and between institutions. Classification of surgical wounds by degree of contamination results in significant differences in infection rates.

Clean wounds (see Table 9.3) have a published infection rate of approximately 3%. In this category, wounds most likely to result in postoperative infection are those associated with severe trauma with multiple fractures, traumatic procedures (i.e., carpal arthrodesis), or fractures of the distal radius or tibia that require plating.

Clean-contaminated wounds (see Table 9.3) are identified when nonsterile luminal organs are entered without significant spillage of contents. Included in this category are procedures in which a minor break in aseptic technique occurs, such as perforation of a surgical glove. The most recently reported rate of SSI for this category is 3.8%.

Contaminated wounds (see Table 9.3) have a published infection rate that varies from 0% to 28.6%. Contaminated fractures of long bones and the pelvis and contaminated urogenital procedures most frequently become infected. Antibiotic prophylaxis is indicated for contaminated wounds, and drug selection is based initially on anticipated bacterial flora and modified according to culture and sensitivity results. These wounds are not infected initially but have a real potential to become infected. Delicate debridement, copious lavage, and antibiotic therapy can convert these wounds to clean ones, whereas inadequate therapy often results in a dirty, infected wound.

Dirty wounds (see Table 9.3) are those in which gross infection is present at the time of surgical intervention (e.g., traumatic wounds with retained devitalized tissue, foreign bodies, or fecal contamination). Management of this type of wound requires antibiotic therapy (initial selection being based on anticipated flora and later modified by bacterial culture and sensitivity results), copious lavage, debridement, drainage, and possible use of wet-to-dry bandages to further debride the wound during the early postoperative period. The postoperative infection rate for dirty wounds is approximately 20%.

Patient-Related Factors

The risk of SSI may be increased by gender, body weight, and preexisting medical conditions or existing treatments.

Hypotension. In a 2015 study, dogs experiencing hypotension (i.e., mean arterial pressure <60 mm Hg and systemic arterial pressure <80–90 mm Hg) during surgery were 27 times more likely to develop an SSI.[1] The proposed mechanism is a decrease in oxygenated blood flow to the surgical site.

Endocrinopathies. Endocrinopathies including diabetes mellitus, hyperadrenocorticism, and hypothyroidism have been inconsistently reported to result in an increased risk of SSIs in dogs and cats.

Gender. Androgenic hormones may result in an increased risk of SSI in male dogs and cats. Although not definitively proven, this would be similar to the 50% increased risk that has been identified in men compared with women.

Other patient-related factors. Numerous other patient-related factors may increase the risk of SSI in dogs and cats based on

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TABLE 9.3 Wound Classification System (National Research Council, Division of Medical Sciences)

Classification	Description	Procedure Type (Examples)	Infection Rate
Clean	Nontraumatic, noninflamed operative wounds in which the respiratory, gastrointestinal, genitourinary, and oropharyngeal tracts are not entered	Exploratory laparotomy Elective neuter Total hip replacement PDA	2.0%–4.9%[a]; 2.6% (tibial tuberosity advancement), 3.5% (elective joint surgery),10% (total hip replacement)[b]; 13% tibial plateau leveling osteotomy[c]
Clean contaminated	Operative wounds in which the respiratory, gastrointestinal, or genitourinary tract is entered under controlled conditions without unusual contamination; an otherwise clean wound in which a drain is placed	Bronchoscopy Cholecystectomy Small intestinal resection Enterotomy Renal transplantation	3.5%–4.5%[a]; 26%[d]
Contaminated	Open, fresh, accidental wounds; procedures in which gastrointestinal contents or infected urine is spilled or a major break in aseptic technique occurs	Bile spillage during cholecystectomy or biliary diversion procedures Open cardiac massage Cystotomy with spillage of infected urine Lacerations	4.6%–9.1%[a]
Dirty	Old traumatic wounds with purulent discharge, devitalized tissue, or foreign bodies; procedures in which a viscus is perforated or fecal contamination occurs	Excision or drainage of an abscess Peritonitis Perforated intestinal tract Ruptured gallbladder caused by necrotizing cholecystitis Bullae osteotomy for otitis media	6.7%–17.8%[a]

[a]Eugster S, et al. A prospective study of postoperative surgical site infections in dogs and cats. *Vet Surg.* 2004;33:542.
[b]Weese JS. A review of post-operative infection in veterinary orthopedic surgery. *Vet Comp Orthop Traumatol.* 2008;21:99. (Note: published infection rates for Total Hip Replacement vary from 1.3%–10%.)
[c]Nazarali A, Singh A, Weese JS. Perioperative administration of antimicrobials during tibial plateau leveling osteotomy. *Vet Surg.* 2014;43:966.
[d]Schmiedt CW, Holzman G, Schwarz, et al. Survival complications, and analysis of risk factors after renal transplantation in cats. *Vet Surg.* 2008;37:683.
PDA, Patent ductus arteriosus.

similar evidence in humans. Veterinary studies often do not have adequate statistical power to verify these concerns. Potential factors include obesity, hypothermia, immunosuppressive medications, and blood loss.

Procedure-Related Factors

Procedure-related factors that may increase the risk of infection include hair removal, duration of surgery, duration of anesthesia, OR operating room conditions, and presence of surgical implants.

Surgical site clipping. Clipping of incision sites produces microscopic abrasions in the skin that can be colonized by bacteria. To minimize SSI from bacteria, which colonize skin abraded by clippers, skin preparation should be performed immediately before surgery and no earlier if possible (see Chapter 5).[2]

Surgical time. Prolonged surgical times increase the risk of SSI by suppression of the immune system associated with a transient decrease in lymphocytes that has been correlated with the duration of surgery. Prolonged surgical procedures also increase the risk of SSI through increased tissue manipulation, which further compromises immunity. It has been estimated that each additional hour of surgery time doubles the risk of SSI; however, surgical time was not identified as a risk factor for SSI in a 2015 study.[1]

Tissue handling. Proper atraumatic tissue handling and instrument use are also important in preventing infection. Traumatized tissue better supports bacterial growth and has impaired host defenses. Traumatized or necrotic tissue also has reduced oxygen content, which permits the growth of anaerobic bacteria. Phagocytosis and humoral immunity are significantly diminished when tissue integrity is interrupted during surgery. Inexperienced surgeons cause more tissue trauma than experienced surgeons, resulting in greater susceptibility to infection.

Anesthesia time. It has been estimated that each hour of anesthesia increases the risk of SSI by approximately 30%; however, anesthetic time was not identified as a risk factor for SSI in the study by Turk et al.[1] The duration of anesthesia depends on the actual surgical time, plus any preoperative or postoperative procedures performed (including patient preparation and diagnostics). The potential increased risk of SSI is due not only to associated prolonged surgical times but also to possible immune alterations from inhalant anesthesia and potential bacterial contamination with drugs such as propofol. In older studies, animals that received propofol were more likely to develop postoperative wound infections than animals that did not receive the drug. This may be because the propofol can be contaminated by hospital personnel, particularly when the vial is used for multiple patients.

NOTE Prepare and handle propofol using strict aseptic technique. Discard any unused drug promptly.

Operating room conditions. OR practices (principles of aseptic technique, sterilization and disinfection, preparation of the surgical environment, gowning and gloving, and preparation of the surgical patient, operative site, and surgical team) are

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important in preventing surgical wound infection and are discussed in Chapters 1 through 6. ORs should be designed with surfaces that are easily cleaned and with a minimum of horizontal open surfaces such as shelves. Protocols should be developed for effective cleaning and regular culturing of the OR surfaces (see Chapter 3), as well as other locations in the hospital, to screen for the growth of potential sources of nosocomial infections, particularly those due to resistant bacteria.

Estimates in human surgery suggest that 30% of SSIs are from airborne pathogens that may originate from either the patient, the OR personnel, or equipment and facilities. OR ventilation systems should be designed to provide 15 total air changes per hour with 3 air changes of outdoor air per hour (see Chapter 3). Increased personnel traffic provides increased sources of airborne bacteria and decreases effectiveness of the ventilation system by movement and opening and closing of the OR door; however, a 2015 prospective study based on CDC guidelines demonstrated no increase in SSIs with increases in personnel in the OR.[1]

Contamination rates of personnel (hands and surgical gloves) and facilities during small animal orthopedic procedures has been demonstrated to be as high as 80%.[3] Yet in this study the bacteria responsible for SSI were most often not the same species as those that contaminated the personnel and equipment, suggesting that the pathogenic bacteria that cause SSI are from outside the OR.

Implanted medical devices. Implanted medical devices (IMDs) are foreign substances used to support, rebuild, or in some fashion mimic the function of an anatomic structure (i.e., total hip replacement, polypropylene mesh, nonabsorbable suture, vascular prostheses, metallic implants, or polymethylmethacrylate bone cement). The risk of SSI is 5.6 times greater in dogs with IMDs than in those without.[1] Antibiotic treatment of SSI in patients with IMDs is seldom successful until the implant is removed because implants inhibit medications and defense mechanisms from reaching bacteria, partly because of the formation of biofilms. Biofilm forms when bacteria, particularly *S. pseudintermedius,* adhere to surfaces and begin to excrete an extracellular matrix and form a bacterial aggregate. Once anchored to a surface, biofilm microorganisms are extremely resistant to antibiotics.

PROPHYLACTIC AND THERAPEUTIC USE OF ANTIBIOTICS

Prophylactic Use of Perioperative Antibiotics

Surgical antibacterial perioperative prophylaxis is defined as a brief course of antibacterial medications administered immediately before a surgical procedure in a patient without a current infection.[4] The mechanism of prevention of SSIs is the reduction in the bacterial load at the surgical site to aid the patient's normal defenses. For this to happen, the drugs must be present at the surgical site during the time of potential contamination. Antibiotics are not a substitute for proper aseptic technique, meticulous and atraumatic tissue handling, careful hemostasis, judicious use of sutures, preservation of blood supply, elimination of dead space, and anatomic apposition of tissues.

Indications for Prophylactic Antibiotics

Clean Wounds

Prophylactic antibiotics are not indicated for most clean procedures; exceptions include placement of an implant or where infection could be catastrophic. Infection may be life-threatening in an athletic animal that can no longer perform. In small animal patients undergoing surgeries where the procedure is categorized as "clean," no significant difference is generally seen in the SSI rate between those that receive appropriate perioperative antibiotic prophylaxis and those that receive no antibiotic prophylaxis; however, additional studies are needed. There are conflicting data on whether prophylactic antibiotics reduce infections in clean procedures when an implant is being placed; however, most studies suggest that their use may reduce infections in these circumstances, and their use is seemingly warranted.

Clean-Contaminated and Contaminated Wounds

Prophylactic antibiotics are indicated for clean-contaminated and contaminated procedures. Surgical procedures warranting use of prophylactic antibiotics are listed in Box 9.2.

Obviously, the bacteria most likely to be responsible for infection should be susceptible to the antibiotic used. Antibiotic selection should be based on clinical experience plus what is known about the microbiology of tissues involved. The goal is to use an antibiotic that is effective against at least 80% of the probable pathogens. Unfortunately, the ability to predict what antibiotic bacteria will be sensitive to is becoming increasingly limited due to widespread resistance.

BOX 9.2 Examples of Surgical Procedures That Warrant Perioperative Prophylactic Antibiotics

General Indications
- Surgery time >90 minutes
- Prosthesis implantation (e.g., mesh, pacemaker, vascular prosthesis, bone cement)
- Patients with a preexisting prosthesis (e.g., total hip, pacemaker, bone cement) undergoing surgical procedures (e.g., dental prophylaxis, traumatic wounds, colorectal surgery)
- Severely infected or traumatized wounds

Orthopedic Procedures
- Total hip replacement
- Open fracture repair
- Extensive fracture repair
- Other elective procedures

Respiratory Procedures
- Resection of infected lung lobe or lobes
- Closure of esophagobronchial fistula

Gastrointestinal Procedures
- Colonic anastomosis or colectomy
- Strangulation or obstruction
- Pancreatic abscess
- Gastric resection for gastric dilation-volvulus
- Anal and rectal surgery
- Esophageal surgery
- Perineal herniorrhaphy
- Hepatobiliary surgery with infection

Urogenital Procedures
- Renal, ureteral, bladder, or urethral surgery with infected urine

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Prophylactic use of antibiotics should also be considered for surgical patients predisposed to infections at sites other than the surgical incision. Specifically, the use of perioperative antibiotics may decrease the incidence of urinary tract infections in dogs undergoing surgery for intervertebral disc disease.

Dirty Wounds

Dirty wounds are considered to be infected at the time of surgery. Prophylactic antibiotics are indicated, and selection should be based on the anticipated infecting organisms. Dirty wounds may also be treated with antiseptics such as 0.05% chlorhexidine solution (see Chapter 5), although knowledge of the individual tissue response to antiseptics is important prior to their use.

Selection and Timing of Prophylactic Antibiotics

Antibiotics chosen for surgical prophylaxis should have demonstrated efficacy against the anticipated bacterial contaminants. This will vary with the type and location of the surgical wound. The pathogens usually responsible for postoperative wound infection in small animal surgical patients are *Staphylococcus* spp. (especially *S. pseudintermedius*), *E. coli*, and *Pasteurella* spp. (especially in cats). In a prospective study of 846 dogs, culture-confirmed SSIs were comprised of 64% MRSP, 21% MRSA, and 15% methicillin-susceptible *S. pseudintermedius*. Only 26% of the culture-confirmed SSIs were susceptible to perioperative antibiotics.[1] In another study where *S. pseudintermedius* was also the most common isolate, most isolated pathogens were without acquired microbial resistance and MDR was uncommon.[5] The later study found only three cases of MRSP (1% of all isolates), one of extended spectrum β-lactamase producing *E.coli* infection, and no MRSA infections. The organisms most likely associated with surgical procedures are listed in Table 9.4 according to body system. Special considerations in the selection and administration of prophylactic antibiotics are presented in Box 9.3. Cefazolin has no adverse effects on platelet aggregation, bleeding time, platelet count, platelet size, prothrombin time, or activated partial thromboplastin time and therefore is a good choice for use as a perioperative antibiotic in dogs with conditions predisposing them to hemostatic complications. Intravenous prophylactic antibiotics should be given no earlier than 30 to 60 minutes before the first surgical incision, and they should optimally be discontinued at or within 24 hours of the end of the procedure (see Box 9.3). Because many bacterial isolates that are "sensitive" now have an MIC approaching the threshold of susceptibility, it is generally appropriate to use the higher end of the antibiotic dose. For time-dependent drugs (e.g., β-lactams), the interval for dosing may need to be shortened. For example, cefazolin should probably be readministered at less than 2-hour intervals during the procedure.

Route of Administration

In general, IV administration of prophylactic antibiotics is indicated; however, there is increasing use of topical and local antimicrobial prophylaxis in human medicine. Advantages of local antibiotic administration include high concentrations at the site of infection, a potential for reduced systemic toxicity, and perhaps a reduced risk of antimicrobial resistance.[4] Methods of delivery include installation of ointment, creams, powders, or gels; intraoperative flushing of the wound or cavity, local injections, and antibiotic impregnated or coated beads or implants. Many of these techniques appear promising; however, clinical trials in veterinary medicine are scarce and efficacy is as yet undetermined. The use of antibiotic-impregnated bone cement (PMMA) is common in cemented total joint arthroplasty in dogs. Antibiotic elution from bone cement is bimodal with a rapid initial release followed by a slower release that may last for several months. The efficacy of antibiotic impregnated cement in veterinary total joint replacement has not been evaluated, but its continued use is justified based on human meta-analysis studies demonstrating a significant decrease in the risk of SSIs. The use of antibiotic-impregnated PMMA or calcium sulfate beads in small animals for the treatment of infections is uncommon, and there is limited information regarding efficacy;

TABLE 9.4 Organisms Most Commonly Isolated From Various Body Systems

Procedure, System, or Condition	Likely Pathogens
Thoracic surgeries (pulmonary and cardiovascular procedures)	*Staphylococcus* spp., gram-negative bacilli
Orthopedic surgeries (e.g., total hip replacement, prolonged internal fixation)	*Staphylococcus* spp.
Gastric and upper intestinal surgeries (high-risk patients)	Gram-positive cocci, enteric gram-negative bacilli, anaerobes
Biliary tract surgeries (high-risk patients)	Enteric gram-negative bacilli, anaerobes
Colorectal surgeries	Enteric gram-negative bacilli, anaerobes
Urogenital system (e.g., with pyometra, endometritis)	*Escherichia coli, Streptococcus* spp., anaerobes
Deep, penetrating wounds (e.g., wounds <6 hours old, bite wounds)	Anaerobes, facultative bacteria
Dentistry (patients with valvular heart disease)	*Staphylococcus* spp., *Streptococcus* spp., facultative bacteria, anaerobes

BOX 9.3 Considerations for Selection and Administration of Prophylactic Antibiotics

Antibiotic Selection

- Determine system involved and most likely organism (see Table 9.4)
- Cefazolin attains appropriate concentrations to prevent bacterial growth of the most common contaminants

Timing of Antibiotic Administration

30 minutes to 1 hour before first surgical incision

Cefazolin Dose

22 mg/kg

Routes of Antibiotic Administration

Intravenous; may repeat every 90 minutes depending on length of surgery

Duration of Antibiotic Administration

Discontinue immediately after closure of surgical wound or within 24 hours

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however, their use may be justified in cases of severe orthopedic infections. Caution is advised when using antibiotic-impregnated collagen sponges, as they may increase the risk of SSI. In humans undergoing abdominal surgery, the evidence suggests that the application of topical antibiotics should only be considered in morbidly obese patients undergoing laparotomy where the local application of kanamycin has been shown to be of potential benefit.[6]

Use of Prophylactic Antibiotics Postoperatively

Administering prophylactic antibiotics postoperatively is commonplace in both human and veterinary medicine. The widespread use of medications in this manner likely contributes to the development of antibiotic resistance. The CDC and the European Center for Disease Prevention and Control both recommend against the use of prophylactic postoperative antibiotics because of conflicting studies regarding their efficacy and their potential contribution to antibiotic resistance.

Three recent studies[7–9] demonstrated a reduction in SSI in dogs following clean procedures where a medical device was implanted when prophylactic antibiotics were used postoperatively. One study of 97 dogs demonstrated a reduction in SSI from 21% to 4% when prophylactic antibiotics were used postoperatively (oral cephalexin or amoxicillin for 7 days[9]). A study by Andrade et al. showed no difference in SSI when prophylactic antibiotics were used postoperatively (cephalexin, cefpodoxime, amoxicillin, metronidazole, or clindamycin for 10–14 days).[3] The increased risk of SSI in clean procedures with IMDs may justify the use of postoperative prophylactic antibiotics in these cases; however, the risk/benefit ratio of such use should be considered, especially given the negative effects of such usage on the local (hospital and patient bacterial resistance) and global environment.

Therapeutic Antibiotics for Existing Infections

Therapeutic use of antibiotics is based on clinical judgment, knowledge of the antibiotic's mechanism of action (see previous discussion), and microbiologic factors. The goal is to choose a drug that (a) is selectively active for the most likely infecting microorganism(s), (b) has minimal toxicity to the host, (c) kills bacteria at the site of infection, (d) does not negatively influence the host's immune system, and (e) does not promote generation of resistance. Therapeutic antibiotics are indicated in surgical patients with a systemic infection (i.e., septicemia or bacteremia); when infection is present at the surgical site or in a body cavity (i.e., wound infection, pyothorax, or abdominal abscess); or with any contaminated or dirty surgical procedure listed in Box 9.2. In general, antibiotic therapy is instituted before surgery and continued for at least 2 to 3 days after apparent resolution of the infection; the maximum duration of therapy depends on the drug's toxicity and the disease being treated.

The larger the inoculum at a given site, the less likely the success of the antibiotic therapy. More bacteria mean (a) there may be more enzymes present promoting resistance (e.g., β-lactamases), (b) it will require a greater volume of antibiotics to affect all the bacteria, and (c) there is more possibility of resistance developing to the drugs used. Hence it is important to make the first exposure of the bacteria to the antibiotics be as effective as possible (i.e., achieve as high a drug level at the site as possible). Special considerations in the selection and administration of therapeutic antibiotics are listed in Box 9.4.

BOX 9.4 Considerations for Selection and Administration of Therapeutic Antibiotics

Antibiotic Selection

- Determine system involved and most likely pathogen, to establish primary therapy (see Table 9.4).
- Obtain representative samples for cytologic studies and culture and susceptibility testing (e.g., fluid, tissue, implants, necrotic debris). It is best to obtain samples for culture before administering antibiotics, if this wait does not put the patient at inappropriate risk.
- Ensure that antibiotic reaches target tissue.
- If several antibiotics are effective, select the one that is least expensive, least toxic, and most convenient to administer. If the patient will be discharged while still receiving antibiotics, it is often best to choose an orally administered drug.

Timing of Antibiotic Administration

As soon as samples have been obtained, begin empiric antibiotic therapy.

Dose

Follow recommended doses carefully.

Routes of Antibiotic Administration

Treat for 2–3 days and then reassess animal's condition; if improving, continue therapy; if not improving, reevaluate and consider changing antibiotics.

Duration of Antibiotic Administration

Duration depends on effect of antibiotic, toxicity, and disorder being treated.

The success of antibiotic therapy is assessed by observing the patient's response for the first 2 to 3 days of therapy. If the animal's condition has not improved by then, the antimicrobial therapy must be questioned. One should investigate whether the original diagnosis is correct; the culture and sensitivity results are accurate; the pathogen is susceptible to the antibiotic; the proper dosage, route, and frequency are being used; a foreign body or undrained focus of infection is present; a new infection is superimposed on the original infection; and/or the host's defense mechanisms are compromised. With most surgical infections, antibiotic therapy needs adjunctive therapy to be effective. This may mean drainage of accumulations of serum, pus, or blood from surgical wounds or body cavities; concurrent debridement of necrotic tissue; continued lavage of infected wounds; removal of foreign bodies or infected implants; removal of urinary calculi; removal of pus from an abdominal abscess; debridement of chronic osteomyelitis; or drainage of suppurative arthritis.

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10

Nutritional Management of the Surgical Patient



Care of the surgical patient does not end when the procedure is finished. Postoperative care of surgical patients impacts the ultimate outcome; it may even determine survival with critical patients. Postoperative care involves monitoring vital signs, normalizing homeostasis, controlling pain (see Chapter 13), and recognizing complications early. Early recognition of potentially catastrophic conditions facilitates treatment and outcome. Recommendations for postoperative care are included throughout this textbook, with information on the management of specific diseases included in the relevant sections on postoperative care and assessment.

An important component of postoperative care is nutritional support of debilitated or hyporexic patients. Malnutrition is the progressive loss of lean body mass and adipose tissue because of inadequate intake of or increased demand for protein and calories. Possible consequences of protein-calorie malnutrition (PCM) are organ and muscle atrophy, impaired immunocompetence, ineffective wound healing, anemia, hypoproteinemia, diminished resistance to infection, and death. For these reasons, patients with PCM require nutrient supplementation.

Many conditions cause PCM, including starvation, hyporexia, malabsorption syndromes, severe trauma, surgical stress, sepsis, large surface area burns, and various malignancies. Surgery, postoperative complications, and surgically induced hyporexia also increase metabolic demand for protein and calories. PCM shows no age, sex, or breed predisposition; it is common in severely ill animals, with an incidence ranging from 25% to 65%.

Diagnosis of PCM is possible if three or more of the criteria listed in Box 10.1 are present. Physical examination may reveal poor hair coat, pressure sores or wounds that will not heal, tissue wasting, skeletal muscle atrophy, and/or emaciation. Additional physical findings depend on the cause of malnutrition. Thoracic and abdominal radiographs of malnourished patients are generally nonspecific. Imaging techniques occasionally reveal an underlying cause for the patient's hyporexia or emaciation (e.g., intestinal obstruction, abdominal or thoracic mass). Biochemical changes with PCM may include hypoproteinemia, anemia, hypoglycemia, hyperglycemia, and/or hyperlipidemia. Other changes may be related to the underlying disease.

Nutritional supplementation should not be limited to malnourished patients. Acutely ill patients with severe systemic disease (e.g., peritonitis) are unlikely to eat in the postoperative period. Inadequate food intake in critical patients causes catabolism of lean muscle tissue and leads to an acute malnourished state, as described previously. Patients that are mechanically unable to eat (e.g., maxillary/mandibular fractures, oropharyngeal tumors) should also be evaluated for methods to provide adequate nutrition.

Hyperalimentation is the administration of adequate nutrients to malnourished patients or those at risk of malnutrition. *Enteral hyperalimentation* provides nutrients to a functional gastrointestinal tract by means of a nasoesophageal, pharyngostomy, esophagostomy, gastrostomy, or enterostomy tube. *Parenteral hyperalimentation* provides nutrients intravenously; *total parenteral nutrition* (TPN) provides 100% of an animal's nutrient, protein, and caloric requirements; and *partial parenteral nutrition* provides only part of an animal's nutritional needs. In small animal medicine, *parenteral nutrition* (PN) solutions are rarely formulated to fulfill all essential nutrient requirements. Therefore the terms *central parenteral nutrition* (CPN) and *peripheral parenteral nutrition* (PPN) have been generally accepted to reflect the route that PN is administered.

Specific treatment depends on the patient's calculated energy needs, dietary formula chosen, and route of administration (i.e., enteral or parenteral). *Basal energy requirement* (BER) (also called *resting energy requirement*) is based on body weight. Traditionally, *maintenance energy requirement* is then determined by multiplying the BER by an arbitrary factor (Fig. 10.1) to accommodate for the presumptive increase in metabolism associated with the severity of the clinical problem (e.g., cage rest, postsurgical stress, trauma, cancer, sepsis, major burn). Recently, a more conservative approach of starting with BER is advocated to avoid overfeeding and subsequent complications. Such complications include hyperglycemia, gastrointestinal upset, hepatic dysfunction, and increased carbon dioxide production.

DIETS FOR ENTERAL USE

The ideal enteral dietary formula should be well tolerated, easily digested and absorbed, contain essential nutrients, be readily available and inexpensive, have a long shelf life, and be easy to use. The most cost-effective, well-balanced diets for enteral administration are those blenderized from prescription pet food or homemade diets. Caloric density and protein content vary with the diet chosen. Examples of blenderized diets and their compositions are given in Table 10.1. Blenderized diets can be administered through tubes with a diameter of size 8 French (Fr) or larger; a commercially available liquid diet is recommended when feeding through smaller diameter tubes (e.g., 5 Fr).

Liquid diets should be isotonic (i.e., approximately 300 mOsm/L), have a caloric density of approximately 1 kcal/mL, include fiber at 1 to 1.5 g/100 kcal, provide approximately 16% of total calories as protein (i.e., protein content of at least 4 g/100 kcal), and approximately 30% as fat. Liquid diets for enteral use are generally categorized as monomeric (elemental) or polymeric. *Monomeric diets* use small-molecular-weight compounds for nutrients such as crystalline amino acids as the protein source, glucose and oligosaccharides as the carbohydrate source, and medium- or long-chain triglycerides as the essential fatty acid source. These diets generally have approximately twice normal osmolality and are primarily used in patients with malabsorptive gastrointestinal disorders (e.g., short bowel

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ABSTRACT

Providing nutrition to surgical patients that are debilitated or hyporexic may influence survival of these critical patients; however, nutritional supplementation should not be restricted to these patients. Acutely ill animals who are unlikely to eat in the postoperative period should also have adequate nutrition provided.

KEYWORDS

parenteral nutrition
total parenteral nutrition
partial parenteral nutrition
peripheral parenteral nutrition
enteral nutrition
mirtazapine
ghlerin receptor agonist
nasoesophageal tube
nasogastric tube
esophagostomy tube
gastrostomy tube
enterostomy tube

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BOX 10.1 Diagnosis of Protein-Calorie Malnutrition[a]

- Weight loss of more than 10% normal body weight
- Anorexia or hyporexia (i.e., suboptimal intake of nutrients) for >5 days or an expected decrease in nutrient intake of >5 days
- Increased nutrient loss (e.g., through vomiting, diarrhea, severe wounds, or burns)
- Increased nutrient needs (e.g., due to trauma, surgery, infection, burns, or fever)
- History of chronic illness
- Serum albumin concentration ≤2.5 g/dL

[a]These are findings that suggest protein-calorie malnutrition (PCM). The more of these findings that are present, the more likely it is that PCM is present. However, not all of these will be found in a patient with PCM, and not every patient with one of these findings will have PCM.

syndrome, severe inflammatory bowel disease). A commonly used commercially available monomeric elemental diet for humans is Vivonex T.E.N. (Nestle Health Science; Table 10.2). Vivonex is low fat and lactose free, but it should only be used in dogs for short-term treatment due to protein levels that are too low to support the nitrogen and amino acid requirements of dogs and cats.

Polymeric enteral diets contain complex large-molecular-weight proteins, carbohydrates, and fats. These diets approach isotonic osmolality, require normal gastrointestinal digestive processes, supply approximately 1 kcal/mL, and are more economical than monomeric diets. They include blenderized diets, commercially available partially hydrolyzed diets, and commercially available liquid diets (see Tables 10.1 and 10.2). Commercial polymeric

TABLE 10.1 Blenderized Diets for Dogs and Cats

Homemade Diets

Liquid Diet for Dogs

Ingredients	*Nutrient Availability*
1 jar (2.5 oz) baby food	1 kcal/mL
1 cooked egg	
15 mL corn oil	
15 mL corn syrup	
100 mL water	

Liquid Diets for Cats

Ingredients	*Nutrient Availability*
Equal parts egg yolk, strained baby food, and water	1.1 kcal/mL
3 oz egg yolk	1.5 kcal/mL
3 oz strained baby food	
3 oz water	
1 tsp cooking oil	
1 T corn syrup	

Prescription Diets

Product	*Amount*	*Amount of Water Added*	*Protein Content*		*Fat Content*	
			(kcal/mL)	*(g/100 kcal)*	*(g/mL)*	*(g/100 kcal)*
Feline a/d	1 can[a]	none	1.2	8.75	0.105	5.5
	1 can[a]	1 can[c]	0.6	4.38	0.053	2.75
Feline p/d	½ can[b]	¾ cup[d]	0.8	9.29	0.074	6.22
Feline k/d	½ can[b]	1¼ cup[e]	0.9	4.36	0.039	7.54
Feline c/d	½ can[b]	1¼ cup[e]	0.62	8.87	0.055	5.96
Canine a/d	1 can	None	1.2	8.75	0.105	5.5
	1 can	1 can	0.6	4.38	0.053	2.75
Canine k/d	½ can	1¼ cup[e]	0.62	3.06	0.019	5.29
Canine i/d	½ can	1¼ cup[e]	0.57	5.86	0.033	3.41

Preparation of Prescription and Homemade Diets

Mix the appropriate quantity of each ingredient (see chart).
Blend at high speed for 60 seconds.
Strain twice through kitchen strainer (1-mm mesh).

Advantages

Provides all required nutrients, low cost, appropriate protein and branched-chain amino acid content, normal stool consistency, manageable viscosity for 8 Fr catheter or larger.

Disadvantages

Cannot be used if the lumen diameter of the feeding tube is smaller than 8 Fr.

[a]1 can is equal to 156 g.
[b]½ can is equal to 224 g.
[c]1 can is equal to 156 mL.
[d]¾ cup is equal to 170 mL.
[e]1¼ cup is equal to 284 mL.
Modified from Zsombor-Murray E, Freeman LM. Peripheral parenteral nutrition. *Compend Cont Educ Pract Vet.* 1999;21:512–523.

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Calculate basal energy requirement (BER)

Animals less than 2 kg body weight:

$$70 \times (\text{body weight (kg)})^{0.75} = \text{BER (kcal/day)}$$

Animals more than 2 kg body weight:

$$30 \times \text{body weight (kg)} + 70 = \text{BER (kcal/day)}$$

Calculate maintenance energy requirement (MER)

Associated clinical problems	Factor
Cage rest	1.00 - 1.25
Postsurgical stress	1.25 - 1.35
Trauma or cancer	1.35 - 1.50
Sepsis	1.50 - 1.70
Major burns	1.70 - 2.00

$$\text{factor chosen} \times \text{BER (kcal/day)} = \text{MER (kcal/day)}$$

Calculate volume of formula required (See Tables 10.1 and 10.2 for formula composition.)

$$\text{MER (kcal/day)} \div \text{kcal/mL} = \text{mL of formula per day}$$

name of formula chosen

Calculate protein requirement

Species	Maintenance	Hepatic or renal failure
Canine:	5.0 - 7.5 g/100 kcal	< 3.0 g/100 kcal
Feline:	6.0 - 9.0 g/100 kcal	< 4.0 g/100 kcal

$$\text{protein requirement chosen g/100 kcal} \times \text{MER (100 kcal/day)} = \text{protein requirement (g/day)}$$

Calculate needed protein supplementation if required

name of formula chosen

$$\text{protein content (g/mL)} \times \text{mL of formula per day} = \text{protein provided (g/day)}$$

$$\text{protein requirement (g/day)} - \text{protein provided (g/day)} = \text{supplemental protein required (g/day)} \div 0.76 \text{ g protein per g ProMod} = \text{grams of ProMod per day}$$

FIG. 10.1 Formulas used to calculate basal and maintenance energy requirements for dogs and cats.

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TABLE 10.2 Commercially Available Diets and Their Composition[a]

Product	Caloric Content (kcal/mL)	PROTEIN CONTENT (g/100 kcal)	PROTEIN CONTENT (g/mL)	Fat Content (g/100 kcal)	Osmolality (mOsm/kg)
Polymeric Diets					
Clinicare canine/feline	1.00	8.2	0.082	4.6	340
Monomeric Diets[b]					
Vivonex RTF	1.00	5.0	0..05	1.16	630
Vital HN	1.00	4.7	0.042	1.08	500

[a]These figures should be used for the energy requirement calculations in Fig. 10.1.
[b]There are no monomeric or elemental liquid diets available for dogs and cats. The products listed are for human use and, although they might be indicated for severe gastrointestinal disease or demonstrated intolerance of a polymeric diet, they lack essential nutrients for dogs and cats and should only be used short-term or they require supplementation. They are rarely used in veterinary medicine.

diets are available in a variety of osmolalities, caloric densities, and compositions (see Table 10.2). These diets are indicated for malnourished patients with intact digestive and absorptive functions or those suspected of having food allergies. These diets also should be used for patients that must be fed through small-diameter tubes, such as nasoesophageal, gastroduodenostomy, or enterostomy tubes. Polymeric liquid enteral diets have been shown to be effective for nutritional support in critically ill and injured animals.

DIETS FOR PARENTERAL NUTRITION

Diets available for CPN should be customized to meet an animal's protein, carbohydrate, and fat requirements; caloric needs are calculated as described in Fig. 10.1. A common composition is 8.5% amino acids with electrolytes (protein source), 10% to 20% lipids (fat source), and 50% dextrose (carbohydrate source). B-complex vitamins are added at 1 to 2 mL/L.

Diets for PPN are made with the same ingredients used for CPN except that the osmolarity of PPN solutions is diluted by using sterile water or by reducing the amount of dextrose in order to prevent thrombophlebitis. As a result, the caloric density of these solutions is reduced.

The clinical use of ready-made PN solutions in veterinary medicine has recently been described. These formulas do not require compounding and tend to be less expensive. Vamin 9 (Fresenius Kabi) is a lipid-free formula that was evaluated in 70 dogs.[1] Metabolic complications were frequent, but only hyperkalemia was associated with a negative outcome. Mechanical complications were common when this solution was administered through a peripheral vein. Procalamine is a lipid-free, moderately hyperosmolar solution that was evaluated in 36 dogs.[2] Hyperkalemia was not noted in this study; however, other electrolyte disturbances were common, particularly hyponatremia and hyperglycemia.

In vitro studies have shown that PN formulas do not directly contribute to colloid oncotic pressure; however, PN has been shown to positively affect nitrogen balance, wound healing, and the patient's recovery. Possible problems include catheter management (i.e., sterile placement, maintaining sterility of the catheter entrance, and routine changing of infusion sets), expensive equipment (i.e., infusion pump), expensive feeding formulas, technical problems (i.e., routine patient monitoring during administration, proper diet preparation, and storage), metabolic complications, and sepsis. Hyperglycemia was the most common metabolic complication noted in one veterinary study.[3] In addition, if the gastrointestinal tract is not adequately stimulated by luminal nutrients and hormonal or neurovascular mechanisms, the intestines and pancreas may atrophy. Intestinal mucosal compromise predisposes the intestinal mucosa to bacterial translocation into the portal circulation and could theoretically lead to sepsis. In general, these problems make parenteral hyperalimentation much less desirable than enteral hyperalimentation.

BOX 10.2 Drugs Used as Appetite Stimulants

Mirtazapine (Remeron)[a]
Cats: 1.9 mg/cat PO q24h: range is 1.875–3.75 mg PO q24–48h
Dogs: 0.5 mg/kg PO q24h: this usually equates to 3.75–7.5 mg PO q24h

Cyproheptadine (Periactin)
Cats: 2 mg/cat PO

Capromorelin (Entyce)
Dogs: 3 mg/kg PO qd for 4 d
Cats: FDA approval expected in 2018[b]

[a]Preferred pharmacologic appetite stimulant in cats and dogs. This drug may have dangerous interactions with other drugs, especially those that can cause a "serotonin storm" such as cytochrome P450 inhibitors (ketoconazole, cimetidine, macrolides), furazolidone, monoamine oxidase inhibitors, amitriptyline, and warfarin.
[b]Emesis, hypersalivation, lethargy/depression, head shaking, and lip smacking may be seen in cats after administration, but these signs generally resolve within 5 minutes.
PO, Orally; *q,* every.

ENHANCING APPETITE

Oral feeding is preferable to parenteral nutrition if adequate nutrients can be consumed to meet protein and caloric requirements. Several techniques have been used successfully to encourage an animal to eat. If the owners can manage the patient at home, the likelihood of eating is higher in a familiar environment. Petting and vocal reassurance are also helpful, albeit time consuming. Highly palatable foods or food coverings, such as gravy, may stimulate the appetite, and warming foods increases aroma and palatability. Supplementing potassium (i.e., 2–44 mEq/day potassium gluconate for dogs, depending on size; 2–5 mEq/day for cats), vitamin B complex (in maintenance fluids), and/or zinc may also increase appetite. Pharmacologic appetite stimulants are available; currently only mirtazapine and cyproheptadine are recommended for use (Box 10.2).[4] Cyproheptadine stimulates appetite through antagonism of serotonin (5-HT2) receptors in the ventromedial hypothalamus. It is metabolized in the liver and therefore not recommended for cats with hepatic lipidosis. Mirtazapine antagonizes presynaptic alpha-2 receptors,

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causing an increase in norepinephrine, but also antagonizes 5-HT2 receptors. Mirtazapine has been shown to be an effective appetite stimulant and antiemetic in cats with chronic kidney disease.[5] Ghrelin receptor agonists (e.g., capromorelin) have recently been approved as appetite stimulants in dogs. Initial observations suggest they are usually effective.

METHODS OF PROVIDING HYPERALIMENTATION

Parenteral Nutrition

For placement of CPN catheters, patients should usually be sedated or anesthetized.

Insert a 16-gauge, 18-cm, single- or double-lumen silicone elastomer catheter into the right or left external jugular vein. Position the catheter tip in the cranial vena cava and create a subcutaneous tunnel such that the catheter hub emerges on the dorsum of the neck. Anchor the catheter to the vein, to subcutaneous tissue along the tunnel, and to skin at the exit point with 4-0 to 5-0 nonabsorbable monofilament suture. Attach an extension set to the catheter hub, and bandage the catheter in place with sterile gauze, cast padding, and self-adherent wrap. After each use flush the catheter with heparinized saline (0.9% sterile saline with 1 IU/mL heparin). Remove the neck bandage every 2 days, clean the catheter entrance site with povidone-iodine or chlorhexidine solution, change the administration set, and apply a new bandage.

NOTE Designate the catheter as a dedicated-use or sole-use catheter and do not use it for obtaining blood samples or administering other medications unless there are severe extenuating circumstances.

PPN is administered through a catheter in a small peripheral vein. Although a dedicated catheter is ideal, it is not as critical with PPN as it is with CPN. PPN is generally administered short term (e.g., <5 days) to nondebilitated patients that are unable to tolerate full enteral feeding.

Enteral Hyperalimentation

Enteral hyperalimentation is practical, safe, easy, economical, physiologic, well tolerated, and has minimal morbidity in patients with a functional gastrointestinal tract. Enteral hyperalimentation is indicated in any animal with overt or impending PCM. Such patients include those that are hypermetabolic (e.g., patients with severe burns, sepsis, postsurgical stress, trauma, or cancer) and those with chronic hyporexia or malnutrition, as evidenced by a greater than 10% loss of normal body weight. Enteral hyperalimentation can also be used whenever 3 to 5 days of anorexia are anticipated, such as after oral, pharyngeal, esophagogastric, duodenal, pancreatic, or biliary tract surgery; during postoperative management of septic or cancer patients (particularly if chemotherapy is instituted); and when the patient's mental status prevents self-feeding, such as with head trauma or after brain surgery.

Although enteral hyperalimentation is desirable for most patients with actual or pending PCM, infusion of nutrients into the intestines may be ineffective or even contraindicated in patients with small bowel obstruction that cannot be resolved and those with very severe and persistent adynamic ileus. In these patients, enteral feeding may worsen vomiting or diarrhea. Parenteral nutrition should be considered in such instances.

In general, oral alimentation is more efficient, easier, safer, and allows greater flexibility in formula composition. The further aboral materials are delivered, the less efficient the assimilation and digestion of nutrients and the greater the care necessary in choosing formula composition. Route of administration dictates feeding tube diameter (Box 10.3), which in turn dictates the usable feeding formulas. The most common routes of administration for enteral hyperalimentation are nasoesophageal, nasogastric, esophagostomy, gastrostomy, gastroduodenostomy, and enterostomy tubes. The need for pharyngostomy tubes have been largely replaced by esophagostomy tubes. Each route has its indications, contraindications, advantages, disadvantages, and complications.

BOX 10.3 Tube Diameter Used Based on Route of Administration of Enteral Diets

5 Fr Tube Diameter
- Nasoesophageal (cats, dogs <15 kg)
- Enterostomy (cats, dogs <15 kg)

8 Fr Tube Diameter
- Nasoesophageal (dogs >15 kg)
- Enterostomy (dogs >15 kg)

16 Fr or Larger Tube Diameter
- Pharyngostomy
- Esophagostomy
- Gastrostomy

BOX 10.4 Recommended Tube Sizes for Nasoesophageal Placement

Cats, dogs <15 kg: 5 Fr × 91 cm
Dogs >15 kg: 8 Fr × 91 cm

Nasoesophageal and Nasogastric Intubation

Nasoesophageal (NE) and nasogastric (NG) tubes are a primary means of short-term enteral nutrition in most veterinary patients. NE and NG intubation is easy, effective, and efficient. The availability of small-bore, soft rubber (polyvinyl chloride), and Silastic feeding tubes (i.e., 5 Fr) and of low-viscosity, nutritionally complete liquid diet formulations (see Table 10.1), in addition to patient tolerance of tube placement, have made NE and NG feeding popular. Advantages of NE and NG tubes include ease of placement, acceptance by most patients, ease of tube care and feeding, patients' ability to eat and drink around the tube, and flexibility that allows tube removal an any time after placement. In addition, NG tubes provide a means for gastric decompression. The major disadvantages of NE and NG tubes are the small tube size, inadvertent tracheal placement, and premature removal by the patient. Feeding can be accomplished through continuous rate infusion or bolus feedings because no difference has been shown with the incidence of gastrointestinal or mechanical complications. A patient may occasionally vomit out or regurgitate the tube. Light general anesthesia may be necessary to place these tubes, but a topical anesthetic or light sedation is usually sufficient.

Instill proparacaine hydrochloride (0.5–1 mL; 0.5%) into the nasal cavity and elevate the head to encourage the anesthetic to coat the nasal mucosa (Video 10.1). Repeat the application to ensure adequate anesthesia of nasal mucous membranes. If the patient will not tolerate nasal intubation, consider topical lidocaine (e.g., 1–2 mL of 2% lidocaine), heavy sedation, or light general anesthesia. Select an appropriate-sized feeding tube (Box 10.4). For NE tubes, estimate the length of tube to be placed in the esophagus by measuring the tube from the nasal planum, along the patient's side, to the seventh

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FIG. 10.2 Direct the nasoesophageal tube from ventrolateral in the external nares to caudoventral and medial as the tube enters the nasal cavity.

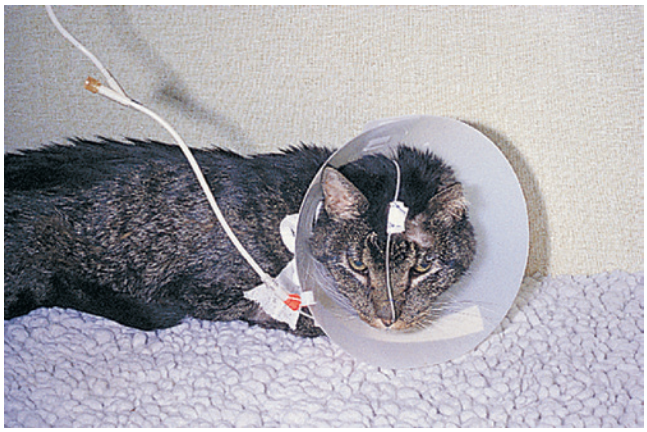


FIG. 10.3 In cats, secure nasoesophageal feeding tubes to the dorsal nasal midline and forehead.

or eighth intercostal space. For NG tubes, measure the tube from the nasal planum, along the patient's side, to the last rib. Place a tape marker on the tube once the appropriate measurement has been taken. Before passing the tip of the tube, lubricate it lightly with 5% viscous lidocaine and hold the patient's head in a normal functional position (i.e., avoid hyperflexion or hyperextension). Identify the prominent alar fold and direct the tube from a ventrolateral location in the external nares to a caudoventral and medial direction as it enters the nasal cavity (Fig. 10.2). When the tube has been introduced 2 to 3 cm into the nostril, contact with the median septum at the floor of the nasal cavity can be felt. *Push the external nares dorsally to facilitate opening of the ventral meatus. Elevate the proximal end of the tube and advance it into the oropharynx and esophagus.* It will generally "drop" into the oropharynx and stimulate a swallowing reflex. Several methods can be used to confirm placement in the esophagus or stomach: (1) check for negative pressure, (2) inject 3 to 5 mL of sterile saline through the tube and see if a cough is elicited, (3) inject 6 to 12 mL of air and auscultate for borborygmus at the xiphoid, (4) connect the tube to a capnograph, (5) check the pH of aspirated fluid, or (6) confirm tube placement using a lateral thoracic radiograph. If the patient requires general anesthesia, correct tube placement can be visually confirmed using a laryngoscope. *Once satisfied that the tube has been properly placed, suture it to the nose and head to prevent removal by the patient using 3-0 monofilament nonabsorbable suture (e.g., nylon). In cats, it is important that the tube is not in contact with the whiskers; position it directly over the dorsal aspect of the nose and forehead (Fig. 10.3), and secure it with a Chinese finger-trap friction suture (see p. 924). In dogs, secure the tube to the lateral aspect of the nose and dorsal nasal midline with a Chinese finger-trap friction suture or cyanoacrylate glue. Place a column of water in the tube before capping it to prevent air intake, reflux of esophageal contents, or occlusion of the tube by diet.*

NOTE Use an Elizabethan collar immediately after surgery until it is determined if the patient will tolerate the NE or NG feeding tube.

When comparing the use of NE versus NG tubes in dogs, NE tubes tended to be implemented in younger dogs.[6] There was no difference in the overall low complication rate between NE and NG tubes.

Nasojejunal Tubes

Direct intestinal feeding can be accomplished through placement of long, weighted nasojejunal tubes for patients where gastric feeding is contraindicated or poorly tolerated. Fluoroscopy or endoscopy is required to ensure placement of the end of the tube beyond the duodenal flexure. As the tubes are slightly larger than what would be used for NG tubes, complications such as sneezing, nasal discharge, and vomiting are common.

Esophagostomy Tubes

Esophagostomy tubes are now the primary means of long-term enteral feeding in most veterinary patients. Esophagostomy feeding is indicated in hyporexic patients with disorders of the oral cavity or pharynx and those with a functional gastrointestinal tract distal to the esophagus. These tubes are contraindicated in patients with primary or secondary esophageal dysfunction, such as with esophageal stricture, postesophageal surgery, removal of an esophageal foreign body, or with esophagitis or megaesophagus. Advantages of esophagostomy tubes include ease of placement, acceptance by patients, placement of large-bore tubes that allow blenderized diets, ease of tube care and feeding, patients' ability to eat and drink around the tube, and flexibility that allows tube removal any time after placement. Esophageal tube placement eliminates the coughing, laryngospasm, partial airway obstruction, and aspiration that can be associated with improperly placed pharyngostomy tubes. The major disadvantage of esophagostomy tubes is the need for general anesthesia for tube placement.

Anesthetize the animal and place it in right lateral recumbency (left side uppermost) (Video 10.2). The tube may be placed on either the right or left side of the midcervical region; however, the esophagus lies left of the midline, making left-sided placement more desirable. *Prepare the midcervical area from the angle of the mandible to the thoracic inlet for aseptic surgery. Premeasure a 16 to 24 Fr polyvinyl chloride feeding tube from its insertion point to the level of the seventh or eighth intercostal space (ensuring mid-esophageal to caudal-esophageal placement), and mark it. Alternatively, have an additional tube of identical length available to assess length of placement of the original tube. Enlarge one lateral opening of the feeding tube to encourage smoother flow of a blended diet (Fig. 10.4) or cut the end of the tube off at an oblique angle.* A curved Rochester-Carmalt is used for esophagostomy tube placement in

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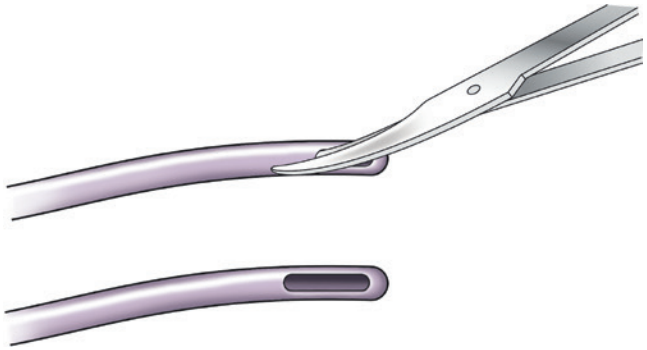


FIG. 10.4 For esophagostomy tube, enlarge the lateral openings of the tubes 3 to 4 mm to encourage a smooth flow of blended diets.

cats and small to medium sized dogs; however, an ELD feeding tube placement device (Fig. 10.5) or similar product can be used in large dogs. *Place the curved tip of the forceps through the oral cavity into the esophagus to the level of the midcervical region (i.e., equidistant from the angle of the mandible and the point of the shoulder). Palpate the tip as it bulges through the cervical skin. Make a small incision over the tip and push the forceps through the esophageal wall, subcutaneous tissue, and skin. Grasp the tube with the forceps, apply a small amount of lubricant, and pull the tube through the incision and out the oral cavity. Redirect the tube down the esophagus using forceps, a stylet, or digital manipulation. Confirm length of placement by location of the premarked area on the tube or by comparing to a tube of identical length. Secure the tube to the cervical skin using a Chinese finger-trap friction suture with 2-0 monofilament nonabsorbable suture (e.g., nylon). Bandage the tube loosely around the neck. Place a column of water in the tube, and cap the exposed end.*

Alternatively, for large dogs, use an ELD device instead of Rochester-Carmalt forceps. The ELD is placed through the oral cavity into the midcervical area of the esophagus. A small skin incision is made over the ELD tip as it protrudes through the skin. The spring-loaded blade is activated by pushing the plunger at the end of the handle. The trocar should be visible through the skin incision, but the tissues around it can be carefully dissected using a scalpel blade such that the entire shaft of the instrument protrudes through the incision (Fig. 10.6A and B). *Place a 2-0 nonabsorbable suture through the side holes of the feeding tube and the hole in the instrument trocar. Tighten the suture until the tip of the instrument blade and the tip of the feeding tube are in close apposition (see Fig. 10.6C). Retract the blade into the instrument shaft so that the tip of the feeding tube enters the instrument shaft (i.e., deactivating the instrument blade), and lubricate the tube and the instrument shaft. Retract the instrument and pull the feeding tube into the oral cavity to its predetermined measurement (Fig. 10.7A). Remove the suture to free the feeding tube from the instrument blade and, in dogs, place a stylet through one of the side holes of the feeding tube and against its tip (see Fig. 10.7B). Lubricate the tube and advance it into the esophagus until the entire oral portion of the feeding tube disappears (see Fig. 10.7C) and the tube passes down the esophagus without twisting or bending. Carefully retract the stylet from the oral cavity to ensure its release from the tube. Secure the tube to the cervical skin with a Chinese finger-trap friction suture with 2-0 monofilament nonabsorbable suture (e.g., nylon; see p. 924). Bandage the tube loosely around the neck. Place a column of water in the tube, and cap the exposed end.*

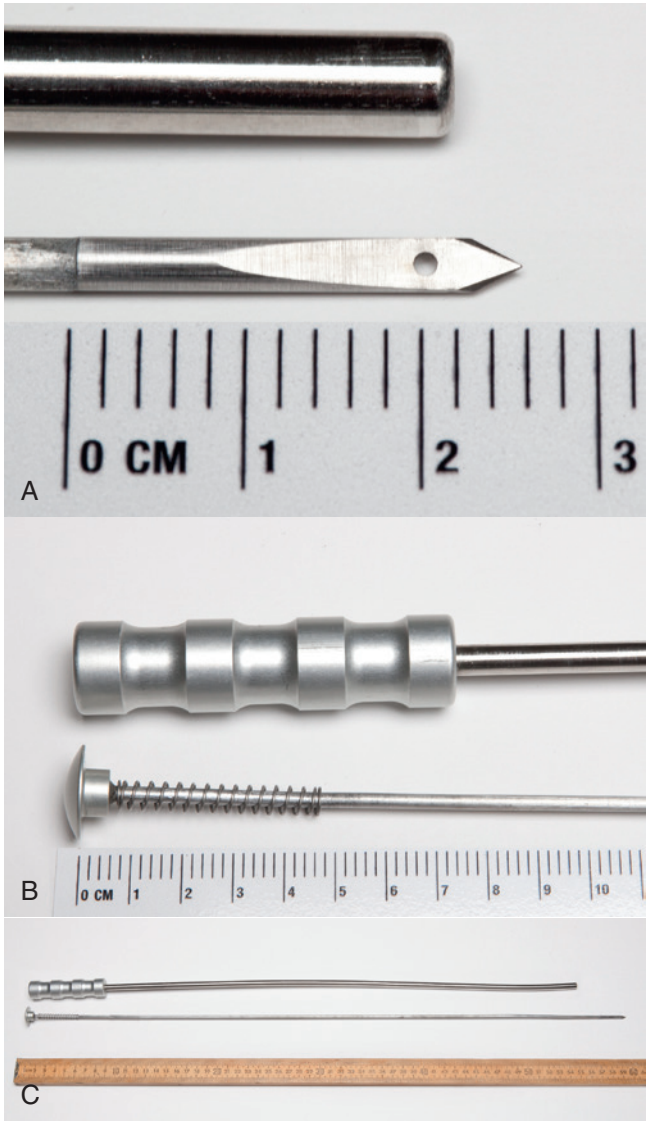


FIG. 10.5 (A) ELD feeding tube placement device and stylet. (B) Activated spring-loaded blade *(top)* and stylet *(bottom)*. (C) Complete trochar and cannula, disassembled, with a ruler to show length. (From Devitt CM, Seim HB III. Clinical evaluation of tube esophagostomy in small animals. *J Am Anim Hosp Assoc.* 1997;33:55.)

A one-step technique for esophagostomy tube placement in cats has been described using a specific applicator (von Werthen esophageal feeding tube applicator; B. Braun Vet Care) or using a large gauge, over-the-needle catheter. The disadvantage of the latter technique is the small diameter of the tube that can be placed by this method.

NOTE In dogs and cats, a small diameter, rigid endoscope can be used to direct the tube into the esophagus and ensure that there are no kinks or other problems. A lateral thoracic radiograph can also confirm appropriate placement.

Most patients tolerate esophagostomy tubes, and Elizabethan collars are seldom necessary. Esophagostomy tubes can be safely removed immediately after placement or can be left in place for several weeks or months. The tube exit site may require light

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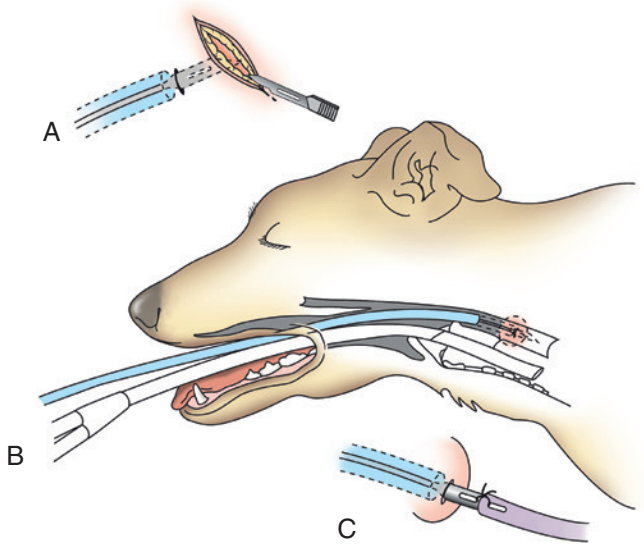


FIG. 10.6 Esophageal tube placement. (A) Place the instrument shaft in the oral cavity and palpate the tip as it bulges the cervical skin; make an incision over the tip. (B) Activate the instrument blade until it is visible through the skin incision; enlarge the incision to allow penetration of the instrument shaft. (C) Use 2-0 nonabsorbable suture (e.g., nylon) to secure the tip of the feeding tube to the tip of the blade; retract the blade so that the tip of the feeding tube contacts the instrument shaft.

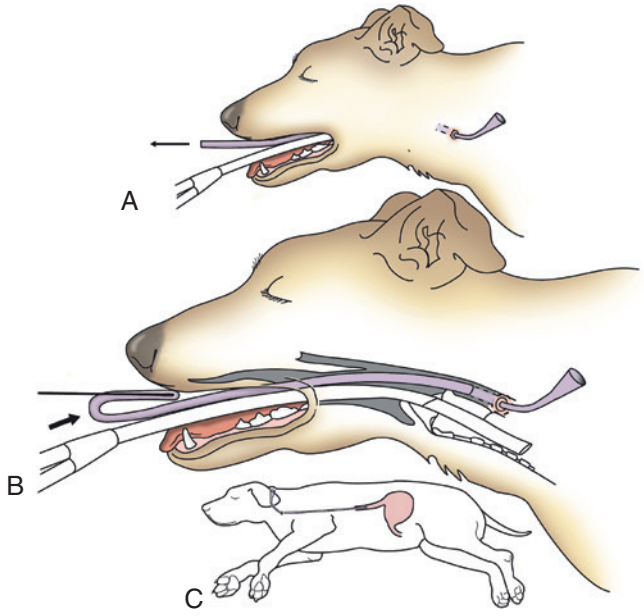


FIG. 10.7 Esophageal tube placement (continued from Fig. 10.6). (A) Retract the instrument and pull the feeding tube into the oral cavity. (B) In dogs, place a stylet into one of the lateral holes of the feeding tube and against its tip; advance the tube into the esophagus. (C) The esophageal tube should lie in the mid-esophageal region.

bandaging and periodic cleaning with an antiseptic solution. The tube is removed by cutting the finger-trap suture and gently pulling the tube through the skin. No further exit wound care is necessary; the hole seals in 1 or 2 days and is healed by 4 to 5 days.

Complications associated with esophagostomy tube placement include early removal by the patient or vomiting the tube up and chewing off the end. Esophageal perforation has occurred in cats but is unlikely if a stylet is not used. Esophageal perforation is not expected in dogs. Significant complications (e.g., jugular vein laceration, carotid artery laceration, esophagitis, esophageal stricture, esophageal diverticulum, subcutaneous cervical cellulitis) are rare. Reflux esophagitis can occur from improper tube placement (i.e., through the lower esophageal sphincter), or the tube itself can irritate esophageal tissue. Making sure that the distal tip of the soft rubber or Silastic tube is in the midesophageal region notably reduces the incidence of esophageal injury and reflux esophagitis.

Gastrostomy Tubes

Gastrostomy tubes are indicated in patients with a functional stomach and gastrointestinal tract that are anorexic, at risk for PCM, or are undergoing operations of the oral cavity, larynx, pharynx, or esophagus. These tubes are avoided in patients with primary gastric disease (e.g., gastritis, gastric ulceration, gastric neoplasia). Advantages of gastrostomy tubes include ease of placement, patient tolerance, availability of large-bore feeding tubes, ease of feeding and tube care, and that oral feeding can begin or continue while the gastrostomy tube is in place. Disadvantages include the need for general anesthesia and potentially specialized equipment (e.g., an endoscope), invasion of the peritoneal cavity, and inability to remove the tube for at least 7 to 14 days postplacement (i.e., the time needed to allow adhesion formation between the stomach and abdominal wall). Premature removal puts the animal at risk for peritonitis from gastric contents leaking into the abdomen. Very large dogs may be problematic as it can sometimes be difficult to achieve a good adhesion between the stomach and the abdominal wall when placing a gastrostomy tube with a device or an endoscope. In these circumstances, it may be best to place the gastrostomy tube surgically. In addition, tubes placed blindly using a large stomach tube or spring-loaded device can easily be malpositioned such that they are placed at the lower esophageal sphincter (resulting in vomiting and gastroesophageal reflux) or so far caudally that the stomach may pull off the tube when the stomach is filled with food. Rarely the tube may be associated with gastric bleeding. Low-profile gastrostomy ports are sometimes used in animals for which long-term nutritional management or medication administration is needed.

Methods of placement of gastrostomy tubes include blind (nonendoscopic) percutaneous gastrostomy, percutaneous endoscopic gastrostomy (PEG), or surgical placement through a left flank approach or midline laparotomy.

Percutaneous Endoscopic Gastrostomy Tube Placement

Advantages of endoscopic placement is that tube placement occurs under direct visualization, and gastric insufflation displaces omentum, mesentery, spleen, and small intestines, which decreases risk of iatrogenic damage. This technique may not be appropriate for larger animals (e.g., >25 kg) because it can be hard to ensure that an adequate adhesion will form between the stomach and the abdominal wall at the ostomy site. Recently it was shown that animals receiving a PEG tube while concurrently on corticosteroid therapy had a significantly higher rate of major complications when compared to animals with PEG tubes not on corticosteroid therapy.[7] There are various commercial kits available containing the necessary equipment for placing

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gastrostomy tubes. The general idea of placement (which may vary slightly, depending upon the kit) is as follows:

Place the animal under general anesthesia. Position the animal in right lateral recumbency and clip and prep the area caudal to the last rib. The animal is positioned in right lateral recumbency, and the area caudal to the last rib is clipped and aseptically prepped. Pass the endoscope into the stomach, and insufflate the stomach with air. Make a 1 mm skin incision in the left flank 1 to 2 cm caudal to the last rib and 2 to 3 cm distal to the transverse spinous processes of lumbar vertebrae two or three. Pass an 18-gauge needle through the skin incision and into the stomach lumen. Pass the strand of the suture material provided by the kit through the needle and into the stomach, retrieve it using endoscopic grasping forceps, and bring it out through the mouth. Once the strand of suture is entering the left flank and exiting the oral cavity, attach the suture to the feeding tube as directed by the kit instructions. In large dogs, a percutaneous suture of No. 1 monofilament nonabsorbable material can be placed from through the skin and abdominal wall, into the gastric lumen, back out of the skin, and tied under direct visualization with the endoscope. This keeps the stomach in apposition with the body wall so it is more likely for a strong adhesion to form, protecting the peritoneal cavity from leakage.

Blind Percutaneous Gastrostomy Tube Placement

This technique should only be performed after one has done several gastrostomy tube placements using endoscopic guidance to help better understand the anatomy of the stomach and configuration within the abdominal cavity. Advantages of this technique are that no special instrumentation is required for placement and it is easy to perform. Disadvantages are that the stomach is not surgically pexied to the body wall (early removal by the patient could result in peritonitis), novices can easily malposition the tube too far orad or too far caudad in the stomach, it is possible to penetrate and trap omentum, mesentery, or spleen, and tube placement must be confirmed endoscopically or radiographically.

Placement of the blind percutaneous gastrostomy tube is performed similarly to PEG tubes described earlier except that entrance of the hypodermic needle into the stomach is performed blindly. *Place the animal under general anesthesia. Position the animal in right lateral recumbency and clip and aseptically prepare the area caudal to the last rib. The animal is positioned in right lateral recumbency, and the area caudal to the last rib is clipped and aseptically prepped. Pass a stiff, large-bore stomach tube or feeding tube placement device (e.g., ELD; Fig. 10.8A) into the stomach until it can be palpated bulging against the left body wall 1 to 2 cm caudal to the last rib and 2 to 3 cm distal to the transverse processes of lumbar vertebrae two or three. If a stomach tube is used, pass an 18-gauge hypodermic needle through the skin into the lumen of the stomach tube. Place the suture strand through the needle, into the stomach tube, and out through the mouth. Remove the stomach tube. If a feeding tube placement device is used (see esophagostomy tube placement earlier), activate the device and thread the suture through the hole in the instrument blade (see Fig. 10.8B). Retract the blade into the instrument shaft and withdraw the instrument out through the mouth (see Fig. 10.8C).* In each case the suture enters the stomach through the left flank and exits through the oral cavity. *Attach the end of the suture to the gastrostomy tube. Pull the No. 1 suture exiting the left flank until the catheter tip exits the skin (see Fig. 10.8D). Enlarge the skin incision to 3 to 4 mm, allowing easy delivery of the catheter. Pull the catheter until the mushroom tip is snugly against the body wall, ensuring a seal between the stomach wall and the body wall (see Fig. 10.8E). Secure the tube to the skin with a Chinese finger-trap suture using 2-0 nonabsorbable suture (e.g., nylon). Alternatively, use the piece of tube previously cut off to create an external flange.*

Flank Incision Gastrostomy Tube Placement

Advantages of this technique include ease of tube placement, ease of finding the stomach in an anorexic patient, quick placement, no need for special equipment, creation of an initial seal between the stomach wall and the body wall, and confirmation of proper placement during the procedure. The disadvantage is lack of visualization of gastric anatomy, which may cause malpositioning of the tube.

Prepare the patient as described previously, but in addition, place quarter-towels and a fenestrated drape over the left flank to create a sterile field. Have a nonsterile assistant pass a large-bore, stiff plastic tube into the stomach. The surgeon, who is sterile, palpates the left flank area until the end of the stomach tube can be palpated and grasped (Fig. 10.9A). Manipulate the tube to a location 2 to 3 cm caudal to the thirteenth rib and 2 to 3 cm distal to the transverse processes of the lumbar vertebrae. Hold the tube stable and make a skin incision over the end of the stomach tube. Bluntly dissect the subcutaneous tissues and abdominal muscles to expose the wall of the stomach over the tube; take care not to enter the lumen of the stomach (see Fig. 10.9B). Place a purse-string suture in the stomach wall around the tube (see Fig. 10.9C). Use a No. 11 scalpel blade to make a stab incision into the lumen of the stomach in the middle of the purse-string suture; make the incision large enough to easily accommodate the selected tube. Place a 20 to 24 Fr Foley catheter into the lumen of the stomach and inflate the bulb (see Fig. 10.9D). Place traction on the purse-string suture and slowly withdraw the stiff stomach tube from the oral cavity. Place gentle traction on the Foley catheter to bring the inflated bulb against the stomach wall (see Fig. 10.9E). Snugly tie the purse-string suture around the Foley catheter. Place three to four simple interrupted 2-0 absorbable sutures (e.g., PDS or Maxon) from the stomach wall to the body wall to firmly pexy the stomach in place. Close the subcutaneous tissue and skin around the existing Foley catheter, push the Foley catheter 1 to 2 cm into the stomach lumen, and secure the Foley catheter to the skin with a Chinese finger-trap friction suture with 2-0 nonabsorbable suture (see Fig. 10.9F).

Gastrostomy Tube Placement by Midline Laparotomy

This procedure is generally performed when tube placement is ancillary to another abdominal procedure, such as biopsy or removal of an abdominal mass. Advantages to this method are that placement is directly visualized, and a secure surgical gastropexy can be performed. The disadvantage is that placement is more invasive when compared to the techniques listed previously due to the surgical approach.

Pass the distal end of a 20 Fr Foley or Pezzer catheter (i.e., bulb or mushroom tip) into the abdominal cavity through a stab incision in the left body wall. Exteriorize the stomach and place a purse-string suture in the ventrolateral wall of the body of the stomach. Make a stab incision in the center of the purse-string suture with a No. 11 scalpel blade and place the distal end of the feeding catheter in the lumen of the stomach. Tighten the purse-string suture around the catheter and inflate the catheter bulb (i.e., Foley catheter) with saline. Place gentle traction on the catheter to bring the body of the stomach in close apposition to the left body wall. Pexy the stomach wall to the abdominal wall with four 2-0 or 3-0 synthetic standard absorbable sutures (e.g., polydioxanone or polyglyconate). Secure the feeding tube to the skin with a Chinese finger-trap suture of 2-0 nonabsorbable suture (e.g., nylon). Close the abdomen routinely.

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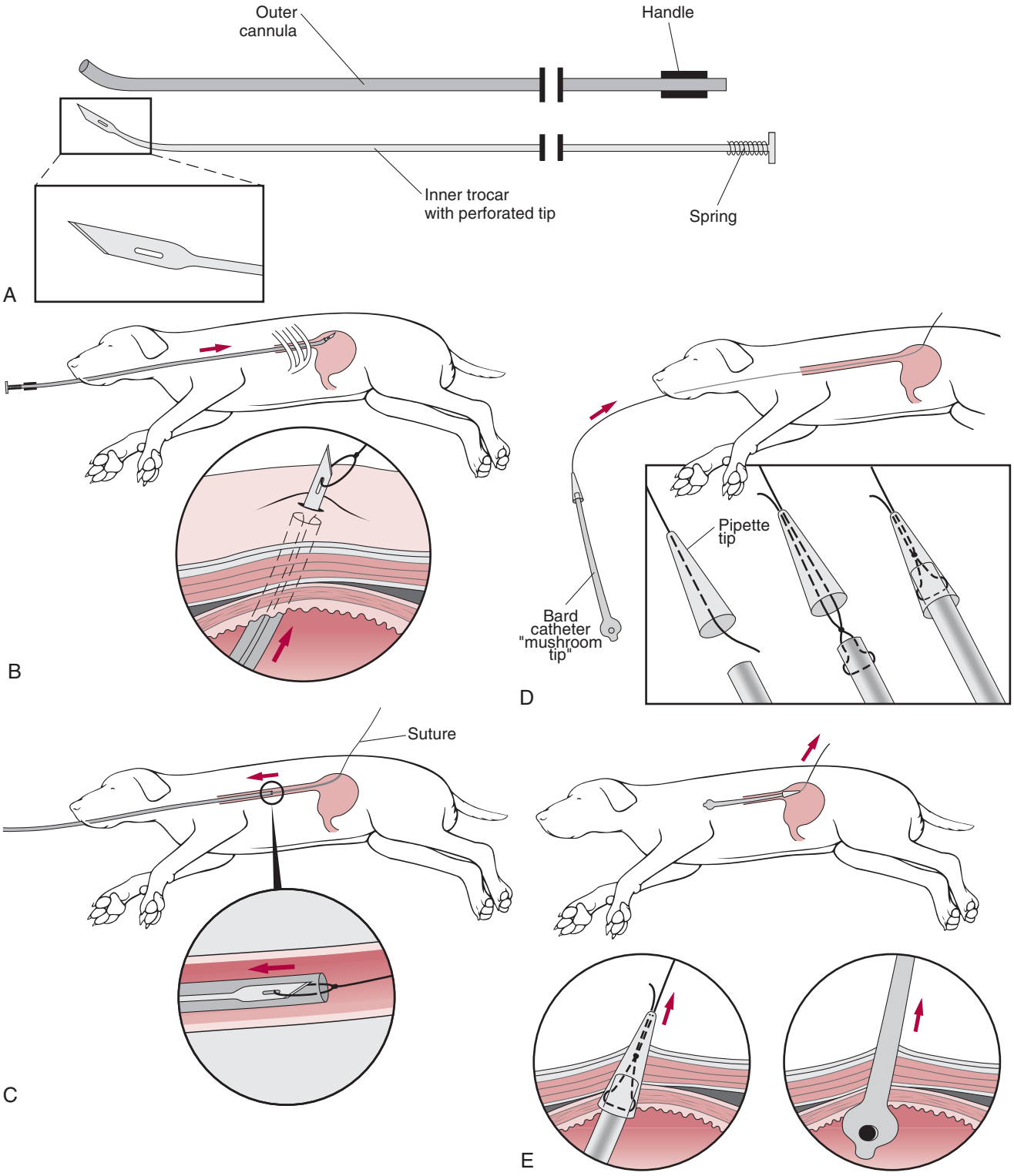


FIG. 10.8 (A) Schematic view of a device designed to place gastrostomy tubes without endoscopy. The trocar *(lower)* is placed through the cannula *(upper)*. The cutting tip *(inset)* is not extended until the device has been properly placed. (B) Schematic view showing how the device is placed in an animal receiving a gastrostomy tube. Note that the tip of the device ultimately arrives at a point 2 to 3 cm caudal to the last rib and 2 to 3 cm distal to the transverse processes of lumbar vertebrae two or three. Push the trocar tip through the cannula so that the blade tip extends through the skin *(insert),* and tie a suture to the tip. (C) Retract the entire device (cannula and trocar) through the mouth, bringing the suture with it. (D) Fasten the end of the suture that is brought out of the mouth to the tip of a mushroom-tip catheter. Note how a pipette tip is placed where the suture attaches to the tip of the catheter. (E) Pull the suture through the skin, pulling the mushroom-tip catheter into the stomach. The pipette tip facilitates passage of the catheter through the abdominal wall. Withdraw the catheter until the mushroom tip is against the gastric mucosa; the stomach is pulled snugly against the abdominal wall.

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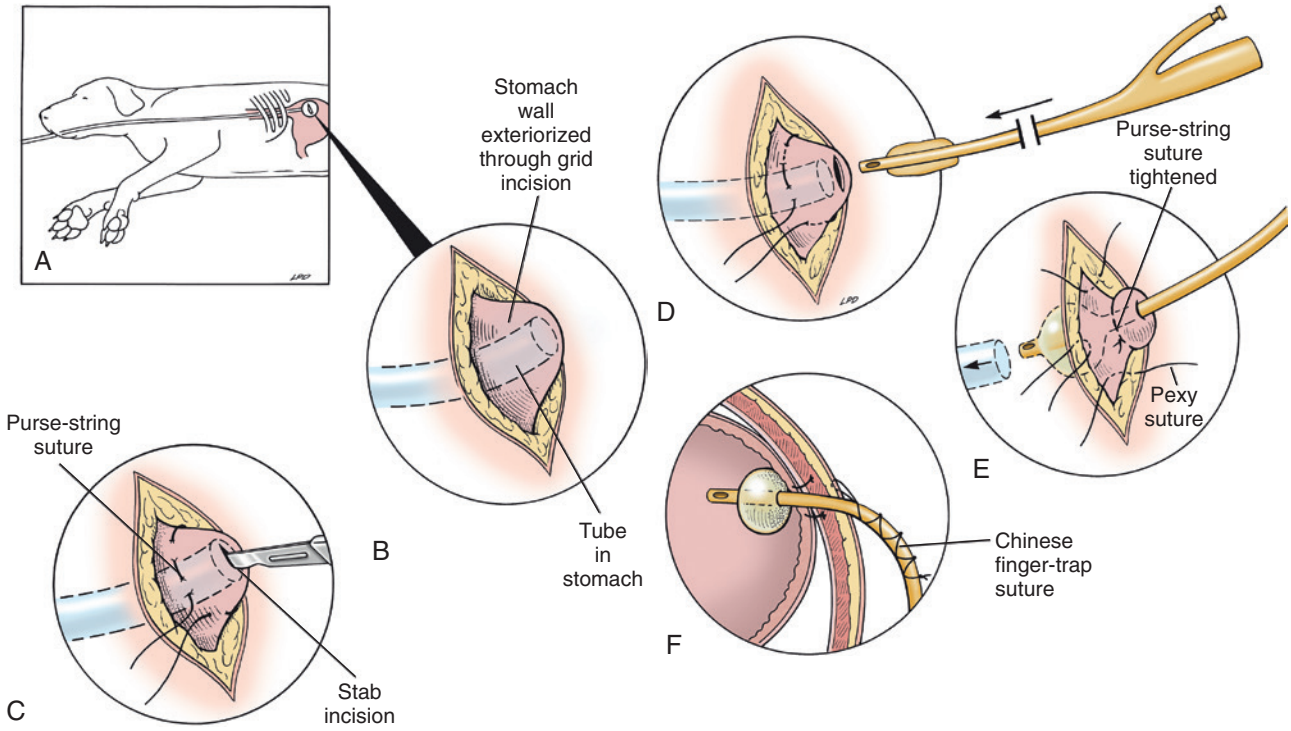


FIG. 10.9 Percutaneous gastrostomy tube placement with gastropexy. (A) Pass a large-bore, stiff plastic stomach tube into the stomach. Palpate the end of the tube at the flank. (B) Grasp the tube and move it to a point 2 to 3 cm caudal to the thirteenth rib and 2 to 3 cm distal to the transverse processes of the lumbar vertebrae. Secure the tube with thumb and finger, make an incision through the skin and subcutaneous tissue, and bluntly dissect the abdominal muscles to expose the gastric wall over the tube. (C) Place a purse-string suture in the gastric wall around the tube and puncture the wall with a scalpel blade. (D) Place the Foley or Pezzer catheter into the lumen of the stomach and into the tube. (E) Tighten the purse-string suture, remove the stomach tube, inflate the bulb of the Foley catheter, and suture the gastric wall to the abdominal wall. (F) Note the proper tube placement of the inflated Foley catheter, the gastropexy, and the Chinese finger-trap friction suture to secure the tube in place.

Low-Profile Gastrostomy Tube Devices

Various low-profile gastrostomy tube devices are available in different configurations and generally serve two main functions. First, some owners prefer to use them when a PEG tube will be required for a lengthy period because the low-profile devices do not have a long tube exiting the body, which makes them aesthetically more appealing. Tubes that do not "dangle" from the body may also be easier to care for (i.e., they are less likely to be pulled out or chewed out by the patient). Second, and perhaps the major value of low-profile devices, is that they allow quick and easy replacement of gastrostomy tubes that have inadvertently been pulled out or have so deteriorated that they must be replaced. The low-profile device is inserted into the stoma made by the first gastrostomy tube. However, these stomas close rapidly (i.e., <24 hours) after removal of the gastrostomy tube; therefore speed is of the essence. If the patient cannot be seen within several hours after removal of the first gastrostomy tube, a relatively large-diameter sterile red latex male urinary catheter or similar tube should be placed in the stoma to prevent it from closing down until the low-profile device can be placed.

NOTE If you need to replace a gastrostomy tube but do not have a low-profile device, simply insert the string through the existing gastrostomy tube before removal and use it to place a second, regular gastrostomy tube.

The most catastrophic complication associated with gastrostomy tubes is leakage of gastric contents into the abdominal cavity with subsequent generalized peritonitis. This may occur with premature tube removal, when overfeeding large breed dogs (i.e., a very heavy stomach can pull the stomach off the tube), or secondary to improper placement of a low-profile tube when trying to replace an old gastrostomy tube. This complication can generally be prevented by using a technique that results in a sutured pexy of the stomach to the body wall (flank or midline surgical approach). Other complications of gastrostomy tubes include vomiting, peristomal infection (relatively common, although often minor and easily managed), and migration of the tip of the catheter into the pylorus. The balloon on Foley-type catheters usually disintegrates over time; hence, these catheters are not always optimal for long-term use. There are many

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dedicated gastrostomy catheters that have mushroom tips that can last for months. As noted previously, low-profile gastrostomy devices may be better tolerated by owners when tubes are needed to remain in place for some time (e.g., dogs with megaesophagus, esophageal stricture, chronic renal failure).

Enterostomy Tubes

Enterostomy (jejunostomy) feeding tubes are indicated in patients with gastric, intestinal, or pancreatic disease and in patients having biliary tract surgery in which the intestinal tract distal to the disease or surgical site is functional. Immediate feeding of a highly digestible, low-bulk diet in patients undergoing colonic surgery or that will undergo colonic surgery can be accomplished using an enterostomy feeding tube. Patients with preexisting PCM that must have major abdominal surgery are candidates for early enteral hyperalimentation by means of an enterostomy tube.

The main reason to use enterostomy tubes is to bypass a dysfunctional stomach or duodenum. Flexible endoscopy, laparotomy, or laparoscopy is required for placement of an enterostomy feeding tube. A 5 Fr, 90-cm infant feeding tube is recommended for cats and for small and medium size dogs. An 8 Fr feeding tube may be used in large and giant breed dogs. Due to the small size of the tubes, liquid diet formulations are used for nutritional supplementation. Enteral feeding through enterostomy tubes is typically performed in a hospitalized setting as a continuous rate infusion to minimize the chance of discomfort associated with overdistension of the intestine. Although premature removal and associated peritonitis is the most significant complication associated with jejunostomy tubes, this event is uncommon. A 2016 study of 64 dogs with jejunostomy tubes placed during abdominal surgery found a 22% rate of complications, none of which was life-threatening.[8] Diarrhea (high fecal score) is expected following intestinal feeding regardless of diet chosen.

Enterostomy Tube Placement by Flexible Endoscopy

Nasoenterostomy tubes may be placed by flexible endoscopy. *Place a guidewire, flexible end first, through the nose and into the oropharynx. Grasp the tip of the guidewire with flexible endoscopic forceps placed through the endoscope, and then pass the endoscope into the duodenum, "dragging" the guidewire into the distal duodenum with it. Then, release the tip of the guidewire and withdraw the endoscope from the patient. Next, introduce the feeding tube over the guidewire and advance it until the tip of the feeding catheter is present in the distal duodenum or proximal jejunum. Carefully remove the guidewire and fix the tube to the external nares as described under nasoesophageal tubes.*

Surgical Placement of Enterostomy Tube

Bring the distal tip of the feeding tube into the abdominal cavity through a 2- to 3-mm stab incision made on the right or left body wall with a No. 11 scalpel blade or a 10-gauge hypodermic needle (Fig. 10.10 and Video 10.3). Select a segment of proximal jejunum and identify the normal direction of flow of ingesta (i.e., aboral and oral end). Ensure that the selected intestine is easily mobilized to the feeding tube entrance on the body wall. Make a 1- to 1.5-cm linear incision in the seromuscular layers of the antimesenteric border of the selected jejunal segment (Fig. 10.11A). Use a No. 11 scalpel blade to enter the lumen of the jejunum at the most aboral end of the incision. Insert the distal end of the feeding tube through the incision and pass 10 to 12 inches (25 to 30 cm) of the tube in an aboral direction into the jejunal lumen. Position the exiting

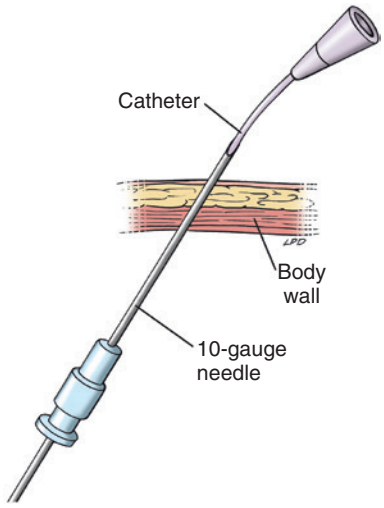


FIG. 10.10 For placement of an enterostomy tube, a 10-gauge hypodermic needle facilitates transabdominal placement of a 5 Fr feeding tube.

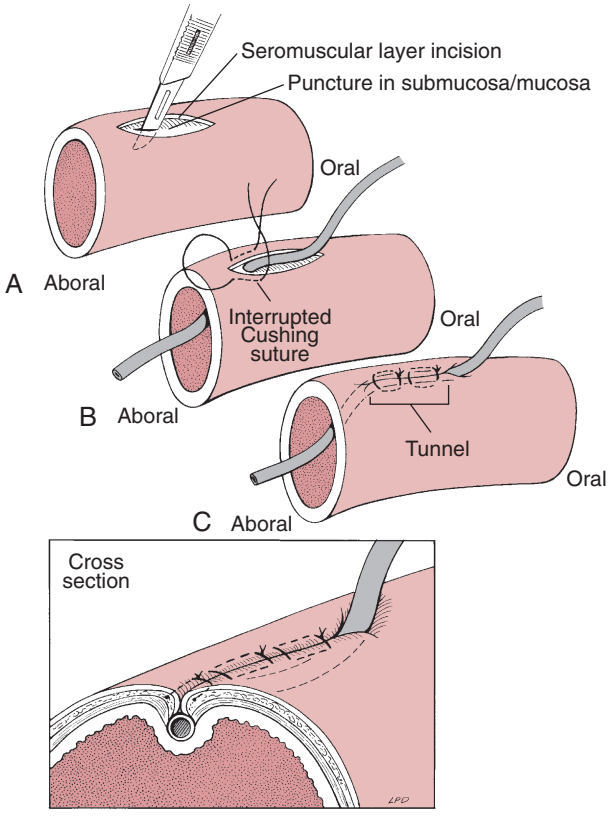


FIG. 10.11 Placement of an enterostomy tube. (A) Make a 1- to 1.5-cm linear incision in the seromuscular layers of the antimesenteric border of the selected jejunal segment; use the tip of a scalpel blade to puncture a hole in the aboral aspect of the seromuscular incision. (B) and (C) Place the distal end of the feeding tube through the incision; lay the exiting portion of the tube in the 1- to 1.5-cm seromuscular incision and construct a "tunnel" by inverting the seromuscular layer over the tube with three or four Cushing sutures of 4-0 absorbable material.

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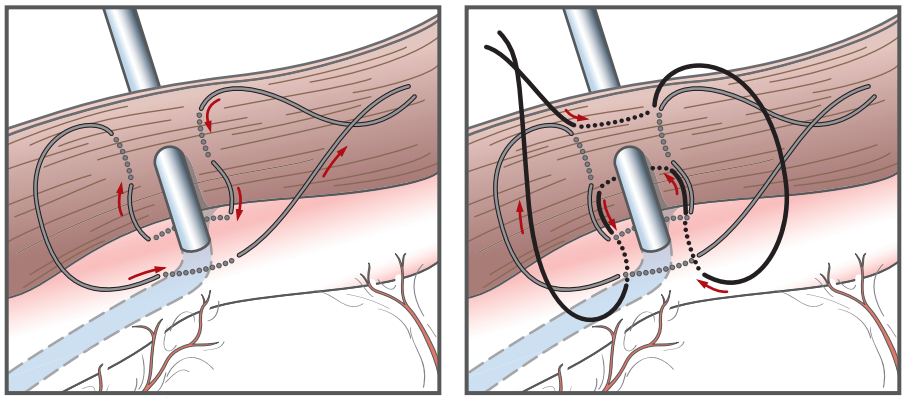


FIG. 10.12 Interlocking box technique for securing enterostomy tubes to the abdominal wall.

portion of the tube in the 1- to 1.5-cm seromuscular incision, and suture it in this "tunnel" by inverting the seromuscular layer over the tube with three or four Cushing or horizontal mattress sutures of 4-0 or 5-0 synthetic monofilament absorbable suture material (e.g., polydioxanone or polyglyconate) (see Fig. 10.11B–C). Alternatively, place a small purse-string suture in the antimesenteric border using 4-0 or 5-0 synthetic monofilament absorbable suture material. Use a No. 11 scalpel blade to make a stab incision in the middle of the purse-string suture and insert the feeding tube as described earlier. Tighten the purse-string suture.

In an in vitro study, no significant difference in bursting pressure was found between purse-string and seromuscular incision techniques.[9]

Pexy the jejunal tube exit site to the exit site at the body wall with four to five simple interrupted sutures of 4-0 standard absorbable suture (e.g., polydioxanone or polyglyconate); secure the feeding tube to the skin using a Chinese finger-trap friction suture of 3-0 nonabsorbable material (e.g., nylon). Alternatively, an interlocking box technique (Fig. 10.12) has also been described in which two complete suture boxes are placed and secured between the jejunal segment and abdominal wall. This technique is thought to decrease the risk of leakage and allow for earlier tube removal.

Laparoscopic-assisted jejunostomy tube placement has been described. Standard laparoscopy procedures for abdominal exploratory are followed with ultimately a segment of jejunum exteriorized through an instrument portal. Jejunostomy tube placement is performed similarly to what was described earlier.

Patients with enterostomy feeding tubes can be fed immediately after surgery. The exit point of the feeding tube should be incorporated into a body bandage to prevent premature removal by the patient, technical staff, or client. A column of water should be kept in the tube between uses. Possible complications include premature removal, tube-induced jejunal perforation, peritoneal leakage, and subcutaneous leakage. Theoretically, enterostomy tubes can be removed almost immediately after placement, but experience suggests that they should be left in place for at least a week before removal to minimize the chance of post-removal peritonitis. Subcutaneous leakage can be prevented by securely fixing the tube to the skin, and peritoneal leakage is prevented by taking care to include a 360-degree jejunal-abdominal wall pexy.

Gastrojejunostomy Tubes

Several products (e.g., Kangaroo gastrostomy feeding tube with Y-ports) are available that combine gastric and intestinal feeding tubes. These tubes provide access to the stomach and small intestine without the need for an enterotomy. The most significant advantage over other tubes is the ability to institute postgastric feeding while simultaneously allowing for gastric decompression.

CALCULATION OF RATE AND VOLUME OF FEEDING

Once the number of calories necessary to meet the patient's total caloric requirement has been calculated, the rate and volume of feeding are determined based on the route of administration (e.g., oral, nasoesophageal, pharyngostomy, esophagostomy, gastrostomy, or enterostomy). With gastric feeding (i.e., oral, nasoesophageal, esophagostomy, pharyngostomy, and gastrostomy tubes), the amount fed is determined by the patient's stomach capacity. The normal canine and feline gastric capacity is approximately 80 mL of fluid per kilogram of body weight. However, anorexic patients can typically accommodate only 30 to 40 mL/kg of body weight when feeding begins. A gradual increase over 2 to 3 days allows the stomach to accommodate progressively larger volumes. The patient should have a minimum of three feedings daily; however, if vomiting and abdominal distention occur, the daily volume should be reduced and the number of daily feedings increased. In animals that routinely vomit after administration of the diet through a nasoesophageal, esophagostomy, or gastrostomy tube, it is often advantageous to drip commercially prepared liquid diets into the tube as a constant-rate infusion (CRI). Simultaneous administration of antiemetics and/or gastric prokinetic agents may also be beneficial.

With enterostomy feedings, the rate and volume must be carefully regulated to prevent overdistention. Each patient is unique in the amount of fluid the small intestine will accommodate; guidelines for feeding by enterostomy tube are presented in Box 10.5. These are only guidelines; some patients require a longer adjustment time (5–7 days), whereas others allow total volume feeding in 2 to 3 days. Signs of overfeeding include vomiting, diarrhea, abdominal distention, cramping, or all of these. Diluting the diet concentration and reducing the rate and volume of administration generally resolve these complications.

PARENTERAL FEEDING COMPLICATIONS

Complications associated with PN include catheter occlusion, catheter displacement, phlebitis, thrombosis, sepsis, hyperglycemia, hypophosphatemia, hyperlipidemia, azotemia, and electrolyte

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BOX 10.5 Guidelines for Feeding via an Enterostomy Tube

- Calculate total caloric requirement.
- Give ¼ of the calculated volume during the first 24 hours; a minimum of four to five feedings per day is recommended.[a]
- Give ½ of the calculated volume during the second 24 hours in four to five feedings.[a]
- Give ¾ of the calculated volume during the third 24 hours in four to five feedings.[a]
- Give the entire calculated volume during the fourth 24 hours in four to five feedings.[a]

[a]Continuous feeding by means of an infusion pump is preferred.

imbalances. If sepsis is suspected, it is usually best to replace the IV catheter and submit the tip of the old catheter for culture and susceptibility testing.

Rarely, insulin may be needed to control persistent hyperglycemia. In the rare case in which insulin seems required, a nutritionist with experience with PN should be consulted. Initially, a very low dose (e.g., 0.25 unit of regular insulin/4.5 kg body weight in dogs) may be tried and the patient closely monitored. One must anticipate that the need for insulin will disappear as the patient adapts. Cats are much more sensitive to the effects of insulin than dogs, and insulin should be avoided, if possible. Life-threatening hypophosphatemia and/or hypokalemia may occur secondary to administration of insulin to emaciated patients.

Complications associated with PPN have been infrequently reported. Most complications are mild and do not require discontinuation of PPN.

ENTERAL FEEDING COMPLICATIONS

Three types of complications can occur with enteral hyperalimentation: mechanical complications, gastrointestinal complications, and metabolic complications. In most cases complications can be prevented by proper tube placement technique, by using an appropriate-diameter, soft rubber feeding tube and the proper diet, by carefully calculating feeding schedules, and by proper tube management during and between feedings.

Mechanical Complications

Mechanical complications include inadvertent placement of the tube into the trachea (nasoesophageal, esophagostomy, and pharyngostomy tubes) or peritoneal cavity (gastrostomy or enterostomy tubes), gastrointestinal perforation by the feeding tube (gastrostomy and enterostomy tubes), regurgitation or vomiting of the tube (nasoesophageal, esophagostomy, and pharyngostomy tubes), esophageal irritation (nasoesophageal, esophagostomy, and pharyngostomy tubes), gastric outflow obstruction (gastrostomy tubes), infection at the tube exit site, occlusion of the tube, or premature tube removal by the patient. Inadvertent placement of feeding tubes in the trachea or peritoneal cavity can be prevented by paying careful attention during tube placement. If there is any question about the tube's location, a small amount of sterile aqueous contrast material should be injected through the feeding tube and a radiograph taken. Gastrointestinal perforation has been virtually eliminated by use of small-bore, Silastic, or soft rubber enterostomy feeding tubes. In cats, esophageal perforation (esophagostomy tube) has largely been eliminated by discontinuation of the use of a stylet during tube placement. Premature tube removal by the patient can usually be prevented by adequate mechanical restraint (e.g., bandaging, Elizabethan collar) and secure attachment of the tube to its exit site (i.e., Chinese finger-trap friction sutures). Use of small-bore, soft rubber feeding tubes has improved patient tolerance. Esophagitis secondary to nasoesophageal, esophagostomy, or pharyngostomy tubes occurs; however, use of Silastic or soft rubber feeding tubes has reduced the frequency. Midesophageal placement of the tip eliminates reflux esophagitis.

Stoma site infection can be minimized by proper tube management. The area should be kept clean and covered with a loose bandage. Care should be taken when feeding the patient to prevent diet formula from contaminating the exit site. Rhinitis secondary to placement of a nasoesophageal tube occurs, but use of small-bore, soft-rubber tubes makes this uncommon.

Small-bore feeding tubes (5–8 Fr) can become occluded with the diet formula. This is best prevented by using a commercial liquid diet rather than a blenderized diet preparation. Flushing material out of the tube when feeding is finished and capping the tube to maintain a column of water help prevent occlusion by gastrointestinal reflux. Large-bore feeding tubes accept blenderized diets, but similar precautions should be taken to prevent occlusion. If a tube becomes occluded, flexible endoscopy forceps can be passed into the tube to remove the clogged material. If endoscopy forceps are unsuccessful, a carbonated liquid (e.g., cola) can be infused into the tube; the effervescence of the liquid and its acid pH may encourage removal of clogged material. If this is unsuccessful, tube replacement may be necessary.

Gastrointestinal Complications

Common gastrointestinal complications of enteral nutritional therapy include vomiting, cramping, abdominal distention, or diarrhea. The most common causes are feeding too rapidly, feeding too large a volume, and feeding diets with a high osmolality. Treatment is aimed at reducing the rate and volume fed or diluting the diet formula.

Metabolic Complications

The most common metabolic complications of enteral nutritional therapy are hyperglycemia and hypophosphatemia. However, they tend to be less common and usually less severe than is seen with parenteral nutrition.

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11

Fundamentals of Physical Rehabilitation

DEFINITIONS

Osteokinematics is the branch of biomechanics concerned with the description of bone movement when a bone swings through a range of motion (ROM) around the axis in a joint (i.e., flexion, extension, abduction, adduction, rotation). **Arthrokinematics** is the manner in which adjoining joint surfaces move in relation to each other during bone movements. Arthrokinematic movements occur as glides, rolls, or spins and accessory motions such as distraction, shear, and compression. A **roll** is a rotary movement (one bone rolling on another); a **spin** is also a rotary movement (one body spinning on another). A **slide or glide** is a translatory movement (sliding of one joint surface over another). **Sarcopenia** is a gradual loss of muscle mass as a result of aging. **Proprioceptive neuromuscular facilitation** (PNF) uses the body's proprioceptive system to facilitate or inhibit muscle contraction. By stimulating the distal segments, the proprioceptors in the more proximal segments are stimulated. The purpose of PNF is to enhance and create movement in areas where the neurologic system has been compromised.

OVERVIEW OF REHABILITATION THERAPY

Human physiotherapy, or physical therapy, is a relatively new medical discipline that began in Sweden in the early 1800s; modern physical therapy was developed in Britain in the early 1900s. Physiotherapy was initially focused on the treatment of polio victims, but after the outbreak of World War I, the field concentrated on helping to restore mobility to injured soldiers. The first school of physiotherapy was established at Walter Reed Army Hospital in Washington, DC.[1]

Interest in the veterinary derivative of human physiotherapy began initially in the 1970s with the publication of a book called *Physical Therapy for Animals: Selected Techniques* by Ann Downer, PT. Shortly thereafter, national presentations to such groups as the American College of Veterinary Surgeons, American Physical Therapy Association, and the American Veterinary Medical Association led to increased interest in canine rehabilitation. Interest in canine sports medicine began at about the same time with publication of *Canine Sports Medicine and Surgery* and with the formation of the International Racing Greyhound Symposium, associated with what was then known as the Eastern States Veterinary Conference (now the North American Veterinary Conference). Rehabilitation was a frequent topic at this conference and was expanded to include all sporting dogs.[2] Today, physical rehabilitation is becoming common in small animal practices. These exercises and modalities are used not only for recovery from orthopedic and neurologic conditions, but also for wellness care and preventive medicine in the form of weight management and maintenance of muscle strength and conditioning, particularly for athletes and geriatric animals. Current practices combine the knowledge and skills of veterinarians, veterinary technicians, human physiotherapists, and physiotherapy assistants. Formal training and accreditation are now available for veterinarians and veterinary technicians, and advanced specialty training is available through the American College of Veterinary Sports Medicine and Rehabilitation.

With advances in surgery and with an increasing number of owners who regard their pets as family members, demand for rehabilitation for veterinary patients is increasing. Postoperative care focusing on decreasing pain, inflammation, and joint stiffness while improving mobility and quality of life may result in shorter hospital stays and improved patient and client well-being. Rehabilitation for the veterinary patient with orthopedic or neurologic disease has as its overlying principle a return to normal mobility and neuromuscular function. The goals of rehabilitation are first and foremost to relieve pain and ameliorate discomfort while ensuring that the patient's condition does not worsen, particularly in the light of spinal instability or surgical repair. Once pain control has been adequately addressed (see Chapter 13), therapists can focus on restoring normal muscular function, minimizing muscular atrophy and other secondary problems, and improving and restoring mobility.

Rehabilitation therapy emphasizes return to function and maximum recovery from the disease process or surgical procedure, as well as the improvement of overall well-being, rather than the treatment of a specific disease or diagnosis. Postoperative orthopedic or neurologic surgery patients are among the many patients that benefit from physical rehabilitation (Box 11.1). Other patients include those with chronic osteoarthritis, severely debilitated patients, oncology patients, obese patients (to aid in weight loss), canine athletes and working dogs (to enhance condition or performance), and patients with orthopedic or neurologic disease that require nonsurgical management. Improvement in pain control, joint ROM, muscle strength and coordination, balance and proprioception, and decrease in contracture and fibrosis are some of the objectives of physical rehabilitation. Return to function and recovery are achieved through the use of the many different modalities and techniques discussed in this chapter.

ROLE OF EXERCISE PHYSIOLOGY IN REHABILITATION

Exercising animals rely on skeletal muscles to perform; skeletal muscle performance is dependent on muscle fiber type. Traditionally, muscles are classified as type I (oxidative or slow twitch) or type II (glycolytic or fast twitch).[3] Postural muscles (i.e.,

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ABSTRACT

Physical rehabilitation is the newest specialty in veterinary medicine and encompasses a variety of fields, including exercise physiology, orthopedics, neurology, myology, nutrition, and pain management, among others. It is crucial for optimal postsurgical, nonsurgical, and long-term management of many orthopedic and neurological conditions. Rehabilitation assessment of orthopedic or neurologic conditions involves collecting subjective and objective data about the patient, including goals for rehabilitation, posture, gait analysis, range of motion, strength, and specialized tests. An individualized rehabilitation plan is formulated based on the primary diagnosis, taking into consideration the tissue types involved and chronicity of the injury. Treatment interventions may include manual therapies, therapeutic exercises, hydrotherapy, modalities, and neurologic reeducation.

Keywords
Canine rehabilitation
orthopedic rehabilitation
neurologic rehabilitation
physical modalities
therapeutic exercises
aquatic therapy

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BOX 11.1 Benefits of Rehabilitation Therapy

- Decreased pain
- Increased rate of recovery following injury or surgery
- Improvement in quality of movement and performance
- Increased strength and endurance
- Improvement in flexibility and biomechanics
- Reduction in the incidence of future injury both due to restoration of normal biomechanics and through client/owner education
- Positive psychological effect for both the pet and for the owner
- Minimally invasive approach
- Minimal complications if administered properly

Data from Niebaum K. Rehabilitation physical modalities. In: Zink MC, Van Dyke JB, eds. *Canine Sports Medicine and Rehabilitation.* Ames, IA: Wiley-Blackwell; 2013:115–131.

BOX 11.2 Modified Numerical Frankel Scale for Dogs With Thoracolumbar Intervertebral Disc Disease

Grade 0: Paraplegia with absent deep nociception
Grade 1: Paraplegia with absent superficial nociception
Grade 2: Paraplegia with intact nociception
Grade 3a: Nonambulatory paraparesis: ability to bear weight on pelvic limbs without support
Grade 3b: Nonambulatory paraparesis: inability to bear weight on pelvic limbs without support
Grade 4: Ambulatory paraparesis
Grade 5: Normal gait with paraspinal hyperesthesia

From Lee CS, Bently RT, Weng HY, Breur GJ. A preliminary evaluation of the reliability of a modified functional scoring system for assessing neurologic function in ambulatory thoracolumbar myelopathy dogs. *BMC Vet Res.* 2015;11:241.

quadriceps femoris) are capable of slow and sustained contraction and contain more type I fibers than mobilizing muscles (i.e., gracilis muscle), which contain more type II fibers. Type I muscle fibers may be considered the "endurance muscle fibers" and are prominent in dogs that run long distances, such as sled dogs. Type II fibers provide the power and speed muscles fibers prominent in sprinting dogs, such as greyhounds. When compared to humans, dogs have a high oxidative capacity in their muscles because they are adapted for endurance activities[4]; nonetheless, certain breeds (e.g., greyhounds) have more type II muscle fibers than other dogs, making them better sprinters.

When muscles are immobilized, such as in casts or splints, or following long periods of cage rest, muscle strength decreases rapidly with as much as 50% of strength lost within the first week. With disuse, postural muscles that contain more type I fibers atrophy more than the mobilizing muscles containing type II fibers. However, with geriatric sarcopenia the opposite is true; more type II muscle fibers atrophy than type I fibers. This is an important consideration in designing a rehabilitation program for an athlete with muscle loss due to injury versus a geriatric patient with age-related atrophy. Historically, veterinary orthopedic postoperative care consisted of 6 to 12 weeks of cage rest after surgery. This may be acceptable for fracture healing; however, muscle physiology research has shown that the longer a muscle is immobilized or not moved in a normal manner, the more difficult it is to return that muscle to full strength and function. All postoperative patients can benefit from a rehabilitation program that starts as soon as possible after surgery to minimize muscle atrophy and return the patient to full function as quickly as possible.

Muscle contractions can be described as having two variables: force and length. *Force* is either tension or load. *Load* is the force exerted on the muscle by an object, and muscle tension is the force the muscle exerts on an object. *Isometric* contractions occur when muscle tension changes without a change in muscle length; *isotonic* contractions occur when muscle tension remains the same but muscle length changes. Canine rehabilitation is concerned with isotonic contractions. Isotonic contractions occur as either concentric or eccentric contraction. *Concentric contraction* occurs when the muscle contracts, tension in the muscle increases, and shortening occurs. *Eccentric contraction* occurs when the muscle contracts but lengthens because the tension generated in the muscle is insufficient to overcome the load pulling down on the muscle. The eccentric contraction is controlling the movement: it is the natural braking force that occurs during movement.[5] Eccentric contractions tend to preferentially strengthen type II muscle fibers, whereas concentric contractions favor type I fibers. In general, in an exercise program concentric exercises are performed first to help accustom the muscle to movement. Eccentric exercises are added later because, although they help develop strength, they have the potential to cause muscle damage and delayed onset muscle soreness. A balanced program between concentric and eccentric muscle contractions is desirable.[6]

REHABILITATION EVALUATION

An ideal rehabilitation evaluation involves a thorough history, physical examination, and a complete orthopedic and neurologic examination, followed by a specific rehabilitation examination. Historical data of interest to the rehabilitation professional include onset and duration of the problem, body part involved, trauma or activities associated with the injury, current pain score, lifestyle and exercise habits, weight loss or gain, and type of surgery (if any) performed. A general physical examination focusing on any pathology affecting the cardiovascular, neuromuscular, pulmonary, endocrine/metabolic, or integumentary system is critical because all these systems can affect muscular performance. It is also important to evaluate the patient's psychological state and willingness to perform any exercises, as well as the experience of the owner or handler. Orthopedic and neurologic examinations are discussed in Chapters 31 and 38, respectively; only the rehabilitation examination is outlined here. A rehabilitation examination is comprised of a thorough myofascial examination, including muscle girth assessment, examination of each joint, goniometric measurements, subjective and objective (force plate, pressure mat, stance mat) lameness assessment, gait analysis, pain score assessment, body condition score, muscle condition score, modified Frankel score (Box 11.2), and—where available and desired—dual energy x-ray absorptiometry assessment and advanced imaging (i.e., computed tomography, magnetic resonance imaging).[7]

Understanding gait and canine locomotion is vital to diagnosing many neurologic and orthopedic conditions. When possible the lameness evaluation and gait analysis should take place before the other physical examinations because palpation of the muscles, limbs, and joints may alter subtle lamenesses. Subjective lameness evaluation involves visualizing the patient from all angles at both a walk and a trot. It is often easier to detect lameness when the patient is walking because it is a slower gait; however, subtle

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BOX 11.3 Numerical Rating Scale

0 = Normal gait pattern
1 = Mild intermittent lameness
2 = Moderate lameness, normal stride length, partial weight bearing
3 = Moderate lameness, shortened stride length, partial weight bearing
4 = Severe lameness with toe touching and minimal limb use
5 = Non–weight-bearing lameness

lameness may be more visible at the trot because this is the only gait that allows individual limb evaluation.[6,8] Patients should be examined on a nonslippery, flat surface such grass or a cement or rubber flooring. The patient should be examined walking toward and away from the examiner, as well as from the side, and while sitting and standing. Having the patient walk through a cavaletti course (see p. 118) and perform weaves and figure eights around cones (see p. 119) is an excellent way to evaluate intermittent lameness. Videotaping these sessions can be helpful and allows the therapist to compare sessions. Either a numerical rating score (Box 11.3) or a visual analogue scale can be used if they are used consistently.[8] Visual gait observation is inferior to objective gait analysis; however, it is still a useful tool in clinical practice.[8]

Validated objective gait analysis is the preferred method of lameness evaluation and is often used to detect subtle lameness because it allows quantification of gait parameters. Currently there are three validated methods of objective gait analysis: kinematic analysis, kinetic analysis (force plate analysis), and temporospatial gait analysis (pressure-sensing walkways). *Kinematic analysis* (involving markers on joints, 3D cameras, and special software) and force plate analysis are used primarily by research institutions due to the cost of the equipment. Pressure-sensing walkways are more affordable but are not widely used by most rehabilitation practitioners; however, these methods do provide an objective outcome measure that quantifies lameness and improvement with rehabilitation.[9]

A myofascial examination includes palpation of the body, origin, and insertion of each muscle; and assessing for functional muscle disorders (acute indirect muscle disorders without macroscopic evidence of a muscle tear), as well as structural muscle injuries (indirect muscle disorders with macroscopic evidence of a muscle tear). Particular attention should be paid to the identification of myofascial trigger points. *Myofascial trigger points* are defined as a hyperirritable spot within a taut band of skeletal muscle that is a source of pain and disability. Identification of trigger points is by manual palpation through either flat or pincher palpation (Video 11.1).[10]

Central to a myofascial examination is the determination of muscle circumference of the major muscles of front and back legs. The girth of the affected leg is compared with the unaffected leg on the opposite side to determine extent of muscle atrophy and to give a baseline for therapeutic outcome measures. This measurement is done using a weighted tape measure. If an ordinary tape measure is used, the measurement will depend on how tightly the tape is pulled. If the examiner pulls tighter, tissue compression will be greater and the measured circumference may be smaller; thus two consecutive measurements may be quite different. By applying a constant tension, accurate and repeatable measurements can be obtained. If multiple examiners are evaluating the patient, it is important that each uses the same anatomic landmarks to ensure accurate measurements. Other variables such as coat length and position of animal (standing or lying) should be taken into consideration when measuring girth circumference (Fig. 11.1).



FIG. 11.1 Measurement of thigh circumference using a gulick.

BOX 11.4 Calculating Energy Requirement

Resting energy requirement (RER) = 70 × (Ideal body weight in kg)$^{0.75}$
Nonobese energy requirement = RER × 1.0 to 1.3 (depending on activity)
Obese energy requirement = 0.7 × RER
Goal: provide sufficient energy to achieve 1% weight loss per week until ideal weight is achieved

Data from Brooks D, Churchill J, Fein K, et al. 2014 AAHA weight management guidelines for dogs and cats. *J Am Anim Hosp Assoc.* 2014;50(1):1-11.

Pain is considered to be the fourth vital sign and is frequently overlooked.[11] Pain scoring is an important part of the rehabilitation evaluation and should be done consistently at every patient visit. Using a validated pain scale such as the Glasgow Chronic Pain Index or the Helsinki Chronic Pain scale (see Chapter 13) can ensure that the patient is comfortable and willing to participate in rehabilitation. Exercises and modalities should be adjusted according to the patient's pain score.

Body condition scoring (BCS) and muscle condition scoring (MCS) are two vital pieces of information required from all patients (Figs. 11.2 and 11.3). Many veterinary patients are obese, and some are also sarcopenic. BCS and MCS should be documented at the initial evaluation and can be used to document progress through rehabilitation. Calculation of ideal body weight and number of calories needed to achieve that weight, as well as grams of protein required in the diet for optimal weight loss and muscle integrity, should be addressed with all patients (Box 11.4; see also Chapter 10). Most rehabilitation patients require 3.75 g of dietary protein per kilogram of body weight for maintenance of lean body mass, muscle mass, weight loss, and healing. Senior dogs require 5 g of dietary protein per kilogram of body weight to maintain muscle integrity.[12,13] If this protein requirement is not met by the dog's diet, additional supplemental protein, such as whey protein powder, may be required.

The rehabilitation evaluation concludes with a complete set of goniometric measurements. This is best accomplished with the patient lying on its side, restrained by an assistant. A commercial goniometer should be used and measurements taken at the most comfortable degree of flexion and extension for each joint with one arm of the goniometer along each long axis of

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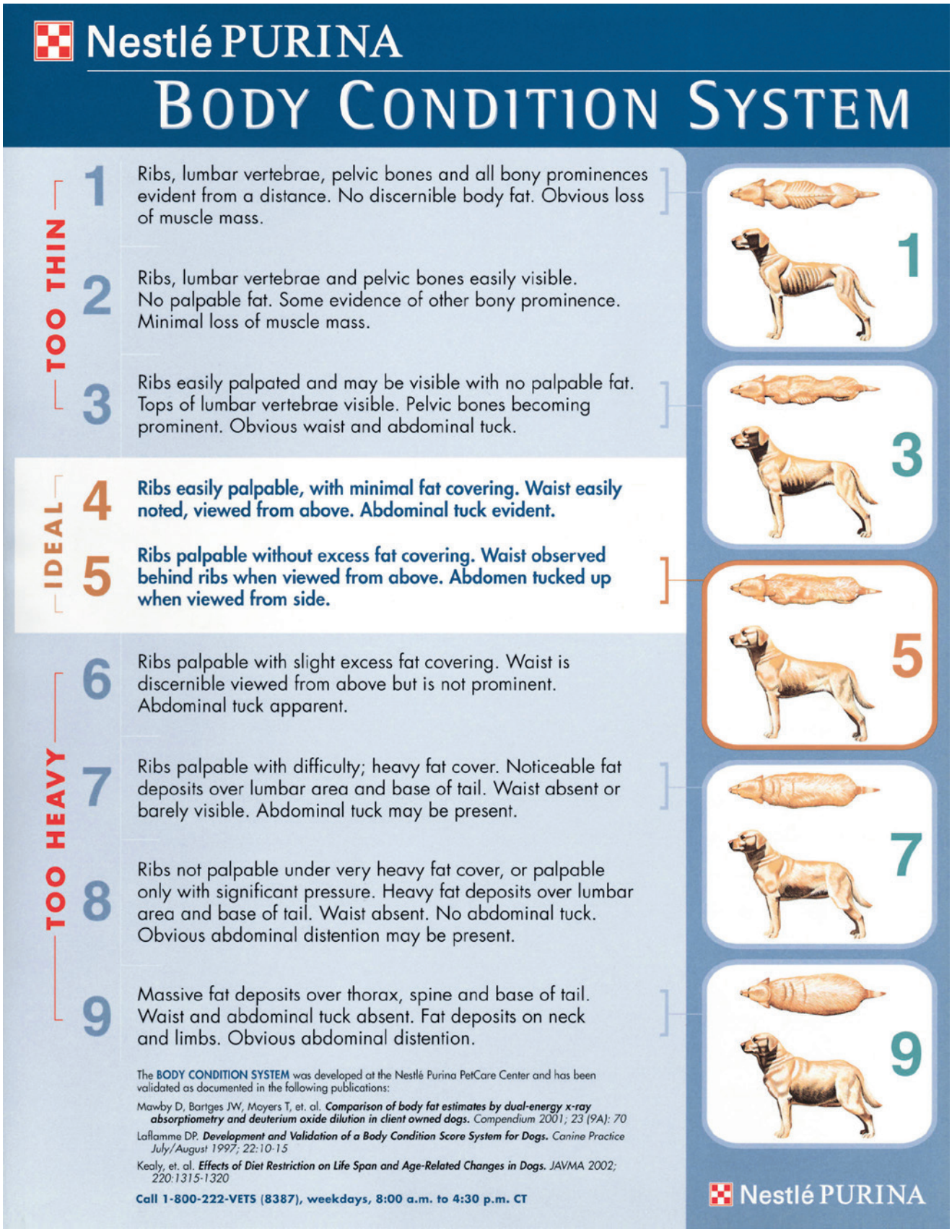


FIG. 11.2 Body condition scoring (BCS). (Courtesy Nestlé Purina PetCare Center.)

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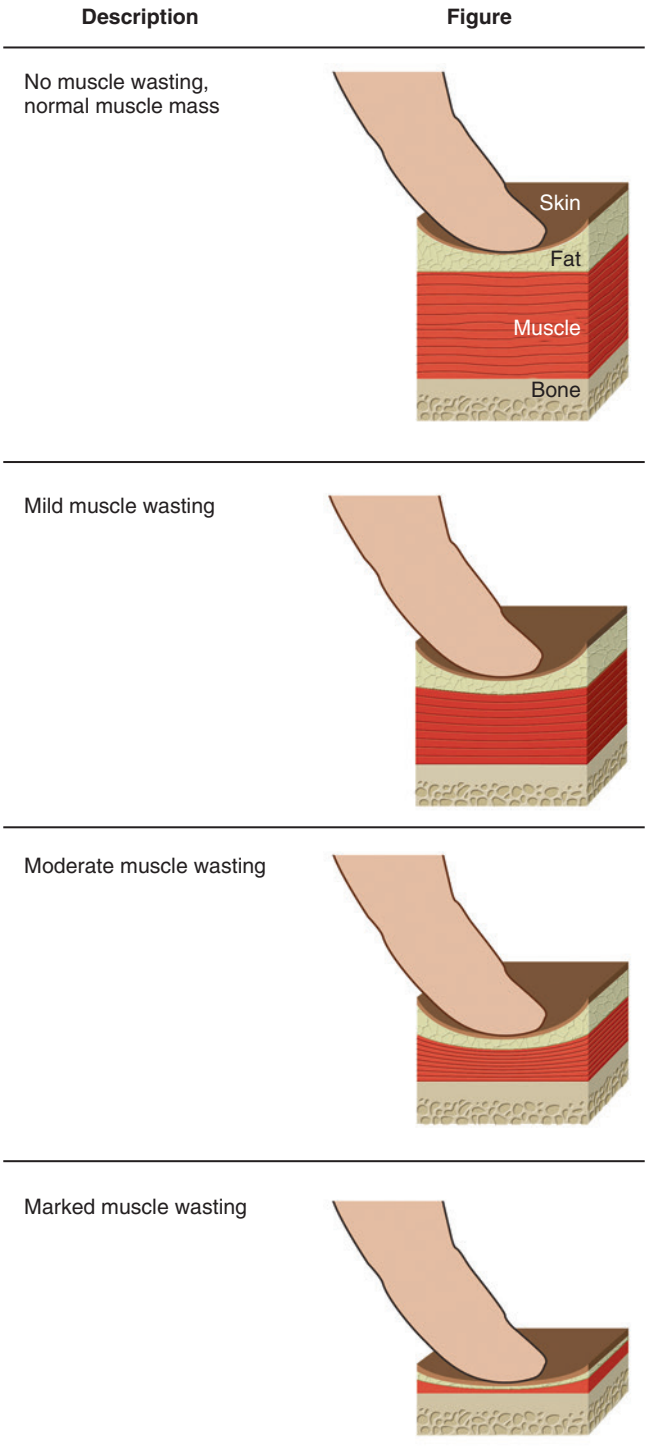


FIG. 11.3 Muscle condition scoring. (Modified from Baldwin K, Bartges J, Buffington T, et al. AAHA nutritional assessment guidelines for dogs and cats. *J Am Anim Hosp Assoc.2010;*46[4]: 285–296.)

the bone proximal and distal to the joint in question. The center of the goniometer is placed over the axis of joint rotation as determined by flexion and extension (Fig. 11.4). Measurements are recorded for each limb and compared with a set of normal values, as well as to the contralateral limb (Table 11.1). The



FIG. 11.4 Measurement of tarsal flexion with a goniometer.

TABLE 11.1 Goniometry Normal Values for Labrador Retrievers

Joint	Position	Mean in Degrees
Carpus	Flexion	32
	Extension	196
	Valgus	12
	Varus	7
Elbow	Flexion	36
	Extension	165
Shoulder	Flexion	57
	Extension	165
Tarsus	Flexion	39
	Extension	164
Stifle	Flexion	42
	Extension	162
Hip	Flexion	50
	Extension	162

Modified from Jaegger GJ, Marcellin-Little DJ, Levine D. Reliability of goniometry in Labrador retrievers. *Am J Vet Res.* 2002;63(7):979–986.

published normal values are for Labrador retrievers. Variation may occur among breeds; therefore calculating the angles of the contralateral, unaffected limb is important for comparison.[14]

DEVELOPING A REHABILITATION PLAN

The purpose of the rehabilitation assessment is to determine the patient's major problems, both structural and functional. Once the problems are identified, the therapist develops a plan specific to the patient's needs and that meets the owner's expectations, financial and time commitments, and desired outcome. The rehabilitation plan for a geriatric, sarcopenic dog with a long bone fracture will be different from that of a canine athlete that has undergone a tibial plateau leveling osteotomy (TPLO) procedure. The plan should be specific, with described outcomes and timelines. Regular scheduled evaluations should be a part of the plan. The therapist must be able to modify the plan based on the patient's progress. Table 11.2 shows an example of a detailed rehabilitation plan for a TPLO procedure in a young dog.

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TABLE 11.2 Example Rehabilitation Protocol for a Young Dog After TPLO

Treatments/ Modalities	Days 1–7	Days 7–21	3–4 Weeks	5–7 Weeks	8–12 Weeks
Pain medications	As directed	As directed	PRN	PRN	PRN
Cryotherapy	10–15 minutes 3 times daily before walks or exercises	Use after exercise for 15 minutes	PRN	PRN	PRN
Heat therapy		Use heat before exercise or if dog is stiff	PRN	PRN	PRN
Massage	5 minutes 3 times daily prior to starting exercises; massage from toes to hip	Continue twice daily	Twice daily	Twice daily	Only if desired
PROM	10 repetitions 3 times daily, all joints affected limb—start above and below knee	Continue twice daily. If dog is using leg really well then decrease	Discontinue if dog is walking well and ROM is normal		
Laser therapy	Daily or every other day for first week	Every other day for first week then twice weekly	Twice weekly	Twice weekly	Discontinue
Walks	5-minute supported leash walk 2–3 times daily	Increase each walk by 2–3 minutes each week	Increase by 5 minutes each week	Increase by 5 minutes each week	15–20-minute walks 2–3 times daily
NMES	10 minutes twice daily	10 minutes twice daily	Discontinue if dog is doing well		
Balancing		5 minutes twice daily	5 minutes twice daily	One-leg standing up to 5 minutes	Replace with hill walks
Cavalettis/obstacles/ sit-to-stand		5 minutes twice daily	5 minutes twice daily	5 minutes twice daily	5 minutes twice daily
Weaving/circles		2–3 minutes once daily	5 minutes twice daily	5 minutes twice daily	5 minutes twice daily
Hills				Zigzag low, slow hills 5 minutes up and down	Increase to 10 minutes twice daily
Underwater treadmill		10 minutes 3 times weekly after sutures are removed	10 minutes 3 times weekly	15–20 minutes twice weekly	15–30 minutes twice weekly until released from rehab
Swimming					8 weeks post-TPLO for 5–10 minutes if all healed

NMES, Neuromuscular electrical stimulation; *PROM,* passive range of motion; *ROM,* range of motion; *TPLO,* tibial plateau leveling osteotomy.

THERAPEUTIC MODALITIES: GENERAL CONSIDERATIONS

Therapeutic modalities are used to augment medications and reduce pharmaceutical dosages, as well as to speed and direct healing in order to return the patient to full function as quickly as possible. The most common modalities used for pain relief include heat, cryotherapy, manual therapies such as massage, joint mobilizations and stretching, laser therapy, transcutaneous electrical nerve stimulation (TENS), and therapeutic ultrasound (TUS). Muscle strengthening and reeducation are accomplished using neuromuscular electrical stimulation (NMES), therapeutic exercises, hydrotherapy, and PNF. Assistive devices, such as harnesses, slings, boots, carts, orthotics, or incontinence aids, are often essential to the well-being and recovery of surgical or neurologic patients (Fig. 11.5).

Cryotherapy

Cryotherapy or cold packing has historically been the most common nonpharmaceutical method of treating postoperative or acute postinjury pain and inflammation. It is most effective when applied immediately after acute trauma and for up to 72 hours afterwards. It is a superficial treatment, but the depth of tissue cooling can be as great as 4 cm. Cryotherapy may also be used after exercise and before painful modalities.[15–17] Benefits of cryotherapy are listed in Box 11.5.

Cryotherapy can be delivered in several ways, including reusable ice packs, circulating cold-water blankets, ice packs,

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FIG. 11.5 A dog with custom orthotics. Rehabilitation patients may require custom orthotics for recovery.

BOX 11.5 Benefits of Cryotherapy

- Local vasoconstriction, which decreases blood flow thereby decreasing swelling, interstitial hemorrhage, and inflammation.
- Reduction of enzymatic tissue activity and damage through inhibition of the release of histamine, protease, hyaluronidase, and collagenase.
- Reduction of cellular metabolism. This is important because decreased tissue oxygen requirements prevent secondary cellular hypoxic injuries.
- Reduction in pain perception. Analgesia and a reduction in muscle spasm occur due to a decrease in nerve conduction velocity.

Data from Otte JW, Merrick MA, Ingersoll CD, et al. Subcutaneous adipose tissue thickness alters cooling time during cryotherapy. *Arch Phys Med Rehab.* 2002;83(11):1501–1505; and Millard RP, Towle-Millard HA, Rankin DC, et al. Effect of cold compress application on tissue temperature in healthy dogs. *Am J Vet Res.* 2013;74(3):443–447.

cold compression devices, ice cups, iced frozen towels, or vapocoolant sprays. Cold compression devices are frequently used after surgery. Circulating cold-water blankets can be used to decrease total body temperature and thereby decrease intracranial pressure in head trauma patients, but ice packs and ice massage are most commonly used for acute injuries in clinical practice (Fig. 11.6).

Ice packs can be either bags of crushed ice, ice-alcohol slushes, or gel packs. Gel packs, particularly human physiotherapy gel packs, are extremely convenient because they can be kept in the freezer yet still retain the ability to mold well to the desired area. Canine-specific cold packs that fit into preformed sleeves are available commercially. A thin, moist towel should be placed between the patient and ice pack to improve cooling and avoid damaging the skin.[18] The length of time to apply the ice pack



FIG. 11.6 A dog with a cold compression system placed after surgery.

BOX 11.6 Precautions and Contraindications for Cryotherapy

- Avoid any previous areas of frostbite.
- Apply with caution in very old, very young, very small, or hypertensive patients, as blood pressure can be affected.
- Avoid superficial wounds, superficial nerves, areas of decreased sensation, and open fractures.
- Check the patient's skin for redness or blanching after the first 5 minutes to avoid cold injury. Do not apply ice for more than 20 minutes per session.

Data from Niebaum K. Rehabilitation physical modalities. In: Zink MC, Van Dyke JB, eds. *Canine Sports Medicine and Rehabilitation.* Ames, IA: Wiley-Blackwell; 2013:115–131.

BOX 11.7 Tips for Using Ice Packs

- Make your own ice gel packs using 3 parts water to 1 part rubbing alcohol and freezing this mixture in resealable freezer bags of various sizes. These packs will stay malleable in the freezer.
- Bags of frozen peas can be used because they are malleable, but they will not cool as well as other methods because they contain air.
- Duration of ice pack application (for an average dog) is 10 to 15 minutes on, then 10 to 15 minutes off, repeated twice. Repeat every 4 hours for the first 24 to 48 hours.

varies with patient size, size and depth of the ice pack, and the target tissue. The average size dog will require 10 to 15 minutes of cooling, whereas a large dog may require 15 to 20 minutes and a very small dog may only need 5 to 8 minutes of icing. Fatter dogs will require more time to cool the target tissue because adipose tissue is a poor conductor of heat.

Ice massage can be used for small areas and may be used before other manual therapies. Water can be frozen in paper cups and the paper torn away as the ice melts. An ice popsicle can be made by placing a tongue depressor in the center of the cup before freezing, which provides the therapist a handle for applying the ice. Treatment duration for ice massage is generally 5 to 10 minutes. Although cryotherapy is generally safe, certain precautions and contraindications exist (Box 11.6). Tips for using ice packs are provided in Box 11.7.

Thermal Therapy

Superficial heating or thermal therapy is one of the oldest physical modalities. It is generally used after icing, before exercise or

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BOX 11.8 Benefits of Heat Therapy

- Vasodilation increases circulation to tissues, which increases tissue metabolic rate, improves tissue oxygenation, and decreases tissue edema and inflammation.
- Pain relief, which is mediated by an increase in the pain threshold.
- Decreased muscle spasm and relaxed muscle tone, which are thought to occur due to decreased muscle ischemia.
- Increased extensibility of soft tissue. Hot packing before stretching promotes flexibility and increases range of motion. When soft tissue is heated before stretching, the effect of the stretch lasts longer, and less force is required to maintain an effective stretch.

Data from Niebaum K. Rehabilitation physical modalities. In: Zink MC, Van Dyke JB, eds. *Canine Sports Medicine and Rehabilitation.* Ames, IA: Wiley-Blackwell; 2013:115–131.

BOX 11.9 Precautions and Contraindications When Applying Heat Therapy

- Protect the patient's skin by wrapping the hot pack in a towel and checking the skin frequently. Discontinue heating if skin has white areas or red mottled areas.
- Do not use heat in areas of impaired sensation or with acute inflammation.
- Use with caution in obese, pregnant, and cardiac patients, as well as very young or very old patients.
- Absolute contraindications include active bleeding, malignancy, fever, and open wounds.

Data from Niebaum K. Rehabilitation physical modalities. In: Zink MC, Van Dyke JB, eds. *Canine Sports Medicine and Rehabilitation.* Ames, IA: Wiley-Blackwell; 2013:115–131.

BOX 11.10 Tips for Using Hot Packs

- If using commercial gel packs or oat bags, be sure to mix the gel or oats well within the bag before applying to the patient to avoid hot areas. Always place a towel between the heat source and the skin.
- To determine if the hot pack will be comfortable for the patient, check the temperature by applying it to your inner arm or neck. Put your hand between the hot pack and skin at least every 5 minutes to monitor the temperature.
- If using towels, fold in thirds and roll (if using around a joint) or accordion fold (if using for a larger area). Immerse in hot water, squeeze the water out, and apply to affected area. Cover with a large dry towel to keep the heat in.
- Always use hot packing before stretching.

stretching, or to relieve pain and muscle spasms. Benefits of heat therapy are provided in Box 11.8.[15] Thermal therapy can be delivered by hot packs, hydrotherapy, infrared lamps, heating blankets, and TUS. Moist hot packs are most commonly used. Commercial physical therapy gel packs or oat or bean bags can be heated in the microwave, covered with a thin towel, and used. A hot, wet towel covered by a dry towel works well to apply moist heat. Duration of treatment depends on the patient and condition treated; however, heat treatments generally last 15 to 30 minutes and may be applied up to four times daily. Hot packs work well in conjunction with other forms of therapy, such as massage and exercise therapy.

Another way of applying thermal therapy is by using warm water baths. The patient or the affected area can be immersed in a warm bath, a whirlpool, or a hydro treadmill. The advantage of immersion therapy is that the increased hydrostatic pressure of the water decreases edema and improves lymphatic circulation. The water is often circulated with jets, and the patient receives the benefit of heat, massage, and increased hydrostatic pressure. Heating pads and infrared lamps have the potential to burn the patient and are not recommended, particularly for patients with neurologic disease who may be unable to move away from the source of heat. Precautions and contraindications for thermal therapy are listed in Box 11.9.[15] Tips for using hot packs are provided in Box 11.10.

Therapeutic Ultrasound

Human physical therapists have used TUS to treat patients with pain for many years. The major indication for use of this modality



FIG. 11.7 Therapeutic ultrasound being applied to the paraspinal muscles in a dog.

is deep tissue heating; however, other uses such as enhancement of wound and fracture healing by means of tissue modulation and modification of cellular function have been investigated (Fig. 11.7). Researchers have demonstrated nonthermal effects of TUS that include increased nerve conduction velocities of both sensory and motor nerves and improved nerve healing.[19–21] TUS works by the conversion of electricity to sound waves through the piezoelectric effect on the crystal in the transducer head of the machine. It is used to heat tissues from 1 to 5 cm in depth without superficial heating. The frequency, intensity, and mode of the treatment can be modified by the therapist according to the patient's condition. The thermal effects of TUS mirror those of superficial heating and include vasodilation, pain relief, increased flexibility of soft tissue, and decreased muscle spasm.[19–21] TUS requires a coupling medium to be placed between the sound head and the skin, as air attenuates the ultrasound beam. In general, a water-soluble ultrasound gel is smoothed over the skin, with the sound head having direct contact with the gel. TUS has some very specific contraindications and precautions (Box 11.11).[15]

Phonophoresis is the delivery of pharmaceutical agents using TUS. It is commonly used in human physiotherapy, but rarely in veterinary medicine. It can deliver local anesthetics or dexamethasone into painful muscles after surgery or injury.

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BOX 11.11 Precautions and Contraindications of Therapeutic Ultrasound

- Avoid therapeutic ultrasound over cardiac pacemakers, eyes, the gravid uterus or testes, spinal cord after laminectomy, open epiphyseal plates, bleeding areas, and areas of infection or malignancy as this may damage tissue, induce parturition, or cause metastasis.
- Use caution over fractures; in areas of decreased circulation, temperature, or pain sensation; in sedated animals; in areas of boney prominences; and over metal implants as this may damage tissue.

Data from Maxwell L. Therapeutic ultrasound: its effects on the cellular & molecular mechanisms of inflammation and repair. *Physiotherapy.* 1992;78:421; Raso VV, Barbier CH, Mazzer N, Fasan VS. Can therapeutic ultrasound influence the regeneration of peripheral nerves? *J Neurosci Methods.* 2005;142(2):185–192; and Kramer JF. Sensory and motor nerve conduction velocities following therapeutic ultrasound. *Aust J Physiother.* 1987;33(4):235–243.

Phonophoresis is used with muscle strain or painful muscles secondary to intervertebral disc disease surgery or medical therapy. Phonophoresis for these conditions is done using a compounded gel (dexamethasone 0.4%/lidocaine 0.1%). The settings currently used for phonophoresis treatments are 0.9 W/cm^2, 1 mHz, 100% duty cycle, and 4 minutes per probe head that will fit in the area treated.[22] Because veterinary patients are hairy, some therapists shave the area before TUS, whereas others use larger amounts of gel. However, Steiss and Adams have shown that ultrasound treatment applied through an intact coat considerably warms the coat with loss of thermal effects in underlying tissues.[23]

NOTE Clipping the hair, whether it is long or short, provides more consistent results with TUS and reduces the amount of heat lost in the hair coat.

Laser Therapy

Laser is an acronym for *light amplification by simulated emission of radiation*. Laser therapy is used in veterinary medicine to manage pain and inflammation and improve healing,[18,24] including promotion of healing following nerve and spinal cord injuries (Fig. 11.8). Research has shown laser therapy is associated with increased axonal sprouting and growth, increased myelination, reduced neuronal degeneration, and improved nerve recovery after injury.[18] Physiologic effects of laser stimulation are provided in Box 11.12.

Lasers used for therapy are different than surgical lasers and are considered low-level lasers or therapeutic lasers, depending on the laser's power. A laser is by definition collimated and monochromatic. The wavelength determines the penetration of the laser energy. Laser power is measured in milliwatts or watts. There are four classes of lasers that are divided according to the power produced. Class 1 lasers produce less than 0.5 mW (e.g., a supermarket scanner). Class 2 lasers produce up to 1.0 mW (e.g., laser pointers). Class 3a, 3b, and 4 are the classes into which most therapy lasers fall. Classes 3a, 3b, and 4 lasers produce 1.0 to 100 mW, 100 to 500 mW, and greater than 500 mW, respectively. Surgical lasers are class 4.

NOTE Laser therapy is beneficial after spinal surgery in dogs with intervertebral disc disease lesions of the T3–L3 region. If laser is applied daily for 5 days after surgery, time to ambulation is decreased.[8]

BOX 11.12 Physiologic Effects of Laser Stimulation

- Chromophores in the cell mitochondria absorb the laser light energy, causing increased production of oxygen, adenosine triphosphate, and DNA; increased cell permeability; and decreased cyclooxygenase and prostaglandin E2 production.
- Stimulation of stem cells.
- Promotion of tissue repair through angiogenesis, stimulation of fibroblast production and collagen synthesis, and growth factor release.
- Increased activity of leukocytes.
- Increased healing of nerves, connective tissue, and ligaments.
- Pain reduction through change in nerve conduction and increased levels of endogenous opiates or through stimulation of acupuncture points.

Data from Draper WE, Schubert TA, Clemmons RM, Miles SA. Low-level laser therapy reduces time to ambulation in dogs after hemilaminectomy: a preliminary study. *J Small Anim Pract.* 2012;53(8):465–469; and Looney A, Huntingford JL, Blaeser L, et al. A comparison of sham versus actual low level light therapy (LLLT) in treatment of canine elbow osteoarthritis pain and lameness. 44th Annual Veterinary Orthopedic Society Conference, Snowbird, UT, March 11–18, 2017.



FIG. 11.8 A dog with a shoulder injury receiving laser therapy. Note the protective eyewear that the persons administering the therapy and the dog are wearing.

NOTE To observe beneficial effects and mitigate pain, dogs with elbow arthritis and lumbosacral disease may need increased dosages of laser (i.e., 18–20 J/cm^2).[24]

Laser therapy is measured in joules (J) of energy, which is the power emitted over time. One joule is the amount of energy delivered by one watt of laser energy in one second. For example, it takes 2 seconds for a 500-mW laser to deliver 1 J of energy (500 mW × 2 seconds = 1 W second or J). A 10 W laser will deliver 1 J in ⅒ of a second. From a therapeutic standpoint, a higher-power laser will deliver a therapeutic dose in a shorter period. Research has shown different laser energy doses for therapy; however, for tissue healing the dose is usually 1 to

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BOX 11.13 Precautions and Contraindications for Laser Therapy

- Use protective eyewear and do not treat the eye area; laser light can damage the retina.
- Use caution over metal implants.
- Use with caution in seizure patients.
- Do not treat over the gravid uterus, neoplasia, growth plates, or open fontanels; this may cause metastasis or induce parturition.
- Dark-colored skin can absorb light and undergo excess heating, thus burning the patient.

Data from Niebaum K. Rehabilitation physical modalities. In: Zink MC, Van Dyke JB, eds. *Canine Sports Medicine and Rehabilitation.* Ames, IA: Wiley-Blackwell; 2013:115–131; Draper WE, Schubert TA, Clemmons RM, Miles SA. Low-level laser therapy reduces time to ambulation in dogs after hemilaminectomy: a preliminary study. *J Small Anim Pract.* 2012;53(8):465–469; and Looney A, Huntingford JL, Blaeser L, et al. A comparison of sham versus actual low level light therapy (LLLT) in treatment of canine elbow osteoarthritis pain and lameness. 44th Annual Veterinary Orthopedic Society Conference, Snowbird, UT, March 11–18, 2017.

5 J/cm^2 and for pain 4 to 10 J/cm^2. With lower-power lasers, such as class 3a and 3b lasers, spot treatment is generally done, whereas with a class 4 laser the dose is administered in a sweeping fashion. Precautions when using lasers are provided in Box 11.13.

NOTE When using class 3b and class 4 lasers in dogs with dark hair, hold the head of the laser off the skin by 1 inch to avoid heating the hair. Be sure the head is perpendicular to the skin.

Transcutaneous Electrical Nerve Stimulation and Neuromuscular Electrical Stimulation

TENS is one of two electrical stimulating modalities used in rehabilitation therapy for pain control, the other being NMES. TENS works on the sensory nerves to provide pain relief by applying high-frequency current to the skin and mediating pain relief through the gate theory of pain inhibition. TENS is generally applied immediately postoperatively, but it may be used to provide pain relief during treatment. The effect of TENS is transient in veterinary patients; however, longer-acting relief may be achieved using a high-intensity low-frequency current, known as *acupuncture-like TENS*, which is applied to acupuncture points and causes endorphin release. Precautions and contraindications when using any type of electrical stimulation, whether for pain relief or strengthening, are provided in Box 11.14.[15]

Different equipment is used for TENS compared with NMES. NMES is most frequently used in rehabilitation of neurologic patients (particularly tetraparetic or paraparetic animals) because it stimulates motor nerve and muscle fibers (Fig. 11.9). NMES contracts muscles, reduces edema, and assists with wound healing. In a weight-bearing animal it can be used to increase the force of contraction. It can also be used to assist gait retraining after surgery or injury. It is generally performed daily, or at least three times weekly, and with an amplitude sufficient to cause contraction. Postoperative NMES works best if done in the first 1 to 4 weeks after surgery or injury and is of questionable value in cases of lower motor neuron disease. Parameters for NMES are provided in Box 11.15.

Electrical stimulation is applied from the unit by leads and electrodes. These must be flexible and conform to the skin. The electrodes need to have low resistance and be highly conductive, inexpensive, and reusable. Most TENS and NMES machines have disposable electrodes meant for human use, which are of limited use in veterinary patients due to their hair. Nonsticky carbon electrodes that can have gel applied and are taped to the skin are preferable. Treatment time for both modalities is generally 15 to 20 minutes.

BOX 11.14 Precautions and Contraindications When Using Electrical Stimulation

- Do not use over the heart or in animals with pacemakers; this may induce abnormal rhythms.
- Do not apply over the pregnant uterus; this may induce parturition.
- Do not use over areas of neoplasia or thrombosis; this may cause metastasis or a thromboembolism.
- Do not use in animals with seizures.
- Do not apply over the carotid artery.
- Use with caution in areas of skin damage or decreased sensation.

Data from Niebaum K. Rehabilitation physical modalities. In: Zink MC, Van Dyke JB, eds. *Canine Sports Medicine and Rehabilitation.* Ames, IA: Wiley-Blackwell; 2013:115–131.

BOX 11.15 Parameters for Neuromuscular Electrical Stimulation

- Frequency: 25–50 Hz
- Pulse duration: 100–400 μs
- Ramp up and down: 2–4 seconds
- On/off time ratio: 1:3–1:5

Modified from Niebaum K. Rehabilitation physical modalities. In: Zink MC, Van Dyke JB, eds. *Canine Sports Medicine and Rehabilitation.* Ames, IA: Wiley-Blackwell; 2013:115–131.



FIG. 11.9 Proper application of neuromuscular electrical stimulation for muscle stimulation after surgery.

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Extracorporeal Shock Wave Therapy

Extracorporeal shock wave therapy (ESWT) has been used extensively since the 1970s to treat musculoskeletal problems in horses. In human medicine it has been used for lithotripsy and such musculoskeletal conditions as plantar fasciitis, calcifying tendonitis, and delayed unions. In dogs it has been used in the treatment of osteoarthritis, particularly elbow arthritis, supraspinatus tendinopathy, and patellar ligament desmitis.[25] Although the exact mechanism by which ESWT acts on bone is not fully understood, studies have shown greater osteogenic activity and strength in treated bone in addition to decreased inflammation, short-term analgesia, and increased vascularization of surrounding tissue. Extracorporeal shock wave generators produce acoustic waves of high pressure and velocity. These waves are characterized by high-amplitude acoustic pressures and differ from ultrasound waves because of their lower frequency, minimal tissue absorption, and lack of a thermal effect.[26]

ESWT can be delivered as focused or radial shock waves. Focused shock waves converge on a small target point and do not expose surrounding tissues to the same peak pressures as the focused point. There are three types of focused shock wave generators: *piezoelectric*, which uses crystals to generate the shock wave; *electromagnetic*, where magnetic fields produce the shock wave; and *electrohydraulic*, which uses a high-voltage spark gap that causes plasma bubble generation. In the latter, expansion and collapse of the bubble produces the shock wave.

Radial shock waves are generated pneumatically and move more slowly through the tissue with a lower pressure, reduced depth of penetration, and exposure of all tissues to the same pressure. The stimulation effects and therapeutic mechanism of focused verses radial shock waves are similar; however, radial shock waves are well suited for indications closer to the surface of the body, whereas for deeper target areas, focused shock wave therapy seems to be more favorable.[26]

Application of ESWT requires heavy sedation or light anesthesia because the procedure is painful. The area to be treated needs to be clipped and ultrasound gel applied as a coupling agent. The direction of the application and length of time depends on the condition and body part treated. In general, most conditions require 500 to 1000 shocks, with two treatments 3 to 4 weeks apart. Adverse effects can include bruising, petechiae, and pain. These patients commonly require nonsteroidal antiinflammatory drugs after treatment. Precautions and contraindications for ESWT are provided in Box 11.16.[26]

Manual Therapies for Pain Relief

Manual therapy techniques are used in canine rehabilitation practice to modulate pain; induce relaxation; improve tissue extensibility; increase ROM; mobilize joints and soft tissues; and reduce soft tissue swelling, inflammation, and edema. Common manual therapies include joint mobilizations, massage, passive ROM, and stretching. Some manual therapies require minimal training, whereas others, such as joint mobilization, require a high level of skill to be performed safely and effectively. Specific canine indications for manual therapy include pain and restricted motion secondary to canine hip dysplasia, elbow dysplasia, intervertebral disc disease, and osteoarthritis. Mobilizations, whether joint or soft tissue, are passive movements that are either oscillatory or a sustained stretch performed in such a manner that the patient can prevent the motion if so desired.[25,27] These motions can be performed anywhere within the available ROM.

BOX 11.16 Precautions and Contraindications for Extracorporeal Shock Wave Therapy

1. Do not expose the lung fields, brain, heart, major blood vessels, nerves, or a gravid uterus to shock waves; this may cause disruption in the organs, damage the fetus, or induce parturition.
2. Do not use near gas-filled cavities or organs because the pressure waves may enhance gas bubbles and damage the organs.
3. Do not apply over areas that are infected or over neoplasia or unstable fractures.
4. Do not use in dogs with immune-mediated joint disease or in those with neurologic deficits.

Data from Adamson CP, Taylor RA. Preliminary functional outcomes of extracorporeal shockwave therapy on ten dogs with various orthopedic conditions. *Proceedings of the Second International Symposium on Rehabilitation and Physical Therapy in Veterinary Medicine, Aug 10-14*, Knoxville, TN: University of Tennessee; 2002:195; and Ogden JA, Alvarez RG, Levitt R, Marlow M. Shock wave therapy (orthotripsy) in musculoskeletal disorders. *Clin Orthop Relat Res.* 2001;(387):22–40.

TABLE 11.3 Maitland Grades of Joint Mobilization

Grade	Description	Use
1	Fast, small amplitude at beginning of ROM	Pain relief
2	Slow, large amplitude, first half ROM	Provide early ROM, pain relief
3	Slow, large amplitude, mid to end ROM	Stretches joint capsule and ligament
4	Fast, small amplitude at end of ROM	Resolve inappropriate fibrous tissue adjacent to joint
5	Manipulation: small amplitude quick thrust	Chiropractic adjustment

ROM, Range of motion.
Data from Coates J. Manual therapy. In: Zink MC, Van Dyke JB, eds. *Canine Sports Medicine and Rehabilitation.* Ames, IA: Wiley-Blackwell; 2013:100–114.

Joint mobilization is used to assess the joint and improve the actions between joint surfaces (*joint arthrokinematics*). It involves stretching the joint capsule, ligaments, and any fibrous tissue that may be present. Joint mobilizations include glides or slides, oscillations (rolls, spins), distractions, and compressions of the joint. The goals of joint mobilization are to improve joint arthrokinematics, decrease pain, improve joint alignment, and reduce muscle spasm.[27] Joint mobilizations are graded according to the scale provided in Table 11.3.

Joint mobilizations are used after surgery or injury in any joints that are stiff and painful. The effectiveness of these manual therapy techniques depends on the skill level of the therapist. Joint mobilizations should only be practiced by those who have had sufficient training in these techniques. Contraindications to joint mobilization are provided in Box 11.17.[25]

Massage or Soft Tissue Mobilization

Soft tissue mobilization (STM) or massage involves tissue manipulation not associated with bones or joints. The pain-relieving effects of massage are produced through the release of endorphins and activation of neuropeptides, the intercellular messages within the nervous system. Manual manipulation of tissue can increase circulation, loosen stiff muscles, reduce excess interstitial fluid, and help minimize muscle atrophy (Fig. 11.10).

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BOX 11.17 Precautions and Contraindications for the Use of Joint Mobilization

- Do not use in areas of spinal instability.
- Use with caution if there is a neoplasm or infection (osteomyelitis) present in the adjacent bones.
- Be cautious if there are sutures over the joint.
- Perform with care if there is a recent, nonhealed, or partially healed fracture.
- Perform cautiously if the animal has unmitigated pain.
- Beware if the joint feels "bony" or empty or if there are muscle spasms.

Data from Coates J. Manual therapy. In: Zink MC, Van Dyke JB, eds. *Canine Sports Medicine and Rehabilitation*. Ames, IA: Wiley-Blackwell; 2013:100–114.

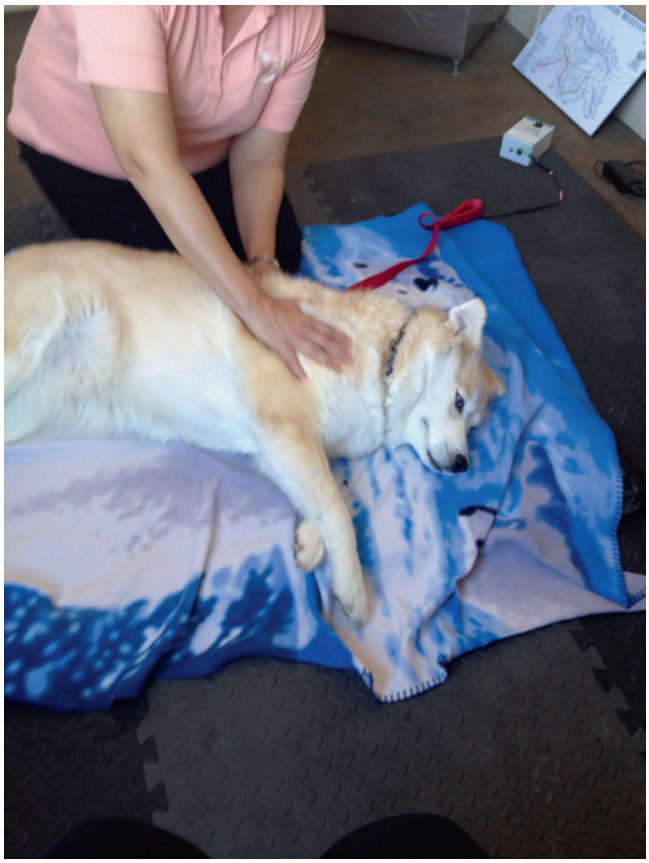


FIG. 11.10 Chronic pain patient receiving a massage.

Massage is particularly important in recumbent neurologic or orthopedic patients with paresis or painful and stiff muscles. If the dog is confined or on restricted exercise, STM can help retain muscle tone and condition. Teaching STM techniques to the owner provides psychological benefit to the dog and the owner associated with increased touch; in addition, the owners feel that they are participating in the dog's recovery. Several massage techniques can be used depending on the patient needs. Massage is generally done for 15 to 20 minutes and can be done several times a day, if needed (Video 11.2).

Various STM techniques may be chosen depending on the treatment goal and the patient's condition. *Effleurage* is long slow strokes with light to moderate pressure that runs parallel to the muscle fibers. It is often used at the beginning of the massage. *Petrissage* is short, quick strokes with moderate to deep pressure that is parallel perpendicular, or across the direction of the muscle fibers. Wringing, skin rolling, and kneading are all part of petrissage. Tapping on the muscle, or *tapotement,* is done with fingertips to stimulate weak muscles (Video 11.3). Friction or cross friction massage is performed by applying moderate pressure to scars, tendons, and ligaments to break up scar tissue. It can be performed perpendicular to the tendon, along the tendon, or circularly over the tendon and is usually done for 5 to 10 minutes. *Trigger point therapy* is a technique that applies digital pressure to a trigger point, which is a firm, tender area of muscle. Trigger points can be treated with pressure, dry needling, or injection.[28] Precautions and contraindications for massage are provided in Box 11.18.[28]

BOX 11.18 Precautions and Contraindications for Massage

- Fever or shock
- Areas of acute inflammation
- Infectious skin diseases
- Acute viral infections

Modified from Sutton A, Whitlock D. Massage. In: Millis DL, Levine D, eds. *Canine Rehabilitation and Physical Therapy*. 2nd ed. Philadelphia: Elsevier; 2014:464–483.

Passive Range of Motion and Stretching

ROM exercises and stretching are important after surgery, in neurologic patients, or for those with chronic disease. The normal ROM of a joint is affected by the joint structure and integrity and the type of soft tissue surrounding it. Normal ROM can be measured by a goniometer, with each joint having characteristic angles of flexion and extension (see Fig. 11.4). When joint motion is restricted, normal muscle ROM or functional excursion may also be restricted. Functional excursion of a muscle is defined as the amount the muscle can shorten after having been maximally elongated. Muscles that cross two joints, such as the biceps brachii, may lose functional excursion more quickly than muscles that cross only one joint. In these muscles in particular, passive range of motion (PROM) exercises are important to prevent disuse atrophy and mitigate the effects of immobilization.[28]

PROM involves taking the joint through its normal, comfortable ROM. It is performed by a therapist to increase muscle extensibility, prevent and treat stiffness, improve and maintain joint arthrokinematics, and to improve lymphatic drainage and circulation. PROM is necessary for all rehabilitation patients but is particularly useful for the nonambulatory patient. It can be accomplished with the patient in lateral recumbency, over a therapy ball or peanut, or in a sling or cart to maintain a standing position. The latter two allow gravity to pull the limbs down and provide sensory input to the limbs. Each limb should be done separately and at a minimum of three times a day. It is important to move all the joints of each limb when doing PROM, not just the joints on the affected limb. When performing PROM, the origin of the muscle is stabilized and the insertion moved (Fig. 11.11). A 30-second passive stretch is usually adequate. The stretch must be effective and not painful. Neurologic and postoperative patients are at risk for contractures of tendons and ligaments, which can be mitigated by PROM. Caution should be used when performing PROM in animals that are geriatric, have decreased sensation, or are in pain or when it is done immediately after surgery. Forceful PROM can damage bone and cartilage (Video 11.4).

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FIG. 11.11 Passive range of motion being performed in a patient with joint disease.

NOTE PROM works best on warmed muscles that have been heated with hot packs.

To avoid pain and spasm, flex and extend each joint of the affected limb approximately 10 times before flexing the entire limb.

Special Considerations for Neurologic Patients

Proprioceptive Neuromuscular Facilitation

After stretching and PROM, it is important to reeducate unresponsive muscles. In general, these are muscles that are antagonistic to those shortened through contracture, or by spasticity. This reeducation process takes place through PNF patterning. PNF is used to simulate movement patterns that are ingrained and functional. Common patterns include walking, running, scratching, sit to stand, movement from lateral to sternal recumbency, kicking back, and turning. PNF patterns can be done in any position depending on the disability. One example of PNF patterning is passive walking or running, which is often performed in paretic dogs in an underwater treadmill (UWTM) with a therapist moving the legs and placing the dog's feet. Use of therabands to assist in gait patterning is another example of PNF patterning. To be effective these patterns should be repeated multiple times per day.[29] Tips for stimulating a neurologic patient are given in Box 11.19 (Videos 11.5 to 11.7).

THERAPEUTIC EXERCISES

Therapeutic exercises are a crucial component of any patient's rehabilitation program, regardless of problem or diagnosis. Exercises need to be tailored to the individual patient considering

BOX 11.19 Tips for Stimulation of the Neurologic Patient

- Try a small vibrator or an electric toothbrush to stimulate weak muscles.
- Tap over muscle bellies to stimulate muscles to contract.
- Consider postural reflexes such as wheelbarrowing or hopping.
- Wrap a tensor around the patient from the front to the back to create awareness and connect front and rear of the animal. Alternatively, use a medical shirt or storm coat to do this.
- Remember that any sensory stimulation you can provide will stimulate the superficial receptors.
- Be sure to counsel clients regarding lifestyle changes (e.g., keep the animal on nonslip flooring, raise the food and water bowls, and don't allow the animal to use stairs or jump.)

the patient's age and physical condition, condition treated, and resources available. Exercises can be used for weight loss; to improve a patient's ROM; to improve muscle strength, weight bearing, flexibility, balance, and proprioception; to improve aerobic capacity (endurance) and performance; and to decrease pain and improve healing after surgery. Therapeutic exercises, as part of a home exercise program, allow owners to become involved in their pet's recovery and often strengthens the bond with a disabled pet.

After evaluating the patient, identifying problems and structure(s) involved, as well as the amount of tissue healing that has taken place, the therapist must set both long- and short-term goals. It is important that the owner's goals are in line with those of therapist. Each part of the program (proprioception, strength, endurance, and flexibility) should have specific goals, which will vary with the patient and injury. Once the goals are set and the program is designed, it is important for the therapist to evaluate the patient at each visit. Asking and documenting questions about soreness or activity after the last session is crucial. This allows the therapist to adjust the program as needed so the patient continues to progress through the rehabilitation process. At no time should the patient be harmed or uncomfortable. If soreness persists longer than 2 hours after exercise, the exercise may be too intense or the patient may need to be reevaluated. Prophylactic pain control and ice application after exercise may be needed, particularly for postoperative patients. In weak, ataxic, or nonambulatory patients, assistive devices must be used for exercise; this can include booties to protect feet from scuffing the floor or harnesses or slings to assist in ambulation. Frequency, intensity, and duration of exercise are increased as the patient heals to further challenge them and to strengthen muscles. It is usually safe to increase the activity by 10% to 15% each week if the patient does not experience an increase in pain or a loss of function.[23] It is important to monitor the patient for signs of fatigue (e.g., lagging behind, refusing to do exercises, panting, laying down, having a spade-shaped tongue, drooping of the tail and ears, or developing an elevated heart rate or muscle trembling). If these signs occur, one additional rep should be requested from the patient to preclude avoidance behavior. The patient's behavior the next day should be noted and the exercise program adjusted if necessary.[30–32]

Harnesses, safety vests, and leashes should be used to control the patient during exercise. Harnesses that do not restrict shoulder motion are used for rehabilitation. For patients that are not ambulatory or ataxic, front and back end harnesses may be used to assist both the patient and therapist (Fig. 11.12). Booties should be used on slippery surfaces or if the dog is weak and drags its

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FIG. 11.12 A poodle in an assistive harness on a BOSU ball. This harness has back and front end handles, making it easier to safely perform exercise.

feet. Doing exercises over balls, foam rollers, or peanuts or while in a sling can be useful for neurologic patients.

Equipment Required for Therapeutic Exercise

Many therapeutic exercises require minimal equipment; however, others require specific pieces of equipment such as physioballs or rolls, cavellettis, weave poles and cones, planks/blocks or stairs, balance boards or discs, therabands, mini-trampolines, or land treadmills (Table 11.4).

Balance and Proprioceptive Exercises

Balance and proprioceptive exercises are used to encourage limb use and improve body awareness and proprioception; some also strengthen selected muscles.[30–32] These exercises allow the patient to recognize that the affected limb is no longer painful and that it is possible to regain a normal posture. Some of these exercises can be started as early as 24 hours postoperatively.

Assisted Standing Exercises

These exercises are used for nonambulatory patients. *Support the patient with a harness or sling. Using the pelvis, assist the patient from a seated to a standing position. Encourage the patient to support weight for as long as possible. Increase the time as the animal gains strength and regains balance (Video 11.8).*

Rhythmic Stabilization/Weight Shifting

These exercises are used to increase weight bearing and balance. *Perform while the dog is standing on a flat, even surface. Place your hands on either side of the dog's ilium and slowly sway the dog from side to side, making sure both legs are weight bearing. Do not use enough force to cause the dog to lose its balance.*

TABLE 11.4 Equipment Required for Therapeutic Exercise Typically Found in a Rehabilitation Center

Equipment	Used To
Physio balls or rolls	• Build strength in all quadrants • Develop core strength, balance, and proprioception
Cavalettis or ground poles	• Train gait, improve proprioception, and strengthen the forelimb and hind limb flexor muscles • Improve active range of motion of all joints
Weave poles and cones	• Create lateral flexion of the spine • Strengthen the adductor and abductor muscles • Improve balance and proprioception
Planks/blocks or stairs	• Strengthen limb muscles • Develop core musculature • Improve proprioception
Balance boards or discs	• Develop strength and balance, particularly in the stabilizer muscles
Therabands	• Can be used as part of an exercise program to facilitate limb movement in neurologic cases, or • To facilitate muscle strengthening and gait training
Mini-trampolines	• Balance and proprioceptive training in cats and small dogs • Particularly useful following spinal surgery to regain core muscle strength
Land treadmills	• Can be used in place of outdoor walking to build cardiovascular fitness and strengthen limb muscles

Weight Shifting, Unsteady Surface

These exercises are used to improve weight bearing and proprioception. This exercise, which can be done in many ways, is performed on an unsteady surface such as a ball, air mattress, or cushion. *Start with two paws on the uneven surface and move the patient around it, then have the patient walk across the surface. Progress to standing on the surface while you apply gentle pressure to each hip or shoulder in a back-and-forth motion to challenge balance. Be sure to use a harness to control the patient so it does not lose balance and fall.*

Balance Boards, Balance Discs, or BOSU Ball

In addition to balance and proprioceptive training, this exercise also strengthens limb muscles (Fig. 11.13). *Place either both front or hind legs (the affected leg and contralateral unaffected leg) on the disc, or alternatively place all four feet on it. Control the patient with a harness and rock the board back and forth. To encourage the dog to walk and stretch while maintaining their balance, have it take treats from different locations on the board. Attempt to have the dog maintain balance despite movement of the board or disc. Start with 3 to 5 minutes twice daily and increase the duration as the patient gains strength. To increase the challenge, place two legs on a peanut or physio roll or BOSU ball.* For home exercises, cushions or air mattresses can be used.

Cavaletti Course

A cavaletti course strengthens limb muscles, improves joint ROM, and benefits all ambulatory patients, including geriatric patients,

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FIG. 11.13 A border collie on a rocker board used to improve balance and proprioception.



FIG. 11.14 A rottweiler using a cavaletti course to improve proprioception, increase joint flexion, and strengthen muscles.

canine athletes, and those who have had surgery (Fig. 11.14). Cavaletti courses come in many sizes and shapes and have been used extensively for training in the equine world. The distance between poles and their heights can vary depending on the animal's size and mobility, but the poles should be spaced an even distance apart. *Walk the animal very slowly over the poles while attempting to keep it from touching the poles. In general, start with 5 minutes twice daily and work up to 10 minutes twice daily.* For canine athletes, this exercise may eventually be done at a trot and as part of gait retraining. Other obstacles such as hockey sticks, pool noodles, or broomsticks can be used by the client at home (Video 11.9).

Weave Cones

For this exercise, place six to eight objects 1.5 to 3 feet (0.5 to 1 meter) apart (depending on patient size) and weave the dog in and out of the objects (Fig. 11.15). Use an odd number of cones to improve proprioception and flexibility on only one side of the body; use an even number to work both sides of the body. Pivot the dog sharply at the end of the pattern. Perform this exercise for 5 minutes twice a day. Postoperative orthopedic patients can start this exercise once the fixation is stable; neurologic patients can start once ambulatory (Video 11.10).

Figure Eights

Figure eight exercises are used to improve balance, coordination, spinal ROM, and weight bearing and can also be done with cones. They are completed by walking the outline of the number 8 around two cones. The figure eight is normally twice the height of the dog. *Perform this activity at a slow speed, which allows for an increase in spinal ROM. Do it for a few minutes at a time to avoid making the animal (or the therapist) dizzy.*

Planking on Blocks and Balls

These exercises are used to strengthen core muscles. *To perform, place one paw on each block with the animal in a standing position and have it hold the stance for increasing periods of time. Over time, move the blocks closer to midline or place both paws on one block. Alternatively, use peanut balls to simulate a plank exercise with the dog holding the plank for a period of time.*

Ball Work

Ball work increases proprioception, allows for advanced strengthening and balance, and increases core and trunk stability. Most exercises can be performed on a theraball. *Start the dog on solid ground; once the dog has mastered these exercises, slowly introduce balls. Use a large egg-shaped ball to work on balance and proprioception. Have the dog balance and do sit-to-stands on the ball. Start with four to five reps and work up to 10 to 12 reps, with three sets of each exercise (Video 11.11).*

Core Strengthening

Exercises that help strengthen the core include crawling, sit up and beg, and diagonal leg lifts.[30–32]

Crawling

Crawling improves spinal mobility and ROM and helps strengthen both fore and hind limbs. Dogs can be taught to crawl under beds, boxes, chairs, through an agility tunnel, or simply along the floor. The higher the crawling tunnel that is created, the easier the exercise is for the patient. *Begin with short distances and put treats along the tunnel to encourage the dog to move forward. Gradually increase the distance. Remember to add a command word and always reward while they are learning. Aim to have the dog crawl for 5 minutes per session twice daily (Video 11.12).*

Sit Up and Beg

This exercise helps core strengthening and hind leg strength. *Initially, have the dog sit squarely and then get up onto the back*

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FIG. 11.15 A German shepherd using weave poles to improve proprioception and flexibility.

legs as though it were begging. Use treats if necessary. You may need to hold one front leg if the dog is unstable until it is stronger (higher is easier for the dog). To strengthen the hind limbs once the dog can hold the beg position, ask it to stand on its hind limbs by holding the treat higher. Then ask the dog to get back into the beg position. The up and down from beg to stand is similar to performing squats for humans. *Eventually work up to two sets of 8 to 10 reps, twice daily (Video 11.13).*

NOTE Sit up and beg exercises should not be done if the dog has back or hip issues; this may aggravate already injured areas.

Diagonal Leg Lifts

These exercises help with core strengthening as well as balance, weight shifting, and leg strengthening (Fig. 11.16). *Lift one front leg off the ground along with the diagonal hind leg. Lift both legs simultaneously and provide minimal support to the limbs. Hold this pose for 10 to 30 seconds and repeat 8 to 10 times. When this exercise is easy for the dog, progress to having the dog stand on four blocks and then lift the diagonal legs. Finally, add some weight shifting to cause the dog to balance further.* Diagonal leg lifts on a therapy ball can be done as a further challenge for the dog (Video 11.14).

Hind Limb Exercises

These exercises include sit-to-stand, backwards walking, side stepping, incline walking, standing with elevated front legs, and ladder walking.[30–32]

Sit to Stand

This exercise helps improve strength in the pelvic limbs, particularly in the gluteal and hamstring muscles. *Ensure that the dog sits squarely and that he uses both pelvic legs to propel the body up.* A square sit occurs when the hips, stifles, and tarsi are in a straight line with the shoulders, which are perfectly aligned with the carpal joints. *If the dog kicks one leg out to the side, have the dog stand again and sit while placing your leg against the dog's paw, thus ensuring a square sit. Alternately, ask the dog to sit against the wall with the affected leg against the wall, thus preventing the leg from moving laterally. Repeat this exercise often for increased strengthening (Video 11.15).*



FIG. 11.16 A cat undergoing diagonal left lifts for core strengthening.

Backwards Walking

This exercise helps strengthen the pelvic legs (particularly the hamstring muscle group) and increase balance, coordination, and proprioception. *Start with the dog parallel to a wall, sofa, or anything straight. Hold a treat at chest level and walk toward the dog, saying "back."* Most dogs will try to turn while they are learning to walk backwards; thus it is important to reward the dog when it takes a step backwards. *Alternatively, have a couch parallel to the wall just far enough from the wall that the dog cannot turn around. Entice the dog forward with a treat, then say "back" and encourage it to walk backwards for another treat.* This is an important exercise to teach all dogs and puppies because at some time in their lives all dogs will need more hind limb strength. *Perform a backwards walking session for 5 to 10 minutes, twice daily (Video 11.16).*

Side Stepping

This exercise helps with both the fore and hind limb strengthening. *Stand with the front of your body facing the side of the dog, grasp the dog's collar in one hand, and hold the other hand on the dog's opposite hip. Then walk toward the dog, encouraging it to side step. If there is a problem controlling the back end of the dog during this exercise, use a front and back end harness for extra back end control. Perform this exercise both ways to strengthen the adductor, abductor, and pectoral muscles.*[47]

Incline Walking

This exercise helps build muscles in the rear legs due to the weight being shifted towards the back. *Start with shallow inclines and then add steeper inclines when the pet is comfortable and adjusting well. Incorporate zigzag hill walking, where the patient walks in a zigzag pattern across the hill, up and down. Start with 5 minutes and gradually work up to 20 minutes twice daily, incorporating hills (Video 11.17).*

Standing With Elevated Front Legs

This exercise is used to improve rear leg weight bearing and strength, ROM, and hip extension. *Have the owner sit on the stairs or a couch. With the dog's back legs firmly on the ground, place the front legs one or two stairs up or on the couch cushion (height depends*

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on dog's body length). This causes the dog's weight to shift to the pelvic limbs, thus causing the weaker hind legs to support the majority of the body weight. *Start with 10 seconds and work up 30 seconds at a time. Aim for 10 reps at a time (Video 11.18).*

Ladder Walking

This exercise strengthens legs and improves ROM, balance, coordination, and proprioception. The exercise is performed with a ladder flat on the ground and is similar to cavalettis but can be modified to be easy or difficult. *Initially, walk the dog forward over the rungs. Over time teach the dog to walk backwards and sidestep through the ladder. Start with 5 minutes twice daily and work up in frequency.*

Front Limb Exercises

Exercises specific for the front limbs include the high-five salute, wheelbarrow, stairs, play bow, and digging.[30–32]

High-Five Salute

This exercise helps with ROM and strengthening of the forelimbs. *Start with the dog sitting, then have the dog bring its paw up to its head and away as though saluting.* This particular exercise takes some training for most dogs. When "giving a paw," dogs usually only move their forelimb distal to the elbow. In this exercise, they need to move their shoulder to strengthen the extensor, abductors, and adductor muscles of the shoulder. *Start with a treat in your hand. When the dog paws at the treat, give the treat. Move your hand higher and into the position you wish the dog to reach (Video 11.19).*

Wheelbarrow

This exercise helps strengthen the front limb and the core. This may seem like an easy exercise, but it is quite challenging and needs to be done slowly and cautiously. *Never pick the dog up by the pelvic limbs. Start with lifting the patient by the caudal part of the abdomen so that the rear legs are a few inches off the ground. Observe contractions of the core muscles. Keep the stationary position for a while until the dog is comfortable with being lifted. Next, slow movement forwards and backwards can be added, making sure to monitor the animal's spinal alignment. Keep the spine perfectly straight to prevent injury. Do not push the patient too quickly; this can cause tripping and inevitably the dog's head will hit the ground. Do not do this exercise in a patient with spinal issues, shoulder problems, or carpal hyperextension.*

Stairs

Descending stairs helps build muscle in the front legs, as most of the weight is forced to the front half when walking down stairs. This exercise is very similar to walking on a decline. *Start with a shallow decline and gradually use a steeper decline as the patient progresses. Add changes only when the animal is comfortable and adjusting well. Start with slow stair walking 5 minutes twice daily and increase the time as the patient becomes stronger.*

Play Bow

This exercise helps increase forelimb strength and flexibility and promotes core strengthening. *Hold a treat in one hand and place your other hand under the patient's abdomen. Move the treat to the floor, allowing the dog's head and shoulders to follow while holding the abdomen up so that the back legs are standing and front legs are down. Do 8 to 10 reps initially and add more sets as the patient becomes stronger.*

Digging

Digging helps strengthen the front limbs and improves ROM, proprioception, and core strength. *When performing this exercise, have a command and a designated area where digging can occur, such as soft sand, soil, or light snow when in season. Do not use stones, which may hurt the paws. Bury a cookie or favorite bone so that the dog can dig it up.* Caution: some owners do not wish to encourage digging!

NEUROLOGIC REHABILITATION EXERCISES

Assisted Standing

This exercise builds and maintains muscles needed for balance, proprioception, and locomotion. These muscles quickly become atrophied if they are not used, which frequently occurs with paresis. *Place a therapy ball, rolled towel, cushion, foam roller, or other device (depending on the size of the dog) under the dog's abdomen.* While performing assisted standing, it is crucial to keep the pet's feet in a normal, anatomically correct standing position without knuckling. *Provide larger dogs with additional abdominal support from a sling or back end harness. If needed, have an assistant help place the feet.* Smaller dogs and cats generally require only one person if their abdomen is supported by a theraball. *Continue only until you see the first signs of fatigue. After a brief rest, repeat the procedure but do not exhaust the patient (Videos 11.20 to 11.22).*

Physio Ball

Physio ball work is particularly important for rehabilitation of neurologic patients. It can be used for assisted standing as described earlier, for assisted walking, and to regain balance and coordination. *Place the front end of the patient on the ball and rock it back and forth. If needed, have an assistant help place the feet on the ball. The dog may progress to sit-to-stands on the ball with assistance* (Fig. 11.17).



FIG. 11.17 A dog on a ball performing a sit-to-stand exercise. This exercise may need two people for a large dog.

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Weight Shifting

Weight shifting is used for neurologic patients that need to practice balancing on three limbs. *To perform this exercise, place the dog in a standing position and lift one limb. When the dog starts to sway, replace the limb. Rotate all four paws through this exercise.*

Treadmill Gait Training

Land and water treadmills are used for gait retraining for neurologic patients. Walking on a treadmill with a 5% incline has been shown to improve hamstring strength. The patient often must be supported with a sling. *Move the dog's legs in a proper gaiting pattern (right rear to right front, left rear to left front).*[10] *To overcome adductor spasticity, tickle the caudomedial aspect of the pelvic limb to prevent crossing over. As the gait becomes more normal, add walking and hills to the exercise (see Video 11.21).*

Tapping

Tapping is used as part of the nursing care for animals recovering from paralysis. *Tap over a muscle belly to elicit muscle contractions and stimulate the muscle spindle fibers and Golgi tendon organs in muscle and tendon. Perform for 3 to 5 minutes a few times a day (see Video 11.3).*

Tensor Bandaging or Medical Shirts

These items are used in dogs with neurologic conditions to connect the front and back end of the dog and create body awareness. *Place the wrap such that it exerts slight pressure on the body to help with the neurologic coordination.* Many of these dogs can wear such shirts or wraps throughout recovery.

Tactile Stimulation

Touch is used to provide stimulation to the superficial receptors in the patient's skin. *Brush the dog, tap, pinch, or use a vibrator to provide additional sensory stimulation to increase input into the nervous system.*

Mental Stimulation of Neurologic Patients

Mental stimulation is used to engage the patient mentally, which speeds recovery. Be aware that the patient's mental state plays a big role in recovery. Dogs can become depressed if they cannot move, go outside, or interact with their family. *Stimulate the dog by taking it outside or on "walks" in a wagon or stroller, moving around the house frequently if the patient is unable to move, or provide new treats or squeaky toys.*

AQUATIC THERAPY

Aquatic therapy can consist of deep water swimming or UWTM therapy. Water offers a safe and effective medium for improving strength, muscular endurance, cardiovascular fitness, ROM, agility, psychological well-being, and pain reduction. Metabolic energy requirements for exercise are greater when the same exercise is performed in the water versus on land. Shmalberg and colleagues have demonstrated that a dog trotting in a UWTM burns 5 to 10 times the energy of an equivalent trot on land.[33] Benefits of aquatic therapy are provided in Box 11.20.

Aquatic Therapy Equipment

Most rehabilitation services will have a pool, a UWTM, or both. However, aquatic therapy can take place in a wading pool, hot tub, bath tub, or a lake depending on the patient and the skill of the therapist. Studies have shown that a dog immersed in water to the level of the greater trochanter only bears 38% of the weight on his joints compared with the same dog on land.[34]

Life vests are needed for most patients and help ensure that the therapy is safe and effective. Ramps or other devices may be needed to get the patient into the water. Therabands, floating toys, small floating surf boards, pool noodles, and children's water wings are all essential pieces of equipment. The disadvantage of aquatic therapy is the initial cost and upkeep of a pool or UWTM. Precautions and considerations for aquatic therapy are listed in Box 11.21.

UWTM therapy is the preferred method of strengthening rehabilitation patients, whether the patient is ambulatory or not. UWTM therapy assists with both joint kinetics and kinematics. By increasing or decreasing the level and speed of the treadmill, joint flexion and extension are maximized, unlike swimming, in which normal kinematics are altered. Active ROM is also improved because the patients lift their legs higher while walking in the treadmill (Fig. 11.18). Gait retraining and sequencing is possible using the treadmill whether the patient is ambulatory or not. A therapist can assist the patient in ambulation by manual placement of the feet to stimulate muscle memory and retraining. Tetraparetic animals can be assisted in the UWTM by means of slings or harnesses. Two therapists may be required to assist these animals (see Video 11.6).

NOTE All dogs should have a therapist with them in the UWTM. This allows the therapist to quickly adjust if the patient requires it and provides added safety and enhanced efficacy of therapy.

BOX 11.20 Benefits of Aquatic Therapy to the Rehabilitation Patient

- Water provides buoyancy, which creates an environment of reduced gravity, thereby allowing greater joint range of motion with decreased pain, earlier postoperative intervention, early ambulation for neurologic patients, and a controlled exercise environment.
- Water exerts hydrostatic pressure, which reduces edema and nociceptor activity, which reduces pain.
- Water creates resistance due to its viscosity. This resistance strengthens the muscles as the patient pushes against it and provides stability. Dogs who are unable to walk on land often can stand and ambulate when in the water. Dogs with weak or atrophied muscles or obese patients that struggle to move on land often do well in the underwater treadmill.
- Warm water provides muscle relaxation.
- Water provides muscle stimulation for atrophied muscles.

Data from Levine D, Millis DL, Flocker J, MacGuire L. Aquatic therapy. In: Millis DL, Levine D, eds. *Canine Rehabilitation and Physical Therapy*. 2nd ed. Philadelphia: Elsevier; 2014:526–542.

BOX 11.21 Precautions and Contraindications for Aquatic Therapy

1. Prevention of wound infection—make sure all surgical incisions are healed before beginning aquatic therapy.
2. Panic by the patient—some dogs are afraid of water. In general, most patients tolerate the underwater treadmill better than swimming as their feet are on the ground.
3. Assessment of cardiovascular fitness—as it requires more energy to swim, postoperative, geriatric, and obese patients may tire easily. The therapist needs to schedule a number of breaks in each session to allow the dog to rest.

Data from Levine D, Millis DL, Flocker J, MacGuire L. Aquatic therapy. In: Millis DL, Levine D, eds. *Canine Rehabilitation and Physical Therapy*. 2nd ed. Philadelphia: Elsevier; 2014:526–542.

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FIG. 11.18 A dog with joint disease using an underwater treadmill.



FIG. 11.19 A dog swimming in a therapy pool. This is great cardiovascular conditioning for canine athletes.

With deep water swimming, life vests should be used and a therapist should assist the patient in the pool. A trained therapist can help the patient recreate movement patterns in the water that may not be able to be performed on land. Swimming is a demanding exercise and may not be appropriate for the postoperative patient (Fig. 11.19). The length of time that the patient swims depends on condition and degree of disability. Thirty-minute sessions are often used, with many rests between swim cycles. Vital signs need to be monitored by checking pulse, tongue, facial expression, and speed of swimming, as well as any change in emotional state. Pool noodles or flutter boards can be used for increased buoyancy, if needed. Deep water swimming is often added to the rehabilitation plan if the patient is a canine athlete that needs further cardiovascular conditioning. Tips for performing hydrotherapy are provided in Box 11.22.

SPECIAL CONSIDERATIONS FOR COMMON CONDITIONS

Common conditions that may require special consideration for rehabilitation include fracture patients, neurologic patients, patients post–joint surgery, and patients with osteoarthritis. Tips for dealing with each condition are included in Boxes 11.23 to 11.26.

BOX 11.22 Tips for Performing Hydrotherapy

- Maintain the water temperature between 82°F and 88°F, (27.5°C–31°C) depending on the time of year. This is comfortable for most patients.
- Fill the water level in an underwater treadmill such that it is at the level of the greater trochanter for most patients.
- Treadmill speed is dependent on dog size and condition. Generally, neurologic patients are started at 0.1–0.4 mph (0.16–0.6 kph) and adjusted accordingly. Most orthopedic patients start at 0.3–1.0 mph (0.5–1.6 kph) depending on the condition and the size of the dog. The dog needs to be able to walk at a normal pace to perform the appropriate gait pattern.
- Start most patients with three short sessions of 1–3 minutes with breaks for massage and passive range of motion.
- Use swim diapers for all neurologic patients. Express the bladder for patients with urinary incontinence and stimulate them to defecate by using a cotton swab in the anal area.
- Do not use hydrotherapy in animals with diarrhea, poor anal tone, or open sores.

Data from Levine D, Millis DL, Flocker J, MacGuire L. Aquatic therapy. In: Millis DL, Levine D, eds. *Canine Rehabilitation and Physical Therapy*. 2nd ed. Philadelphia: Elsevier; 2014:526–542.

BOX 11.23 Clinical Tips for Rehabilitation of Fracture Patients

- Consider the well-being of the entire patient. In early fracture healing, overuse can lead to implant failure; however, excessive cage rest can lead to delayed healing, contracture, decreased range of motion, and muscle atrophy. Remember safety but strike a happy medium.
- Delay hydrotherapy until incisions are healed to prevent infection.
- Use slings, harnesses, or assistive devices at all times in early rehabilitation to ensure safety and protect the fracture repair.
- Limit active exercise to 15 minutes twice daily for the first 4 weeks, increasing gradually.

BOX 11.24 Clinical Tips for Neurologic Patients

- Begin therapy as soon after surgery as possible.
- Use stimulation, proprioceptive neuromuscular facilitation patterns, and neuromuscular electrical stimulation early and often with these patients.
- Acupuncture, as discussed in Chapter 13, works well for stimulation of these patients.
- Use an underwater treadmill and laser therapy early in neurologic patients to reduce time to ambulation.
- Gait patterning is extremely important for these patients. See text on p. 117 regarding stimulation of, and exercises for, the neurologic patient.

BOX 11.25 Clinical Tips for Patients After Joint Surgery

- Work closely with the surgeon and have an understanding of the procedure performed and the limitations that it puts on the patient.
- Primary goal after joint surgery is to restore weight bearing to the affected limb as safely and quickly as possible.
- Animals with extracapsular cruciate repairs and femoral head and neck osteotomies can begin rehabilitation immediately after surgery.
- Patients who have undergone a tibial plateau leveling osteotomy procedure should start rehabilitation immediately, but water therapy should be delayed to prevent patellar desmitis. Treat these patients similarly to patients after fracture repair.

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BOX 11.26 Clinical Tips for Patients With Osteoarthritis

- Many of these patients are obese and calorie and protein requirements should be calculated. A dietary consult should be included for all patients with osteoarthritis.
- This is a waxing and waning disease that needs to be treated with multimodal therapy. Therapeutic exercises, laser, acupuncture, massage, heat therapy, and underwater treadmills are appropriate for these patients.
- These patients benefit from a home exercise program to help to manage their disease.

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12 Principles of Anesthesia and Anesthetics

GENERAL CONSIDERATIONS

Anesthesia of the small animal patient can be done safely when care and consideration are used in the selection and administration of anesthetic and analgesic agents and when anesthesia equipment is used correctly. The use of an anesthesia/surgery checklist is recommended to improve safety and to avoid potential errors. A routine checklist has been provided in Fig. 12.1.

Anesthesia (general and local), sedation, and analgesia (see Chapter 13) are used to alleviate pain, produce muscle relaxation and amnesia for humane and safe care of veterinary patients, and to create a quiet, nonmoving surgical field for surgeons. Before anesthesia or sedation the patient should have a complete physical exam (see Chapter 4). The American Society of Anesthesiologists has developed a physical classification system that is subjectively used to help determine the risk factor a patient may face when undergoing anesthesia (see Table 4.1). Fluid therapy (see later and Chapter 4) and blood replacement (see Table 4.5 and Box 4.1) are also important considerations during anesthesia.

General anesthesia is drug-induced unconsciousness that is controlled by reversible depression of the central nervous system (CNS) and perception. *Balanced anesthesia* is achieved by the use of multiple agents and techniques, with the goal of minimizing high drug doses and potential complications associated with the use of any one single agent. *Total intravenous (IV) anesthesia*, as the name implies, denotes the maintenance of general anesthesia without the use of inhalants. The classic stages of anesthesia (disorientation, excitement, anesthesia, and moribund) are provided in Box 12.1.

NOTE Be aware that when newer inhalant agents are given, or balanced or total IV anesthesia are used, the classic stages of anesthesia are not always reliable indicators of anesthetic depth.

Premedication is commonly given intramuscularly (IM) or subcutaneously (SC) to alleviate stress to the animal, provide analgesia and restraint, and decrease the amount of inhalant anesthesia that will be needed. In an animal that already has an IV catheter in place, premedication agents may be given intravenously or can be part of an induction protocol.

ANESTHETIC AGENTS

Numerous anesthetic agents are used in dogs and cats. Table 12.1 lists drugs and their class, doses, onset and duration of action, and their uses and precautions.

Antimuscarinics

Antimuscarinics (**atropine, glycopyrrolate**) competitively inhibit acetylcholine at parasympathetic muscarinic (cholinergic) receptors. Indications for their use are provided in Table 12.1. Antimuscarinics can cause transient slowing of the heart rate (including atrioventricular block) before the onset of the peripheral anticholinergic effect. This occurs because of inhibition of presynaptic cholinergic receptors that are part of the sympathetic pathways. Bradycardia is most common after IV administration and can last up to 3 minutes before an increase in heart rate occurs. It is more likely to occur with atropine than with glycopyrrolate because of the greater anticholinergic effects of atropine on the heart and because of its rapid onset of action. Usually this paradoxical bradycardia quickly resolves, but if necessary the dose can be repeated to cover additional cholinergic receptors to obtain the desired cardiac effect.[1] Glycopyrrolate is a quaternary ammonium that does not readily cross the blood-brain and placental barrier. The antisialagogue effect is more potent than that of atropine and, it is less likely to cause tachycardia.[1] See Table 12.1 for doses, onset and duration of action, and precautions.

Tranquilizers

Tranquilizers (e.g., acepromazine, diazepam, midazolam, zolazepam) are used to reduce anxiety and are not reliable by themselves for sedation or chemical restraint. It is important to remember that they do not have analgesic effects.

Phenothiazines

Acepromazine, a phenothiazine, antagonizes dopamine and serotonin neurotransmission (see Table 12.1 for dose and onset and duration of action). Dopamine antagonism creates calming and antiemetic effects. Acepromazine also blocks central norepinephrine and epinephrine transmission. The peripheral action is blockade of alpha-1 vascular receptors leading to vasodilation and potential hypotension. See Table 12.1 for precautions associated with the use of this drug. Acepromazine is metabolized in the liver and excreted by the kidneys and therefore should be used cautiously in patients with hepatic or renal dysfunction. In dogs with preexisting seizure disorders there is no clinical evidence that administration of acepromazine increases seizure activity.[2,3] It does cause marked relaxation of the splenic smooth muscle, leading to pooling of red blood cells in the spleen. It has minimal respiratory depressive effects, so it can be used to provide tranquilization in animals with respiratory distress. It is not a controlled drug, and there is no antagonist. Acepromazine has been used in combination with opioids, alpha-2 agonists, and dissociatives. It is generally only recommended for use in young and healthy patients. In this patient population, it can provide profound sedation that may assist in quieting animals, both preoperatively and postoperatively (see later).

Text continued on p. 131

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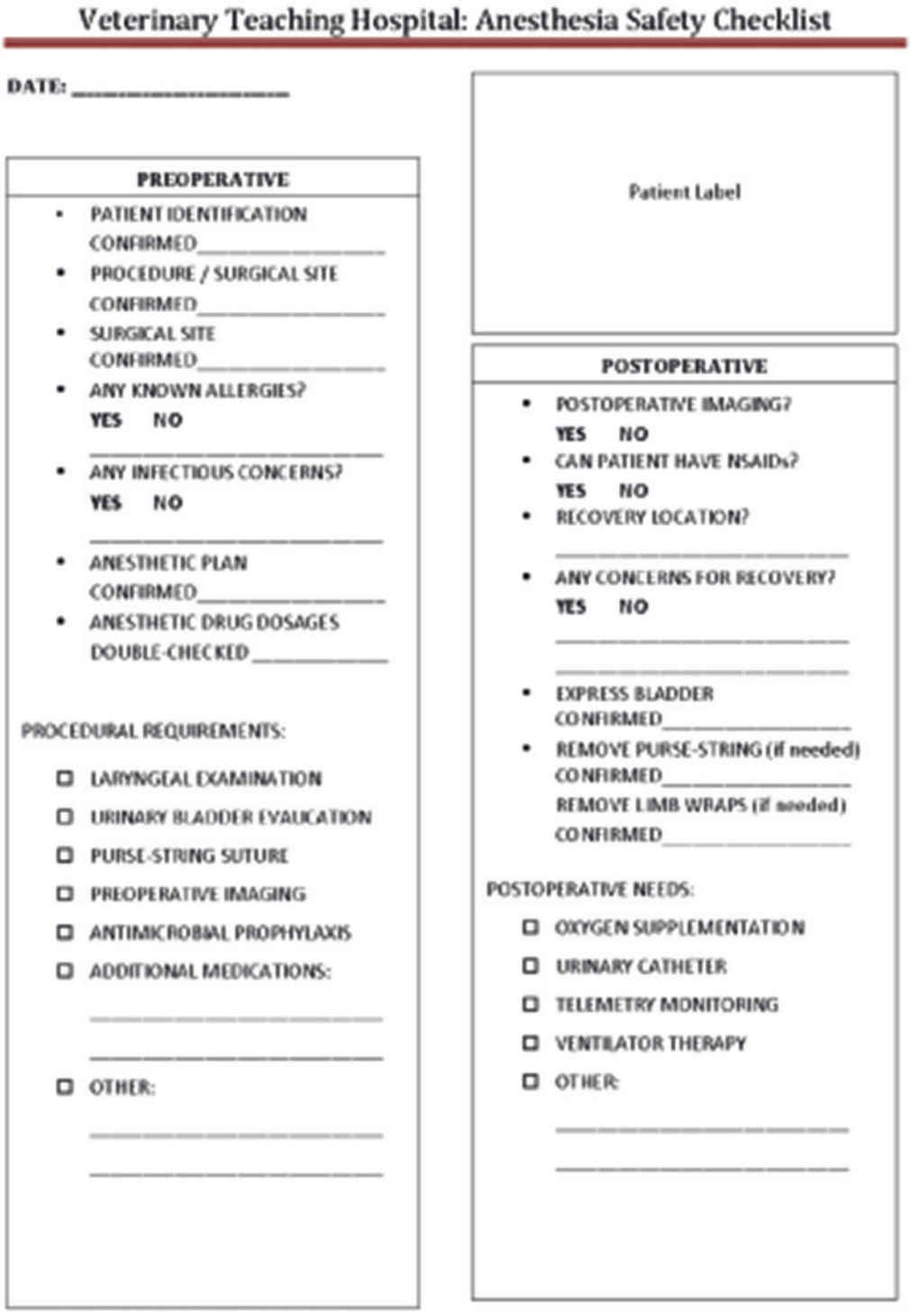
ABSTRACT

Safe anesthesia is part of a successful surgical procedure. Anesthesia can consist of general anesthesia, intravenous anesthesia, and local or regional blocks to supplement the anesthetic episode. Various drug classes are utilized to provide anesthesia and analgesia, including tranquilizers, alpha-2 agonists, dissociatives, opioids, and local anesthetics.

KEYWORDS

tranquilizers
alpha-2 agonists
dissociatives
opioids
local anesthetics

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Veterinary Teaching Hospital: Anesthesia Safety Checklist

DATE: _____

Patient Label

PREOPERATIVE

- PATIENT IDENTIFICATION CONFIRMED _____
- PROCEDURE / SURGICAL SITE CONFIRMED _____
- SURGICAL SITE CONFIRMED _____
- ANY KNOWN ALLERGIES? YES NO _____
- ANY INFECTIOUS CONCERNS? YES NO _____
- ANESTHETIC PLAN CONFIRMED _____
- ANESTHETIC DRUG DOSAGES DOUBLE-CHECKED _____

PROCEDURAL REQUIREMENTS:

- ☐ LARYNGEAL EXAMINATION
- ☐ URINARY BLADDER EVALUATION
- ☐ PURSE-STRING SUTURE
- ☐ PREOPERATIVE IMAGING
- ☐ ANTIMICROBIAL PROPHYLAXIS
- ☐ ADDITIONAL MEDICATIONS: _____ _____
- ☐ OTHER: _____ _____

POSTOPERATIVE

- POSTOPERATIVE IMAGING? YES NO
- CAN PATIENT HAVE NSAIDs? YES NO
- RECOVERY LOCATION? _____
- ANY CONCERNS FOR RECOVERY? YES NO _____ _____
- EXPRESS BLADDER CONFIRMED _____
- REMOVE PURSE-STRING (if needed) CONFIRMED _____
  REMOVE LIMB WRAPS (if needed) CONFIRMED _____

POSTOPERATIVE NEEDS:

- ☐ OXYGEN SUPPLEMENTATION
- ☐ URINARY CATHETER
- ☐ TELEMETRY MONITORING
- ☐ VENTILATOR THERAPY
- ☐ OTHER: _____ _____

FIG. 12.1 Example of a routine anesthetic safety checklist.

NOTE A rare idiosyncratic reaction of profound aggression has been reported in dogs following oral or parental acepromazine administration. The reported dogs did not manifest aggression prior to drug administration, nor on recovery from the drug effect. Though this is a rare event, due to the potential for serious injury, it is recommended that treated dogs be supervised when around other animals or children, especially if the dog has received acepromazine for the first time.[4]

NOTE Use acepromazine cautiously, if at all, in boxer dogs due to possible cardiovascular collapse.

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BOX 12.1 Stages of Inhalant Anesthesia

Stage I: Voluntary movement and excitement from initial administration until loss of consciousness. Release of epinephrine can occur during this stage.

Stage II: Delirium and involuntary movement until the onset of a regular pattern of breathing. Release of epinephrine can occur during this stage.

Stage III: Surgical anesthesia. Muscle relaxation develops. In this stage planes of anesthesia have been described:

- Light plane (plane 1): has eye movement reflexes.
- Medium plane (plane 2): has progressive intercostal paralysis.
- Deep plane (plane 3): has diaphragmatic respiration.

A normal surgical level of medium depth would be stage III, plane 2.

Deep surgical anesthesia would be stage III, plane 3, with a centered and dilated pupil.

Stage IV: Extreme central nervous system depression and a shock state. Death will ensue unless the anesthesia is withdrawn and resuscitation instituted.

Modified from Tranquilli WJ, Grimm KA. Introduction: use, definitions, history, concepts, classification, and considerations for anesthesia and analgesia. In: Grimm KA, Lamont LA, Tranquilli WJ, et al, eds. *Veterinary Anesthesia and Analgesia.* 5th ed. Ames, IA: Wiley-Blackwell; 2015:3–10.

TABLE 12.1 Anesthetic Agents

Anesthetic Agent/ Class	Dose	Onset and Duration of Action	Uses/Precautions
Antimuscarinics			• Used to prevent vagal-induced bradycardia and decrease excessive salivations Side effects include: • Tachycardia • Decreased respiratory secretion; makes secretions more viscous • Bronchodilation • Decreased GI motility • Transient bradycardia may occur before anticholinergic effect • May increase myocardial work and oxygen consumption
Atropine	0.04 mg/kg IM, 0.02 mg/kg IV	Onset of action: 15–20 min IM Duration of action: 1–1.5 h	• Bradycardia is more likely than with glycopyrrolate • Causes mydriasis • Can cause tachyarrhythmias
Glycopyrrolate (Robinul-V)	0.01 mg/kg IM, IV	Onset of action: 30–45 min IM Duration of action: 2–4 h	• Less likely to cause tachycardia than atropine • Does not readily cross the blood-brain barrier or placenta • More potent than atropine
Tranquilizers			• Used to produce a calming effect • Not reliable by themselves for sedation or chemical restraint (mild sedatives) • Do not have analgesic effects • Wide margin of safety • Can be given IM, IV, PO, transmucosally, or rectally
Benzodiazepines			
Diazepam (Valium), Schedule IV	0.2–0.5 mg/kg IM, IV CRI: 0.1–0.5 mg/kg/h	Onset of action: allow at least 20 min after intramuscular administration Duration of action: 1–4 h	• Reversible • Contains propylene glycol • Can cause phlebitis; use large vein for CRIs • Painful when given IM • Increasing the dose does not produce a proportional increase in sedation
Midazolam (Versed), Schedule IV	0.07–0.4 mg/kg IM, IV CRI: 0.1–0.5 mg/kg/h	Onset of action: 5–10 min Duration of action: 1–2 h	• Water soluble • Excellent absorption
Flumazenil (Romazicon)	0.01–0.02 mg/kg IV	Onset of action: rapid Duration of action: 1 h	• Used to reverse benzodiazepine effects • Expensive • Is not a scheduled drug
Phenothiazines			• Vasodilatory • Long duration of action • Does not provide analgesia

Continued

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TABLE 12.1 Anesthetic Agents—cont'd

Anesthetic Agent/ Class	Dose	Onset and Duration of Action	Uses/Precautions
Acepromazine (PromAce)	0.01–0.1 mg/kg IM, IV	Onset of action: IM, 35–40 min; IV, 5–15 min Duration of action: dose dependent, 3–8 h	• Do not give more than 3 mg total IM • Use with caution in boxers and animals with hepatic or renal dysfunction • Do not use in animals with hypovolemia, hypotension, or shock • May cause collapse when given intravenously to an extremely excited or stressed animal
Opioids			• Antidiuresis is due to release of ADH (which can lead to urinary retention) • Cause analgesia, antidiuresis, decreased GI motility, and sedation
Mu Agonists *Schedule II Drugs*			• May cause respiratory depression and bradycardia • Good analgesic • Minimal effect on CV parameters • Monitor cats for hyperthermia, most commonly occurs with hydromorphone • Reverse with naloxone • May need an antimuscarinic when given as a CRI
Morphine	*Dogs:* 0.2–2 mg/kg IM, SC CRI 0.1–0.3 loading dose then 0.1 mg/kg/h *Cats:* IM 0.05–0.4 mg/kg	Onset of action: IM, 5–10 min Duration of action: 3–6 h	• May cause emesis • Give IV slowly as it can cause histamine release
Oxymorphone (Numorphan)[a]	*Dogs:* 0.02–0.2 mg/kg IM, IV, SC *Cats:* 0.02–0.17 mg/kg IM, 0.02–0.05 mg/kg IV	Onset of action: IM, 5–10 min Duration of action: 1–3 h	• Safe to give IV; no histamine release
Meperidine (Demerol, Validus, Pethidine)	*Dogs:* 1–5 mg/kg IM, SC *Cats:* 0.5–5 mg/kg IM, SC	Onset of action: 5–10 min Duration of action: 1–2 h	• IV not recommended due to histamine release
Methadone (Dolophine, Methadose, Amidon)	*Dogs:* 0.2–1.5 mg/kg IM, IV *Cats:* 0.1–0.5 mg/kg IM, IV	Onset of action: 10–15 min Duration: 3–4 h	• Safe to give IV; no histamine release • Minimal incidence of vomiting • Poor sedation when given alone
Hydromorphone (Dilaudid)	*Dogs:* 0.05–0.2 mg/kg IM, IV, SC CRI 0.025–0.05 mg/kg IV loading dose, then 0.01–0.04 mg/kg/h *Cats:* 0.02–0.2 mg/kg IM, IV	Onset of action: 15 min Duration of action: 1–3 h	• Safe to give IV; no histamine release • Less likely than morphine or oxymorphone to cause vomiting
Fentanyl (Sublimaze)	0.005–0.015 mg/kg IM, IV, SC CRI for dogs 5–10 µg/kg IV loading dose, then 0.7–1 µg/kg/min, or 5–10 µg/kg/h CRI for cats 2–3 µg IV loading dose, then 0.3–0.4 µg/kg/min, or 2–5 µg/kg/h	Onset of action: 5–10 min Duration of action: 15–30 min Transdermal patch: time of drug uptake to effective drug concentrations is variable, depending on skin temperature, blood flow to the skin, and patch adherence to the skin: from 12 hours in cats to up to 24 hours in dogs. The duration of effect is approximately 4 days	• Safe to give IV; no histamine release • Large volumes needed for IM or SC • May cause bradycardia and respiratory depression
Remifentanil (Ultiva)	*Dogs:* CRI 0.25–0.5 µg/kg/min has been used in conjunction with a propofol CRI at 0.2 mg/kg/min *Cats:* 3 µg/kg IV; CRI 0.1–0.3 µg/kg per minute alone or in combination with ketamine at 0.5 mg/kg IV followed by 1.8 mg/kg/h	Onset of action: rapid Duration of action: short, must be given as CRI, half-life 6 min	• Elimination is independent of hepatic or renal function • Bradycardia may occur and may require treatment with an antimuscarinic • It is a potent respiratory depressant and may necessitate controlled ventilation

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TABLE 12.1 Anesthetic Agents—cont'd

Anesthetic Agent/ Class	Dose	Onset and Duration of Action	Uses/Precautions
Mu Antagonist/ Kappa Agonist			• Partial reversal of mu agonist drugs • Minimal CV effects • Mild analgesic
Butorphanol (Torbugesic,Torbutrol), Schedule IV	0.1–0.8 mg/kg IM, IV, SC CRI 0.1–0.2 mg/kg IV loading dose, then 0.1–0.2 mg/kg/h	Onset of action: 5–7 min Duration of action: 45 min–1.5 h	• Can be used to partially reverse mu opioid agonist but maintain kappa effects
Nalbuphine (Nubain)	0.25–2 mg/kg IM, IV, SC	Onset of action: 5–10 min Duration of action: 1–2 h	• Minimal to no sedation; use with a tranquilizer or sedative • Not a scheduled drug
Partial mu Agonists: Schedule III Drugs			
Buprenorphine (Buprenex, Simbadol)	0.005–0.02 mg/kg IM, IV, SC *Cats:* 0.24 mg/kg once a day for up to 3 days, SC	Onset of action: 20–30 min Duration of action: 3–6 h Onset of action: 1 hr Duration of action: 24–48 h	• Effects difficult to reverse • Good for moderate pain • Large safety profile • Adverse side effects may include hyperactivity, difficulty in handling, disorientation, agitation and dilated pupils; but more commonly cats become quiet, euphoric, purring, rubbing and kneading with their forepaws.[7]
Opioid antagonist: Naloxone (Narcan)	0.001–0.04 mg/kg IM, IV	Onset of action: 5 min Duration of action: 30–60 min	• Reverses opioid effects • May cause tachycardia, hypertension, pain, and excitement • Renarcotization may occur when used to reverse a long-acting opioid
Opioid agonist and inhibits reuptake of serotonin and norepinephrine: Tramadol; Schedule IV	2–10 mg/kg every 6–12 h	Onset of action: variable Duration of action: variable, hours	• Mu agonist • Metabolite m1 has activity • Centrally acting • Naloxone will only partially antagonize effects of tramadol • Unpleasant taste • May see sedation at higher dose
Dissociatives			
Ketamine (Ketaset, Ketaved, Vetalar); Schedule III	4–11 mg/kg IV, IM CRI 0.5 mg/kg IV loading dose then 0.1–0.5 mg/kg/h	Onset of action: 10 min Duration of action: dose dependent, 20–30 min	• Can cause excess salivation, so it is commonly used with an antimuscarinic • May increase heart rate and ICP and IOP • Renal elimination in the cat • Well absorbed, but stings on injection • Can cause seizure-like activity in dogs when given alone, so it is commonly used with benzodiazepines • Will cross the placenta and lead to depression of neurologic reflexes in cesarean-delivered neonates
Tiletamine (dissociative) and zolazepam (benzodiazepine; Telazol); Schedule III	2–4 mg/kg IM, IV as part of premed or induction Diagnostic procedures: *Dogs:* 6.6–9.9 mg/kg IM *Cats:* 9.7–11.9 mg/kg IM Minor procedures (e.g., laceration repair): *Dogs:* 9.9–13.2 mg/kg IM *Cats:* 10.6–12.5 mg/kg IM	Onset of action: 5 min but can vary Duration of action: dose dependent, 20–30 min	• As given for ketamine • Profound hypothermia and prolonged recovery may occur • Most useful for restraint or very short procedures • Because onset of action is variable, observe the patient carefully after administration • May cause respiratory depression • Do not use in animals with pancreatic disease, renal pathology, or severe cardiac or pulmonary dysfunction • Do not use for caesarean section • Do not use with phenothiazine-derivate drugs • Do not exceed maximum allowable dose for supplementation of increment sedation or total anesthesia of 26.4 mg/kg IM for dogs and 72 mg/kg IM for cats

Continued

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TABLE 12.1 Anesthetic Agents—cont'd

Anesthetic Agent/ Class	Dose	Onset and Duration of Action	Uses/Precautions
Barbiturates			
Thiopental (Pentothal); Schedule III	Titrate to effect: 4–20 mg/kg IV	Onset of action: ultra-short acting; 20–30 s Duration of action: short (15 min), dose dependent	• Can cause CV and respiratory depression • Can decrease ICP and IOP pressure • Effects may be potentiated by concurrent acidosis or hypoproteinemia • Tissue necrosis if given perivascularly • See caution in text regarding sighthounds, greyhounds, wolfhounds, and Afghan hounds
Propofol (Diprivan); Schedule IV	2–8 mg/kg IV only CRI 0.05–0.4 mg/kg/min	Onset of action: rapid acting Duration of action: 5–10 min	• May cause respiratory and myocardial depression, peripheral vasodilation, and Heinz body anemia with repeated use in cats • Not an analgesic • Use it with caution in patients with volume depletion or CV compromise
Propofol 28 (PropoFlo); Schedule IV	2–8 mg/kg IV only CRI 0.05–0.4 mg/kg/min	Onset of action: rapid acting Duration of action: 5–10 min	• Can be used in healthy cats as a single induction dose, not as a CRI • Cumulative toxic effects include ataxia, hyperesthesia, fasciculations, blindness, aggression, convulsions, respiratory failure, and death
Etomidate (Amidate)	0.5–4 mg/kg IV dog dose 1–2 mg/kg IV cat dose	Onset of action: rapid Duration of action: single IV dose 10–20 min	• Contains propylene glycol, may cause hemolysis • Maintains CV stability • Suppresses adrenocortical function for 2–6 h following a single bolus dose • Side effects include involuntary muscle movements, hypertonus, and tremors, which can be minimized by giving IV benzodiazepine beforehand • Additional side effects are vomiting and excitement
Sedatives			
Alpha-A Agonists			• Cause sedation, analgesia, muscle relaxation • Pancreatic insulin inhibition causes hyperglycemia • ADH inhibition causes diuresis
Dexmedetomidine (Dexdomitor)	2–40 µg/kg IM, IV, transmucosally CRI 1 µg/kg loading dose, then 0.5–2 µg/kg/h	Onset of action: 10–20 min Duration of action: 3 h	• Initially causes vasoconstriction and hypertension, followed by hypotension • May cause cardiovascular collapse • May cause vomiting and/or aerophagia • Provides good sedation and analgesia • Commonly combined with opioids or ketamine • Use in healthy animals only; avoid using in very debilitated, old, or young animals
Xylazine (Rompun)	0.1–1 mg/kg IM, IV, SC	Onset of action: 5–10 min Duration of action: 20–30 min	• Causes emesis in cats and occasionally in dogs • Causes bradycardia • Dogs may develop bloat from aerophagia
Alpha-2 Antagonist			• Side effects include tachycardia, systemic and pulmonary hypertension, and anxiety • IM administration is preferred to avoid rapid vasodilation • If giving a reversal agent intravenously, it must be given slowly to avoid sudden awakening and excitement or potential cardiac arrest because the patient may experience sudden pain with resultant catecholamine surge
Atipamezole (Antisedan)	0.04–0.5 mg/kg IM, IV, SC Equal volume of dexmedetomidine	Onset of action: 10 min Duration of action: 2–3 h	
Tolazoline	0.5–4 mg/kg IV, IM, SC	Onset of action: within 5 minutes of IV administration Duration of action: short, may need to be redosed	
Yohimbine	0.05–0.1 mg/kg slow IV, 0.25–0.5 IM, SC	Onset of action: within 3 min when given IV Duration of action: 1.5–2 h	• Doses higher than 0.1 mg/kg are recommended to be given IM or SC • High doses can cause excitement in dogs

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TABLE 12.1 Anesthetic Agents—cont'd

Anesthetic Agent/ Class	Dose	Onset and Duration of Action	Uses/Precautions
Neuroactive Steroid			
Alfaxalone (Alfaxan); Schedule IV	1–5 mg/kg IV; CRI 0.07–0.11 mg/kg/min	Onset of action: rapid Duration of action: dose dependent, 14–50 min	• Can cause respiratory and CV depression, rapid onset, and is not analgesic • Sedation may be needed to improve recovery • Does not contain a preservative
Other			
Lidocaine	CRI 1–2 mg/kg IV loading dose, then 1–3 mg/kg/h	Onset of action: rapid Duration of action: IV bolus, 10–20 min	• Can be used to provide analgesia and decrease MAC • Is antiarrhythmic and can scavenge free oxygen radicals • Not recommended for use in cats due to CV depression
Inhalants		Anesthetic depth can be adjusted rapidly; rapid uptake and recovery	• Produce dose-dependent CV depression and peripheral vasodilation • Decrease metabolic rate, mean arterial blood pressure, and cerebral perfusion pressure
Isoflurane MAC: dog = 1.2; cat = 1.63	MAC: dog = 1.2; cat = 1.63		
Sevoflurane (Ultane)	MAC: dog = 2.1; cat = 2.58		
Desflurane	MAC: dog = 7.2; cat = 9.8		• Requires a special heated vaporizer due to high vapor pressure

[a]At the time of publication, oxymorphone was not readily available; however, a dose has been provided throughout in the event it is compounded or becomes available.
ADH, Antidiuretic hormone; *CRI,* constant-rate infusion; *CV,* cardiovascular; *GI,* gastrointestinal; *ICP,* intracranial pressure; *IM,* intramuscularly; *IOP,* intraocular pressure; *IV,* intravenous; *MAC,* minimum alveolar concentration; *SC,* subcutaneously.

Benzodiazepines and Benzodiazepine Antagonists

Benzodiazepines potentiate the primary inhibitory neurotransmitters of the CNS, gamma-aminobutyric acid type A ($GABA_A$) receptors. There are five main pharmacologic effects: anxiolysis, sedation, anticonvulsant activity, spinal cord–mediated muscle relaxation, and amnesia. Benzodiazepines undergo hepatic metabolism. Animals receiving benzodiazepines alone become disinhibited; therefore the behavioral response can vary from patient to patient and from species to species. Some animals become quiet and sedate, whereas others can become more vocal, excited, euphoric, dysphoric, and even aggressive. Dysphoria and aggressive behavior limit the use of benzodiazepines, especially when given alone in cats. These unwanted side effects can persist postoperatively when longer-acting drugs, such as diazepam, are used (see Table 12.1). Benzodiazepines are usually used in combination with other agents such as opioids, sedatives, or dissociatives; however, they can be used alone in neonatal or extremely ill or depressed animals. Benzodiazepines are a schedule IV agent. They have minimal effect on the cardiovascular and respiratory systems and do not produce analgesia.

Diazepam (see Table 12.1) is highly lipid soluble and has a prolonged duration of action. Liver metabolism produces active metabolites with prolonged half-lives that undergo renal excretion. Diazepam can cause hypotension if given rapidly intravenously and may be painful on injection; slow administration and dilution with a crystalloid will decrease these side effects. Diazepam is often given at induction with ketamine, barbiturates, propofol, etomidate, or opioids. Its use in these combinations takes advantage of its sedative and relaxation properties and allows reduced dosing of the other induction medications.

Midazolam (see Table 12.1) can be given intravenously and does not create phlebitis or pain on injection. It has a rapid onset of action and rapid metabolism. It is especially useful as a premedication when combined with an opioid in pediatric, geriatric, and ill animals. Midazolam undergoes hepatic metabolism and renal excretion. It is useful in combination with ketamine, propofol, etomidate, opioids, or barbiturates for induction. *Zolazepam* is combined with tiletamine, an *N*-methyl-D-aspartate (NMDA) receptor antagonist, and is most useful for restraint or for very short procedures. See precautions in Table 12.1. *Flumazenil* is a benzodiazepine antagonist (see Table 12.1). It works well but is expensive. It is not a controlled agent.[1]

Recommendations for Handling Aggressive or Dangerous Dogs

Oral medications can be given to aggressive or dangerous dogs when administration of parenteral medications is difficult or not possible. The goal is to quiet and calm the dog to allow safe handling and for subsequent administration of parental agents. Medications given orally require a longer onset time to be effective than when given parenterally. When working with aggressive or dangerous dogs it is important to communicate to the owner the potential risks and side effects of drugs used in a patient that has not had a complete physical exam. Additionally, it is imperative that you know the patient's general health status. Low-stress handling measures, such as avoiding a busy waiting room, going straight to a quiet exam room, and keeping the dog in a quiet environment, are helpful. Tips for administering medications to aggressive or dangerous dogs are provided in Box 12.2. If possible have the owner administer the agent(s) orally into the cheek pouch or squirt it into the mouth (see Box 12.2). Oral-transmucosal administration avoids the first-pass hepatic effect, and the oral mucosa's rich blood supply allows for therapeutic circulating concentrations to be achieved.[5–7] The "chill protocol" developed by Dr. Alicia Karas is useful for anxious pets. The protocol requires that the owner give oral

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BOX 12.2 Drugs That May Be Useful When Handling Aggressive or Dangerous Dogs

Detomidine, Oral Gel

- 0.5–2 mg/m^2 applied to the buccal pouch
- Onset of sedation: ~30–45 min
- Duration of sedation: ~30–90 min
- Considerations and precautions
- Dogs will develop bradycardia

Dexmedetomidine, Injectable

- 16.1–40 µg/kg sprayed into the mouth or buccal cheek pouch via a 3 mL syringe with a 22-gauge needle using forceful pressure from a distance of 0.6 m
- Onset of sedation: ~11–34 min
- Duration of sedation: ~30–70 min (longer sedation occurs with the oral gel)
- Considerations and precautions:
  - Level of sedation is sufficient to allow basket muzzle placement or safe restraint
  - Reversal can be done by injection of atipamezole IM

Telazol, Injectable

- Dose: 10–20 mg/kg in combination with acepromazine at 2 mg/kg sprayed into the cheek pouch, as described for dexmedetomidine
- Onset of sedation: sternal and lateral recumbency will occur at approximately 50 minutes
- Duration of sedation: return to normal ambulation may take up to 5 hours
- Considerations and precautions:
  - Dogs may be slightly to moderately ataxic
  - Not all dogs will become recumbent
  - May have a prolonged effect

"Chill Protocol"[a] (Use in Combination With the Below)

- Dose:
  - Gabapentin, 20 mg/kg PO the night before the visit or early the morning of the visit; consider reducing the dose in elderly dogs
  - Melatonin: 1–5 mg PO (1 mg for small dogs, up to 5 mg for dogs >40 kg), give with gabapentin
  - Acepromazine: 0.025–0.05 mg/kg PO, have the owner give 30 minutes prior to leaving the house (dose is the same as for an injection IM)
- Onset of sedation: see earlier
- Duration of sedation: variable; the dog may be quiet for as long as 24 hours, return to normal ambulation may take up to 5 hours
- Considerations and precautions:
  - The owner must supervise the pet as the pet will be wobbly and may misjudge stairs and jumping from the car.
  - Gabapentin is a sedative and anxiolytic with renal elimination; use with caution in dogs that are dehydrated or that have renal disease.
  - Acepromazine may cause hypotension and is a sympatholytic; use with caution in sick patients; consider giving the owner two syringes without needles in case they spill one.

[a]Courtesy Alicia M. Karas.

medications prior to the visit (see Box 12.2). When the dog arrives, quietly place a muzzle on it and then inject other medications IM, or place an IV catheter for drug injection. Further sedation may consist of opioid, dissociative, or alpha-2 agonist combinations.

NOTE Remember to remove the dog's muzzle after injection of any medication that is known to cause vomiting.

Alpha-2 Agonists and Antagonists

Alpha-2 agonists cause central presynaptic inhibition of norepinephrine release, have vagomimetic actions, and induce peripheral activation of postsynaptic alpha receptors in the vasculature, pancreas, and gastrointestinal tract. They undergo hepatic metabolism. Effects from administration of an alpha-2 agonist are sedation, analgesia, muscle relaxation, bradycardia, peripheral vasoconstriction, and vomiting. There is an initial vasoconstrictive-hypertensive phase followed by a hypotensive phase. Bradycardia is due to vagal stimulation and reflex response to the hypertension, leading to a decreased cardiac output. Heart rate and rhythm should be monitored, as the bradycardia can progress to second-degree atrioventricular block. Use of anticholinergics to treat the bradycardia is controversial. An anticholinergic given when the blood pressure is high may lead to extreme hypertension and tachycardia. In this case, it may be better to reverse the alpha-2 agonist to improve the heart rate. If the blood pressure is normal to low, consider the use of an anticholinergic to treat bradycardia. Animals that are highly stressed or excited may not sedate well. Sedated animals may still respond with a reflex action such as biting. Alpha-2 agonists can be used in combination with opioids and dissociatives. Alpha-2 agonists are not controlled drugs and are reversible.

Dexmedetomidine (see Table 12.1) is the most potent of the alpha-2 agonists and has profound, long-lasting effects. It provides good sedation and analgesia. It is useful as a restraint drug, especially when combined with ketamine and an opioid. If reversed, it is recommended to give the reversal agent (atipamezole; see below) IM to avoid sudden awakening that could lead to excitement or pain, and profound hypotension. Dexmedetomidine can be combined with opioids to decrease the dose of both drugs and create neuroleptanalgesia. When used as premedicant it will decrease anesthetic drug doses. It can be part of a technique for injectable anesthesia for minor procedures or used to treat dysphoria and anxiety during anesthetic recovery.

Alpha-2 antagonists include *yohimbine, tolazoline,* and *atipamezole,* the latter of which was designed for use with dexmedetomidine (see Table 12.1). Of these, atipamezole is the most selective and effective. These reversal agents antagonize both sedation and analgesia. If an alpha-2 agonist has been used in combination with a dissociative, reversal of the alpha-2 agonist may result in unmasking of the dissociative that permits its adverse effects (i.e., muscle rigidity, excitement) to be seen.[1]

Opioids

Opioids can be used as preanesthetics, intraoperatively, and postoperatively to provide sedation and analgesia (see Table 12.1). They are controlled drugs and are reversible. The opioid receptors are mu (µ), kappa (κ), and delta (δ). Mu receptors can also cause emesis, (which is most likely when given IM or SC), euphoria, and respiratory depression. Opioids have minimal cardiovascular compromise, so they are commonly used in unstable patients. Bradycardia can occur and may be treated with antimuscarinics. They can cause a dose-dependent respiratory depression. Dogs may pant excessively due to thermoregulatory center disruption or initial stimulation of the respiratory center. Euphoria and dysphoria can occur in some dogs and is common in cats, but occurs less commonly when combined with a tranquilizer or sedative. Opioids should be used cautiously in animals with head injury as hypoventilation can increase CO_2, which causes

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cerebral vasodilation and increases intracranial pressure. Opioids can be used safely if intermittent positive pressure ventilation is provided. They undergo hepatic metabolism. All mu opioid agonists are schedule II drugs that require strict record keeping. Full mu agonists are morphine, hydromorphone, oxymorphone, fentanyl, remifentanil, meperidine, and methadone.

Morphine is an excellent analgesic (see Table 12.1) that undergoes 50% hepatic and 50% extrahepatic metabolism. Morphine, when given rapidly intravenously, can result in histamine release leading to profound hypotension. *Oxymorphone* (see Table 12.1) is an excellent premedicant in pediatric, geriatric, and debilitated animals and is often combined with midazolam. *Hydromorphone* (see Table 12.1) is less likely to cause vomiting as compared with morphine or oxymorphone. Hyperthermia may be seen in cats during recovery following anesthesia, and hydromorphone may make them more prone to this complication. Cats that are hypothermic during surgery or the procedure seem to rebound and become hyperthermic in recovery; thus all cats should have their temperature monitored closely in recovery.[8]

NOTE Hyperthermia seen in cats during recovery is usually self-limiting, but remove heating sources as a precaution. You may need to place them on a metal cage floor. A low dose of acepromazine (0.01 mg/kg IV), if safe to give, will calm them, and may decrease body temperature due to vasodilation.

Fentanyl is a potent mu agonist commonly used intravenously as a constant-rate infusion (CRI) to provide analgesia (see Table 12.1). It can be used in intensive care settings at the lower dose range to provide analgesia. Fentanyl also comes as a transdermal patch. The skin should be clipped and cleaned prior to patch application. In some animals pain may not be completely controlled and adverse opioid effects may occur. The patch is easily removed and may be consumed by the animal or children, which may be toxic. The potential for human abuse is also present. *Methadone* is a synthetic mu agonist with some NMDA antagonist effects. It is less likely to cause vomiting than morphine; however, it does not create good sedation when given alone. *Meperidine* can cause tachycardia because it has a chemical structure similar to atropine. Meperidine may produce serotonergic effects, so it should not be used with other serotoninergic drugs or monoamine oxidase inhibitors.

Remifentanil is a synthetic opioid with direct action on mu opioid receptors. It has an ultrashort duration of action and is metabolized by nonspecific esterases in blood and tissue. The metabolite is excreted by the kidneys. Remifentanil has been used with propofol as a CRI to provide total IV anesthesia in dogs. It provides intraoperative analgesia when given as a CRI. Remifentanil does not affect myocardial contractility or cause histamine release but may lead to hypotension. It is good for use in animals with liver compromise or for short procedures and has been used in dogs for liver biopsy.[9] Its use decreases the amount of inhalant needed for general anesthesia, which may help preserve hepatic blood flow. Remifentanil has been used in cats as a CRI to decrease the level of inhalant needed for general anesthesia.[10] Recovery is very rapid, within 5 to 10 minutes, even after prolonged infusions as there is no drug accumulation. There is no residual analgesia, so a longer-duration analgesic should be given to patients that have had a painful procedure. This analgesic should be given prior to recovery from remifentanil. There is no residual respiratory depression following recovery from remifentanil.

Buprenorphine is a partial mu agonist and kappa antagonist. It is commonly used in cats and can be given transmucosally on the oral mucus membranes. Buprenorphine has a large safety profile. A new proprietary formulation of buprenorphine (Simbadol) has been developed for use in cats.[11] The first dose should be administered approximately 1 hour prior to surgery. It has a high affinity for the mu opioid receptor. Adverse side effects may occur (see Table 12.1), but cats more commonly become quiet and euphoric, and they purr, rub, and knead with their forepaws.[11] Buprenorphine in metabolized and excreted by the liver. Reversal with naloxone may be difficult. It should *not* be dispensed to the owner for administration.

Butorphanol is a kappa agonist and mu antagonist. It creates mild analgesia. It does not give reliable sedation by itself, so it is often combined with a tranquilizer or sedative. Butorphanol is used to treat mild to moderate pain; it is not expected to be completely effective for severe pain. It is an effective antitussive. It can be used to reverse mu agonist effects, but maintain some analgesia at a dose of 0.1 mg/kg IV.

Nalbuphine provides mild analgesia but minimal to no sedation. It can be used to reverse mu agonist effects, but maintain some analgesia. It is not scheduled. *Tramadol* (see Table 12.1) is metabolized to O-desmethyltramadol (M1), which has mu agonist activity; in addition it acts as a serotonin and norepinephrine reuptake inhibitor. The analgesic effects are weak in dogs because they do not make substantial amounts of O-desmethyltramadol. Cats, however, produce substantial amounts of O-desmethyltramadol, so it may be an effective analgesic in this species, but the bitter taste makes oral administration difficult. It is not recommended to give tramadol with meperidine, tricyclic antidepressants such as amitriptyline or clomipramine, or selective serotonin reuptake inhibitors such as fluoxetine and paroxetine, as there is a risk of serotonin syndrome, clinical signs of which include fever, seizures, muscle tremors/fasciculations, hyperthermia, salivation, and rarely death.

Barbiturates

Thiopental, an ultra-short-acting thiobarbiturate, is used for induction. It comes as a crystalline powder and is reconstituted to 2.5%, 5%, or 10% solutions with sterile water or saline. In small animals a 2.5% solution is most commonly used to avoid vascular irritation. The resulting solution is alkaline (pH 10–11) and can cause tissue necrosis if injected perivascularly. If perivascular administration occurs, dilution of the drug with saline injected into the tissue should be done to help minimize tissue damage. Lidocaine can also be injected into the site to produce vasodilation and local anesthesia.[12]

Barbiturates produce CNS depression by activation at the $GABA_A$ receptor. When given intravenously they equilibrate rapidly across the blood-brain barrier to the well-perfused brain, inducing anesthesia. The drug concentration in the blood and brain falls rapidly as the drug redistributes to less perfused muscular tissue, allowing for the return of consciousness. Repeated dosing will result in a prolonged recovery as muscle tissue reaches equilibrium with blood concentrations. Metabolism is hepatic, with hepatic enzyme P450 system induction and renal excretion. The barbiturates are cerebral protectants; cerebral metabolic oxygen, cerebral blood flow, and intracranial pressure are decreased. These properties make thiopental an appropriate choice for induction of patients with intracranial disease and

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seizures. Thiopental can decrease stroke volume and myocardial contractility. Venodilation and sequestration of red cells occurs in the spleen with an accompaniment increase in splenic size. Ventricular arrhythmias and bigeminy may occur, and thiopental sensitizes the myocardium to epinephrine-induced arrhythmias. There is a dose-dependent respiratory depression, which may result in apnea. Barbiturates are a good choice for laryngeal exam as the laryngeal reflexes are minimally affected. They cross the placenta, causing more respiratory depression in puppies following a cesarian section than does the barbiturate-like sedative propofol (see later). Barbiturates do not produce antinociception, and the only analgesia seen is during unconsciousness; therefore analgesics should be given with painful procedures.

NOTE Greyhounds can have prolonged recoveries following thiopental administration due to a relative deficiency in hepatic microsomal enzymes needed for metabolism, and also because of their lean bodies and low fat stores. Barbiturates are not commonly recommended for use in sighthounds, greyhounds, wolfhounds, or Afghan hounds but can be used if there is appropriate premedication to decrease the amount of barbiturate needed.

Propofol is highly lipid soluble and is formulated as an emulsion. It is used for IV induction and, because it has a short duration and does not accumulate in the body, it is safe to use as a CRI for total IV anesthesia. The original formula does not contain a preservative and will support bacterial and fungal growth; therefore strict aseptic technique in drug handling is required. An open drug vial should be discarded after 6 hours, and when giving as a CRI the administration set should be changed every 12 hours. The anesthetic effect is through interaction at the $GABA_A$ receptor. Propofol must be given intravenously to be effective, though if given perivascularly it does not cause tissue necrosis. It has a rapid onset and reduces cerebral metabolism of oxygen and intracranial pressure. Propofol does have anticonvulsant effects and has been used in seizure control. It is a dose-dependent cardiovascular depressant and vasodilator leading to hypotension. A dose-dependent respiratory depressant effect often causing apnea also occurs. Propofol crosses the placenta but has a rapid clearance from the neonatal circulation. It does not provide analgesia.

PropoFlo 28 is new formulation of propofol that has a shelf life of 28 days due to the preservative benzyl alcohol. It has the same properties and dose as regular propofol. It comes in a multiuse vial at 10 mg/mL and is an emulsion. It is labeled for IV use in dogs; it is not labeled for use in cats due to the benzyl alcohol, which may be toxic to cats because they lack adequate glucuronic acid conjugation, which results in a decreased rate of metabolism and cumulative toxic effects (see Table 12.1). This new formulation has been used in healthy cats as a single IV induction dose with no adverse effects.[13] In those cats that are ill, have liver disease, will need multiple anesthetic episodes, or require a CRI, the original propofol is recommended. Repeated administration of propofol, daily or as a CRI, may induce oxidative injury to feline red cells.

Nonbarbiturate Hypnotics

Etomidate (see Table 12.1) is an imidazole derivative with agonist activity at the GABA receptor. Etomidate causes vasoconstriction of the cerebral vasculature leading to reduced cerebral blood flow and metabolism of oxygen. It is given intravenously for induction and is quickly hydrolyzed by esterases in the liver and plasma to inert metabolites. It has a rapid onset and may be painful upon injection. This pain may be reduced by IV administration of an opioid prior to etomidate, use of a large vein, and administration through a running IV line. Hemolysis can occur due to an osmolality of 4640 mOsm/L from the propylene glycol in the formulation. Hemolysis may be more likely in cats due to the fragility of their red blood cells. There is a lack of cardiovascular depression, and thus minimal effect on cardiac function, that makes it useful for induction of unstable cardiac patients. Etomidate has a minimal effect on the respiratory system. The drug does cause adrenocortical function suppression of 2 to 6 hours following an induction dose. Care should be taken if using this drug in patients with adrenocortical disease and if highly stressed. Long duration of this drug as a CRI is not recommended. There are no analgesic properties.

Neuroactive Steroids

Alfaxalone (Alfaxan) is a clear, aqueous solution for IV injection, registered for the induction and maintenance of anaesthesia in dogs and cats. It is a neurosteroid injectable anesthetic. It does not contain a preservative, so an open vial must be discarded after 6 hours. It causes CNS depression by binding to GABA receptors. It has rapid liver metabolism and elimination. There is a wide margin of safety. It is labeled for IV use only in dogs and cats. It is water soluble, nonirritating, and therefore not painful when given IM, which is useful in cats and small dogs. It is useful combined with an opioid for intramuscular sedation in the aggressive ill cat. Cerebral blood flow, intracranial pressure, and cerebral metabolic oxygen demands are decreased. There is dose-dependent cardiovascular depression, respiratory depression, and apnea. There is good muscle relaxation and rapid recovery. The animal may have a rough recovery, with paddling, muscle rigidity, myoclonus, and excitement. Adding a sedative or analgesic may help avoid this. Alfaxalone may be an alternative to propofol. Alfaxalone has been used for safe induction of anesthesia in dogs undergoing cesarean section; it had a negligible effect on the puppies.[14]

Dissociatives

The dissociatives, *ketamine* and *tiletamine*, interfere with transmission of nervous impulses between the thalamocortical and limbic systems, causing a dissociation between subconscious and conscious systems. There is noncompetitive antagonism of NMDA-type glutamate receptors. They provide dose-dependent effects ranging from mild chemical restraint to general anesthesia with a wide margin of safety. They can be used IM to sedate dogs and cats, especially aggressive animals (see Box 12.2). They can be used for IV induction and IM or intravenously for injectable anesthesia. Ketamine is used as a racemic mixture; the positive (S) isomer produces more intense analgesia, has more rapid metabolism, and has a lower incidence of emergency reactions than the negative I isomer. Tiletamine is only available in 1:1 combination with the benzodiazepine zolazepam (Telazol). It comes in powder form, is reconstituted with 5 mL of diluent, and loses potency over time. Dogs may have a rough recovery due to slow metabolism of tiletamine, causing muscle rigidity, sympathetic stimulation, and emergence delirium; a tranquilizer may need to be given to smooth recovery. In cats, the action of the benzodiazepine zolazepam lasts longer. The tiletamine/zolazepam powder can be reconstituted with 100 mg of xylazine and 400 mg of ketamine to give a potent combination used for

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chemical restraint/anesthesia. Careful drug administration is necessary due to the small volume used; profound hypothermia and prolonged recovery may occur. The dissociatives do not have a reversal agent, but zolazepam can be reversed with flumazenil and an alpha-2 antagonist can be used to reverse xylazine.

The dissociatives have a rapid onset of action with a short duration. There is hepatic metabolism, with ketamine producing the active metabolite norketamine. In dogs, norketamine is inactivated prior to renal excretion, whereas in cats, norketamine is excreted unchanged in the urine. Emergence delirium can be seen on recovery; treatment with agents such as benzodiazepines, acepromazine, or an alpha-2 agonist may decrease the incidence and severity of the delirium. CNS effects include an increase in cerebral metabolism of oxygen and intracranial pressure. The stimulation of the CNS leads to increased sympathetic outflow, resulting in increased blood pressure, heart rate, cardiac output, myocardial work, and oxygen requirements. When the catecholamine stores and sympathetic nervous system compensatory system are exhausted, ketamine has direct negative inotropic effects. Dissociatives can cause excess salivation and are therefore commonly used with an antimuscarinic. The dissociatives do not cause significant respiratory depression. They do not produce muscle relaxation; increased tone of extraocular muscles can create increases in intraocular pressure. Muscle relaxation is improved with the addition of a benzodiazepine or alpha-2 agonist. Dissociatives do provide somatic analgesia. The blockade of the excitatory neurotransmitter glutamate at the NMDA receptor plays a role in the prevention or minimization of central sensitization and wind-up. These agents are most commonly used in combination with tranquilizers, opioids, and alpha-2 agonists.

Inhalants

Inhalants are widely used to provide anesthesia. They do require special equipment for use but have a rapid onset and recovery via the lungs. The two commonly used inhalant agents are isoflurane and sevoflurane (see Table 12.1). The delivery gas for inhalants is oxygen. Anesthetic potency is defined by the minimum alveolar concentration of an anesthetic that produces immobility in 50% of patients exposed to a supramaximal noxious stimulus. These potent inhalants cause vasodilation that leads to a loss of cerebrovascular autoregulation, and the loss of vascular resistance increases cerebral blood flow. There is a subsequent increase in intracranial pressure. There can be a decrease in cardiac output and stroke volume due to a dose-related depression in myocardial contractility. The inhalants cause respiratory depression, which may necessitate intermittent positive-pressure ventilation (IPPV).

Anesthesia Equipment and Devices

Animals needing general anesthesia for longer procedures should be intubated to provide an airway for delivery of oxygen and inhalant gases, protect the lung from aspiration, and prevent contamination of the environment with waste gases. Intubation also allows for IPPV to help prevent hypoxemia and overcome respiratory depression; ventilation is required during a thoracotomy or diaphragmatic hernia repair.

Endotracheal Tubes, Endobronchial Blockers, and Supraglottic Devices

Endotracheal tubes are used to seal the trachea during inhalant anesthesia or to protect the airway in an anesthetized dog or cat. The most common type used in veterinary medicine is the **Magill tube**, which ranges in size from 3 to 40 mm (internal diameter). These tubes have a beveled tip and are curved and may be cuffed or uncuffed, although cuffed tubes are most commonly used. Uncuffed tubes may be used in very small animals, where the cuff may be sufficiently large to prevent placement of a tube of adequate diameter. **Cole tubes,** originally designed for pediatric anesthesia, are less commonly used in veterinary medicine but have been used in cats. These tubes are designed such that the shoulder of the tube touches the larynx and provides an air-tight seal. The size of the tube is based on the weight of the animal (Table 12.2); however, remember that weight guidelines are based on lean body weight and that overweight animals do not have larger tracheas than thinner animals. Brachycephalic breeds typically have narrower tracheas than dogs or cats of similar size that are mesocephalic or dolichocephalic. It is wise to have a tube one size larger and one size smaller readily available.

TABLE 12.2 Suggested Endotracheal Tube Size Based on Animal Body Weight

Body Weight (kg)	Endotracheal Tube Size (mm Internal Diameter)
Cats	
1	3
2	4
5	4.5–5
Dogs	
2	5
4	6
7	7
9	7–8
12	8
14	9–10
16–20	10–12
30	12
40	14–16

From McKelvey D, Hollingshead KW. *Small Animal Anesthesia: Canine and Feline Practice.* St. Louis, Mosby; 1994.

NOTE Lube the tube before inserting, being careful to not occlude the "eye" of the tube. Use a cuffed tube if possible and inflate the cuff gently to avoid traumatizing the trachea. Check for leaks in the tube cuff prior to induction.

One lung ventilation (OLV) can be achieved using endobronchial blockade, selective intubation, or a double-lumen tube (DLT). Ventilating one lung is done to improve surgical conditions for thoracoscopy, to control contamination or hemorrhage, or when there is unilateral pathology. Endobronchial intubation can be done with a long endotracheal tube and is relatively easy to perform; the disadvantage is that it provides less direct control to make changes in the nonintubated lung.

A **DLT** is two single-lumen tubes bonded together and comes as either a right- or left-sided tube, corresponding to the right or left mainstem bronchus where the tube is designed to fit. The

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tube has two cuffs; one occludes the trachea and the other occludes the bronchus. The later cuff and pilot tube are usually colored blue to differentiate them from the tracheal cuff. Correct tube placement is confirmed by direct visualization using a small-diameter bronchoscope. Correct placement should result in lung sounds only in the ventilated lung field. A DLT is capable of providing gas flow directly to a bronchus isolated from the rest of the lung by an expandable cuff or through a tracheal port that provides gas flow cranial to the obstructed bronchus. They are designed with an angled bronchial tip that can be inserted into either the left or right mainstem bronchus. This allows for ventilation of the lung field in which the bronchial tip has been placed into that mainstem bronchus. If the fresh gas flow is directed to the tracheal lumen only, then ventilation of the contralateral lung field will occur. Thus when the DLT is correctly positioned, there is flexibility in choosing to ventilate left, right, or both lung fields. Blind placement of the DLT with thoracoscopy assistance is possible.

There are three DLTs available that have been used in dogs: the Robertshaw left-sided tube, Carlens left-sided tube, and Dr. White right-sided tube. The Robertshaw left-sided tube is the simplest design with a tip that is angled to the left approximately 30 degrees to the main body of the tube. It has a tracheal lumen that opens just before the tip angulation point. The bronchial tip of the tube has a near-spherical balloon that inflates just oral to the termination of the tube, which has an end-hole design. The Carlens tube is similar to the Robertshaw tube but incorporates a small hook just distal to the opening of the tracheal lumen. This hook is designed to catch on the carina and prevent passage of the tube deeper into the bronchial tree. The Dr. White right-sided tube is similar in design but has a right-sided angulation to the tube tip. It incorporates the carinal hook similar to the Carlens tube but has an L-shaped balloon with a side- and end-hole bronchial tube tip. This bronchial tube tip is aimed at providing ventilation to the more cranially positioned right cranial lobar bronchus. A thoracoscope can be used for tube placement allowing for direct observation of lung lobe inflation during ventilation. It is possible to have the tubes positioned too far cranially, resulting in partial prolapse of the balloon into the trachea. Complete balloon prolapse into the trachea obstructs the lumen and results in total airway obstruction in some cases (Fig. 12.2).[15]

An advantage of the DLT is that they allow for alternating hemithorax OLV by simply switching the anesthetic gas supply from the bronchial lumen to the tracheal lumen without having to use intraoperative bronchoscopy to guide tube manipulation. The carinal hook may not offer an advantage when used in dogs, as they are more difficult to guide through the larynx and have greater potential to create laryngeal trauma as they are advanced through the rima glottidis.[15]

Endobronchial blockers (EBBs) are long catheters tipped with an elliptical or round inflatable cuff, which is blue. Bronchial blockers are used coaxially or in parallel with a standard endotracheal tube. The bronchial blocker is placed with fiberoptic–assisted direct visualization; a single lung lobe or the entire hemithorax can be blocked. Once the blocker is correctly placed, the cuff is inflated to prevent ventilation to that lung segment. EBBs are used in small dogs as the smallest DLT are 28 Fr, which precludes their use in animals weighing less than 10 kg. EBBs have been described for creation of OLV in small patients as they are available in a 5 Fr size.

Supraglottic airway devices (SGAD) include laryngeal mask airways and V-gels (Fig. 12.3). These devices do not require the use of a laryngoscope for placement. They are an alternative to the standard endotracheal tube and may be easier to place. When properly placed, they fit over and form a seal around the glottis. A specific SGAD, V-gel, has been designed for cats. It is relatively easy to place and has been used to maintain inhalant general anesthesia in cats undergoing abdominal surgery.[16] Placement of the V-gel may require a more superficial level of anesthesia and less induction agent when compared with a standard endotracheal tube. Cats with a V-gel in place have undergone controlled mechanical ventilation up to 16 cmH_2O pressure with no leakage.[17] The endotracheal tube or SGAD is attached to the breathing circuit, which, in turn, is connected to the anesthesia machine.

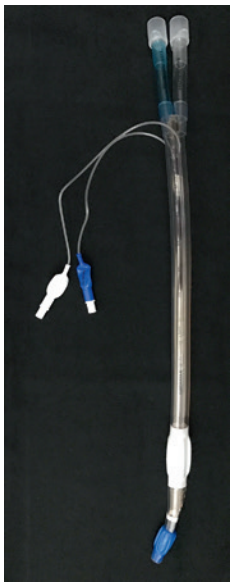


FIG. 12.2 A left-sided Robertshaw double lumen endotracheal tube. Double lumen tubes can be used to selectively ventilate one or both lung fields.



FIG. 12.3 Supraglottic airway device, feline V-gel (Docsinnovent Ltd.).

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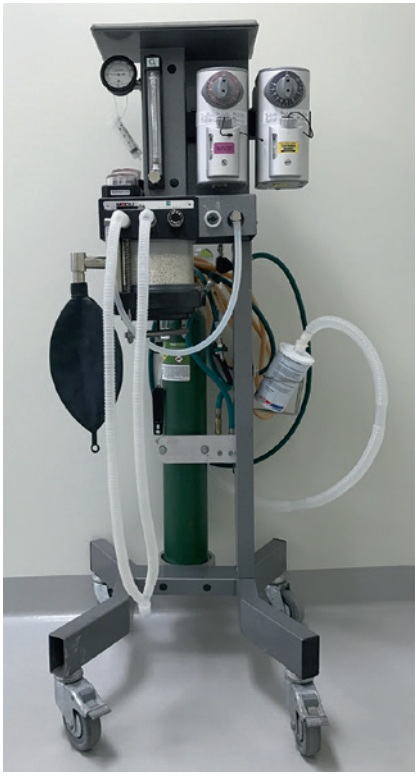


FIG. 12.4 The circle rebreathing system on a small animal anesthesia machine.



FIG. 12.5 Nonrebreathing circuit (Norman Elbow), Mapleson F, Jackson-Rees with the relief valve located on the bag. Note the circuit connection to the common gas outlet.

Anesthesia Machines

The anesthesia machine consists of a source of oxygen, an oxygen regulator and flowmeter, and a precision vaporizer. All waste gases should be scavenged. The breathing circuit directs oxygen and inhalant to the patient, removes carbon dioxide or prevents rebreathing, and provides a means of controlling ventilation.

The **circle or rebreathing system** is the most commonly used system in veterinary anesthesia (Video 12.1; Fig. 12.4). The circle system produces a unidirectional flow of gas and absorbs carbon dioxide from exhaled gas. The components are a fresh gas inlet, inspiratory one-way valve, breathing tubes, expiratory one-way valve, adjustable pressure limiting valve or "pop-off" valve, reservoir bag, and carbon dioxide absorber. The fresh gas flow determines how much rebreathing occurs. For complete rebreathing, the oxygen flow rate is metabolic oxygen consumption of the patient, 3 to 14 mL/kg per minute. Partial rebreathing is greater than metabolic oxygen consumption, 20 mL/kg per minute. Nonrebreathing to minimal rebreathing occurs when the oxygen flow is greater than 200 mL/kg per minute and is not normally used. Initially high gas flows are used to cause rapid inspired concentration within the system and then flow rates are decreased for the maintenance phase; it is more economical to use lower flow rates when possible. Higher gas flows are used in the recovery phase to help wash the inhalant gases out of the system to speed awakening.

Nonrebreathing systems are characterized by the absence of the unidirectional valves and the carbon dioxide absorber (Fig. 12.5). The carbon dioxide is removed by being flushed from the system with a high gas flow. Flow rates vary from 130 to 300 mL/kg per minute. This system is used is patients that weigh less than 10 kg as the high gas flow is not economical in larger patients. The nonrebreathing system has lower resistance to breathing and a smaller total circuit volume. The Bain system is a coaxial design, with the inner tube being the inspired gas and the outer tube containing exhaled gases that have less bulk, and provides a means for potentially warming the inspired gas as the warmer exhaled gas passes around the inner tube (Figs. 12.6 to 12.8).[18]

Monitoring

Monitoring the anesthetized patient is necessary to avoid potential complications, provide an early warning of life-threatening developments, and to ensure a safe outcome following anesthesia. Monitoring should begin during the induction phase and continue into recovery. There are three main areas to monitor in the anesthetized patient: depth of anesthesia, cardiovascular and respiratory parameters, and body temperature. Physiologic parameters to asses include heart rate, blood pressure, respiratory rate, mucous membrane color, capillary refill time, hemoglobin saturation, and end-tidal CO_2. Spontaneous movement is a reliable sign of light level of anesthesia. An abrupt increase in heart rate, blood pressure, and respiratory rate are indicators of a response to surgical stimulus and a light level of anesthesia. Loose jaw tone, absence of palpebral reflex, and muscle relaxation are observed during medium to deep anesthesia levels.

Cardiovascular System

The cardiovascular system is evaluated via the heart rate and rhythm, peripheral pulse rate and rhythm, tissue perfusion, and blood pressure. Heart rate is an important determinant of cardiac output; excessive bradycardia will result in a decreased cardiac output. Excessive tachycardia will also decrease cardiac output due to decreased filling time and reduced stroke volume. An ECG is important for the identification of potential arrhythmias.

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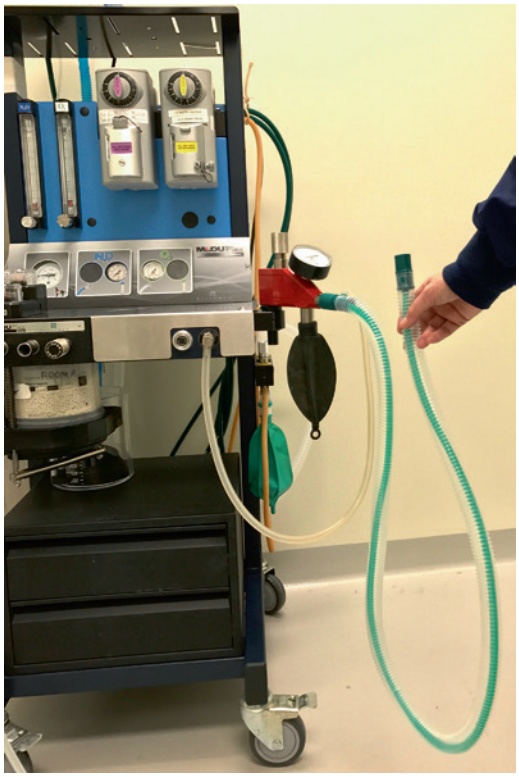


FIG. 12.6 The Bain, coaxial nonrebreathing, Mapleson D circuit attached to a small animal anesthesia machine. The inner green tube carries the fresh inhaled gases to the patient, and the clear outer tube removes the exhaled gases from the patient. Note the connection of the circuit to the common gas outlet.

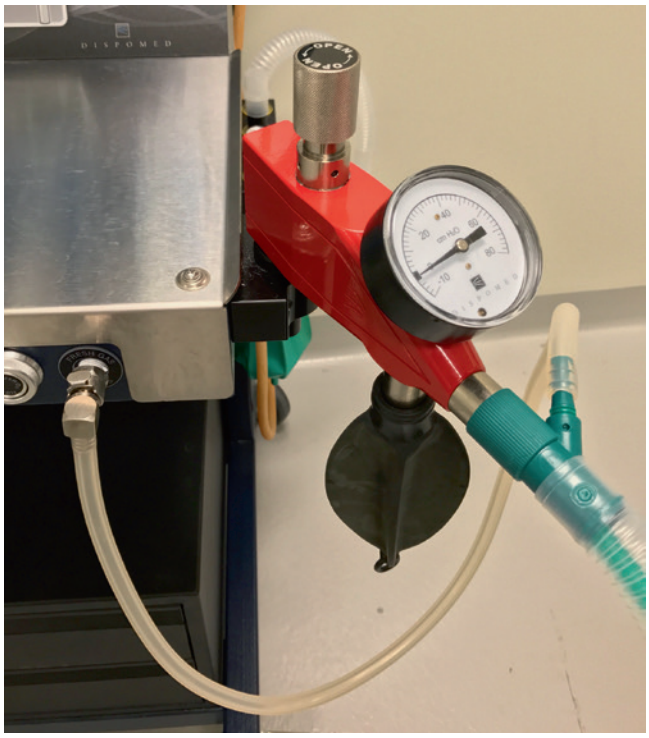


FIG. 12.7 Bain mount for the nonrebreathing circuit. It provides a fixed connection point for the bag, the adjustable pressure-limiting valve, and a pressure gauge. Note the circuit connection to the common gas outlet.

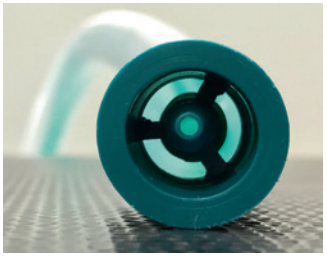


FIG. 12.8 The patient end of the coaxial Bain nonrebreathing circuit. The inner port is for the fresh inhaled gas, and the exhaled gas flows out the larger port holes around the inner port.

The most common intraoperative complication is hypotension. When blood pressure is low, tissue perfusion becomes compromised. Hypotension is a mean arterial blood pressure less than or equal to 60 mmHg or a systolic pressure of less than or equal to 80 mmHg. Common causes of hypotension are hypovolemia, poor cardiac output, and vasodilation. Blood pressure can be measured indirectly with sphygmomanometry, placing an occlusive cuff over an artery on a forelimb, hind limb, or tail. The width of the cuff should be 40% of the circumference of the appendage. A Doppler ultrasound piezoelectric crystal is placed over an artery distal to the cuff on the appendage. The cuff is manually inflated and then deflated to the first audible sound, which is systolic blood pressure. The oscillometry device will automatically inflate/deflate the cuff and record the systolic, diastolic, mean blood pressure, and heart rate at determined time intervals. Oscillometry determines blood pressure by analyzing fluctuations of pressure within the cuff. The gold standard for blood pressure measurement is direct catheterization of an artery. This gives beat-to-beat blood pressure measurements. Commonly used arteries for direct arterial measurements are the dorsal metatarsal, radial/carpal, coccygeal, lingual, femoral, and auricular.

Respiratory System

The respiratory system is evaluated by observation of the respiratory rate and depth of breathing via chest, abdominal, or reservoir bag excursions. A respiratory rate of 6 breaths per minute or less is indicative of hypoventilation. Chest wall movement with no corresponding movement of the reservoir bag is indicative of an airway obstruction or a tension pneumothorax. An arterial blood gas is the gold standard for evaluation of ventilation and oxygenation. Noninvasive techniques are capnometry for ventilation and pulse oximetry for oxygenation. Normal carbon dioxide partial pressure ($PaCO_2$) is 35 to 45 mmHg. A $PaCO_2$ of more than 60 mm Hg is respiratory acidosis due to hypoventilation and would be an indication for IPPV. A $PaCO_2$ below 20 mm Hg is indicative of respiratory alkalosis and decreased cerebral blood flow, seen with hyperventilation. The end-tidal CO_2 gas sample is taken at the end of exhalation and is a surrogate marker for $PaCO_2$. The end-tidal CO_2 is usually 3 to 6 mm Hg lower than $PaCO_2$. Capnography is the waveform of the exhaled CO_2; this capnogram curve, if abnormal in shape, can be used to identify complications. A sloping inspiratory waveform indicates a leak during inspiration, dilution with fresh gas during a long pause between breaths, or prolonged inspiration. A waveform with a sloping expiration indicates a partial airway obstruction or impaired lung deflation. A normal PaO_2 on room air should be between 80 and 110 mm Hg. Hypoxemia is a PaO_2 of less than

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80 mm Hg, and potentially life-threatening hypoxemia is a PaO_2 less than 60 mm Hg. When on an oxygen-enriched gas the PaO_2 should be above 110 mm Hg. The pulse oximeter measures the hemoglobin saturation with oxygen (SpO_2), and this is used as a surrogate for PaO_2. The normal value for SpO_2 is 98% to 99%. Hypoxemia is a SpO_2 less than 95%, which corresponds to a PaO_2 of 80 mm Hg, with severe hypoxemia at less than 90%, which is a PaO_2 of 60 mm Hg. The pulse oximetry probe is placed across an arterial bed such as the tongue, ear pinnae, lip, or toe web.[19]

Body Temperature

Body temperatures of 90°F to 94°F (32°C–34°C) result in reduced anesthetic requirements, impaired cardiovascular function, hypoventilation, decreased resistance to infection, and a prolonged recovery. Hyperthermia (105°F [40.5°C]) can occur with overzealous application of warming devices and in dogs with thick hair coats and warming devices in place; cell damages occur with temperatures at or above 108°F (42°C). It is important to monitor body temperature during anesthesia and into recovery. Cats given opioids as part of their anesthetic episode seem prone to hyperthermia in recovery. Hyperthermia is inversely related to the body temperature at extubation; the colder the cats were at extubation, the warmer they become during the postoperative period. This exaggerated rebound from hypothermia may be enabled by the resetting action of opioids upon the hypothalamic thermoregulatory threshold.[5] The hyperthermia is usually self-limiting but can be treated by giving naloxone at 0.01 mg/kg IM or SC; this would also result in reversal of opioid-induced analgesia. If a mu opioid agonist has been given, partial reversal to retain some analgesia can be achieved with butorphanol at 0.1 mg/kg IV or IM. Acepromazine at 0.01 to 0.05 mg/kg can be given to provide calming and may improve heat loss through vasodilation. Fans, removal of any warming devices, and placing the cat on the uncovered cage floor also aid in heat loss.

Fluid Therapy

IV fluid therapy should be tailored to the individual animal based on its needs, taking into consideration acute versus chronic conditions, pathology such as electrolyte abnormalities, acid-base disturbances, changes in volume such as dehydration or blood loss, and changes in distribution such as pleural effusion. If possible, correct fluid and electrolyte imbalances prior to anesthesia and surgery (see Chapter 4). In healthy patients the suggested initial rate for fluids during surgery is 3 mL/kg per hour in cats and 5 mL/kg per hour in dogs. If the patient requires replacement and maintenance fluid therapy, the fluid rate may need to be 10 mL/kg per hour. The fluid rate should be reduced in procedures that are of long duration, provided the patient is stable, to avoid overhydration.

Fluid bolus administration is often used to treat hypotension during anesthesia. A fluid bolus may be appropriate if there is relative hypovolemia caused by peripheral vasodilation contributing to the hypotension. Anesthetic depth should be assessed and the level of inhalant decreased if possible. A crystalloid bolus, such as lactated Ringer's solution, can be given at a rate of 3 to 10 mg/kg IV and repeated once. If the response is inadequate, a colloid such as hetastarch may be considered. It is given at a dose of 5 to 10 mL/kg in dogs and 1 to 5 mL/kg in cats, titrating to effect to avoid vascular overload. Blood pressure should be monitored every 3 to 5 minutes. If, following fluid bolus therapy, the animal remains hypotensive and is not hypovolemic, other methods should be considered, such as inotropes/vasopressors or balanced anesthetic techniques to further reduce the inhalant levels. Cats require close monitoring to avoid volume overload.[20]

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13

Pain Management and Acupuncture



Pain perception is the unpleasant sensory and emotional experience associated with either actual or potential tissue damage; pain management is an important aspect of surgery in all animals. Because animals cannot express pain in the same manner as humans, it is important that surgeons be able to recognize pain in their patients and that they preemptively do everything possible to avoid or limit pain and distress prior to, during, and after surgery. Behavioral and physiologic manifestations of pain are provided in Box 13.1. Pain management guidelines have been established for dogs and cats and are detailed in this chapter. Pain management can involve a variety of techniques and agents to provide multimodal analgesia for the care of the surgical patient.

DEFINITIONS

There are varying types of pain; *nociceptive pain* occurs when peripheral neural receptors are activated by noxious stimuli, such as a surgical incision or trauma, whereas *inflammatory pain* is due to activation of the immune system in response to injury or infection. *Pathologic* or *maladaptive pain* occurs when pain is amplified and sustained, leading to peripheral and central hypersensitization, and is characterized by hyperalgesia, allodynia (pain, generally on the skin, caused by something that would not normally cause pain), expansion of the painful field beyond the normal boundaries, and protracted pain. *Neuropathic pain* is a disease of the central nervous system (CNS) and occurs under conditions that create genomic and phenotypic changes.

NEUROANATOMY OF NOCICEPTION

The simplified pathway for transmission of nociceptive information from the periphery to the brain consists of a three-neuron system. There is a primary peripheral nociceptive neuron (cell body in dorsal root ganglion), a spinal cord projection neuron (in the dorsal gray matter), and a thalamic relay neuron (that subsequently transmits information to the somesthetic cerebral cortex) (Fig. 13.1).[1–4] Roughly half of all afferent axons in cutaneous nerves are related to nociception (pain sensation). Most of the nociceptive receptors are the free nerve endings (dendritic zones) of general somatic afferent (GSA) neurons, whose respective cell bodies are located in the dorsal root ganglia of all spinal nerves, as well as the ganglia of some cranial nerves (CNs; primarily CN V). An area of skin that is innervated by a specific dorsal root is called a *dermatome*, and there is some degree of overlap of dermatomal regions.[1,3,4] The types of axons conveying nociceptive signals include Aδ (delta) and C fibers. Aδ fibers are lightly myelinated and larger than the C fibers; they are thought to transmit "fast" painful stimuli, such as sharp, pricking pain that is localizable to a particular body area. The much more numerous C fibers are unmyelinated and conduct more slowly; the nature of pain impulses carried by C fibers is often described as a dull, aching, or throbbing pain.[2,3]

The axons of nociceptive neurons travel through peripheral nerves, spinal nerves, and dorsal nerve roots to reach the spinal cord in the region of the dorsolateral sulcus. At this point, these afferent axons traverse the spinal cord in a white matter tract called the *dorsolateral fasciculus* (Lissauer's tract), within which collateral branches of the telodendria traverse the spinal cord cranially and caudally over several spinal cord segments. Axonal terminals (telodendria) from this tract then synapse either on interneurons or projection neurons in the dorsal gray column (Fig. 13.2). The gray matter of the dorsal horn is arranged in layers or laminae, and the majority of the primary nociceptive telodendria synapse on neurons in layers I to IV. The *marginal nucleus* (lamina I), *substantia gelatinosa* (lamina II), and *nucleus proprius* (primarily lamina III) are most relevant to this discussion (see Fig. 13.2). Projection neurons from the dorsal gray column provide axons that form the ascending pain pathways of the spinal cord. Interneurons from these laminae have a number of roles, including segmental reflex activity and modulation of other synapses within the spinal cord gray matter.[1–4]

Clinically relevant spinal cord pain projection pathways include the *spinothalamic tract,* the *spinocervicothalamic tract,* the *spinomesencephalic tract,* and the *spinoreticular tract* (Fig. 13.3). Of most clinical importance in terms of conscious perception and reaction to noxious stimuli are the spinothalamic and spinocervicothalamic tract. The spinomesencephalic tract and spinoreticular tract are important in the understanding of several central pain modulating mechanisms (discussed later). Axons of dorsal gray horn projection neurons that give rise to the spinothalamic tract travel in the ventral aspect of the lateral funiculus of the spinal cord; these axons eventually reach neuronal cell bodies of the ventrocaudolateral (VCL) nucleus of the thalamus. These VCL nuclei provide axons to neurons in the somesthetic cerebral cortex. In primates, the spinothalamic tract is lateralized; the majority of the projection neuron axons cross to the opposite side of the spinal cord before ascending to the brain stem. In dogs and cats, the spinothalamic tract is thought to be more of a bilateral, multisynaptic pathway. The spinocervicothalamic tract may be a more clinically important nociceptive pathway in dogs and cats, and does appear to be more lateralizing than the spinothalamic pathway. Axons of dorsal gray horn projection neurons for this pathway ascend the spinal cord ipsilaterally in the dorsal aspect of the lateral funiculus. These axons terminate in another gray matter nucleus located in the first two cervical spinal cord segments (on the lateral side of the dorsal gray column), called the *lateral cervical nucleus* (LCN). Projection

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ABSTRACT

Analgesia is necessary for patient comfort and to improve outcomes. Analgesia can be achieved by various techniques and drugs. Multimodal analgesia allows for decreased systemic side effects by minimizing the amount of any single drug given. Local and regional blocks provide additional analgesia.

KEYWORDS

analgesia
acupuncture
local anesthetics
opioids
dissociatives
alpha-2 agonists
constant rate infusions

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BOX 13.1 Signs of Pain in Dogs and Cats

Abnormal posture: Hunched up, sitting in abnormal position, praying position
Abnormal gait: Stiff, non–weight bearing, limp
Abnormal movement: Thrashing, restlessness, no movement when not sleeping
Vocalization: Whining, crying, screaming, howling, no sound
Miscellaneous: Looking/licking or chewing painful area, reaction when touched
Behavior changes: Low and/or no wagging tail, head down, depressed, not grooming, tachypnea, trembling, shaking, low appetite, bites, recumbent, unaware
Physiologic signs: Tachypnea, tachycardia, hypertension, dilated pupils, increased serum cortisol and epinephrine

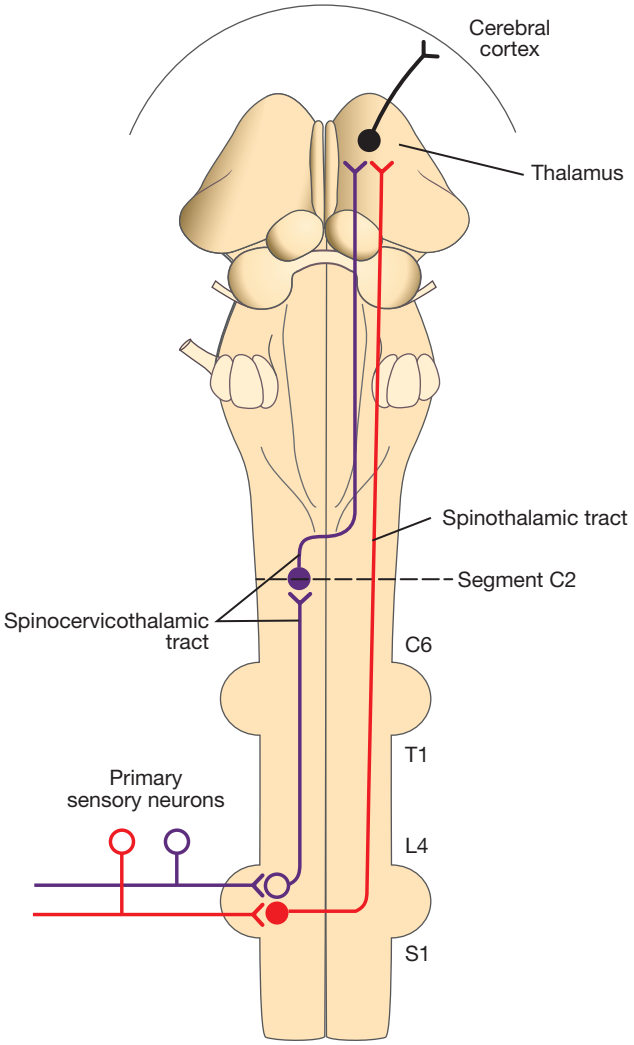


FIG. 13.1 Diagrammatic representation of simplified three-neuron (spinothalamic tract) or four-neuron (spinocervicothalamic tract) nociceptive pathways. (Modified from Uemura EE. Somatosensory system. In: Uemura EE, ed. *Fundamentals of Canine Neuroanatomy and Neurophysiology*. Ames, IA: Wiley-Blackwell; 2015: 128–155.)

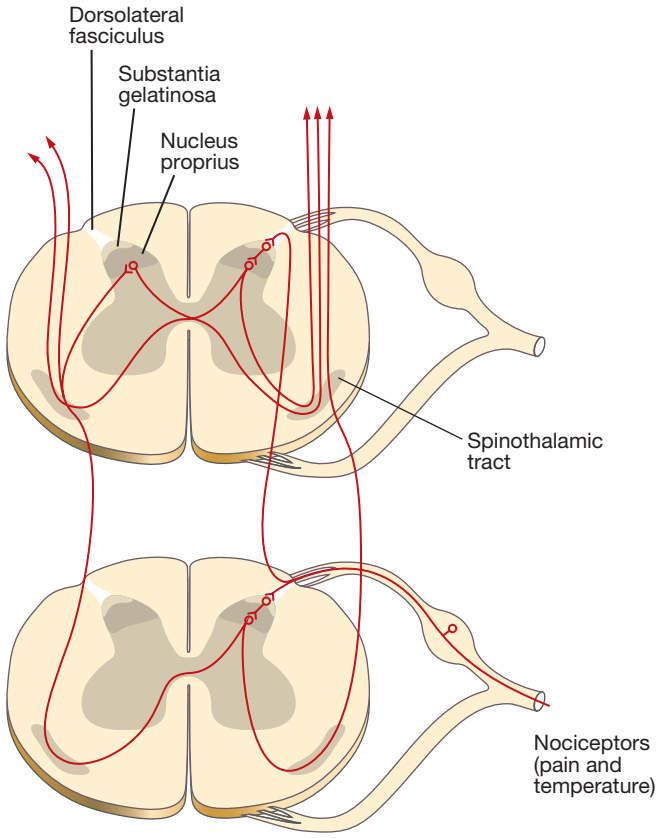


FIG. 13.2 Classic spinothalamic tract nociceptive pathway for dogs and cats. (Modified from Pypendop BH, Barter LS. Pharmacologic management of pain for patients with neurologic disease. In: Dewey CW, da Costa RC, eds. *Practical Guide to Canine and Feline Neurology*. 3rd ed. Ames, IA: Wiley-Blackwell; 2016:585–597.)

neurons from the LCN cross the midline at the level of the medial lemniscus of the brain stem to reach the VCL nucleus of the thalamus.[1,4] The nociceptive pathways (on the whole) in dogs and cats appear to be lateralized, as are many other sensory modalities. Dogs and cats with *hemineglect* (hemi-inattention) syndrome tend to have difficulty localizing (or sometimes even acknowledging) noxious stimuli (e.g., skin pinch with a hemostat) applied to the inattentive side of the body.

Nociceptive information from the face and head is conveyed primarily via CN V and its associated nucleus and tract in the brain stem. The anatomic arrangement is similar to that already described for the body, in that there is a nociceptive first-order neuron with its cell body in a peripheral ganglion (in this case the trigeminal ganglion in the petrosal bone), a white matter tract in the CNS (the spinal tract of CN V), a gray matter nucleus in the CNS (nucleus of the spinal tract of CN V), and thalamic relay nuclei that convey impulses to the somesthetic cerebral cortex (Fig. 13.4). The spinal tract of CN V is continuous with the dorsolateral fasciculus of the spinal cord, and the nucleus of the spinal tract of CN V is continuous with the substantia gelatinosa of the spinal cord. Sensation from the face is also lateralized in dogs and cats. The majority of axons from the nucleus of the spinal tract of CN V from one side of the face will cross at the medial lemniscus to the opposite

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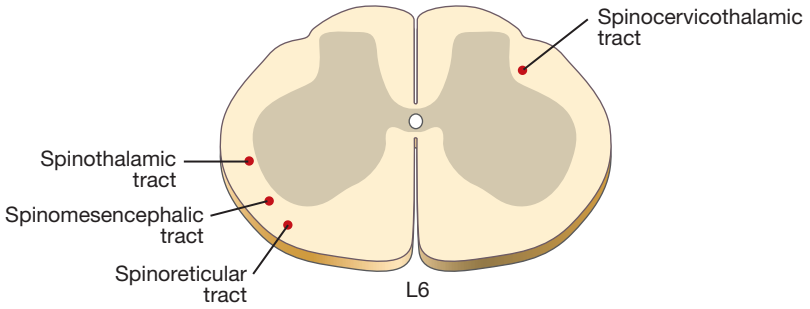


FIG. 13.3 Approximate locations of clinically relevant nociceptive tracts in the lumbar white matter. (Modified from Evans HE, de Lahunta A, eds. *Miller's Anatomy of the Dog.* 4th ed. Philadelphia: Elsevier; 2013.)

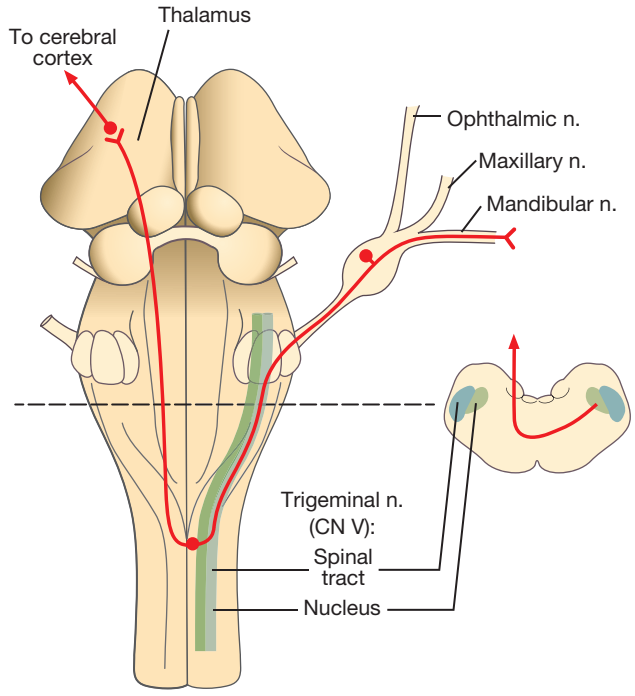


FIG. 13.4 Neuroanatomic pathway for nociception of the face. (Modified from Dewey CW, da Costa RC, eds. *Practical Guide to Canine and Feline Neurology.* 3rd ed. Ames, IA: Wiley-Blackwell; 2016.)

ventrocaudomedial nucleus of the thalamus. This information is then relayed to the cerebral cortex for perception.[1,4]

Visceral nociception is conveyed by axons similarly as already described, but the axons are usually referred to as *general visceral afferent* (GVA) axons. The GVA axons travel to the spinal cord in sympathetic and parasympathetic nerves to reach the dorsolateral fasciculus. At this point, the pathway for nociception is exactly as described for the body. In fact, the GVA axons synapse on the same pool of dorsal horn gray matter neurons as do the GSA neurons in a specific body region. In other words, there is a visceral sensory map that corresponds to the dermatomal map for specific regions of the body.[1,5,6] This concept is important in understanding phenomena such as referred pain and some of the suspected mechanisms of acupuncture.

NEUROPHYSIOLOGY AND MODULATION OF NOCICEPTION

Local inflammatory mediators in peripheral tissue are responsible for creating the signal that will ultimately be perceived as pain. Such mediators released by tissue after injury include bradykinin, prostaglandins, leukotrienes, serotonin, arachidonic acid, histamine, nerve growth factor, tumor necrosis factor, substance P, acetylcholine, adenosine triphosphate, and protons. Many of these inflammatory mediators and cytokines are released from epithelial cells, endothelial cells, mast cells, macrophages, and platelets. Prostaglandin E2 (PGE2) is an arachidonic acid metabolite generated by cyclooxygenase (COX) released from damaged cells. Bradykinin is a potent pain-producing peptide; it directly activates Aδ and C fibers and increases the synthesis and release of prostaglandins by nearby cells.[2,7–10] If unabated, stimulation of peripheral nociceptors by these mediators can lead to release of additional inflammatory mediators by the nociceptors themselves, leading to amplification of the local inflammation via a process of *neurogenic inflammation*. In addition to cytokines already mentioned, nociceptors may release other chemicals such as substance P and calcitonin gene-related peptide. Both these peptides can cause increased tissue edema by direct actions on venules. They also activate local mast cells, leading to histamine release; histamine accentuates the pain response by decreasing the threshold for nociceptor activation. These processes can ultimately lead to *peripheral sensitization*, a phenomenon in which nociceptor afferent neurons become hyperexcitable to stimuli, effectively increasing the gain of the response of these peripheral neurons to potentially noxious stimuli.[2,5,9,10]

In the CNS, similar processes are thought to contribute to the development of hyperesthesia, allodynia, and chronic pain states. *Central sensitization* refers to enhancement of the pain response to a certain level of stimulus over time, due to similar mechanisms of increasing the responsiveness of neuronal pathways in the spinal component of the nociceptive pathway. With constant overstimulation of dorsal horn gray matter neurons by peripheral nociceptors (particularly C fibers), neurotransmitters such as glutamate and substance P that bind to postsynaptic dorsal horn gray matter neurons will eventually increase the efficiency of pain transmission at the spinal cord level. Glutamate is the predominant excitatory neurotransmitter released by Aδ and C fibers in the spinal cord gray matter. Substance P is released mainly by C fibers. Substance P can enhance and prolong the

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BOX 13.2 Examples of Mechanisms of the Central Sensitization Process

- Wind-up via excessive glutamate activation of neuronal NMDA receptors.
- Release of normal inhibition of NMDA receptor overstimulation via co-release of substance P and CGRP from hyperstimulated C-fibers (see earlier). This involves Mg^{++} channels.
- Transcriptional and translational changes in postsynaptic dorsal horn neurons (pain "memory").
- Activation of central nervous system microglial cells (by substance P, glutamate, etc.), which upregulate cyclooxygenase-2, and release other neuroactive substances (which increase excitability of dorsal horn neurons).
- Axonal sprouting, altered neuronal connectivity and cell death; all associated with microglial cell activation.

BOX 13.3 Summary of Specific Regions of the Brain and Information Involved in Suprasegmental Inhibition of Nociceptive Pathways Along With Their Respective Primary Neurotransmitters

- *Dorsolateral prefrontal cerebral cortex* and *cingulate gyrus*: activates periaqueductal gray (PAG) and raphe magnus nuclei via *endorphins*.
- *Hypothalamus*: sends long axons to activate neurons of the *PAG* via *β-endorphins*.
- *Pituitary*: releases *endorphins* into the blood (humoral effect) and cerebrospinal fluid. The pituitary gland also releases *adrenocorticotrophic hormone* and *oxytocin* into the bloodstream, both of which can have analgesic effects.
- *PAG matter of the mesencephalon*: indirectly causes release of *serotonin* and *noradrenaline* in the region of the dorsal horn, via stimulating (via *enkephalins*) the raphe magnus nucleus of the myelencephalon (serotonin) and the locus ceruleus of the pons (noradrenaline). The PAG neurons also have projections to the thalamus and cerebrum.
- *Ventral tegmental area of the mesencephalon:* provides *dopaminergic* and *serotonergic* inhibitory axons to dorsal horn neurons.
- *Locus ceruleus of the pons*: releases *noradrenaline* in the dorsal horn, stimulating alpha-2 adrenoreceptors.
- *Raphe magnus nucleus of the myelencephalon*: releases *serotonin* in the dorsal horn gray matter.

actions of glutamate on dorsal horn neurons. Mechanisms of the central sensitization process include changes in the neurochemical environment as well as cytoarchitectural changes in the spinal cord (Box 13.2). The various physiologic processes described that perpetuate painful conditions form the basis for the development of phenomena like wind-up, hyperesthesia, allodynia, and chronic pain.[5,9,10]

In addition to mechanisms within the peripheral nervous system (PNS) and CNS that serve to exacerbate painful states, there are a number of segmental (spinal cord) and suprasegmental (brain) modulatory systems within the CNS that act to ameliorate the sensation of pain. At the spinal cord level, some interneurons of the dorsal gray column are inhibitory to projection neurons that provide the majority of axons for the various pain-transmitting spinal cord pathways. These interneurons can be stimulated by fast-conducting, nonnociceptive myelinated Aβ fibers (normally convey touch, pressure, and some proprioceptive sensation) as well as by a subset of nonnociceptive Aδ fibers. When stimulated, these fibers can, via excitation of the inhibitory interneuron, prohibit firing of the slower conducting C fibers. In other words, by the time the nociceptive stimulus carried by the C fiber arrives at the dorsal horn, an inhibitory interneuron that has been excited by a faster conducting myelinated fiber has already been given the signal to prohibit C fiber stimulation of dorsal horn projection neurons. This is the basis of the *gate control theory*. In essence, the faster conducting nonnociceptive fibers "close the gate" to slower conducting nociceptive fibers. The inhibitory interneurons involved in this phenomenon are believed to be enkephalinergic and may prohibit substance P release from C fibers.[7,9,11]

Several regions of the brain are involved in suprasegmental modification of nociception. These brain regions give rise to descending tracts that exert an analgesic effect at the level of the dorsal gray column of the spinal cord. They include the periaqueductal gray (PAG) matter of the mesencephalon, ventral tegmental area of the mesencephalon, locus ceruleus of the pons, median raphe magnus nucleus of the myelencephalon, hypothalamus, pituitary, and some areas of the cerebrum. Within the dorsal gray matter of the spinal cord, there are extrasynaptic receptors on the telodendria of primary nociceptive neurons (presynaptic) and dendritic zones of nociceptive projection neurons (postsynaptic). These include adrenergic, serotonergic, opioid, and gamma-aminobutyric acid type B ($GABA_B$) receptors. When these receptors are stimulated by descending pain modulating tracts, transmission of nociceptive stimuli from the dorsal horn gray matter to the respective spinal cord tracts is impaired. Specific information regarding regions of the brain involved in suprasegmental inhibition of nociceptive pathways along with their respective primary neurotransmitters is summarized in Box 13.3.

Although this may seem complicated, it can be summarized as an organized network of neurons and axonal connections that serves to diminish the pain that would otherwise be experienced by patients if the dorsal horn nociceptive projection neurons were not under the control of these brain influences. Once potentially noxious stimuli reach these centers from ascending spinal cord tracts, mechanisms to ameliorate the pain are immediately employed.[5,10,12] For example, a nociceptive stimulus is experienced by a patient, and this stimulus is carried via the spinomesencephalic tract to the PAG and ventral tegmental area of the mesencephalon. These mesencephalic centers, in turn, may activate other regions of this descending pain-alleviating system, such as the raphe magnus nucleus of the myelencephalon and the locus ceruleus of the pons. Eventually, axons from several of these brain centers travel caudally in the dorsolateral funiculus to reach their target dorsal horn projection neurons, which they subsequently inhibit.

Understanding the concept of referred pain from an anatomic and physiologic perspective is important for correctly localizing the source(s) of pain and providing effective treatment. Referred pain does not mean pain that is so intense that it radiates from the initial site of stimulation to other regions. *Referred pain* describes painful stimuli that the brain interprets as emanating from a region different than the actual source. This is an anatomic phenomenon and is based on shared interneuron pools in the dorsal gray matter of the spinal cord. GSA and GVA telodendria synapse segmentally on the same dorsal horn neuronal cell bodies in a certain spinal cord segment. In general, there are not enough dorsal gray horn interneurons to go around, so they are shared by GSA and GVA neurons. In other words, a dermatomal distribution of GSA neurons has a corresponding GVA component. For

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example, pain in the thoracolumbar spine region may, in some cases, be interpreted as abdominal pain in the same cross-sectional region of the patient.[1,5,6] The concept of the brain interpreting a noxious stimulus as coming from a visceral versus somatic site or vice versa due to shared dorsal gray horn neurons is called the *common pool theory*. There is also another theory based on this anatomic arrangement, called the *viscerovisceral reflex arc theory*. According to this theory, a noxious visceral stimulus is carried by GVA neurons to the dorsal horn neurons, resulting in excitation of sympathetic general visceral efferent fibers in that spinal cord segment. The resultant excessive sympathetic outflow in that segment causes peripheral vasospasm. The vasospasm leads to release of inflammatory cytokines, which then stimulate GSAs. The GSAs feed back into the same spinal cord segment dorsal horn neurons. By this mechanism, an initially viscerally located noxious stimulus is converted to a somatically located noxious stimulus.[1,6]

A variety of techniques may be utilized in the treatment of pain states, such as pharmacologic agents, physical therapy, and acupuncture. Treatment of pain may require only one therapy or a combination of different techniques. Pharmacologic agents used include opioids, *N*-methyl-D-aspartate (NMDA) antagonists, local anesthetics, alpha-2 agonists, nonsteroidal antiinflammatory drugs (NSAIDs), gabapentin, amantadine, amitriptyline, selective serotonin reuptake inhibitors, acetaminophen, maropitant, and bisphosphonates. Pain should be treated until the pain has resolved; this requires a pain management plan with reassessment of the treatment to ensure adequate analgesia is being achieved. Knowledge of the severity of pain typically caused by various procedures is helpful in determining treatment (Table 13.1). To be consistent in the diagnosis of a painful state, it is often helpful to use a pain scoring system (Figs. 13.5–13.7).

DRUGS USED FOR ANALGESIA

Opioids (see Chapter 12) are good analgesics and are commonly used both intraoperatively and postoperatively, and as part of a premedication protocol. The full mu agonists provide the most reliable analgesia (see Table 12.2). *Fentanyl* is commonly used as a constant-rate infusion (CRI) for analgesia in dogs and cats because it has an elimination half-life of approximately 45 minutes (Tables 13.2 and 13.3, Box 13.4). A loading dose is given intravenously prior to initiation of the CRI to help maintain plasma levels. When used as a CRI some animals may have a decreased appetite. Fentanyl comes as a transdermal patch that is applied to the skin (see Table 13.2 and p. 133). The other commonly used mu agonists used for analgesia include *hydromorphone, morphine, oxymorphone, remifentanil,* and *meperidine* (see Chapter 12 and Table 13.2).

Ketamine, an **NMDA receptor antagonist**, can be used as part of a multimodal technique for pain management (see Chapter 12 and Table 13.2). Subanesthetic doses of ketamine delivered as a CRI may be helpful in treating acute postoperative and posttraumatic pain, neuropathic pain, and maladaptive pain states. Glutamate, the NMDA receptor, contributes to central sensitization, and hyperalgesia and therapy at this receptor may help minimize "wind-up" and modify the plasticity of the nervous system.[13]

Dexmedetomidine, an **alpha-2 agonist,** is an effective sedative and analgesic (see Chapter 12 and Tables 13.2 and 13.4). Due to its cardiovascular depressant effects, it is typically given as an adjunct with other analgesics. Its effects the alpha-2 receptor in the dorsal horn of the spinal cord, brain stem, and in the periphery. It provides excellent visceral analgesia with a duration of 1 to 2 hours. A microdose CRI (see Table 13.2) can offer a constant and titratable level of analgesia; however, decreased cardiac output may still be seen. Alpha-2 agonists are synergistic with opioids. The effects of alpha-2 agonists can be reversed with *atipamezole* (see Chapter 12 and Table 13.2).[14]

Local anesthetic agents (e.g., lidocaine, bupivacaine) may be given as a local anesthetic (local block), epidural, or used intravenously as a CRI (see Table 13.2). They act mainly by inhibiting sodium influx through sodium-specific ion channels in the neuronal cell membrane. *Lidocaine* can be used in dogs as a CRI to provide analgesia and decrease the amount of inhalant needed; it can be continued into recovery and in the intensive care unit. It also has antiarrhythmic effects and reactive oxygen scavenging properties; this may be useful in gastric dilatation volvulus or gastrointestinal (GI) surgery of compromised bowel. Work in cats has shown that lidocaine causes cardiopulmonary depression

Text continued on p. 150

TABLE 13.1 Characterization of Pain

Type of Pain	Examples
Severe to excruciating	Neuropathic pain, extensive inflammation, bone cancer, meningitis, necrotizing pancreatitis, multiple trauma
Severe	Fracture repair, corneal ulceration, thoracotomy, extensive laparotomies, intraarticular surgery, total ear canal ablation, intervertebral disc disease, thrombosis and ischemia, uveitis, mastitis, limb amputation, gastric/organ torsion, glaucoma
Moderate	Laparotomy, inguinal hernia repair, castration and ovariohysterectomy (old or obese patients), some dental procedures, enucleation, minimally invasive orthopedic procedures, chest drains
Mild to moderate	Ovariohysterectomy (young), lump removal, some dental procedures, cystitis, otitis lacerations

BOX 13.4 Continuous-Rate Infusions

- Intraoperative and postoperative infusions
  - Hydromorphone: 0.025–0.1 mg/kg per hour IV
  - Fentanyl: 5–20 µg/kg IV loading dose, then 2–20 µg/kg per hour
  - Dexmedetomidine: 1–5 µg/kg IV loading dose, then 1 µg/kg per hour
  - Ketamine: 0.5 mg/kg IV loading dose, then 10 µg/kg per min IV CRI intraoperatively and 2 µg/kg per min IV CRI postoperatively
- FLK[a]
  - Fentanyl: 1–5 µg/kg per hour IV, plus
  - Lidocaine: 25–50 µg/kg per min IV, plus
  - Ketamine: 10 µg/kg per min IV
- MLK[a]
  - Morphine[b]: 3.3 µg/kg per min IV, plus
  - Lidocaine: 50 µg/kg per min IV, plus
  - Ketamine: 10 µg/kg per min IV

[a]Not recommended for use in cats.
[b]Give slowly to prevent histamine release.
CRI, Constant-rate infusion; *FLK,* fentanyl, lidocaine, ketamine; *IV,* intravenous; *MLK,* morphine, lidocaine, ketamine.

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SHORT FORM OF THE GLASGOW COMPOSITE PAIN SCALE

Dog's name _____

Hospital Number _____ **Date** / / **Time**

Surgery Yes/No (delete as appropriate)

Procedure or Condition_____

_____

In the sections below please circle the appropriate score in each list and sum these to give the total score.

A. Look at dog in Kennel

Is the dog?

(i)

Quiet	0
Crying or whimpering	1
Groaning	2
Screaming	3

(ii)

Ignoring any wound or painful area	0
Looking at wound or painful area	1
Licking wound or painful area	2
Rubbing wound or painful area	3
Chewing wound or painful area	4

In the case of spinal, pelvic or multiple limb fractures, or where assistance is required to aid locomotion do not carry out section **B** and proceed to **C**
Please tick if this is the case ☐ then proceed to C.

B. Put lead on dog and lead out of the kennel.

When the dog rises/walks is it?

(iii)

Normal	0
Lame	1
Slow or reluctant	2
Stiff	3
It refuses to move	4

C. If it has a wound or painful area including abdomen, apply gentle pressure 2 inches round the site.

Does it?

(iv)

Do nothing	0
Look round	1
Flinch	2
Growl or guard area	3
Snap	4
Cry	5

D. Overall

Is the dog?

(v)

Happy and content or happy and bouncy	0
Quiet	1
Indifferent or non-responsive to surroundings	2
Nervous or anxious or fearful	3
Depressed or non-responsive to stimulation	4

Is the dog?

(vi)

Comfortable	0
Unsettled	1
Restless	2
Hunched or tense	3
Rigid	4

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Total Score (i+ii+iii+iv+v+vi) = _____

FIG. 13.5 Short form of the Glasgow Composite Pain Scale. (Modified from Reid J, Scott M, Nolan A. Development of a short form of the Glasgow Composite Measure Pain Scale [CMPS] as a measure of acute pain in the dog. *Vet Anaesth Analg.* 2005;32:7.)

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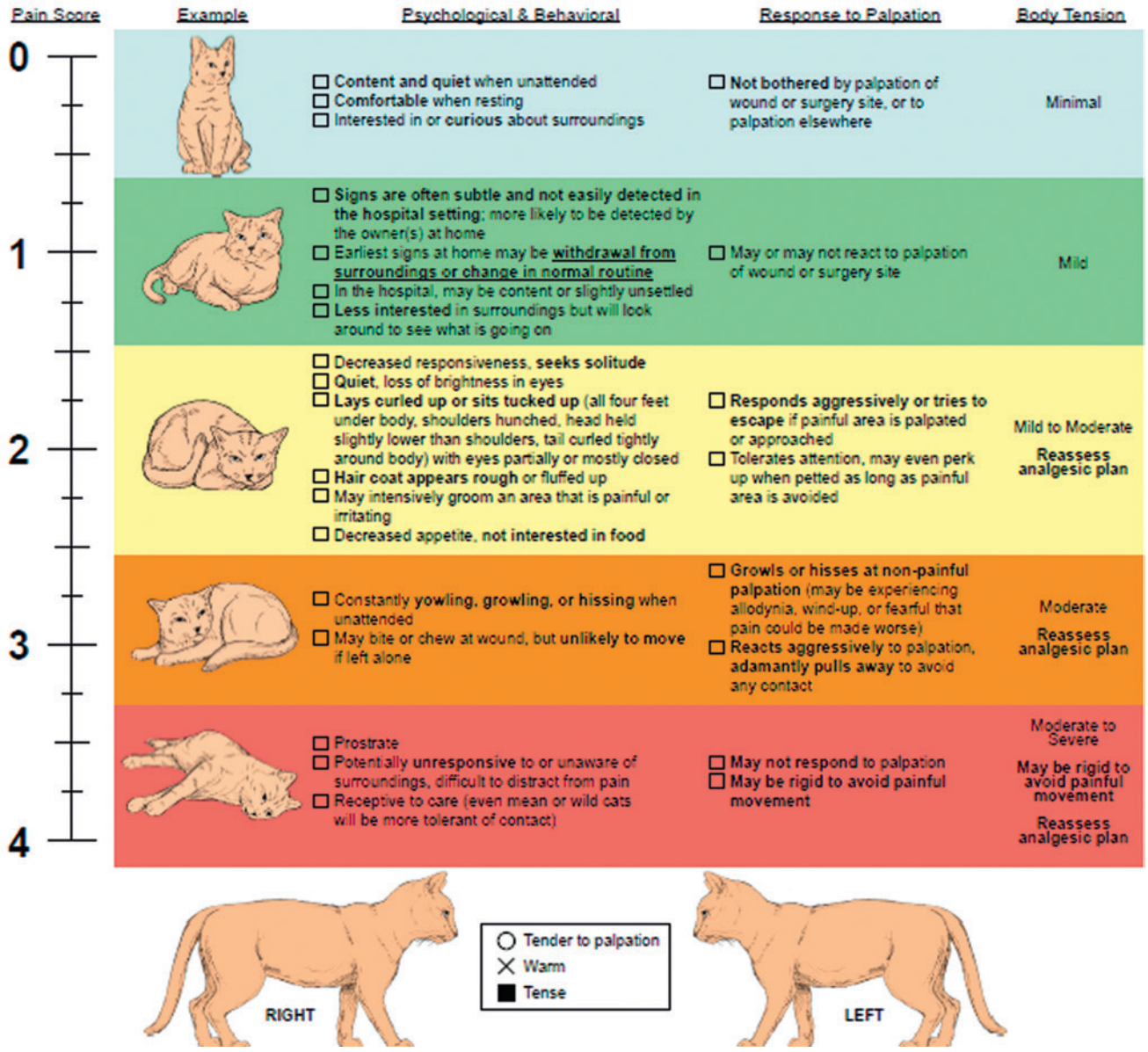


FIG. 13.6 Colorado Acute Pain Scale for the feline. (Modified from Colorado State University Veterinary Medical Center. *Feline Acute Pain Scale*, 2006. Courtesy Peter Hellyer, Samantha Uhrig, Narda Robinson.)

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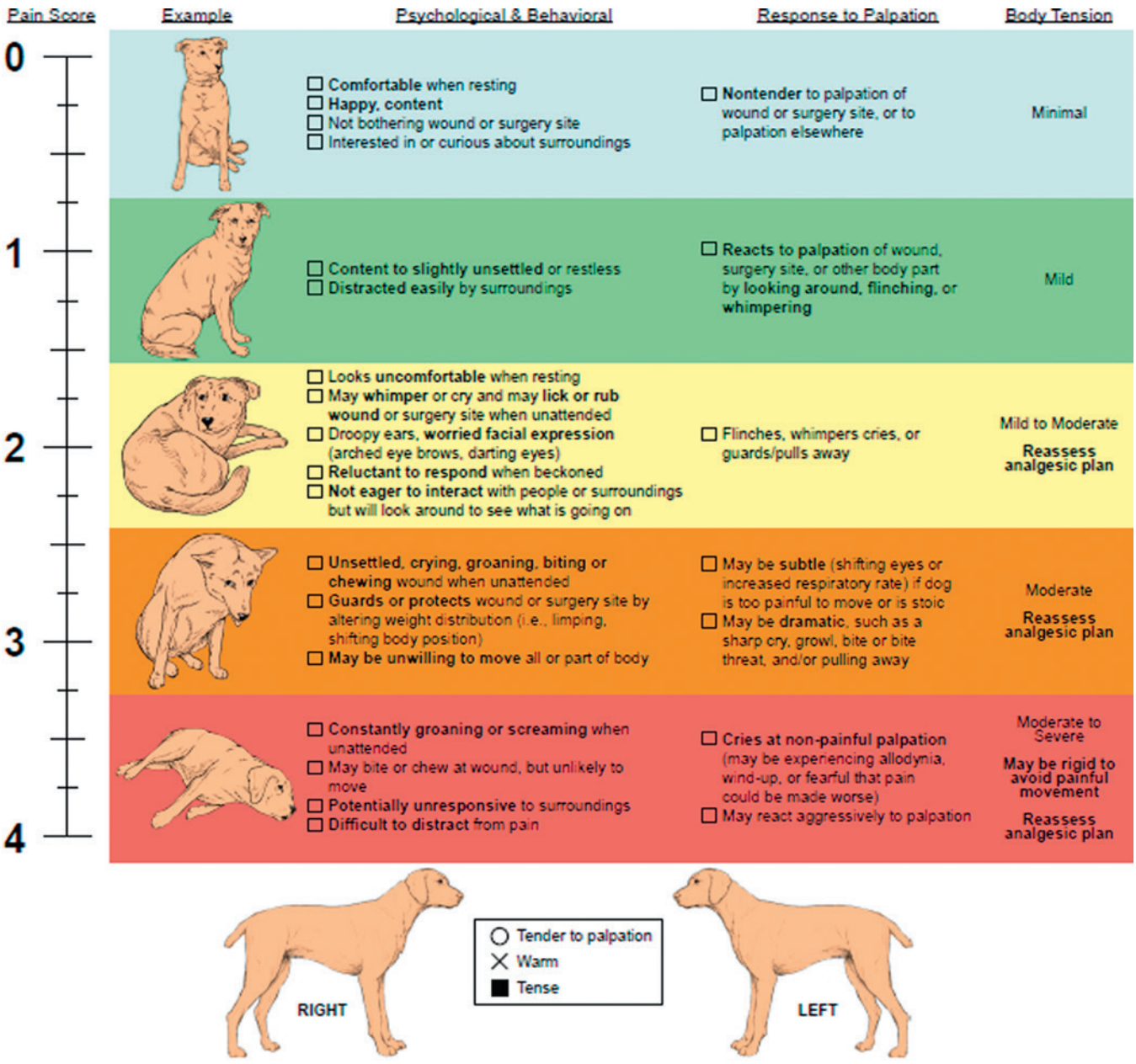


FIG. 13.7 Colorado Acute Pain Scale for the dog. (Modified from Colorado State University Veterinary Medical Center. *Canine Acute Pain Scale*, 2006. Courtesy Peter Hellyer, Samantha Uhrig, Narda Robinson.)

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TABLE 13.2 Analgesics: Indications and Dosages

Analgesic	Use/Indication	Route of Administration	Dose
Amantadine	Chronic pain	PO	*Dogs:* 3–5 mg/kg q24h, *or* 2–10 mg/kg q8–12h *Cats:* 3–5 mg/kg q12–24h
Amitriptyline	Idiopathic lower urinary tract disease	PO	*Cats:* 1–2 mg/kg/day *Dogs:* 1.1–1.3 mg/kg q12h
	Neuropathic pain	PO	Same as above
Atipamezole	Reverse alpha-2 agonists	IV, IM, SC	0.05–0.2 mg/kg once
Bupivacaine	Local anesthetic	Nerve block (SC)	1–2 mg/kg q6h
		Epidural	*Dogs:* 0.5–1 mg/kg *Cats:* 0.5–1 mg/kg
		Soaker catheter	2 mg/kg; total daily dose not to exceed 12 mg/kg
		Ring block (declaw)	0.2–0.3 mg/kg SC divided into 3 doses First dose: lateral and proximal to accessory carpal pad Second dose: medial to accessory carpal pad Third dose: dorsal-medial aspect of the proximal carpus
		Retrobulbar block	Maximum volume of 2 mL
Buprenorphine	Moderate pain	IM, IV	*Dogs:* 0.005–0.02 mg/kg q4–8h *Cats:* 0.02–0.04 mg/kg q4–8h
		Oral transmucosal	*Cats:* 0.01–0.02 mg/kg q6–8h
		Epidural	0.003–0.006 mg/kg
Butorphanol	Mild to moderate pain; mild sedation	IV, IM	0.1–0.4 mg/kg q1–3h
	Partial reversal of a μ opioid agonist	IV	0.05–0.1 mg/kg q1–3h
		CRI (IV)	Loading dose: 0.1 mg/kg Maintenance dose: 0.03–0.4 mg/kg q1h
Carprofen	Antiinflammatory	SC	*Dogs:* 2–4 mg/kg single dose, once; 4.4 mg/kg q24h; *or* 2.2 mg/kg q12–24h
	Mild to moderate pain	PO	
		SC	*Cats:* 4 mg/kg, once
Deracoxib	Chronic pain	PO	*Dogs:* 1–2 mg/kg q24h
	Acute postoperative pain	PO	*Dogs:* 3–4 mg/kg q24h, do not give for >7 days
Dexmedetomidine	Mild to moderate pain	IV	1–10 μg/kg q4h
		CRI (IV)	Loading dose: 1 μg/kg; maintenance dose: 0.5–3 μg/kg/h Dose can be variable depending on level of pain being treated
Etodolac	Antiinflammatory	PO	*Dogs:* 5–15 mg/kg q24h
Fentanyl	Moderate to severe pain	CRI (IV)	*Dogs:* loading dose, 2 μg/kg IV; maintenance dose: 2–10 μg/kg q1h *Cats:* loading dose, 1 μg/kg IV; maintenance dose, 1–4 μg/kg q1h
		IV, IM, SC	Dogs and cats: 5–15 μg/kg q1–3h
		Patch	Dogs and cats: <5 kg, 25 μg patch *Dogs:* 5–10 kg, 25 μg patch 10–20 kg, 50 μg patch 20–30 kg, 75 μg patch >30 kg, 100 μg patch
Firocoxib	Antiinflammatory	PO	*Dogs:* 5 mg/kg q24h
Gabapentin	Neuropathic pain	PO	Dose is variable depending on the level of pain and may need to be increased over time to be effective. Starting dose: 1.25–4 mg/kg q24h; may increase to (see text): *Dogs:* 10–20 mg/kg q8–12h *Cats:* 8–10 mg/kg q8h, or 3 mg/kg q6h
Grapiprant	Pain and inflammation associated with osteoarthritis	PO	*Dogs:* 2 mg/kg q24h
Hydromorphone HCl	Moderate to severe pain	IM, SC	*Dogs:* 0.05–0.2 mg/kg q2–4h *Cats:* 0.03–0.1 mg/kg q2–4h
		IV	*Dogs:* 0.05–0.1 mg/kg q2–4h *Cats:* 0.05–0.1 mg/kg q2–4h
Ketamine	Analgesia without sedation	IV	0.1–1 mg/kg 5–20 min
		CRI (IV)	Loading dose: 0.5 mg/kg Maintenance during surgery: 0.6 mg/kg q1h Maintenance after surgery: 0.12 mg/kg q1h IV for 24 h
Ketoprofen	Antiinflammatory	IV, SC, IM	*Dogs:* 2 mg/kg once, *or* 1 mg/kg q24h
		SC	*Cats:* 2 mg/kg q24h; up to 3 doses

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TABLE 13.2 Analgesics: Indications and Dosages—cont'd

Analgesic	Use/Indication	Route of Administration	Dose
Lidocaine	Local anesthetic	Nerve block (SC)	1–2 mg/kg
		Intratesticular injection for castration	Do not exceed 4 mg/kg; inject into body of each testicle
		CRI (IV)	*Dogs:* loading dose, 1–2 mg/kg; maintenance dose, 2–3 mg/kg q1h Doses as high as 6–12 mg/kg q1h have been used but require close monitoring to avoid toxicity.
		Patch	Animal dose has not been established, but the patch contains 700 mg of lidocaine. Significant systemic absorption has not been found to occur. The patch should be cut to fit the size of the area.
		Soaker catheter	2–4 mg/kg q2–3h
		IV regional block for extremities	2–4 mg/kg
		Retrobulbar block	Maximum volume of 2 mL
Maropitant	Mild to moderate pain	CRI (IV)	*Dogs:* loading dose, 1 mg/kg; maintenance dose, 30 or 150 µg/kg q1h
	Premedicant for ovariohysterectomy	SC	*Dogs:* 1 mg/kg
		IV	*Cats:* 1 mg/kg
Meloxicam	Antiinflammatory	IV, SC, PO	*Dogs:* 0.2 mg/kg once, then 0.1 mg/kg SC or PO q24h
		SC, PO	*Cats:* 0.2 mg/kg once, *or* 0.1 mg/kg q24h for up to 3 doses
Morphine, preservative free	Mild to moderate pain	Epidural	0.1 mg/kg
Morphine sulfate	Moderate to severe pain and sedation	IM, SC	*Dogs:* 0.5–2 mg/kg q4h *Cats:* 0.05–0.4 mg/kg q3–4h
		CRI (IV)	*Dogs:* 0.18 mg/kg q1h
Naloxone	Reverse opioid agonists	IM, IV, SC	0.002–0.1 mg/kg
Nocita	Postoperative pain from cranial cruciate surgery	Tissue infiltration	5.3 mg/kg administered by tissue infiltration just prior to closure (see text)
Oxymorphone	Moderate to severe pain and sedation	IM, IV	*Dogs:* 0.03–0.1 mg/kg q2–4h *Cats:* 0.01–0.05 mg/kg q2–4h
Pregabalin (see also p. 1140)	Neuropathic pain	PO	*Dogs:* 2.0 mg/kg q8–12h *Cats:* 1.0–2.0 mg/kg q12h
Remifentanil	Short-term analgesia	CRI (IV)	Loading dose: 3 µg/kg Maintenance dose: 0.1–0.3 µg/kg/min
Robenacoxib	Antiinflammatory	SC between the shoulder blades	*Cats:* 2 mg/kg
		PO	*Cats:* up to 3 days (based on weight of cat): 2.5–6 kg: 6 mg/cat q24h 6.1–12 kg: 12 mg/cat q24h *Cats:* 3–11 days: 1 mg/kg q24h
Tepoxalin	Analgesia and antiinflammatory	PO	*Dogs:* 10 mg/kg q24h

CRI, Constant-rate infusion; *IM,* intramuscular; *IV,* intravenous; *PO,* orally; *q,* every; *SC,* subcutaneous.

TABLE 13.3 Guidelines for Intermittent Bolus Administration of Select Opioids

Drug	Route	Dose	Frequency
Morphine	IV, IM, SC	Dogs: 0.5–2.0 mg/kg Cats: 0.05–0.4 mg/kg Give IV slowly to avoid histamine release	3–4 h
Hydromorphone	IM, SC	Dogs: 0.05–0.2 mg/kg Cats[a]: 0.05–0.1 mg/kg	2–4 h
	IV	Dogs: 0.05–0.1 mg/kg Cats[a]: 0.03–0.05 mg/kg	
Fentanyl	IV, IM, SC	5–15 µg/kg	Titrate to effect by giving boluses every 3–5 min PRN
Butorphanol	IV, IM, SC	0.1–0.4 mg/kg	1–3 h
Buprenorphine	IV, IM	Dogs: 0.005–0.02 mg/kg Cats: 0.02–0.04 mg/kg	4–8 h

[a]Hyperthermia is a risk in cats, even at recommended doses.
IM, Intramuscular; *IV,* intravenous; *PRN,* when necessary; *SC,* subcutaneous.

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TABLE 13.4 Dexmedetomidine-Butorphanol-Ketamine Combination in Healthy Cats[a]

Level of Sedation or Procedure	Dexmedetomidine (0.5 mg/mL) mL (μg/kg)	Butorphanol (10 mg/mL) mL (mg/kg)	Ketamine (100 mg/mL) mL (mg/kg)	Atipamezole[b] (5 mg/mL) mL (μg/kg)
Profound sedation-analgesia	0.1 (11)	0.1 (0.22)	0.1 (2.2)	0.1 (111)
Castration or laceration repair	0.2 (22)	0.2 (0.44)	0.2 (4.4)	0.2 (222)
Ovariohysterectomy or onychectomy[c]	0.3 (33)	0.3 (0.66)	0.4 (6.6)	0.3 (333)

[a]Based on a 4.5 kg (10 lb) cat. All drugs (except atipamezole) can be mixed in one syringe and administered as a single intramuscular (IM) injection. If given as intravenous (IV), the drug doses in this combination should be halved; however, if a deeper plane of anesthesia is desired, the full IM dose can be given IV.
[b]If reversal is necessary.
[c]More invasive procedures should have a stronger opioid chosen for postoperative analgesia.
From Ko JC, Knesl O, Weil AB, et al. FAQs: analgesia, sedation, and anesthesia: making the switch from medetomidine to dexmedetomidine. *Comp Cont Edu Vet.* 2009;31[suppl 1A]:1.

and should be used cautiously, if at all, as a CRI. Lidocaine has been used in combination with opioids (i.e., morphine, fentanyl) and ketamine as part of a multimodal therapy. These agents can be mixed together in a common bag or syringe, but separate infusions allow dose tailoring to better meet the patient's needs.[14]

A soaker catheter can be placed in a wound to provide continuous analgesia (see p. 149). Lidocaine can be given at 2- to 3-hour intervals (see Table 13.2). A lidocaine patch, developed for treating human postherpetic neuralgia, has been used in animals. The patch is applied to the skin or over the surgical incision. It can be left in place for 3 to 5 days and provides peripheral analgesia with minimal systemic side effects. The patch is not a controlled substance and can be cut to fit the site without affecting drug delivery.[15] *Bupivacaine* can be used perineurally or via the soaker catheter (see Table 13.2) but should never be given intravenously due to its cardiotoxicity.[14] It has a longer onset time of 15 to 20 minutes compared with lidocaine, with a duration of 4 to 6 hours (see Chapter 12).

NSAIDs are useful for pain management because inflammation plays a significant role in the pain process. There are several NSAIDs available for use in dogs and cats, including carprofen (Rimadyl), deracoxib (Deramaxx), meloxicam (Metacam), etodolac (Etogesic), tepoxalin (Zubrin), and robenacoxib (Onsior) (see Table 13.2). The analgesic and antiinflammatory effect is related to the inhibition of the COX enzyme isoforms. COX-1 is responsible for basal prostaglandin production for normal homeostatic processes in the body, including gastric mucus production, platelet function, and hemostasis. COX-2 is found at sites of inflammation and some basal production of constitutive prostaglandins. Selective inhibition of COX-2 would provide analgesic and antiinflammatory effects without the adverse effects of COX-1 inhibition. No available drug is a pure COX-2 inhibitor. NSAIDs should be used cautiously in patients with hypotension, hypovolemia, and renal disease. NSAID use with renal disease has the potential for renal vascular vasoconstriction, which could worsen renal insufficiency. NSAIDs are metabolized in the liver and excess dosing may lead to hepatotoxicity. Grapiprant (Galliprant) is a new, first-in-class piprant (PGE2 EP4 receptor antagonist, non-COX inhibiting) NSAID that is indicated for the control of pain and inflammation associated with osteoarthritis in dogs (see p. 1140 and Table 13.2). It is a once daily oral medication.

NOTE All NSAIDs (including the newer COX-2 selective drugs) have the very real potential for GI ulceration and perforation, even when used at recommended doses.

NSAIDs have a slow onset time of 45 to 60 minutes but provide analgesia for an extended duration, 12 to 24 hours. *Carprofen* is widely used in dogs; there are reports of idiosyncratic hepatic toxicity that will resolve if detected early and if carprofen is discontinued (see Table 13.2). Dogs should be screened for liver dysfunction prior to carprofen use. Labrador retrievers have been suggested to be at increased risk. This might be because many Labrador retrievers have a propensity to accumulate toxic amounts of copper in the liver. *Ketoprofen* in dogs seems to have an increased incidence of the adverse side effects of prolonged bleeding times and gastric lesions (see Table 13.2). A proton pump inhibitor (e.g., omeprazole; 1–2 mg/kg PO every 12 hours) may be given if the patient has GI problems or has had such in the past when receiving NSAIDs. They may also be of benefit when a NSAID that is known to have a distinctly high incidence of problems (e.g., piroxicam) is being administered. Renal insufficiency has been reported in cats. *Meloxicam* has been used in dogs and is safe for perioperative use; it can also be used in cats at the prescribed dose and duration (see Table 13.2). *Robenacoxib* (Onsior) is a feline-specific agent with a high safety index in cats (see Table 13.2). It has a terminal half-life of 1.9 hours in cats with an efficacy persisting for 22 hours.[16] *Tepoxalin* (Zubrin) inhibits COX-1, COX- 2, and lipoxygenase, preventing the synthesis of leukotrienes. It is administered orally as a wafer that dissolves in the mouth (see Table 13.2).

NSAIDs are commonly used in combination with opioids for a combined therapeutic effect. All NSAIDs should be dosed at the lowest effective dose and frequency. All patients should have a thorough exam prior to administration of NSAIDs.[17]

NOTE Do not use NSAIDs and glucocorticoids concurrently due to the substantially increased potential for GI ulceration and perforation.

Piprants are a new class of compounds that block specific prostaglandin receptors without interfering with prostanoid

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production. *Grapiprant* (see Table 13.2) is an EP4 piprant, also known as an EP4 prostaglandin receptor antagonist. This receptor is involved in the mediation of inflammatory pain, especially that associated with osteoarthritis. It is used orally in dogs and cats.[18] Grapiprant binds to the canine EP4 receptor with high affinity and is extremely well tolerated. Dogs can be treated with a dose of 2 mg/kg PO for up to 28 days for osteoarthritis pain.[19]

Gabapentin is a structural analogue of GABA but does not interact with GABA receptors to produce analgesia. It is used to treat neuropathic pain (see Table 13.2). The mechanism of action is not fully understood but may involve inhibition of N-type voltage-dependent neuronal calcium channels. A common side effect in dogs is sedation (see Box 12.2). The effectiveness of analgesia in cats is unknown. Gabapentin is commonly used as part of multimodal analgesic therapy.[20]

Amantadine is a dopamine agonist and NMDA receptor antagonist. The potential analgesic effect is mediated through NMDA antagonism. It appears to be most effective in cases of chronic pain and with central sensitization. It undergoes renal excretion in dogs. When used at higher doses, monitor for CNS excitement (see Table 13.2). CNS stimulation is a side effect of the dopamine agonist activity, and it should be used with caution in patients receiving other dopaminergic agents, such as the monoamine oxidase inhibitor selegiline, or agents inhibiting the reuptake of serotonin (i.e., the tricyclic antidepressant amitriptyline and the selective serotonin reuptake inhibitor fluoxetine). When used together, there is the risk of *serotonin syndrome*. This syndrome can be fatal and is characterized by CNS stimulation, muscle tremors, tachycardia, tachypnea, and hypertension. Hepatic dysfunction may enhance this risk as these agents are metabolized by the CYP 450 pathway. Meperidine, fentanyl, tramadol, and methadone increase the CNS concentration of serotonin and may contribute to serotonin syndrome, although the risk is unknown.[20]

Amitriptyline is a tricyclic antidepressant. It is a selective serotonin reuptake inhibitor leading to increases in synaptic levels of serotonin and norepinephrine. Other actions including sodium channel blockade, NMDA receptor antagonism, and antihistamine activity may contribute to the analgesic effect. Sedation may be a side effect. Overdose may lead to tachycardia, ventricular tachyarrhythmias, hypersalivation, and vomiting. In dogs, hepatic metabolism produces the active metabolite nortriptyline; both it and amitriptyline are excreted unchanged in the urine.[20]

Maropitant is a neurokinin 1 (NK-1) receptor antagonist primarily used for antiemesis. However, it also has anesthetic-sparing effects during visceral stimulation. The NK-1 receptor and its agonist substance P have been reported in pain pathways at the level of the CNS and PNS. The dorsal root ganglia and spinal cord dorsal horn contain large amounts of NK-1 receptors and substance P vesicles. Maropitant has been used in dogs undergoing ovarian stimulation (see Table 13.2)[21] and as a preoperative medication in dogs undergoing ovariohysterectomy. Those dogs receiving maropitant returned to eating sooner.[22]

REGIONAL TECHNIQUES

Local anesthetic agents may be used for regional blocks. Lidocaine or bupivacaine are most commonly used; bupivacaine has the longer duration of action. To enhance the analgesic effect, buprenorphine or dexmedetomidine can be added to the local agent. All blocks are placed following a sterile prep of the area, and the individual placing the block should wear sterile gloves. Prior to any injection of local anesthetic nerve block, aspiration should be performed to decrease the risk of intravascular injection. If there is resistance to the injection, it may indicate that the solution is being injected directly into the nerve; this should be avoided.

An intratesticular injection of lidocaine can be used in dogs and cats to augment analgesia for castration. Lidocaine is injected into the body of each testicle (see Table 13.2).[23] For cats undergoing a declaw procedure, a bupivacaine nerve block can provide analgesia into the postoperative period (see Table 13.2). It is injected subcutaneously, lateral and proximal to the accessory carpal pad to block the dorsal branch of the ulnar nerve, and medial to the accessory pad to block the median nerve and palmar branch of the ulnar nerve. The third site is the dorsal-medial aspect of the proximal carpus to block the superficial branch of the radial nerve.[23]

Animals undergoing rhinoscopies and nasal biopsies will benefit from a caudal maxillary block via the infraobital foramen. The local anesthetic is applied to the maxillary nerve through the infraorbital canal. The needle can be introduced through the skin or the mucosa over the foramen. The foramen is on the lateral side of the maxilla, slightly rostroventral to the eye and dorsal to the distal root of the maxillary third premolar tooth. *Place the thumb of the free hand on the foramen and the needle tip into the foramen. Aspirate and then slowly inject the local anesthetic.*[24]

Dental blocks can be utilized to help provide analgesia for mandibulectomy or maxillectomy procedures. Nerves blocked when performing a *maxillary nerve block* are the maxillary; infraorbital; caudal, middle, and superior alveolar dental; incisivomaxillary; and rostral superior alveolar dental. This provides anesthesia to the ipsilateral maxilla including the teeth, bone, and soft tissues (including the skin of the nose, cheek, and upper lip). This block can be done intraorally or transcutaneously.

Intraoral dental block: *Place the needle behind the last molar tooth and the maxillary tuberosity* (the thin caudal margin of the maxilla covering the roots of the last molar tooth). *Open the mouth and palpate the maxillary tuberosity. Insert a short needle between your finger and the bone. Position it as perpendicular as possible to the hard palate or with a slight medial direction with the needle bevel directed rostrally. Once in place, aspirate and slowly inject the local anesthetic.*

Transcutaneous dental block: *Insert the needle between the caudal margin of the maxilla and the coronoid process of the mandible below the rostroventral border of the zygomatic arch. Palpate the structures on the side of the face just below the ocular globe. Insert the needle perpendicular to the skin and advance a short distance to the pterygopalatine fossa and the maxillary nerve. Aspirate and then slowly inject the local anesthetic.*

Caudal inferior alveolar nerve block: This block is performed to provide analgesia for a mandibulectomy. The mandibular teeth, rostral lower lip, and rostral intermandibular region are anesthetized. If the local anesthetic diffuses to the lingual nerve, desensitization of the soft tissue and periosteum of the lingual side of the mandible, floor of the mouth, and rostral two-thirds of the tongue may develop. This loss of tactile and pain sensation in the tongue can lead to self-inflicted chewing injury in the recovery period until normal sensation returns. If this should happen, the animal may need to be sedated to prevent tongue injury until the block wears off. To help prevent this complication, it is very important to keep the needle bevel next to the mandible and with the bevel directed toward the bony surface with both the intraoral and extraoral techniques.

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Intraoral approach: *Open the mouth and displace the tongue to the contralateral side. Palpate the mandibular foramen and advance the needle through the mucosa medial to the mandible at the level of the last mandibular molar tooth. Direct the needle ventrocaudally towards the angular process between the finger and the mandibular bone to reach the mandibular foramen. Aspirate and then inject slowly;* tissues at the injection site should swell during the injection.

Extraoral approach: *Place the animal in lateral recumbency with the side to be blocked uppermost. Open the mouth and place your hand between the patient's tongue and mandible. Advance your finger ventrocaudally along the mandible until you palpate the foramen at the rostral margin of the medial pterygoid muscle. Advance the needle through the mucosa medial to the mandible, at the level of the last mandibular tooth. Direct the needle ventrocaudally towards the angular process to reach the mandibular foramen. Direct the needle bevel toward the bony surface and slowly advance the tip of the needle medial to the mandible to the mandibular foramen that you are palpating intraorally. Direct the needle tip over the foramen and nerve. Aspirate and then inject;* swelling of the tissues should be detected through the intraoral palpation site.[24]

Intravenous (IV) regional anesthesia or a **Bier block** can be done using lidocaine for analgesia and anesthesia of the extremities distal to the elbow and hock (see Table 13.2). The animal must be under heavy sedation or general anesthesia when using this technique due to the discomfort caused by the tourniquet. This technique should be done for short duration procedures, 90 minutes or less, due to the tissue ischemia that can result from the tourniquet. Anesthesia occurs distal to the tourniquet, with onset beginning distally and progressing proximally. The local anesthetic should be injected as far distally as possible in the limb to be blocked. When the tourniquet is released, the analgesic effects may persist for up to 30 minutes. The tourniquet must prevent blood from entering the limb and prevent the local anesthetic from entering the systemic circulation. The tourniquet can be a rubber or elasticized cloth or pneumatic cuff. When using a nonpneumatic tourniquet the area under the tourniquet should be protected with soft padding to prevent compressive tissue damage. A pneumatic cuff tourniquet should have a pressure 50 to 100 mm Hg above the systolic blood pressure to prevent blood leakage. *Place a distal IV catheter in the limb to be blocked and secure it in place. Use an esmarch or similar elastic bandage to exsanguinate the limb. Wrap the bandage concentrically around the limb starting at the toes and wrapping proximally, being careful not to dislodge the IV catheter. Apply the tourniquet and note the time. Once the tourniquet is in place, carefully remove the exsanguinating bandage. Slowly inject the lidocaine into the catheter over 2 to 3 minutes. Avoid high injection pressures. At the completion of the procedure, remove the tourniquet slowly to allow blood flow to return to the limb. Observe the procedure site for bleeding.* Once the tourniquet is released, any remaining local anesthetic will be delivered to the systemic circulation. *Monitor the patient for any signs of local toxicity such as hypotension and bradyarrhythmias for 10 to 15 minutes after tourniquet removal.*[25]

Intercostal nerve blocks are used to provide analgesia in animals undergoing a thoracotomy. It may also improve ventilation postoperatively. It can be done when the skin incision has been made, prior to incising the intercostal muscles, or through the skin prior to incision. The intercostal nerve lies immediately caudal to the associated rib. Aspiration prior to local injection is important as the intercostal vessels are closely associated with the nerves. Bupivacaine is most commonly used for this block due to its long duration of action. *Place the patient in lateral recumbency with the operative side up. Using a 1.5-inch needle, block the nerve at the incision site and block two nerves on either side of the incision site to provide a wide section of analgesia.*[26] *Palpate the rib immediately cranial to the incision site as far dorsally as possible to target the nerve as proximally as possible. Advance the needle through the skin onto the lateral aspect of the rib. Walk the needle tip off the rib caudally to the rib; do not place the needle beyond the depth of the rib. Aspirate and then inject the local anesthetic.*

Brachial plexus blocks can be used to provide analgesia for structures distal to the elbow. The brachial plexus of the dog consists of the ventral branches of C6, C7, C8, and T1 spinal nerves. The important nerves are the suprascapular, subscapular, axillary, musculocutaneous, radial, median, and ulnar. This block can be done by palpation only or with nerve stimulation guidance. When doing nerve stimulation guidance an insulated needle is used; when done by palpation only an appropriate length spinal needle is used. *Anesthetize the patient and place in lateral recumbency with the leg to be blocked uppermost. Hold the leg in a natural position perpendicular to the longitudinal axis of the body. Inject cranial to the acromion and medial to the subscapular muscle.*[27]

Retrobulbar blocks can be used to provide analgesia for eye enucleation. It will provide analgesia and prevent ocular movement during surgery. Skin infection, ocular neoplasia, or ocular fungal infection may be relative contraindications to placement of the block to avoid seeding abnormal cells into the deeper tissues. An inferior temporal palpebral approach is preferred. Lidocaine or bupivacaine are commonly used; bupivacaine provides a longer duration of action (see Table 13.2). A 1.5-inch (40 mm), 22-gauge spinal needle bent to approximately a 20-degree angle is used. The needle can be passed through the lower eyelid or through the transconjunctiva. *Using the lateral canthus and middle of the lower lid as landmarks, position the needle midway between these landmarks along the inferior eyelid at the level of the orbital rim. Direct the needle along the floor of the orbit. You will note a slight pop when you pierce the orbital fascia; then redirect the needle dorsally and towards the nose to reach the apex of the orbit. Inject the local anesthetic slowly. If there is resistance to injection, this could be due to penetration of the perineural sheath or abutment on the bony orbit; draw back and redirect.* Complications include inadvertent penetration of the globe, intrathecal injection due to penetration of the optic nerve, retrobulbar hemorrhage, or optic nerve damage.[28]

Radial, ulnar, musculocutaneous, and median (RUMM) nerve blocks can be used to provide analgesia to the distal aspect of the thoracic limb in dogs. This is a two-injection technique to obtain a peripheral block of the radial, ulnar, musculocutaneous, and median nerves. *For the radial nerve, place the dog in lateral recumbency with the leg to be blocked upwards. Flex the elbow 90 degrees to allow the soft tissue structures to be less stretched and therefore easier to manipulate. Measure the length of the lateral aspect of the humerus along a line connecting the most prominent craniodorsal point of the greater tubercle and the most prominent point of the lateral epicondyle. Divide this length into thirds. Locate the radial nerve at the junction of the middle and distal thirds, approximately 1 cm caudal to this line and lying between the lateral head of the triceps and the brachialis muscle. Palpate a branch of the radial nerve crossing the humerus slightly distal to this point. Palpate the brachialis muscle and apply craniomedial pressure so that your thumb is on the shaft of the humerus. Move the brachialis*

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muscle cranially away from the lateral head of the triceps. Insert a 22-gauge spinal needle caudal to your thumb at a 45-degree angle, perpendicular to the long axis of the humerus, penetrating the lateral head of the triceps muscle so that the needle contacts the caudolateral aspect of the humeral shaft at the location of the radial nerve. Face the bevel of the needle cranially. Remove the stylet and withdraw the needle slightly so it is no longer contacting the bone. Aspirate and then inject the local anesthetic. Block the ulnar, median, and musculocutaneous nerves with the dog in a lateral position and the leg to be blocked downward. Pull the upper leg caudally and position the down leg with the elbow flexed at 90 degrees. Measure the length of the medial aspect of the humerus along a line connecting the most prominent craniodorsal point of the greater tubercle with the medial epicondyle. The median, ulnar, and musculocutaneous nerves are close to one another, approximately half the distance along this line from the medial epicondyle at the most proximal point at which the humeral shaft can be palpated. *Locate this point, apply digital pressure to the medial aspect of the limb, and palpate the humerus. Insert a 22-gauge spinal needle at a 45-degree angle from a caudal direction, perpendicular to the long axis of the humerus and caudal to the finger applying pressure, until it contacts the caudomedial aspect of the humerus. Face the needle bevel up. Release the digital pressure, remove the stylet, aspirate, and inject half the local anesthetic and then continue injecting as the needle is withdrawn.*[29] The use of ultrasound guidance and a peripheral nerve stimulator may improve the ability to locate the nerves and may enhance the success rate of the block.[30]

Femoral/sciatic nerve blocks can be done in dogs to provide analgesia for rear limb procedures (Videos 13.1 to 13.4). The femoral nerve innervates the femur, stifle, medial stifle joint capsule, intraarticular stifle structures, and skin of the medial aspect of the hind leg. The sciatic nerve innervates the caudolateral stifle, joint capsule, lateral meniscus, tibia, tarsus, digits, and skin of the caudal and lateral aspects of the hind leg. Complete local anesthesia of the stifle can be achieved with a block of both the femoral and sciatic nerves. This block has been shown to be as effective as an epidural for postoperative rear leg analgesia and is less apt to cause urinary retention.[31] The block is best performed with a peripheral nerve stimulator utilizing an insulated stimulator needle. *Position the dog in lateral recumbency with the affected leg up. Block the sciatic nerve by placing the stimulator needle at a point one-third the distance between the greater trochanter and ischiatic tuberosity. Flex the hock to confirm location of the needle close to the nerve. The femoral triangle is defined by the iliopsoas, pectineus, and sartorius muscles. Insert the stimulator needle at a 20-degree angle toward the iliopsoas, immediately cranial to the femoral artery. Extend the stifle to confirm proximity of the needle to the femoral nerve.* The needle stimulates muscle contraction at a current of 0.4 mA. *Aspirate the needle and if you do not see blood, block both nerves with injections of 0.5% bupivacaine until muscle contraction is lost.*

Cranial cruciate surgery local blocks. Nocita, a novel extended-release formulation of bupivacaine, has been approved for use in dogs to treat postoperative pain from cranial cruciate surgery. A single dose is infiltrated into the surgical site. In this formulation, multivescular liposomes composed of nonconcentric bilayers of phospholipids, cholesterol, and triglycerides encapsulate bupivacaine and are designed such that the bupivacaine is gradually released from the vesicles over 96 hours as the lipid bilayers break down (see Table 13.2). The dose is administered by tissue infiltration just prior to closure of the cranial cruciate ligament surgery. Measurable analgesic effect appears to last for up to 72 hours. *Using a 1- to 1.5-inch (25–40 mm) long needle, infiltrate the surgical site by inserting the needle to near the hub and injecting the solution as the needle is pulled out. Insert the needle at varying angles and then approximately parallel to the surface of the skin. Repeat the injections to create an area of infiltrated tissue around and at all levels of the wound. Inject approximately 25%, 50%, and 25% of the solution into the tissues around the joint capsule, fascial tissue, and subcuticular tissue, respectively.* Dilution of the solution with saline can be done with a maximal dilution of 1:1.[32]

Epidurals can be used in dogs and cats to provide anesthesia and analgesia for procedures in the caudal half of the animal (Table 13.5). The local anesthetics work on the nerve root. The pelvic limb is supplied by spinal nerves arising from L3 to S1.

TABLE 13.5 Epidural Anesthesia in the Dog

Epidural Volumes

Low epidural:
- 0.2 mL/kg results in analgesia/anesthesia of pelvis and caudal abdomen

High epidural:
- 0.3 mL/kg results in analgesia/anesthesia of pelvis, abdomen, and thorax
- Usually performed with only an opioid (saline can be used to increase the volume)
- Local anesthetics should be avoided in high epidurals to prevent sympathetic trunk blockade and intercostal nerve paralysis

Drug	Dose (mg/kg)	Onset of Action (min)	Duration of Action (h)
Lidocaine 2%[a]	0.5	10	1–1.5
Bupivacaine (0.25% or 0.5%)[a] (preservative free)	0.5	20–30	4.5–6
Ropivacaine (0.5%)[a]	0.5	20–30	4.5–6
Fentanyl	0.001	15–20	3–6
Morphine (preservative free)[b]	0.1	30–60	10–24
Buprenorphine	0.004	30–45	12–18

[a]A block to T1 leads to intercostal nerve paralysis and bradycardia; a block to C5–C7 leads to phrenic nerve paralysis.
[b]Dosage, onset, and duration are based on clinical experience; each patient should be evaluated individually. Reduce the dose by at least half for spinal administration of local anesthetics; reduce the epidural dose by 25% in geriatric, obese, and pregnant animals and in those with space-occupying lesions of the spinal cord or conditions in which venous engorgement is expected.
Protocols courtesy Dr. Stuart Clark-Price.

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The volume of agent given must be sufficient to reach L3 to achieve sensory and motor block of the entire limb. The drug should be administered slowly over 1 to 2 minutes to keep it from going too far cranially. The injection should flow smoothly and without resistance. To help monitor the resistance, a small air bubble of 1 to 3 mL should be aspirated into the syringe; this bubble should be next to the plunger. Once the syringe is connected to the needle placed for injection into the animal, this bubble should not compress more than 50% of the starting volume as the drug is administered. If the bubble compresses too much, this is suggestive of resistance and the injection should be stopped and the needle readjusted. The air is not to be injected into the epidural space. Contraindications for an epidural can be remembered with the acronym CHINAS as described in Box 13.5. An epidural is done in a heavily sedated animal after a local block has been performed at the site of injection or in an animal under general anesthesia.

Agents commonly used for epidurals include local anesthetics, opioids, and alpha-2 adrenergic agonists; combinations of these agents can also be utilized. Local anesthetics commonly used are preservative-free lidocaine and bupivacaine; these agents block sensory, motor, and sympathetic nerves, which can lead to vasodilation and loss of motor function. The effects are dose dependent, with higher doses leading to a more profound block. Preservative-free morphine is the opioid most often used. Morphine is hydrophilic and less lipid soluble than fentanyl, leading to a longer duration of action. Opioids affect sensory nerves only, preserving motor function. The longest duration of action (12 to 24 hours) is achieved with a combination of morphine and bupivacaine. Opioids can cause urinary retention; thus it is recommended to express the urinary bladder of an animal that has received a preoperative opioid epidural prior to recovery from anesthesia. If urinary retention is present the following morning, an IV dose of naloxone will often result in immediate urination. Pruritus has also been reported as a potential adverse effect of opioid epidurals; IV naloxone may be given to attempt to reverse this affect. Alpha-2 adrenergic agonists (e.g., dexmedetomidine) have been used epidurally; these agents will also have dose-dependent systemic effects such as sedation, supraspinal analgesia, bradycardia, peripheral vasoconstriction, and vomiting.

BOX 13.5 Contraindications for an Epidural: CHINAS

C = Coagulation disorders; due to the increased risk of hemorrhage, potentially leading to a hematoma causing spinal compression and neurologic deficits

H =Hypovolemia and hypotension; hypotension could be exacerbated with a sympathetic nerve blockade; local anesthetics will cause vasodilation

I = Infections; especially of the skin can lead to introduction of infection into the vertebral canal

N = Neoplasia or neurologic deficits. It is recommended to not pass the needle through a neoplastic area as this could see tumor cells into the vertebral canal, and if the animal has preexisting disease, an epidural can make it difficult to monitor resolution or progression of the condition

A = Anatomy; abnormalities of the lumbosacral space will lead to difficulty in palpating the landmarks

S = Sepsis or bacteremia; laceration of a vessel in the epidural space could lead to hematogenous spread of the infection into the epidural/spinal space

The lumbosacral space is the preferred site for epidural injection due to the relatively wide intervertebral space at L7 to S1. *Place the animal in either sternal or lateral recumbency (Video 13.5). Perform a sterile prep wearing sterile gloves. If the animal is in sternal recumbency, pull the pelvic limbs forward to widen the intervertebral space; in lateral recumbency, keep the spine either neutral or flexed.* In animals with pelvic or femoral fractures that are displaced it may be best to perform the epidural with the animal in the lateral position. *Use a 22- to 20-gauge, 1.5- to 2.5-inch (40–60 mm) spinal needle (depending on the size of the animal) or a Tuohy needle (22 to 18 gauge). Palpate the L7-S1 space. Insert the needle perpendicular to the skin on the dorsal midline, caudal to the spinous process of L7. Advance the needle through the skin into the subcutaneous tissue and then through the interspinous ligament (there will be some resistance) and continue slowly until you feel a "pop" as the needle penetrates the ligamentum flavum. You will feel a loss of resistance as the epidural space is entered. If during placement of the needle you contact bony structures, partly withdraw the needle and redirect it caudally or cranially, walking off the adjacent bones to find the intervertebral space.* There are two commonly used methods to confirm correct placement of the needle, loss of resistance and a hanging drop. *With the former method, apply pressure to the syringe plunger as you advance the needle until you feel a "pop" and a sudden loss of resistance as the needle passes through the ligamentum flavum and enters the epidural space. For the hanging drop method, place a drop of saline or the drugs to be injected into the hub of the needle. As the needle penetrates the epidural space watch for the liquid drop to be aspirated into the space due to the subatmospheric pressure within the vertebral canal.* This technique can only be used with the animal in sternal recumbency; use the technique described where a loss of resistance is felt when the animal is in lateral recumbency. *If blood is detected in the needle hub, suggesting that the needle has inadvertently pierced a venous sinus, remove the needle and use a new one for the epidural. Inject slowly and smoothly to avoid excessive cranial spread and an irregular block.* It is possible for the dural sac to be punctured during an epidural; however, dural puncture is more common in cats than in dogs because in dogs the dural sac ends at the level of L6–L7, whereas in cats it ends at S1. *If the dural sac is penetrated, withdraw the needle to the epidural space.*

An **epidural catheter** can be placed to provide longer term analgesia. The Touhy needle is required for catheter placement because the catheter is passed through this needle. *Place the catheter in a sterile fashion with the animal in sternal recumbency. Use the same landmarks as for the single needle injection described previously. Place the Touhy needle into the epidural space, directing the curved needle bevel cranially to assist in catheter placement; tip the needle hub caudally to help in catheter placement. Once in the space, attach an adapter to the needle hub to provide rigidity to the catheter. Remove the needle stylet and thread the epidural catheter through the needle. Once the catheter is at the needle tip, apply slight pressure if required to advance the catheter past this point. Advance the catheter to the desired level. Once the catheter is in place, carefully withdraw the needle over the catheter, keeping hold of the catheter at its point of entry into the skin to avoid displacement as the needle is removed. If desired, tunnel the catheter subcutaneously away from the point of entry to provide additional fixation and minimize catheter contamination. Aspirate the catheter to ensure there is no blood or cerebrospinal fluid present. Connect a bacterial filter, primed with local anesthetic, to the catheter adapter. Secure the catheter in place with a transparent dressing and adhesive.*

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Then slowly inject the anesthetic agents into the epidural space (in the awake animal slow injection is necessary to prevent vomiting). If the catheter cannot be advanced, remove it and the needle together; do not remove the catheter with the needle in place as the catheter could be sheared off and lost in the patient. Resistance to injection will be greater than through the needle because the catheter has a narrower diameter. *Confirm correct catheter placement by ultrasound, radiography, or fluoroscopy. When removing the catheter, grasp it close to the point of entry and gently pull it out. Do so in a sterile fashion (wear sterile gloves and use sterile scissors) so that the tip can be cultured. Make sure the whole catheter has been removed.*

NOTE Warn owners that it has been reported that hair regrowth at the epidural site may be delayed after an epidural has been performed.[33]

Coccygeal epidurals can be done to provide analgesia and relaxation to the perineum, penis, urethra, colon, and anus without loss of motor function to the rear limbs by blocking the pudendal, pelvic, and caudal nerves. Lidocaine has been used in this block to provide analgesia and pain management, as well as assist with catheterization of the obstructed feline urethra. This block could also be used for analgesia for dystocia, tail amputations, or degloving injury and other perineal procedures.[34] *Place the cat in sternal recumbency with the area over the sacrum and first coccygeal vertebra clipped and sterilely prepped. Inject between the first and second coccygeal vertebrae, which is the most mobile joint caudal to the sacrum. Determine the injection site by moving the tail. Use a 25-gauge, 1-inch (25 mm) needle to penetrate the skin at the midline, then direct the needle at a 30- to 45-degree angle and advance it through the interarcuate ligament/ligamentum flavum until you feel a "pop" when the ligament is penetrated. If you encounter bone, redirect the needle or withdraw it slightly. Aspirate and then inject 0.1 to 0.2 mL/kg of 2% lidocaine. Ensure there is minimal resistance to the injection. Withdraw the needle and observe the tail for relaxation. Administer a second dose of lidocaine if the first attempt was unsuccessful.*

ACUPUNCTURE AND ELECTROACUPUNCTURE

Although the appeal of acupuncture as a pain-relieving therapy is increasing in veterinary surgical practice, there remains some reticence among surgeons to accept acupuncture as a viable treatment modality; this reticence persists despite numerous experimental and clinical studies demonstrating both specific biological effects of acupuncture and measurable clinical benefit. Most of these studies have been performed in laboratory animals and humans with painful conditions, but much of the knowledge from this literature can be applied to veterinary surgical practice.[35–38] One of the barriers to integrating acupuncture into modern surgical practice is a commonly held perception that it is based on archaic principles, rather than contemporary medical science. Although the Traditional Chinese Veterinary Medical (TCVM) terminology and mechanistic explanations are indeed based on an antiquated system of understanding, the physiologic principles and rationale for clinical application of acupuncture as a pain-relieving modality can be couched in contemporary medical terms. One of the frustrating aspects of learning how to use acupuncture in modern surgical practice relates to the nomenclature used to describe the acupuncture points; the acupuncture points are found along specific conceptual pathways—meridians or channels—that traverse the patient's body. Because these pathways and respective points are named according to an ancient medical system, they may not make inherent sense to veterinary surgeons. For instance, there are several gallbladder points around the hip joint.

Although acupuncture has been around for thousands of years, many of the mechanisms by which acupuncture is thought to exert beneficial physiologic effects have only recently been elucidated, and this is due mainly to advances in neuroimaging and molecular biology techniques. Although the actual names of the acupuncture points along their respective meridians may cause some confusion, the majority of these points and their respective meridians line up with the PNS. Others correspond to known fascial planes of the body. Some believe that acupuncture signals may also follow vascular routes. It is possible that signals are transmitted through a variety of anatomic scaffolds, but it is likely that the nerves play a prominent role in all the scenarios described.[35,38–41] In general the entire body, including the CNS and the viscera, is accessible via the PNS. Classification of acupuncture points is also based on the location of nerves associated with those points (Box 13.6).

Acupuncture includes manual acupuncture or "dry needling" (DN), aquapuncture (injecting a sterile liquid into an acupuncture point), and electroacupuncture (EA). Aquapuncture may have a greater therapeutic effect than DN; presumptive mechanisms include enhanced stimulation of the injected acupuncture point via spatial distortion of the injected substance and pharmacologic effects of the injected substance. It is generally believed that clinical effects of EA are more potent and longer lasting than DN or aquapuncture. The vast majority of experimental and human clinical literature supports the use of EA as the most likely permutation of this treatment method to exert a measurable and lasting physiologic and clinically appreciable effect on pain alleviation. In short, the authors' recommendation is to employ EA routinely during acupuncture sessions rather than DN alone.[42–44]

An acupuncture point can be thought of as an anatomic unit consisting of free nerve endings, small arterioles, venules, lymphatics, and mast cells. There is some evidence that acupuncture points may be thought of as larger than "points" and similar to receptive regions or fields in the vicinity of the point. Acupuncture points are usually physically in areas of surface depressions and physiologically in cutaneous regions typified by low electrical resistance and high electrical conductivity.[38,45]

The effects of acupuncture on pain relief include local, segmental (spinal cord level), and suprasegmental mechanisms. EA

BOX 13.6 Classification of Acupuncture Points Based on the Location of Nerves Associated With Those Points

Type I (motor points): located where major nerves penetrate muscles. They are the most common and comprise nearly 70% of all acupuncture points.
Type II: located where nerves intersect on the dorsal and ventral midlines.
Type III: located where superficial nerves branch.
Type IV: located where nerves penetrate tendons (e.g., Golgi tendon organs).

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BOX 13.7 Peripheral (Local) Physiologic Effects of Acupuncture, in Particular Electroacupuncture, That Contribute to Alleviating Pain

- Induction of release of endogenous opioids from lymphocytes, macrophages, and granulocytes into tissue. These opioids then suppress the propagation of nociceptive signals by acting at receptors of the peripheral nerves in the tissue.
- Activation of sympathetic nerve fibers, which leads to increased levels of endogenous opioids in the region. Some of this effect is via adrenergic receptor activation of inflammatory cells, which causes them to release β-endorphin into the tissue.
- Sympathetic nerve fiber activation also leads to increased expression of specific intracellular adhesion molecules in blood vessels of inflamed tissue. These adhesion molecules promote migration of neutrophils and mononuclear cells that contain β-endorphin and met-enkephalin.
- Increased levels of cannabinoid CB2 receptors in tissue, which leads to upregulation of endogenous local opioids.
- Decreasing levels of local inflammatory cytokines, including tumor necrosis factor-α, interleukin 1β, and interleukin 6. This is also believed to involve endogenous opioids.
- Inhibition of cyclooxygenase-2 and prostaglandin E2 production.

BOX 13.8 Antinociceptive Effects at the Spinal Level Induced by Acupuncture

- Dampening of glutamate receptor activity in spinal cord dorsal horn neurons, via increasing endogenous opioids, noradrenaline, and serotonin levels.
- Increased serotonin levels in the spinal cord accentuate the ability of gamma-aminobutyric acid (GABA) to inhibit pain signal transmission.
- Inhibition of spinal glial cell activation, thereby decreasing astrocyte and microglial-derived pain-promoting cytokines (interleukin 1β, interleukin 6, tumor necrosis factor-α, cyclooxygenase-2, and prostaglandin E2).
- Blocking of substance P release in spinal cord gray matter (substance P promotes nociceptive signals and activates glial cells).
- Upregulation of nociception/orphanin FQ (N/OFQ) receptors in the spinal cord gray matter. N/OFQ is a powerful opioid-related peptide that has potent analgesic properties and is widely distributed throughout the spinal cord. It inhibits C fiber evoked responses and wind-up.
- Increasing spinal cord dorsal horn levels of acetylcholine and dopamine, both of which are believed to inhibit nociceptive signaling.
- Activation of spinal cord δ opioid receptors, leading to reduced cellular GABA reuptake. Increased levels of GABA in the extracellular space enhance its antinociceptive effects.

is more likely to most effectively recruit all three of these mechanisms, in comparison with DN alone. Acupuncture can be thought of as a method of counterirritation. After providing a stimulus to the tissue with either DN or EA, a local reaction ensues that elicits a number of inflammatory and immune responses. Acupuncture, in particular EA, has a number of physiologic effects peripherally (locally) that contribute to alleviating pain. Many of these processes involve endogenous opioids (Box 13.7).[35,38,41,44]

The segmental (spinal) and suprasegmental (brain) effects of acupuncture cannot be completely separated, as they are anatomically and physiologically connected. However, there are a number of antinociceptive effects induced by acupuncture (again, particularly EA) at the spinal level (Box 13.8).[35,37,38,41] Although the mechanisms for suprasegmental effects of acupuncture are not as well understood as segmental effects, they are based on the descending antinociceptive pathway. There is considerable evidence from functional magnetic resonance imaging and positron-emission tomography studies in people, as well as experimental animal models, that specific acupuncture points, when stimulated, will activate specific regions of the brain. This point specificity for stimulation of antinociceptive regions of the brain stem descending pain-modulating pathway is most convincing for limb (vs. trunk) acupuncture points. In addition, activation of such points with a sufficient stimulus will activate the intended brain stem center, without necessarily producing an appreciable segmental effect. By contrast, stimulating an acupuncture point in the dermatomal segment of interest (e.g., a back *Shu* point for thoracolumbar pain) may produce a mainly segmental analgesic response, or a segmental and descending brain stem response, depending on the stimulus intensity (if intense enough, the ascending pathways to brain stem nociceptive centers may also be recruited). These physiologic phenomena are consistent with the typical TCVM recommendations to treat at both local and distal sites. In this way, local, segmental, and suprasegmental effects can be combined for maximum results. Some specific examples of suprasegmental effects of acupuncture (primarily EA) are included in Box 13.9.[38,41,45]

BOX 13.9 Specific Examples of Suprasegmental Effects of Acupuncture (Primarily Electroacupuncture)

- Activation of serotonergic nucleus raphe magnus neurons in the medulla.
- Activation of noradrenergic neurons in the locus ceruleus of the pons.
- Stimulation of the hypothalamus to release β-endorphin.
- Activation of descending neurons of the periaqueductal gray matter of the mesencephalon.

It has also been demonstrated that low-frequency EA (2 to 10 Hz) is more effective for pain control than high-frequency EA (100 Hz).[38,41] However, there is some rationale to combining modes of acupuncture (DN and EA) and different frequencies of EA to produce a greater analgesic effect than would be possible with only one modality or one frequency. There is some evidence that DN may be more effective at mediating analgesic effects through C fibers than EA, with EA mediating analgesic effects mainly through Aβ and Aδ fibers. EA has been shown to cause the release of different mediators of analgesia, depending on the frequency applied. At low frequencies (2 to 10 Hz), endorphins and enkephalins are mainly released. At higher frequencies (50 to 100 Hz), the primary opioid released is dynorphin. At even higher frequencies (>200 Hz), the most prominent neurochemical mediator released is serotonin. In addition, there are other antinociceptive agents released, according to certain applied frequencies. For example, at low frequencies, there are also noradrenergic and muscarinic cholinergic mechanisms that are recruited. At high frequencies, there are muscarinic cholinergic and GABA-ergic mechanisms that are brought into play to help decrease nociceptive signaling. It would appear that endorphins and enkephalins may be the most effective EA-induced mediators of pain (at low-frequency stimulation); however, it is likely that providing other pain-mitigating agents at higher frequencies may have an additive effect.[38,41,46]

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14

Principles of Minimally Invasive Surgery and Imaging of the Surgical Patient



Minimally invasive surgical procedures (e.g., endoscopy) are diagnostic and therapeutic tools that are increasingly expected by veterinary clients. This chapter provides a brief overview of the endoscopic equipment and techniques used in these procedures. Specific techniques (e.g., thoracoscopic pericardiectomy; removal of osteochondritis dissecans [OCD] lesions) are found in the relevant chapters for that system, disease, or condition.

DEFINITIONS AND TERMINOLOGY

Endoscopy is the use of an instrument (i.e., an endoscope) to visualize the interior of an organ or other area that otherwise cannot be examined without surgery. It can be diagnostic or interventional. **Gastroduodenoscopy** is endoscopy of the esophagus, stomach, duodenum, and occasionally the upper jejunum. **Colonoscopy** is endoscopy of the colon. **Ileoscopy** is endoscopy of the ileum and is performed in conjunction with colonoscopy. **Proctoscopy** refers to examination of the anus and rectum. **Bronchoscopy** is endoscopy of the trachea and bronchi, whereas **laryngoscopy** is examination of the pharynx and larynx. **Rhinoscopy** generally refers to placing an endoscope through the anterior nares and examining the nasal passages. It may or may not include using an endoscope to examine the choanae. **Cystoscopy** is endoscopy of the urinary bladder and may include retrograde cystoscopy (i.e., advancing the endoscope through the urethra and into the bladder) or transabdominal placement (i.e., placing the scope through a cannula that has been inserted through the abdominal wall and the bladder wall). **Vaginoscopy** is endoscopy of the vagina. **Laparoscopy** is endoscopy of the peritoneal cavity, whereas **thoracoscopy** is endoscopy of the pleural cavity. **Arthroscopy** is endoscopy of a joint and may be diagnostic but is usually interventional.

Flexible endoscopy uses an endoscope that has a flexible insertion tube designed to look and/or move around corners. The degree of flexibility depends on the instrument, but being able to make bends of 180 degrees or more is typical for most flexible scopes. Flexible endoscopes have a *handle* (where the scope is held by the operator), an *insertion tube* (the part that is inserted into the patient), and an *umbilical cord* (the part that attaches the scope to the light source and video processor). The *biopsy channel* is the passage that allows one to aspirate air or liquids, as well as place instruments through the scope (e.g., biopsy forceps, foreign body retrieval forceps, aspiration tubes, cytology brushes). Almost all flexible endoscopes are now "*immersible,*" meaning they can have their handles placed in water without risk of damage.

Rigid endoscopy uses a plastic or metal scope, which may be solid or hollow. Endoscopic *telescopes* are generally solid (some have a channel in them) and have a lens at the tip of the scope that points at various angles, but the telescope itself cannot bend. Hollow endoscopes often have an *obturator*—a device placed through the endoscope to facilitate insertion of the scope into the organ desired (e.g., esophagus, colon). Rigid endoscopic telescopes are often placed through a *cannula,* which is a metal or plastic tube. A cannula may have a sharp or blunt-tipped *trocar* designed to facilitate penetration into joints, the abdomen, or the chest.

A "*red out*" or a "*white out*" refers to having the viewing tip of the endoscope so close to the surface of what is being examined that one cannot focus on the surface (i.e., a blur occurs) or to having debris on the viewing end of the scope. In double-puncture laparoscopy, thoracoscopy, and arthroscopy, *triangulation* refers to visualizing the tip of the biopsy and operative instruments through the scope to perform various procedures within body cavities or joints.

In arthroscopy, *instrumentation* refers to the insertion of an endoscope, arthroscope, or other tool into the joint. Equipment inserted into the joint through the skin and soft tissue is done so through *portals*. Portals are defined by their use. The scope is inserted through a *scope* or *camera portal,* whereas power and hand tools are inserted through an *instrument portal.* Arthroscopes are always used through *cannulas.* Fluid flowing into the joint is referred to as *inflow* or *ingress;* fluid flowing out of the joint is referred to as *outflow* or *egress*. Repeat arthroscopic examination of a joint that has been previously scoped is referred to as *second-look arthroscopy.*

Minimally invasive osteosynthesis refers to fracture fixation using small skin incisions and techniques designed to limit the deeper soft tissue surgical trauma. **Minimally invasive plate osteosynthesis** refers to reduction and fixation with a plate of any design, without direct surgical exposure of the fracture site, using small skin incisions and subcutaneous or submuscular insertion of the plate. These techniques are discussed in Chapters 32 and 33.

ENDOSCOPY: GENERAL PRINCIPLES, EQUIPMENT, AND TECHNIQUES

Endoscopy is used to biopsy organs, remove foreign objects, examine the interior surface of hollow structures, and perform procedures typically done by more invasive surgery. As such, endoscopy is valuable only when it eliminates the need for more

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ABSTRACT

Minimally invasive surgery is a growing field of veterinary surgery. A basic understanding of the techniques and possible indications is critical so that doctors can optimize patient care while minimizing invasive techniques when possible. Sophisticated imaging techniques are now readily available in veterinary medicine. An understanding of the physics associated with these imaging modalities is useful for practitioners and will enhance the clinician's ability to evaluate the images and improve knowledge of clinical indications.

KEYWORDS

esophagoscopy
gastroscopy
gastroduenoscopy
colonoscopy
laparoscopy
biopsy
endoscope
arthroscopy
minimally invasive surgery
thoracoscopy
cystoscopy
sophisticated imaging
computerized tomography
magnetic resonance imaging
positive emission tomography with CT

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invasive, classic surgery. If tissue samples are inadequate for diagnosis, unacceptable trauma occurs during removal of foreign objects, or mucosal surfaces cannot be adequately examined, then endoscopy ceases to be useful. Unfortunately, endoscopy is sometimes approached more like a "hobby" than an important procedure to be learned and practiced diligently. Flexible endoscopy of the upper gastrointestinal (GI) tract is more difficult to properly perform than is widely appreciated, especially in regard to biopsying the mucosa. If the clinician is not well trained in the first place and/or is not going to perform the procedure often enough to maintain expertise, then it is usually best if patients are referred elsewhere or classic surgery is elected instead. In addition, endoscopic equipment is complex and expensive. It is imperative that the clinician be adequately trained to use the equipment properly to avoid damage and that setup and maintenance of the equipment is thoroughly understood to direct the hospital staff in handling and cleaning of the endoscopy system. It is best if only one or two staff are responsible for maintenance and setting up the equipment.

INDICATIONS

Flexible Endoscopy

The primary use of flexible endoscopy in the alimentary and respiratory tracts in veterinary medicine is to visualize and obtain tissue or cytologic samples of mucosa. Removing foreign objects, dilating strictures, controlling hemorrhage, and inserting tubes are important but less commonly performed procedures (Box 14.1). Tissue sampling (i.e., biopsy or cytology) should almost always be performed in diagnostic procedures regardless of gross mucosal appearance unless there is a specific reason not to biopsy (e.g., coagulopathy, risk of perforation). Tissue samples obtained by flexible endoscopy are usually limited to the mucosa and adjacent submucosa as opposed to the full-thickness samples

BOX 14.1 Primary Indications for Endoscopic Procedures in Dogs and Cats

Gastroduodenoscopy

- Gastric and intestinal biopsy/cytology for diagnosis of infiltrative and lymphatic disorders
- Identification of a mass, ulceration, erosion, lymphangiectasia, or *Physaloptera* infestation
- Identification and removal of foreign objects
- Location of lesions (e.g., ulcer, site of bleeding)
- Placement of a gastrostomy tube
- Removal of polyps causing clinical signs

Esophagoscopy

- Identification and removal of foreign objects
- Diagnosis of esophagitis
- Diagnosis and dilation of strictures
- Identification of hiatal hernia
- Identification and biopsy of tumors

Proctoscopy and Colonoileoscopy

- Biopsy of the rectum, colon, ileum, or cecum looking for infiltrative disorders
- Identification of occult whipworm infestation
- Removal of polyps
- Diagnosis of cecocolic or ileocolic intussusception
- Diagnosis of colonic vascular ectasia

Laryngoscopy

- Identification of laryngeal paralysis, elongated soft palate, or everted lateral saccules
- Location and removal of foreign objects
- Biopsy of a mass

Cystoscopy

- Diagnosis and laser ablation of ectopic ureters
- Biopsy of proliferative lesions in urethra and bladder, especially carcinomas
- Laser ablation of ureteral/trigonal transitional cell carcinoma
- Removal of stones via basket or lithotripsy
- Identification and dilation of urethral strictures
- Injection of collagen into the urethra to control incontinence

Thoracoscopy

- Identification and biopsy of masses and other infiltrative lesions, including lung biopsy
- Assistance in placing chest tubes in animals with severe pyothorax
- Determination of whether thoracotomy is appropriate, and if so, the best approach
- Performance of minimally invasive surgery, such as pericardiectomy, ligation/resection of persistent right aortic arch, thoracic duct ligation

Bronchoscopy

- Identification of lesions (e.g., collapsed trachea, *Oslerus osleri* infestation)
- Bronchoalveolar lavage or brushing of trachea/bronchus for cytology/culture
- Identification and removal of foreign objects
- Identification of lung lobe torsion
- Biopsy of mucosa (e.g., with chronic bronchitis)
- Aid in placement of stents/evaluate stents that were previously placed

Rhinoscopy

- Identification and removal of foreign objects
- Biopsy/cytology of mass lesions and mucosa for infiltrative disorders
- Identification and biopsy of aspergillomas
- Identification of source of epistaxis or chronic nasal discharge

Posterior Nares (Choanal) Examination

- Identification and removal of foreign objects
- Cytology/culture of the caudal nares
- Identification of and biopsy of proliferative disorders
- Identification, dilatation, stenting of nasopharyngeal stenosis
- Identification of nasal mites

Laparoscopy

- Examination and biopsy of abdominal viscera (especially liver and pancreas)
- Determination of whether celiotomy is warranted (e.g., is there evidence of metastasis that means surgery cannot be curative)
- Performance of minimally invasive interventional surgery (e.g., gastropexy, placement of jejunostomy tube, ovariectomy, cryptorchidectomy)

Arthroscopy

- Identification and biopsy of lesions
- Removal of loose bodies (cartilage fragments, bone fragments, torn meniscus)
- Topical management of osteoarthritis: abrasion arthroplasty, microfracture
- Joint lavage for sepsis
- Arthroscopic-assisted fracture repair
- Arthroscopic-assisted joint stabilization

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obtained surgically. Assuming good technique, endoscopically obtained samples are adequate for diagnosis in probably more than 90% of patients (personal observation of the author) with gastric or intestinal infiltrative disease (e.g., inflammatory bowel disease, histoplasmosis, neoplasia). Endoscopy cannot reliably diagnose disorders beyond its reach (e.g., focal carcinoma of midjejunum), infiltrates too deep in the wall of the viscera for the biopsy forceps to reach, and lesions consisting of hard, densely fibrotic infiltrates (e.g., scirrhous carcinoma) that the biopsy forceps cannot penetrate.

Cytologic studies from brushings or washes are often diagnostic for disorders such as cancer, parasitic infections, fungal infections, protothecosis, and eosinophilic enteritis. Histoplasmosis has occasionally been diagnosed cytologically when it was missed histologically. However, most idiopathic inflammatory bowel diseases, especially lymphocytic-plasmacytic disorders, cannot be definitively diagnosed cytologically. Washings are especially useful in the respiratory tract to diagnose inflammatory or infiltrative problems, but may be useful if looking for gastric *Ollulanus tricuspis* infestation or duodenal giardiasis.

GI, respiratory, and vaginal hemorrhage are often indications for endoscopy. Endoscopy may help determine if surgery is indicated and ensure that all bleeding sites are located. Intraoperative endoscopy can find bleeding gastric mucosal lesions if the surgeon cannot identify them at surgery; even large ulcers may be missed when the stomach is examined through a gastrostomy incision. Severely hemorrhaging lesions may require endoscopic electrocautery or injection with epinephrine. Endoscopy detects small mucosal lesions (e.g., erosions) indiscernible from the serosal surface.

Placement of esophagostomy, gastrostomy, and enterostomy feeding tubes can be done with or without endoscopy (see pp. 95–101). Endoscopic placement of such tubes is indicated when nonendoscopic apparatus for placement cannot be safely passed through the esophagus (e.g., esophageal stricture, esophageal dilation), when the operator is insufficiently trained with nonendoscopic apparatus, or when endoscopy is already in progress for other purposes. Endoscopic placement of gastrostomy tubes includes insufflation of the stomach, which is advantageous because insufflation helps prevent other abdominal organs from becoming trapped between the stomach and the abdominal wall.

Alimentary tract polyps can be removed endoscopically with endoscopic snares adapted for electrosurgery. However, because rectal polyps are usually found close to the rectum, surgical removal is typically easier and more certain to remove the entire polyp. Endoscopic electrosurgery should only be attempted if the clinician has been trained. Inappropriate use of electrosurgical instruments can damage or destroy the endoscopic equipment and seriously injure the patient.

Endoscopic or fluoroscopic dilation of benign esophageal strictures caused by postesophagitis scarring is preferred to surgical resection. Strictures can recur after either method, but surgery may be associated with greater postprocedural morbidity and mortality and has a higher recurrence rate. Endoscopic and fluoroscopic dilation should not be attempted unless the clinician has been well trained in the various techniques used.

Rigid Endoscopy

Rigid endoscopy can be used for the GI, respiratory, and female lower urinary tracts; peritoneal and pleural cavities; and joints. Indications for rigid endoscopy include many of the same as for flexible endoscopy (see Box 14.1), but rigid endoscopy (especially laparoscopy, thoracoscopy, and arthroscopy) is more often used for interventional procedures compared with flexible endoscopy.

Tissue samples obtained by rigid colonoscopy are invariably better than those obtained with flexible colonoscopy. Rigid biopsy instruments typically procure large, deep colonic or rectal samples that include relatively large amounts of submucosa. Therefore rigid endoscopy is appropriate even when dense, fibrotic, submucosal lesions (e.g., carcinoma, pythiosis) are suspected. This is especially true of proliferative processes in the rectal area where it is imperative to obtain generous amounts of submucosa to allow the pathologist to distinguish benign from malignant processes.

Foreign Bodies

Both rigid and flexible endoscopy are useful in removing foreign objects. Rigid endoscopy is the best way to remove most esophageal foreign objects. Most esophageal, nasal, tracheal, and laryngeal foreign objects—and many gastric and duodenal foreign objects—can be removed endoscopically without undue risk to the patient. Objects out of reach of the endoscope, those that cannot be firmly grasped or trapped by endoscopic devices, and those that may cause severe damage if endoscopic removal is attempted (e.g., linear foreign objects that have been present for several days, objects with sharp edges or points that cannot be covered during removal) should usually be removed surgically.

EQUIPMENT

Flexible Endoscopes

Endoscopes

The equipment needed depends on the type of endoscopy and the body system involved, as well as the species to be examined. Rigid and flexible endoscopes are available in a large assortment of sizes and lengths; both types have advantages and disadvantages (Box 14.2). Flexible endoscopes typically used in veterinary medicine are gastroduodenoscopes, bronchoscopes, colonoscopes, and ureteroscopes. Flexible scopes have a handle, an insertion tube, and an umbilical cord (Fig. 14.1). Bronchoscopes usually have a 2- to 6-mm outer diameter, gastroduodenoscopes a 7.9- to

BOX 14.2 Comparison of Flexible and Rigid Endoscopes

Flexible

- Greater access to more sites in viscus organs
- More expensive than rigid scopes
- Easier to damage/requires training to assemble and clean without damaging
- Requires substantial training to use properly

Rigid

- Less expensive than flexible scopes
- Usually more durable
- Easier to learn to use
- Capable of larger biopsies than with flexible scopes
- Excellent for removing foreign objects and protecting mucosa
- Of the viscus organs, can only access the esophagus, gastric fundus, descending colon, larynx, nose, and trachea
- Can be used in the peritoneal, pleural, and joint spaces

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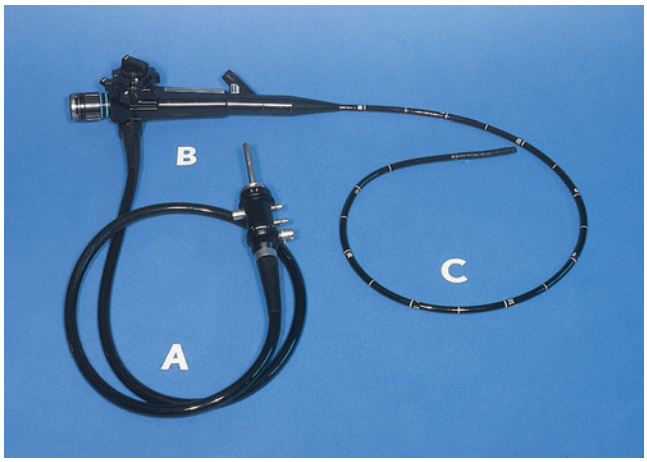


FIG. 14.1 Flexible gastroduodenoscope showing the umbilical cord *(A),* which attaches the scope to the light source; the handle *(B);* and the insertion tube *(C),* which is introduced into the animal.



FIG. 14.2 Detail of flexible biopsy forceps with an ellipsoid, alligator jaw configuration. The upper forceps is used for a 2.8-mm channel, the lower one for a 2.2-mm channel. (From Willard MD. Colonoscopy, proctoscopy, and ileoscopy. *Vet Clin North Am.* 2001;31[4]:657–669.)

10-mm outer diameter, colonoscopes a 10- to 16-mm outer diameter, and ureteroscopes a 2.5- to 3-mm outer diameter. All scopes should have a biopsy-suction channel (usually 2.2 mm in diameter for bronchoscopes, 2.2 to 3.2 mm for gastroduodenoscopes and colonoscopes, 1 to 1.5 mm for ureteroscopes). Gastroduodenoscopes and colonoscopes typically have four-way deflection of the tip of the scope and an air-water channel that is used to insufflate air and wash off the viewing lens; bronchoscopes typically have two-way deflection of the tip and do not have an air-water channel. The insertion tube typically has a working length of 40 to 60 cm in bronchoscopes and ureteroscopes, 100 to 135 cm in gastroduodenoscopes, and 130 to 220 cm in colonoscopes.

The ideal assortment of flexible scopes depends on the expertise of the operator and what procedures are anticipated. Ideally, a bronchoscope (4- to 5-mm diameter), a pediatric gastroduodenoscope (<7.9-mm diameter with a 2-mm channel), and a regular gastroduodenoscope (8.5- to 9.8-mm diameter with a 2.8-mm channel) allow one to perform almost all nonurinary tract flexible procedures. Other flexible scopes that might be considered, depending on ones' needs, are a 1.6-m pediatric colonoscope (helpful in extremely large animals and large, exotic cats) and a ureteroscope for small birds and pocket pets, as well as flexible cystoscopy or rhinoscopy. If one can only have two scopes for both alimentary and respiratory tract work, consider a bronchoscope (4- to 5-mm diameter) and a gastroduodenoscope (8.5- to 9-mm outer diameter with a 2.8-mm channel). If one can only have one scope for respiratory and alimentary tract endoscopy, a 7.9-mm outer diameter pediatric gastroduodenoscope may be the best compromise. If only one scope is possible and it will be used only for alimentary tract endoscopy, an 8.5-mm outer diameter scope with a 2.8-mm channel is preferable.

Biopsy/Cytology Equipment

Biopsy forceps for flexible scopes come in various shapes, sizes, and configurations. The size of the forceps depends on the size of the biopsy-aspiration channel; the larger the channel, the bigger the biopsy forceps that can be used. If possible, a scope with a 2.8-mm channel should be used for most alimentary tract endoscopy in dogs and for cats weighing more than 3 to 4 kg. The tissue sample obtained through a 2.8-mm channel can be more than twice the size of a sample obtained through a 2.2-mm channel. These larger pieces of alimentary tissue often contain the full thickness of the mucosa and some submucosa. Fenestrated biopsy forceps in an ellipsoid, alligator jaw configuration without a needle are widely preferred (Fig. 14.2); however, some endoscopists have other preferences that work for them. Disposable biopsy forceps are widely used in human medicine, but do not necessarily have major advantages in veterinary medicine.

Brushes used to obtain samples for cytology and culture typically consist of a stiff brush in a plastic tube. This brush is manually extended out the tip of the plastic tube once the tip of the brush assembly has been advanced through the endoscopic biopsy channel and is near the tissue to be sampled. Unfortunately, such brush assemblies are easily contaminated as they are passed through the endoscopic channel. There are brush assemblies made for when it is absolutely critical that a noncontaminated sample be obtained (i.e., culture). These brushes consist of an extendable brush that is within an extendable tube that is in yet another tube. The tip of the outermost tube is typically plugged so that there is no chance of contamination when the scope is passed through the biopsy channel of the endoscope. Once the tip of the brush assembly is near the site to be cultured, the innermost tube is extended out the end of the outermost tube, and then the brush is extended out the tip of the innermost tube. Brushes for diagnostic purposes should never be cleaned and reused, as opposed to biopsy forceps, which can be used repeatedly.

Interventional Tools for Flexible Endoscopes

A variety of special retrieval instruments are necessary to reliably remove most commonly encountered foreign objects. The most useful devices are a coin retrieval (W-type) forceps, a shark's tooth forceps (especially useful for firmly grabbing cloth), an alligator jaws, and a 4- or 6-wire basket (Fig. 14.3). The basket should be made of very flexible wire to facilitate passage over and around an object; however, this quality also makes it easier to bend the wire and ruin the basket. Other retrieval devices include wire snares, 3-wire grabbers, magnetic-tip probes, and forceps with nonskid rubber; these instruments are seldom required.

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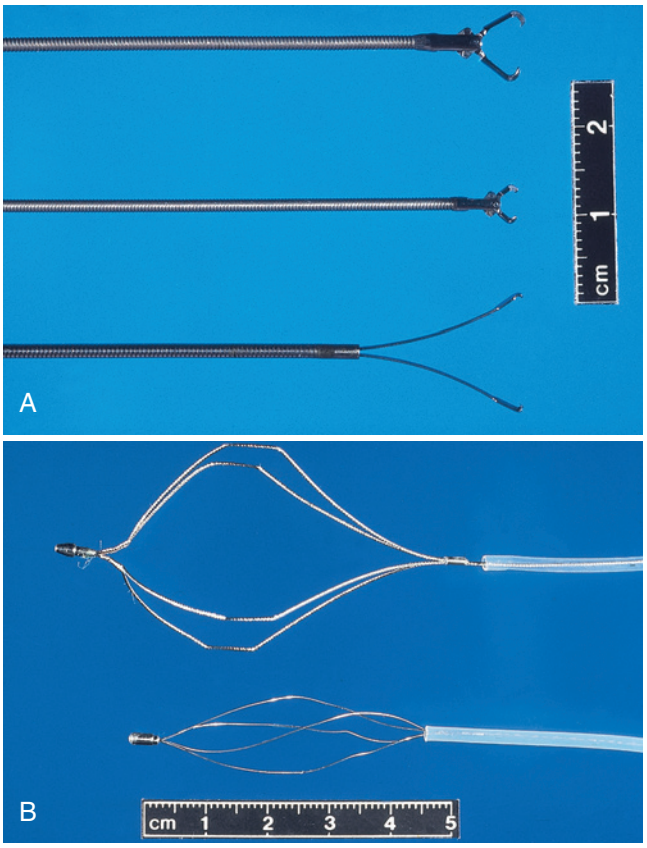


FIG. 14.3 (A) Tips of three commonly used foreign body retrieval forceps. *Top to bottom,* shark's tooth forceps, rat's tooth forceps, and coin retrieval forceps. The coin retrieval and rat's tooth forceps can pass through a 2-mm channel; the shark's tooth forceps requires a 2.8-mm channel. (B) At the top is a 4-wire basket that works well because of the extreme flexibility of the wires. The 4-wire basket below does not open as widely and has wires so firm it is difficult to ensnare the foreign body.

There are various balloons and bougies designed to dilate esophageal strictures. Despite rhetoric that ballooning is superior to bouginage, success is more dependent on the skill and training of the operator rather than the equipment used. Esophageal dilation balloons come in two major configurations: over-the-wire or through-the-endoscope channel. It is important to use these as opposed to a more round balloon, such as is found on endotracheal tubes.

Other equipment includes sheathed needles that allow aspiration or intralesional injection of various lesions through the endoscope. A variety of flexible endoscopic biopsy instruments, snares, knives, biopsy forceps, and probes have electrosurgical capability. The electrosurgical snares are most commonly used because they allow removal of esophageal, gastric, and colonic polyps; they can also be used to make three or four quadrant "cuts" into esophageal strictures to aid in ballooning difficult lesions.

NOTE Use of electrosurgical instruments by untrained individuals can damage or destroy the video processor and cause substantial patient morbidity; therefore they should only be used if the operator is trained in their use.

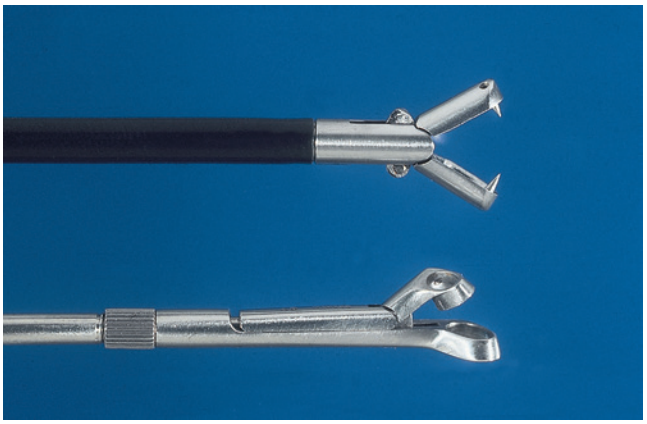


FIG. 14.4 Tips of two types of rigid biopsy forceps. The upper forceps is a human uterine biopsy forceps, also known as a *clamshell* or *double-spoon forceps*. The lower forceps has a smaller upper punch that fits into a larger lower cup with a shearing action, much like the blades of a scissors. (From Tams TR. *Small Animal Endoscopy*. 2nd ed. St. Louis: Mosby; 1999.)

Rigid Endoscopes

Colonoscopes

Rigid colonoscopes (typically human sigmoidoscopes) and proctoscopes are plastic or metal tubes of variable size and length with an obturator and light source. Air can be insufflated into the colonic lumen with a colonoscope, but not with most proctoscopes. A better examination of the colonic lumen is possible with longer scopes of larger diameter. The minimum recommended working length for colonoscopes is 25 cm, and 35 cm is preferred. Most dogs, except the toy breeds, tolerate a 15-mm outer diameter scope, but a 19- to 25-mm diameter scope should be used whenever possible. Rigid scopes must allow simultaneous viewing of the mucosa and insufflation of air. Unlike flexible scopes, rigid colonoscopes do not allow simultaneous insufflation and biopsy. A suction tube that can be inserted through the lumen is important for removing residual feces and debris. A variety of anoscopes and proctoscopes in different lengths (usually 90 to 120 mm) and diameters (usually 14 to 22 mm) should be available. Biopsy forceps used with rigid colonoscopes and proctoscopes should have a shearing or scissor-like tip that cuts the mucosa (i.e., a small punch that fits into a larger diameter cup) rather than the clamshell or double-spoon tip that grasps and tears tissue (Fig. 14.4). Grasping forceps (such as would be used for esophageal foreign bodies) must be strong and preferably have ridges to aid in obtaining a strong purchase on foreign objects.

Laryngoscopes

Laryngoscopy requires an assortment of various sized rigid laryngoscopic blades in straight and curved configurations. Flexible scopes can be used but are seldom necessary.

Rigid Endoscopy With Special Additional Equipment Needs

Laparoscopy, thoracoscopy, and arthroscopy require an outer cannula (usually with a trocar or obturator), which is inserted into the cavity to be examined, plus a viewing telescope (also responsible for directing light), which is inserted through the cannula into the cavity. The same equipment used for arthroscopy can sometimes be used for cystoscopy and rhinoscopy; however,

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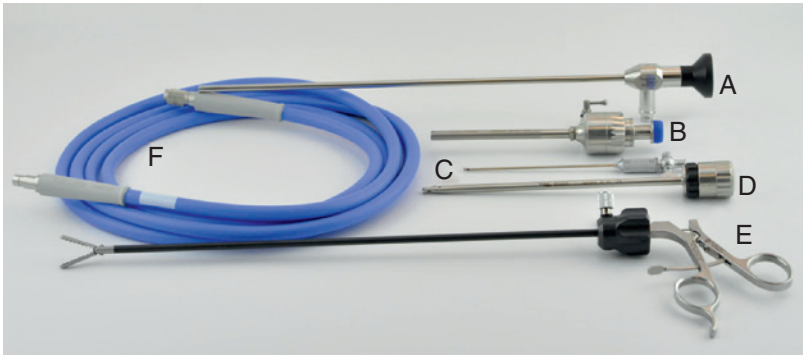


FIG. 14.5 (A) A 5-mm 0-degree autoclavable laparoscope. (B) A 5.5-mm laparoscope sheath with automatic valve and manual valve lever. (C) A 100-mm insufflation Veress needle. (D) A 5.5-mm lacking shielded trocar. (E) A 5-mm 32-cm rotatable grasper with locking handle. (F) A 5-mm fiberoptic light cable.

cleaning and sterilization protocols for these different techniques may vary. Dedicated rigid cystoscopic equipment is also available, and it is often used for rhinoscopy as well.

Laparoscopes/Thoracoscopes

Most telescopes used for laparoscopy and thoracoscopy are 5 to 10 mm in diameter (Fig. 14.5). The larger scopes allow better illumination. There are a variety of angles of view at the tip, ranging progressively from 0 degrees (i.e., looking straight ahead) to 30 degrees (a commonly used telescope that effectively allows one to look behind organs) to scopes that can look back on themselves (i.e., angles of view of 270 degrees or greater). Double- and single-puncture techniques are possible. Double-puncture techniques involve the telescope being placed through one cannula while other instruments (e.g., biopsy forceps, Babcock forceps) are placed through additional cannulas. Some 10-mm diameter telescopes have a channel that allows insertion of an instrument (e.g., biopsy forceps, grasping forceps, scissors) while viewing through the scope. Such scopes are called *operating telescopes* and can be used in single-puncture or double-puncture laparoscopy and thoracoscopy.

There are various modifications of the basic rigid endoscopic techniques, as well as a plethora of one-time use disposable equipment in various configurations, which makes it difficult to succinctly describe how to perform these procedures. Basically, laparoscopy requires a means of insufflating the abdomen (e.g., a Veress needle, a threaded cannula, the Hasson technique), and both laparoscopy and thoracoscopy require a cannula (usually with a trocar or obturator) to insert the telescope into the appropriate cavity. Threaded cannulas are "screwed" into the thoracic or abdominal wall, which eliminates the risk of the cannula inadvertently being withdrawn. The type of biopsy forceps depends on the nature of the biopsy. A liver biopsy is best taken with clamshell or double-spoon forceps (see Fig. 14.4); a biopsy from the pancreas is best accomplished with a punch type of forceps. In general, core needles (e.g., Tru-cut, Becton Dickinson) are only used for the kidney and some masses; they are not recommended for hepatic biopsies unless there is a focal mass (such lesions should generally have been aspirated via ultrasound before endoscopy). Various other forceps (e.g., Babcock), hemostats, aspiration tubes, needle holders, scissors, and so forth should also be available. Electrocautery is particularly important when performing thoracoscopy. Vessel sealing devices (e.g., Ligasure, Medtronic) significantly increase the capabilities and safety of many laparoscopic and thorascopic procedures. Because thoracoscopy is almost always a two-person operation, requiring coordination between endoscopists, video capability is an absolute requirement (as opposed to most other endoscopies in which it is helpful but not critical, the exception being arthroscopy, in which it is also absolutely required).

Cystoscopes

Most cystoscopy is retrograde as opposed to transabdominal, except in cats, in which transabdominal cystoscopy is relatively common. Small-diameter flexible scopes (e.g., bronchoscopes) or rigid telescopes can be used. The rigid telescopes used for retrograde cystoscopy of dogs are usually 10 to 22 Fr in diameter. Cannulas are very useful for rigid cystoscopy because they allow concurrent infusion of a fluid (e.g., physiologic saline) around the telescope during examination, which dilates the urethra and bladder and washes away blood, mucus, and other debris that obscure vision.

Rhinoscopes

Rigid rhinoscopy typically uses the same equipment as cystoscopy and arthroscopy because of the need for small-diameter equipment. The cannulas used for cystoscopy are especially useful because patients presented for rhinoscopy often have mucoid or bloody nasal discharge that obscures visualization. Infusion of cool physiologic saline during the procedure often washes away the mucus, allowing better visualization (as opposed to warm saline, which will dilate vessels and enhance hemorrhage). However, the small size of the nasal cavity in cats and some small dogs limits the use of cannulas because they enlarge the diameter of the instrument being inserted into the nose. Therefore a variety of instruments have been used for rhinoscopy, including flexible and rigid scopes.

Arthroscopes

See later discussion on p. 166.

EQUIPMENT CARE

Endoscopes (especially flexible) are easily damaged. Therefore access to endoscopic equipment should be limited to a few essential individuals, including those who set it up and clean it

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BOX 14.3 Basic Principals of Cleaning Flexible Endoscopes[a]

- Perform leakage test *first*
- Aspirate approved detergent through biopsy channels
- Brush biopsy channels and reaspirate
- If appropriate, aspirate disinfectant solution through biopsy channels
- Aspirate distilled water and then alcohol through biopsy channels
- Aspirate air until biopsy channels are dry
- Clean air-water and suction valves
- Lubricate air-water and suction valves
- Flush all water out of air-water channel
- Clean exterior of scope with approved detergent and water/disinfect or sterilize as appropriate
- Clean and dry biopsy and foreign body retrieval forceps

[a]Always see manufacturer's recommendations.

(Box 14.3). When not in use, flexible scopes should be hung vertically on a rack. If it is absolutely necessary to store a flexible endoscope in its carrying case, care must be taken to ensure that the insertion tube is not caught between the edges of the case (a common cause of damage). Newer flexible scopes are immersible, including the handle; however, older fiberoptic scopes can be severely damaged if water penetrates the handle through a seam during the endoscopic procedure or cleaning. Water must not be splashed on the light source or other electronic equipment, and surge protectors should be used. Only water-soluble jelly should be used as a lubricant on a flexible instrument; petroleum-based substances can shorten the life span of the rubber or plastic coverings.

The insertion tube must not be bent into a severe acute angle, especially at the junction of the insertion tube and the handle, lest the outer covering be cracked and leakage ensues. Care must be taken to prevent biopsying the insertion tube when taking a biopsy with the tip of the scope maximally retroflexed. An instrument should *never* be forced through the biopsy channel, especially when the tip of the scope is deflected more than 70 degrees. If fine, rough material (e.g., sand) is aspirated into the biopsy channel, it can tear the channel and cause a leak when a biopsy instrument is inserted. The insertion tube, especially the tip, should not be allowed to strike a hard surface.

NOTE Always use a mouth gag; *never* introduce the insertion tube into the mouth of an unanesthetized animal.

The scope should be cleaned after each procedure, and the manufacturer's recommendations should be followed explicitly. Generally, a leakage test should be performed first because it is much less expensive to repair a leaking scope before water leaks into the insertion tube and damages fiberoptics or electronics. Next, all water should be expelled from the air-water channel, and the suction and biopsy channels should be cleaned with an approved detergent or cleaner. Only chemicals recommended by the manufacturer should be used as disinfectants on a flexible scope. A brush is used to remove adherent material from the biopsy channel. After these channels have been cleaned, alcohol and then air are aspirated through the channels until they are dry. Cleansing and disinfection of flexible scopes is usually adequate; sterilization is rarely necessary unless the patient is suspected of harboring a specific infectious agent (e.g., parvovirus). If sterilization is necessary, use only manufacturer-approved techniques.

NOTE Review the manufacturer's recommendations and be sure to use ethylene oxide caps, if necessary. Never subject flexible scopes to heat and especially not to autoclaving.

Rigid scopes are usually more resistant to damage than flexible equipment and require relatively simple care, such as washing in an approved detergent solution. Care must be taken to avoid hitting or scratching the lenses in the tips of some rigid scopes, such as cystoscopes. Small-diameter rigid scopes (e.g., cystoscopes and rhinoscopes) are easily bent, so care must be taken when handling and storing them. Sterilization of rigid equipment may involve autoclaving (*if* the scope is designated by the manufacturer as one that can withstand autoclaving), but hydrogen peroxide plasma or ethylene oxide sterilization is preferred.

Care of the arthroscope is given on p. 167.

ANTIBIOTICS

Preprocedural administration of antibiotics is not required for routine endoscopies, including cystoscopy and laparoscopy, unless the patient has valvular cardiac defects or prosthetic implants or is severely immunosuppressed. Esophageal ballooning is associated with bacteremia, and prophylactic use of antibiotics was once advocated; however, this is no longer routinely performed. Use of antibiotics after foreign body removal might be reasonable if significant ulceration/laceration has occurred, but this is unproven. Perforation is often, but not always, an indication for surgery.

For antibiotic usage during arthroscopy, see p. 168.

PROCEDURES

Four basic principles apply to most endoscopic procedures:

1. Advance the scope only if you can see where you are going.
2. If you cannot see what is happening, back the scope out a little rather than advancing it, or insufflate a little air or infuse some fluid into the lumen (or both).
3. Unless you need to look at a specific lesion, aim the scope toward the center of the lumen.
4. Do not insert the endoscope into your patient any harder than you would want a physician to insert it into you.

There are few exceptions to these rules. Beware of excessive insufflation of the alimentary tract or abdomen, as both can impair respiration and/or venous return. Using too large a scope can obstruct the respiratory tract. In general, the largest scope that can *safely* be used allows better visualization, larger biopsy samples, and use of better foreign body retrieval devices. The following sections describe selected techniques that might be performed in a variety of locations.

Biopsies

With flexible instruments, the largest biopsy instrument that can safely be used should be chosen. Better samples of alimentary mucosa can usually be obtained if the organ is not excessively distended with air. Samples of grossly normal and apparently diseased mucosa should be taken. Always note the difficulty

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involved in taking a biopsy of a particular lesion; some infiltrative lesions (i.e., scirrhous carcinomas and pythiosis) characteristically produce so much dense connective tissue that the flexible biopsy forceps cannot "bite" into the tissue and tear off an adequate piece. Such a finding may be an indication for full-thickness biopsy. Proliferative lesions can exist below the mucosa so that only normal-appearing mucosa is seen overlying a mass effect; in such cases repeated biopsies in the same spot (i.e., "drilling for oil") sometimes allows access to underlying lesions. A biopsy is often best performed when the opened biopsy instrument can be pushed against the mucosa at a near 90-degree angle. In the intestines, the turn and suction technique is often used to achieve such an angle.

Biopsy of Gastrointestinal Mucosa

Advance the biopsy forceps through the channel, then open the jaws of the forceps and withdraw it until the opened jaws are flush with the tip of the endoscope. Flex the tip of the scope so that it is turned into the mucosa at as close to a 90-degree angle as possible; suction air out of the intestine, thereby drawing mucosa into the jaws of the forceps. Advance the forceps into the mucosa until moderate resistance is felt (usually 1 to 3 mm) and close the jaws. Do not advance the forceps so much that the cable on the instrument begins to bow excessively or the tip starts to slide along the mucosal surface. Be sure to straighten the tip of the endoscope before withdrawing the forceps.

At this time, it appears best to routinely obtain at least eight adequate biopsies of the duodenum and two biopsies of each part of the stomach to try to ensure that (1) sporadic and scattered lesions are sampled, and (2) enough tissue samples will be oriented optimally to maximize histologic interpretation. The optimal number of ileal and colonic biopsies is undetermined, but generally eight colonic biopsies and three to four ileal biopsies have been suggested. Intestinal, nasal, and gastric fundus and body mucosa are relatively "soft" and easy to biopsy. Pyloric and antral mucosa are tougher, and the mucosa may need to be grasped more firmly to pull off an adequate-sized piece. Normal canine esophageal mucosa is so tough it is almost impossible to obtain an adequate tissue sample with routine flexible forceps. Normal tracheal mucosa is so thin it is difficult to obtain an adequate sample for histopathologic studies.

Using a needle, carefully retrieve mucosal samples from the biopsy forceps and place them mucosal side up on a plastic histologic cassette sponge, cucumber slice, or piece of formalin-soaked paper without distorting them.

Gastric and nasal mucosa are relatively sturdy, but small and large intestinal mucosa is delicate and must be handled carefully to avoid crush and stretch artifacts.

Place cassette plastic sponge, cucumber slice, or piece of paper with the biopsies on it in a vial of neutral buffered formalin. Do not allow the samples to dry out excessively before they are fixed.

Cytologic Preparations

Make cytologic preparations (i.e., squash preps) by putting a small piece of mucosa between two glass slides.

Press the slides together and then pull them apart either vertically or horizontally. As an alternative, use an endoscopic brush; lightly rub the extended brush against the lesion (do not cause hemorrhage) and then rub it against a glass slide. For cytologic studies of very fibrotic lesions that can be obtained only with rigid forceps, scrape the cut surface of the sample with a scalpel blade and make a squash preparation of the material obtained.

BOX 14.4 Advantages and Disadvantages of Endoscopic Removal of Foreign Objects

Advantages

- Often much quicker than surgery
- Often less stressful to the patient
- Reduced tissue trauma, morbidity, and recovery time

Disadvantages

- Cannot remove all objects
- Can hurt animal with careless technique
- Requires an assortment of expensive foreign body retrieval forceps

Washes

Insert a sterile polyethylene or similar tube through the biopsy channel, position the tip of the tube as desired, instill the solution (usually physiologic saline solution or Hank's buffered salt solution), and apply suction. Do not place the tip of the tube too close to the mucosa and use only modest negative pressure, or you may end up occluding the end of the tube with tissue. As an alternative, aspirate the sample directly through the endoscopic biopsy channel into a trap.

Foreign Body Removal

Each foreign object must be considered individually. An ill-planned endoscopic removal may be more damaging to the patient (e.g., perforate the tract) than the foreign object (Box 14.4). It is also possible to "grab" a foreign object and find that it can be neither removed nor released, necessitating surgery to retrieve the endoscope. There are numerous techniques for foreign body removal, but some basic principles always apply:

1. *Always take a radiograph of the animal shortly before inducing anesthesia.* Some foreign objects that have been present for weeks pass out of reach of the scope just before the procedure is performed. In the same manner, some foreign objects will perforate the organ just a few hours after admission to the clinic. Finding air or water in the thorax or abdomen means that perforation may have occurred, and that the fluid should be analyzed (i.e., looking for evidence of sepsis or inflammation) or perform a contrast radiographic procedure (e.g., contrast esophagram) using an iodide-based contrast agent.
2. *Do not just grab the object and pull.* The object may need to be repositioned to allow the forceps to obtain the best grasp on it. It may also be necessary to reorient the object to make it easier and safer to pull it through the sphincters.
3. *Select the retrieval forceps that allows the firmest grasp on the object.* Rigid retrieval forceps typically allow the tightest grasp on an object.
4. *When an object has been snared, do not pull it out against undue resistance.* Resistance is expected at the lower esophageal high-pressure zone (gastric cardia), the base of the heart, the thoracic inlet, and the cricopharyngeal area. If inappropriate resistance is noted, it is usually better to stop and examine the orientation of the object. The clinician may need to reposition the object or release it and use any of various devices (e.g., hoods, overtubes).
5. *Consider an overtube if there are sharp edges or if you need to dilate the lower esophageal sphincter.*

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Postendoscopy Care

Special care is not necessary after routine biopsy of the alimentary or respiratory tracts. Slight hemorrhage is expected after most procedures, especially biopsy procedures. Most hemorrhage is minor or stops spontaneously shortly after the procedure if the coagulation system is normal. Preprocedural coagulation testing is generally done only when (1) there is a reason to suspect the integrity of the coagulation system (e.g., petechiae, spontaneous bleeding, hepatic disease), or (2) laparoscopic or thoracoscopic biopsies will be performed. However, nasal biopsies can be associated with severe hemorrhage, at times requiring ligation of the ipsilateral carotid artery. When laparoscopic biopsy has been performed on a parenchymatous organ (e.g., liver, kidneys), observe the patient closely for 2 to 4 hours to ensure that excessive hemorrhage does not occur (as seen by evidence of progressive hypovolemia). If excessive hemorrhage occurs, either repeat endoscopy or perform surgery to find and stop the bleeding lesion. If excessive hemorrhage continues for an inappropriately long time after routine biopsy, an occult coagulopathy should be considered.

After a foreign object has been removed, the mucosa should be reevaluated for ulceration or perforation, as well as other foreign objects or lesions. If there is any possibility of perforation, radiograph the appropriate body cavity to look for pneumothorax or pneumoperitoneum. Antibiotic regimens effective against aerobic and anaerobic bacteria often are useful in such cases. Such a regimen might be enrofloxacin, 15 mg/kg given intravenously (IV) once daily, plus either ampicillin (22 mg/kg subcutaneously) or clindamycin (11 mg/kg IV) given two or three times a day, respectively. If severe ulceration occurs due to a foreign body, it may be useful to administer either an antacid (e.g., omeprazole [1 to 2 mg/kg] given twice daily) or a gastroprotectant (e.g., Carafate, 0.5 to 1 g given two to four times a day PO), but not both.

It is important to watch for postprocedural dyspnea and/or hypoxia after performing bronchoalveolar lavage in patients with pulmonary disease. In patients that are already marginally oxygenated, it is often best to perform brushings for cytology and culture or a tracheal wash as opposed to a bronchoalveolar wash.

Specialized Interventional Procedures

Other specific interventional procedures (e.g., ballooning benign strictures, intralesional injections, cutting strictures, polypectomy, placing stents, etc.) are discussed under their relevant sections.

ARTHROSCOPY: GENERAL PRINCIPLES, EQUIPMENT, AND TECHNIQUES

The most common arthroscopically performed procedures are fragment removal for diseases such as OCD (see p. 1154) or fragmented coronoid process (see p. 1174), treatment of meniscal injuries (see p. 1227), and assessment of cartilage and intraarticular soft tissue injuries. Other procedures include synovial biopsies, tenotomy (see p. 1169), joint stabilization, and arthroscopic-assisted fracture repair.

INDICATIONS

Arthroscopy may be indicated for either the diagnosis or treatment of joint disease (Boxes 14.5 and 14.6). Arthroscopy is vastly

BOX 14.5 Common Diagnoses With Arthroscopy

Shoulder
Osteochondritis dissecans
Osteoarthritis
Biceps disease
Medial collateral tearing
Lateral collateral tearing

Elbow
Fragmented coronoid process
Osteochondritis dissecans
Ununited anconeal process
Osteoarthritis of the medial compartment

Carpus
Osteoarthritis
Chip fractures

Hip
Osteoarthritis
Labral tearing and avulsion
Tearing of the ligament of the femoral head

Knee
Osteochondritis dissecans
Cruciate disease
Osteoarthritis
Meniscal disease

Tarsus
Osteochondritis dissecans
Chip fractures

BOX 14.6 Common Arthroscopic Procedures

Shoulder
Fragment removal: OCD
Osteoarthritis treatment: microfracture, abrasion
Biceps tenotomy
Soft tissue shrinkage for instability

Elbow
Fragment removal: OCD, FCP
Osteoarthritis treatment: microfracture, abrasion

Carpus
Fragment removal: chip fractures
Osteoarthritis treatment: microfracture, abrasion

Hip
Osteoarthritis assessment

Knee
Fragment removal: OCD
Osteoarthritis treatment: microfracture, abrasion
Meniscal treatment
Cruciate

Tarsus
Fragment removal: OCD
Osteoarthritis treatment: microfracture, abrasion

FCP, Fragmented coronoid process; *OCD,* osteochondritis dissecans.

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superior to radiography in the diagnosis of joint diseases because it allows direct visualization of the cartilage and soft tissue structures, provides magnification, and enables biopsy of virtually all structures within the joint. The most significant diagnostic advantage of arthroscopy is the operator's ability to assess the condition of the cartilage surface. Studies have demonstrated that radiology poorly reflects the condition of cartilage in canine osteoarthritis, whereas arthroscopy enables specific grading and determination of the extent of cartilage injury. Arthroscopy also provides more accurate assessment of intraarticular pathology than open surgery because of the magnification, ability to evaluate more of the joint, and the arthroscopic fluid environment, which improves visualization of surfaces and structures.

EQUIPMENT

Arthroscopes are differentiated by diameter (1.9, 2.3, 2.7 mm, and larger), length (short, long), and angle. Arthroscopes in common use in small animal arthroscopy include any of the diameters and lengths listed earlier, with the vast majority having a 30-degree angle (see Fig. 14.5). The diameter applies to the telescope portion alone and does not include the diameter of the arthroscope cannula, which is necessary for use. The selection of diameter is based on the size of the joint and surgeon preference, with larger scopes providing more rigidity and greater field of view and smaller scopes causing less iatrogenic damage and having greater mobility. Most cameras are "three chip" or high-definition and must be used with a specific camera box that processes the image for the television screen. Medical-grade monitors are recommended to provide a bright, clear, and accurate image. Most new light sources use xenon lamps, which provide increased light intensity and higher color temperature than halogen and therefore provide higher visual clarity and color rendition. Xenon light sources are more expensive than halogen but are generally recommended for superior image quality.

Arthroscopy images and videos may be recorded to digital media (i.e., CDs) or stored on a server or picture archive and communication system.

Fluid flow helps maintain joint distention, aids in clearing blood and other debris from the joint, and decreases the risk of contamination. Fluid may be delivered to the joint either by means of gravity or from an arthroscopic pump. Fluid outflow is provided by either a disposable needle, a specific outflow cannula, or through a portal without a cannula.

Arthroscopic therapy is performed with hand instrumentation and a power shaver (Fig. 14.6). These tools are inserted into the joint through an instrument portal that may be used with or without a cannula. Hand instruments include probes, knives, curettes, and forceps. The most commonly used probes are right angled and may have calibration marks for measurement of lesions. Numerous styles of knives and curettes are available for manipulation of soft tissue. The most common forceps used in small animal arthroscopy are graspers for removal of hard or soft tissue and biters for debridement of soft tissue.



FIG. 14.6 Hand instruments for small animal arthroscopy. (A) Grasping forceps. (B) Right angle probe. (C) Microcurette.

Power instruments increase efficiency and enhance the capabilities of the operator. The most common power instrument used is a shaver. These motorized hand tools have numerous tip designs, including burrs, sharp cutters, and aggressive cutters. Additional power instruments include electrocautery and radiofrequency (RF). Electrocautery tips specific for use in arthroscopy are available for some electrocautery generators. Alternatively, cautery may be performed by use of an RF unit. These units, which are available in both bipolar and monopolar designs, have also been advocated for soft tissue ablation and collagen shrinkage. Extreme care must be taken with the use of electrocautery and RF due to the possibility of severe cartilage and soft tissue damage capable with these instruments.

EQUIPMENT CARE

The telescope is the most fragile and expensive portion of the arthroscopy equipment. It may be damaged during the surgery or at any other time by cracking or scratching the lens or by bending. Specific protocols should be established for handling of the arthroscope to prevent any damage. It is advisable to have small cases for each arthroscope that can secure the arthroscope, cannula, and trocars for sterilization and storage. The cases should be sturdy and have a means of securing the instruments within the case. The arthroscope should be placed back in the case immediately after use to prevent damage while the remainder of the surgical table is cleared. Any junctions on the arthroscope, including those between the light post and scope or between the eye piece and telescope, should be checked regularly for tightness. If these junctions become loose, they may permit fluid to leak in, which may impede light or image transmission. Bending of an arthroscope may be evident by the appearance of a black crescent at the periphery of the field of view. Severe bending will result in complete obliteration of the view. If bending occurs, the instrument should be sent to a qualified repair facility rather than attempting to straighten the instrument, which may result in permanent damage.

Arthroscopes should be cleaned by hand with an enzymatic cleaner and distilled water as soon as possible after the procedure to remove blood or other body fluids or tissue. The lens and eye piece may be gently cleaned with a cotton ball and distilled water. The cannulas and trocars are similarly cleaned. Sterilization may be performed by several methods. Nonautoclavable arthroscopes may be sterilized by ethylene oxide gas or hydrogen peroxide plasma, depending on the recommendations of the manufacturer. Newer arthroscopes are autoclavable.

The camera head, with its associated lens and prism, and the cable may be damaged by dropping or mishandling. The camera cord contains relatively delicate wires, and the connection may have fine pins. The camera cord should never be bent or wound too tightly, and the cap should always be placed back on the connector when not in use. Camera heads may be sterilized by autoclave, ethylene oxide gas, or hydrogen peroxide plasma, depending on the manufacturer's recommendations.

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The light cable may be sterilized by ethylene oxide gas, hydrogen peroxide plasma, or autoclaving, depending on the manufacturer's recommendations. The light cable is composed of numerous glass fibers that may be broken if the cable is bent or wound too tightly. The fiberoptic cable will also heat up significantly and should never be placed directly against the patient because it may cause burning.

Hand instruments should be cared for using routine cleaning and sterilization techniques. These instruments should be inspected during each use for damage or dull edges. The care of power instruments varies based on the tool and manufacturer.

ANTIBIOTICS

The minimally invasive nature and high volume of fluid flow used with arthroscopy result in a very low risk of infection. Antibiotics are typically administered only on a perioperative basis during these procedures. A first-generation cephalosporin (cephalexin, 22 mg/kg, IV) should be given at induction and discontinued after the procedure.

GENERAL PROCEDURES

The most commonly performed arthroscopic procedures are diagnostic visualization and fragment removal (see Box 14.6). When performing diagnostic arthroscopy, it is critical that a complete and repeatable method to document the procedure be routinely performed using either still or video imaging, or preferably both. For best results, make a short video of the entire joint and then take still images of any abnormal findings. This results in a record of all joints examined while producing a minimum of electronic data. The images are valuable for showing lesions to pet owners and for use as a reference for follow-up evaluation of the case.

Fragment removal varies depending on the joint being treated and the specific disease (Fig. 14.7). The most critical points for successful fragment removal include establishment of suitable joint portals, use of appropriate hand instruments, and development of excellent arthroscopic hand-eye skills. The establishment of suitable portals allows good visualization of the fragment, easy removal and insertion of the instrument, and enhanced ability to manipulate the fragment.

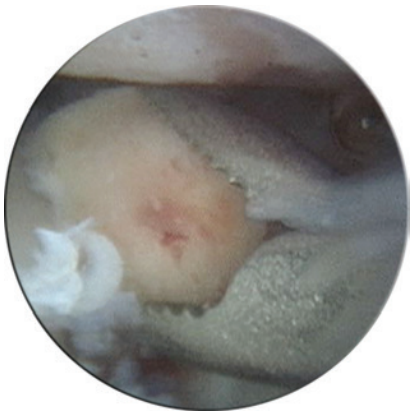


FIG. 14.7 Fragment removal in canine elbow arthroscopy. (From Beale B, Hulse D, Schulz K, Whitney W. *Small Animal Arthroscopy.* Philadelphia: Saunders; 2003.)

SPECIFIC PROCEDURES

Arthroscopy of specific joints can be found in the chapter detailing procedures for conditions affecting that joint. For arthroscopy of the shoulder, see p. 1156; for the elbow, see p. 1177; for the hip, see p. 1211; for the stifle, see p. 1230.

POSTARTHROSCOPY CARE

Because of the minimally invasive nature of arthroscopy, small animal patients undergoing this procedure require little specific postoperative care. Specific instructions depend on the disease being treated and condition of the joint. Arthroscopy portals are generally closed only with skin sutures, although some small portals may require deep closure to limit fluid drainage postoperatively. Regardless of closure method, moderate postoperative drainage from these incisions due to the large volumes of fluid used in the procedure may be seen. Although many animals are administered nonsteroidal antiinflammatory medications for treatment of the primary disease, most patients will not require postoperative analgesics for pain associated with the arthroscopic procedure itself. Exercise restriction is indicated for several days to a week to permit the portals to heal and may be prescribed for longer periods, depending on the underlying disease.

ADVANCED IMAGING

Sophisticated imaging techniques such as computed tomography (CT), magnetic resonance imaging (MRI), and, more recently, positron emission tomography with CT (PET/CT) can be integral parts of the workup of pets with challenging surgical diseases. Since its inception, CT has been used to help resolve complex anatomy. MRI has become the imaging modality of choice for neurosurgery. PET/CT provides important information on both the anatomic location and physiologic function of a variety of diseases. These modalities give the clinician important information on the anatomic or physiologic extent of the disease process.

Medical imaging is no longer just about noninvasive viewing of anatomy. Modern imaging techniques can provide a wealth of information beyond anatomic interpretation. At one end of the spectrum lies pure anatomic imaging; at the other end is pure physiologic imaging. Techniques such as standard planar radiography provide excellent information regarding anatomic structure (especially that of bone), but they do not provide information regarding function of the imaged anatomy. Traditional nuclear medicine (bone scans, thyroid scintigraphy) has provided reliable physiologic imaging and has been commonly utilized in veterinary medicine. Newer modalities such as PET can image glucose metabolism, rates of cell proliferation, and the biodistribution of a drug but do not provide anatomic detail. Hybrid imaging, which provides detail regarding function and anatomy, has become increasingly important in medical imaging. Combining modalities that provide complementary information, such as PET/CT, provide data that are often more useful than the individual information. CT is able to precisely determine anatomy, whereas PET is able to provide information on regional physiology; fusing the two modalities can offer tremendous insight. Other examples of hybrid imaging in use today include excretory urography and upper GI studies, which provide anatomic and limited physiologic information. With ultrasonography, one can look at both anatomy with B-mode and velocity

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with Doppler imaging, giving information regarding structure and function.

Physics: Overview

Magnetic Resonance Imaging

Important concepts when considering the physics of MRI is that all living things have magnetic and electrical properties and that magnetism and electricity are interchangeable. MRI is based on the magnetic properties of living tissue, primarily protons of hydrogen atoms. The majority of atoms in the body are hydrogen, principally as a component of water (H_2O), which makes it a particularly useful atom for MRI. The proton has a spin and a charge; because this is a moving electrical charge, it constitutes an electrical current. This electrical current induces a magnetic field; therefore each spinning proton produces a tiny magnetic field.

The basic sequence of events in MRI involves excitation of protons by administering RF pulses, discontinuing the RF pulses, and collecting the RF energy that is released when the protons "relax" to their original energy states; this released energy is converted into an image.

When the RF pulse is discontinued, the protons tend to "relax" back into alignment with the original magnetic field. In addition, the protons will lose phase coherence. As they relax, they release the previously applied energy as radio waves. These radio waves are picked up at different rates and signal intensities, depending on the tissue type releasing the energy. The waves are picked up by receiver coils in the MRI machine, which convert the energy to electrical signals. The computer uses these electrical signals to form an image.

There are two basic types of relaxation of protons (i.e., T1 and T2) that occur concurrently after the RF pulse is stopped; these types of relaxation are related to the return of the displaced net magnetic vector back to the direction of the static magnetic field (the positive z direction-T1 relaxation) and the loss of phase coherence among individual protons (T2 relaxation). Different tissues have different T1 and T2 relaxation times. Different tissues in the body have varying proton densities (i.e., number of hydrogen protons in a given volume of tissue), and the hydrogen protons in different tissues have varying levels of mobility. These tissue differences account for the variation in signal intensities generated by different tissue types in MRI (e.g., fat vs. cerebrospinal fluid [CSF]). The variation in relaxation times between different tissues in the body is far greater than the variation in tissue density, thus soft tissue contrast resolution is superior in MRI compared with CT.

T1 relaxation refers to the return of excited protons to the original energy state of the static magnetic field. Because this represents energy exchange between spinning high-energy protons and the molecular lattice from which they were excited, it is also called *spin lattice relaxation*. Because this energy transfer occurs as the displaced net magnetic vector relaxes back to the z or longitudinal direction, it is also referred to as *longitudinal relaxation*.

T2 relaxation describes energy exchange between the magnetic fields of individual protons before and after the application of an RF pulse. T2 relaxation is also called *spin-spin relaxation*. Because this type of relaxation occurs in a transverse direction (the x-y plane), it is also referred to as *transverse relaxation*. The phenomenon of T2 relaxation is primarily related to the loss of phase coherence among excited protons as they relax back to the equilibrium or ground state.

The time it takes for 64% of the protons in a particular tissue to regain the original energy state following the application of a 90-degree displacing RF pulse is that tissue's relaxation time (T1 or T2). Water has a very long T1 and T2 relaxation time, whereas fat has short T1 and T2 relaxation times. On a T1-weighted image, water (e.g., CSF, edema) will appear dark (hypointense) and fat will appear bright (hyperintense). On a T2-weighted image, water will appear hyperintense and fat will appear slightly less intense when compared with fat in T1-weighted images. Generally, T2-weighted sequences have the most inherent contrast between the various soft tissues, and the majority of pathologic processes will be associated with an increased signal intensity on T2-weighted images. T1-weighted sequences tend to have better anatomic resolution when compared with T2-weighted images.

Besides T1- and T2-weighted sequences, many additional sequences are commonly utilized in MRI. Proton density (PD) sequencing produces an image based on the density of protons within the tissue rather than T1 and T2 decays. The greater the density of protons, the more hyperintense that tissue contrast will be. PD images also tend to have good anatomy resolution. Fluid attenuation inversion recovery (FLAIR) sequences suppress the signal from free water (e.g., CSF) to discern this from tissue-bound water (e.g., edema), which has a considerably shorter T1 value. In neurologic imaging, FLAIR images are particularly helpful in distinguishing infarcts from cystic structures and delineating periventricular lesions as distinct from the neighboring ventricular CSF. Dual echo sequences combine the acquisition of a T2-weighted and PD image and are used to save time during the scanning process. There are two types of fat suppression sequences, the short tau inversion recovery (STIR) and a chemical fat saturation (FAT SAT). The physics behind these two suppression sequences are different. The important clinical difference between the two suppression sequences is that the physics associated with the STIR sequence will also suppress any gadolinium (Gd)-based contrast enhancement. Contrast enhancement can still be appreciated with FAT SAT sequences. It is extremely important to utilize a fat suppression sequence in musculoskeletal MRI to rule out fat deposition rather than a pathologic process within the bone. A fat suppression sequence should be utilized in any clinical situation where signal from fatty tissues needs to be suppressed to discern other neighboring anatomy or to confirm a pathologic process.

Gd-diethylenetriaminepentaacetic acid (DTPA) is a paramagnetic, IV-administered contrast agent used in MRI. Similar to iodinated intravenous contrast administration in CT imaging, Gd-DTPA is used to demonstrate vascularity and abnormalities of the blood-brain barrier. Gd-DTPA shortens both T1 and T2 relaxation times of tissues in which it localizes, leading to greatest signal intensity on T1-weighted images.

All these sequences are seldom done in one MRI exam. Usually several sequences are established for a standard imaging protocol tailored for a specific organ system, such as a brain, spine, generalized musculoskeletal imaging, or a joint-specific protocol (e.g., shoulder or stifle). Each MRI protocol may vary slightly, being made by each specific practice and tailored to that MRI system. Each magnetic resonance (MR) sequence requires an allotment of time to acquire that sequence. In veterinary medicine the patient is under general anesthesia for MRI. Therefore the typical goal with MRI is to have enough sequences and imaging planes to make an accurate diagnosis, without subjecting the patient to unnecessary or repetitive sequences and increased anesthesia time.

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There are a number of other MRI sequences that are becoming increasingly utilized in veterinary medicine. These include phase-contrast or cine-MRI and diffusion-weighted imaging (DWI). Phase-contrast or cine-MRI utilizes motion-sensitive pulse sequences to evaluate CSF flow. This type of imaging has potential for application in abnormal CSF flow disorders such as Chiari-like malformation with syringomyelia. DWI is based on MR measurement of the diffusion of intracellular water. With ischemic and inflammatory conditions of the central nervous system (CNS), cytotoxic edema leads to abnormalities of water diffusion (damage to ATPase pumps) that can be visualized via DWI. This mode of MRI is particularly applicable to ischemic/vascular events and inflammatory disorders, as abnormalities can be detected earlier in the disease course compared with conventional (e.g., T2-weighted, FLAIR) MR sequences. A variation of DWI, called *diffusion tensor imaging* or *MR tractography,* is used to evaluate white matter tracts via evaluation of the direction of water diffusion along these tracts.

Computed Tomography

CT utilizes x-rays and computers to provide cross-sectional images of the patient. The final images are composed of many small image squares called *pixels*. The thickness of these image squares (voxels) is determined by the chosen slice thickness. The patient is placed in the opening of the CT gantry. The gantry contains the x-ray tube, x-ray collimators, and x-ray detectors. The x-ray tube and detectors are on opposite sides, and the patient is between them. The collimators control slice thickness. The x-ray tube rotates around an object of interest as x-rays are emitted. As the x-ray beam passes through an object, it is attenuated by tissues in its path. Each tissue attenuates the beam to a different degree. The different attenuating abilities of different tissues, or *linear attenuation coefficients*, provide the basis of tissue contrast for CT. The detector receives the attenuated beam, also called a *projection*, of x-ray photons, and the information is fed into the computer. The projections are reconstructed into a 3D volume by using either purely analytic methods such as filtered back-projection (the most common) or iterative procedures such as expectation maximization. The analytic approaches currently dominate; however, iterative methods perform better with less reliable information and will likely see increased usage as the desire to lower the applied radiation dose becomes more important. The computer assigns grayscale numbers (Hounsfield units) to the tissues that the x-ray beam passed through, based on their linear attenuation coefficients.

The resultant image reflects the different grayscale numbers of different tissue types, and therefore the tissues' respective abilities to attenuate x-rays. As one would expect from conventional x-ray procedures, bone appears white, air appears black, and fluid is somewhere in between (gray). The corresponding Hounsfield numbers for these tissues are +1000, −1000, and 0, respectively.

The human eye can discern approximately 20 shades of gray. The number of shades of gray in the image, as well as the central gray color (above which tissues are brighter, below which they are darker), can be manipulated once the computer receives the image information. Choosing the central gray color, above which tissues appear brighter and below which they appear darker, is referred to as setting the window level (WL). The Hounsfield unit of the tissue of interest is typically chosen as the center of the WL. The number of shades of gray assigned to a particular image represents the window width (WW). A narrow WW is chosen for soft tissue (e.g., brain parenchyma) to improve contrast resolution (the ability to discern differences in composition of tissues in close proximity). A wide WW is chosen for tissues in which good inherent contrast already exists (e.g., air in the frontal sinus region) or when imaging a region where a wide range of tissue densities is displayed (e.g., lung). When imaging brain parenchyma, a WL of approximately 35 and a WW of 150 may be assigned. In contrast, when imaging bony tissue, a WL of 420 and WW of 1500 may be used.

After obtaining CT images of the area of interest, iodinated contrast is often given IV and the patient is imaged. The contrast agent will be distributed through the vascular space and excreted by the kidneys. Contrast is used to demonstrate areas of blood-brain barrier disruption, increased areas of vascularity, delineated blood vessels, and anomalous shunts. As the kidneys excrete the contrast, it quickly concentrates in the urine, which also allows for clear delineation of the ureters.

Advantages of CT over other imaging modalities are numerous. CT provides far superior soft tissue contrast in comparison with conventional radiography. The various soft tissue planes are usually delineated with CT imaging. The cost of CT is typically less than MRI. CT is a more rapid imaging modality than MRI, and the mineralized bony matrix and acute hemorrhage are better visualized with CT versus MRI; these attributes make CT preferable to MRI in acute head trauma patients. Currently available multislice CT scanners are so fast that patients can be imaged in seconds, sometimes only requiring sedation. Other advantages of CT imaging are the ability to alter window settings after data acquisition to maximize tissue contrast. Following the initial data acquisition, modern CT has the ability to form multiple 2D and 3D reconstructed images. With newer CT scanners, these reconstructed 3D images are very detailed and artifacts associated with metallic implants are usually not an issue unless precise quantification near the implant is required. The use of CT for postoperative assessment of implants is very efficient and much more detailed than conventional radiography.

There are several disadvantages of CT compared with MRI. CT involves exposure to ionizing radiation (x-rays), whereas MRI does not. MRI provides far superior soft tissue detail compared with CT. CT is usually adequate for visualizing mass lesions in the brain and spinal cord. However, subtle parenchymal lesions (e.g., inflammatory foci in granulomatous meningoencephalomyelitis), as well as brain (especially brain stem) and spinal cord lesions in very small dogs and cats are much more appreciable on MRI than CT.

Positron Emission Tomography

PET is a form of nuclear medicine, in that it relies on an injected radionuclide for the imaging signal. It is different from traditional nuclear medicine in that it is based on positron emission instead of the more typical single-photon gamma emissions. This difference gives PET several advantages. There are several easily producible positrons emitting isotopes such as fluorine (^{18}F), oxygen (^{15}O), carbon (^{11}C), and nitrogen (^{13}N). It is important to note that these elements are all common to the typical organic compounds found in living things. Unlike isotopes used in nuclear medicine such as technetium-99m (^{99m}Tc), the chemistry of the isotopes available to PET are more accurate surrogates for measurement of the function of living tissue. In addition, the physics of PET allows it to be electronically collimated, whereas single-photon nuclear medicine techniques require physical collimation. This results in better efficiency of several orders of

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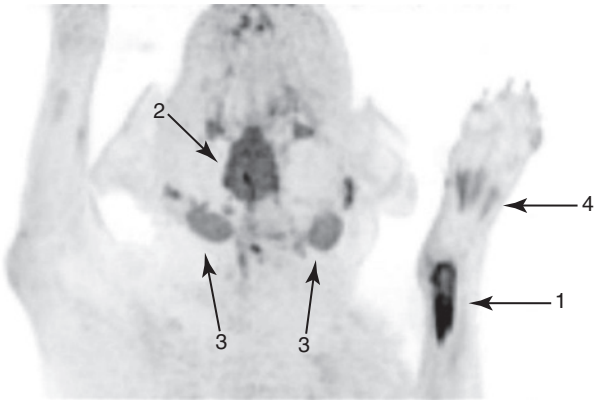


FIG. 14.8 Positron emission tomography imaging of the distal left forelimb and skull of a dog with osteosarcoma of the distal left radius. There is intense uptake of fluorodeoxyglucose (^{18}FDG) in the distal metaphysis and diaphysis of the left radius *(arrow 1)*. There is normal ^{18}FDG uptake in the brain *(arrow 2)* and salivary glands *(arrows 3)*. Mild inflammation in the phalanges of the affected limb is indicated by slightly higher than background FDG uptake *(arrow 4)*.

magnitude. In addition, attenuation correction can be accounted for, resulting in accurately quantified image values. In total, these advantages have led to dramatic changes in the way we view functional physiologic imaging techniques because we are now able to see things that were simply not visible before.

PET relies on positrons to produce an image, but it does not sense them directly.

The application of PET has seen widespread acceptance in human oncology with research applications in neurology and biodistribution for new drug development. In oncology, the primary application is measurement of glucose metabolism with fluorodeoxyglucose (FDG). Imaging of the distribution of FDG gives a good approximation of the overall consumption of glucose (Fig. 14.8). Most cancers are hypermetabolic; thus locating tumors through their glucose metabolism signature has proven very effective.

FDG is one of many PET tracers available that can provide useful information. For example, cells use thymidine during division; thus imaging the cellular use of thymidine using fluorothymidine (^{18}FLT) can be correlated to the rate of cell proliferation. Clearly, for cancers that are growing rapidly, this can be useful information. Knowledge of what parts of the tumor are growing fastest can aid in the development of treatment plans.

PET scanners image the concentration of positron-emitting radioisotopes; therefore any compound that can be labeled can be measured. This feature has had significant impact in the development of new drugs. By labeling a candidate drug with a positron-emitting radioisotope, biodistribution of that drug can be determined without injuring the host. This provides accurate information about dose requirements without the need for large numbers of studies or research animals.

Hybrid Imaging With Positron Emission Tomography/Computed Tomography

From the surgeon's perspective, PET in itself may be interesting, but direct application to surgery is not clearly obvious because reference to recognizable anatomic landmarks is very limited. In fact, the ideal PET scan would show no anatomy at all, only the physiologic parameters of the diseased tissue. The invention of hybrid PET/CT imaging by Drs. Ronald Nutt and David Townsend has allowed correlation of very precise anatomic imaging generated with CT to the functional physiologic information generated with PET, providing an entirely new perioperative surgical planning tool. This application is especially valuable in oncologic surgery, where it may aid the surgeon in obtaining clean margins when resecting a tumor. Although PET/CT is in its infancy in veterinary medicine, its clinical utility will be profound because the state and extent of the disease (the FDG metabolism) is precisely coregistered to the anatomy.

Clinical Applications

As mentioned previously, MRI has become the imaging modality of choice for neurosurgery and the majority of MR studies in veterinary medicine focus on the CNS. The specific clinical applications relating to the CNS are detailed in Chapter 37. In human medicine, the musculoskeletal system is routinely imaged with MRI; it is considered by many to be the most important advanced imaging modality for the assessment of human musculoskeletal injuries and diseases. The advantage of MRI for orthopedics is that it provides an assessment of the entire organ system (e.g., muscles, tendons, ligaments, cartilage, meniscus, joint fluid, fat, and bone). Bone, ligaments, and tendons appear more hypointense on the MR sequences. Many clinicians are hesitant to evaluate bone with MRI because this does not reveal the fine structural detail of the mineralized bony matrix compared with radiography and CT. However, bone is more than a mineralized structure, and MRI is far superior to these other two modalities for evaluating the extent of soft tissue pathology associated with the bone.

Many musculoskeletal MR sequences are reported throughout the literature, some of which focus on a specific tissue type, such as cartilage. The more commonly utilized musculoskeletal sequences include T1-weighted, PD, dual echo, T2-weighted, or PD combined with fat suppression and T1-weighted sequences. One of the most exciting aspects of orthopedic MRI is that the entire articular surface can be imaged. MRI of cartilage in small animals is more challenging because articular cartilage is much thinner in dogs compared with people and large animals (Fig. 14.9). The thinner articular cartilage provides less signal for the MRI, and there is relatively more averaging of the cartilage with subchondral bone and joint fluid.

In musculoskeletal MRI, the terms "bone edema" and "bone bruise" are frequently used to describe a hyperintense lesion on fat suppressed T2-weighted and PD sequences. These lesions are nonspecific and are commonly associated with many types of musculoskeletal injuries and disease processes. When these lesions are evaluated histologically, the findings are quite varied and include inflammatory cells, hemorrhage, hematopoietic cells, necrosis, fibrosis, and edema. A more appropriate term is a "bone marrow lesion."

Although MR allows for superior soft tissue contrast, CT is also a vital imaging modality for the musculoskeletal system. Thinly sliced CT images provide excellent spatial resolution and bony detail, making it the modality of choice to define complex bony anatomy. Some specific examples where this aspect of CT is used frequently include imaging the skull, vertebral bodies, and pelvis following trauma to more fully assess fractures and determine if there is nervous system involvement (Fig. 14.10). Elbows are frequently evaluated with CT to assess for fragmented

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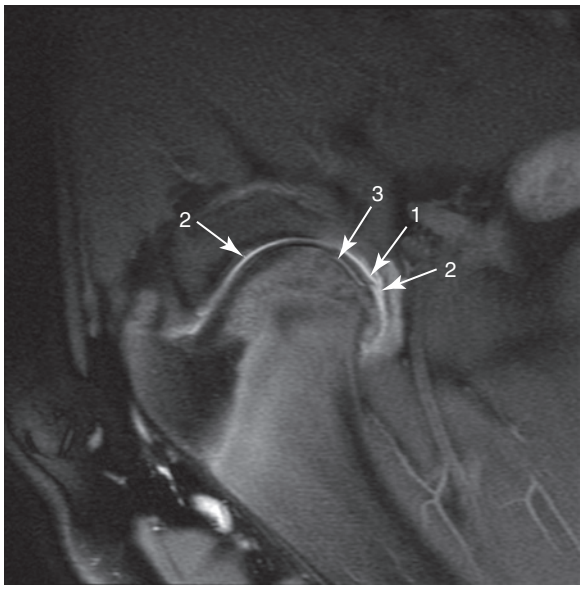


FIG. 14.9 Sagittal magnetic resonance imaging proton-dense, fat-saturated image of the caudal humeral head of a dog with an osteochondrosis lesion. A moderate-sized in situ cartilaginous flap is present *(arrow 1),* which is well delineated by the hyperintense joint fluid *(arrows 2)*. The cartilaginous flap is dorsal to the flattened humeral head *(arrow 3)*.

medial coronoids (Fig. 14.11). MRI of the elbow is also used to assess for coronoid disease and is more sensitive in the identification of cartilaginous coronoid fragments compared with other imaging modalities. CT and MRI are much more accurate and specific in regard to the diagnosis of a fragmented medial coronoid compared with radiography.

For severe angular limb deformities, CT is excellent for presurgical planning. Radiographically, it can be difficult to accurately plan the correctional procedure if the limb deformity is taking place in multiple planes. Not only does CT allow for excellent axial images, modern software can project the limb in all planes and in three dimensions. Additionally, plastic models of the limb can be created from the CT images. These models are highly useful for planning the correctional procedure.

These modalities are also useful for soft tissue surgical planning. The tympanic bullae are frequently evaluated with CT to assess for otitis media and determine whether a bulla osteotomy is indicated. Otitis media is easily recognized on MRI images as a hyperintensity with the bulla on T2-weighted images (Fig. 14.12). Both CT and MRI are useful in the identification and localization of embedded soft tissue foreign bodies, which may or may not be associated with a draining tract. Traditionally, if a draining tract is present, a radiographic fistulogram is performed. Clinically, many fistulograms are of questionable diagnostic accuracy. With MRI, the regional inflammation is readily identified as an atypical hyperintensity on the T2 or PD

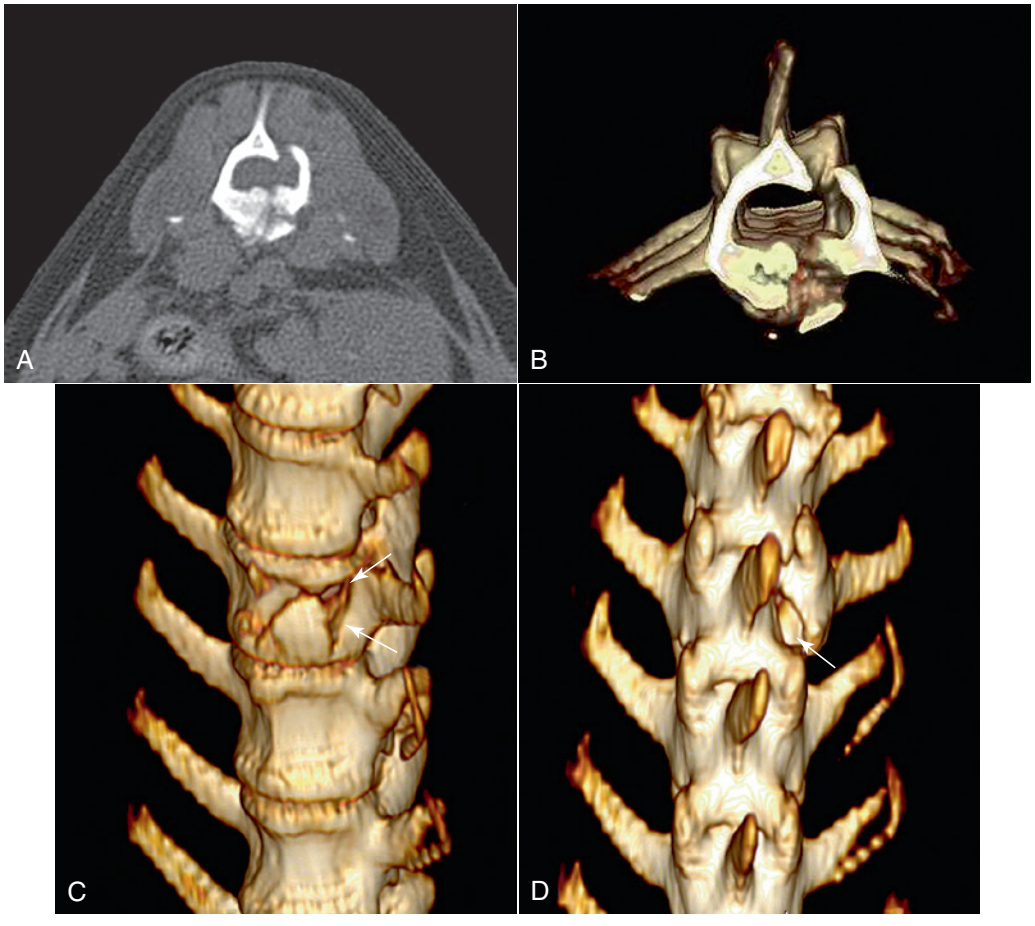


FIG. 14.10 Multiple images of a fractured lumbar vertebral body. (A) Transverse computed tomography image at the level of the fracture site. (B–D) Three-dimensional (3D) surface renderings of the same fracture. (B) A 3D transverse view sliced obliquely to show the extent of the fracture separation. (C) Ventral aspect of the comminuted vertebral body fracture *(arrows)*. (D) Dorsal aspect of the vertebra; note how the left pedicle, part of the lamina, and facet are displaced laterally *(arrow)*. Also note the displaced fracture fragments of the left transverse processes of the two vertebrae, caudal to the vertebral body fracture.

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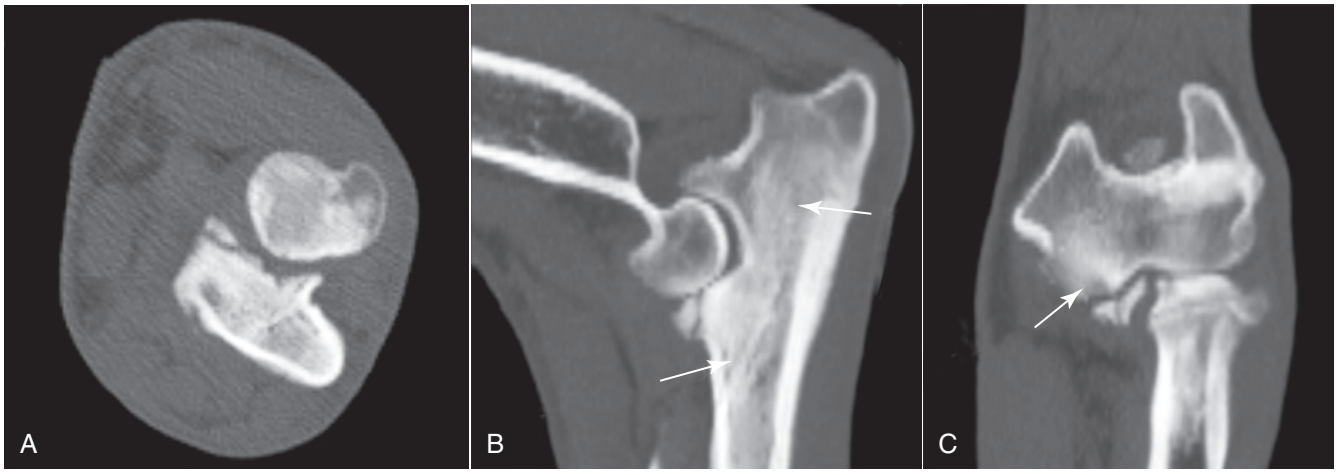


FIG. 14.11 (A) Transverse computed tomography image of a fragmented medial coronoid process (FMCP) (window width = 2000; window level = 400). (B) A sagittal image reformatted at the level of the FMCP. The patient also had concurrent panosteitis *(arrows),* which is best seen on image as sclerosis throughout the proximal ulna. (C) A cranial reformatted image at the level of the FMCP. A small osteochondrosis lesion is present at the medial condyle of the humerus *(arrow).*

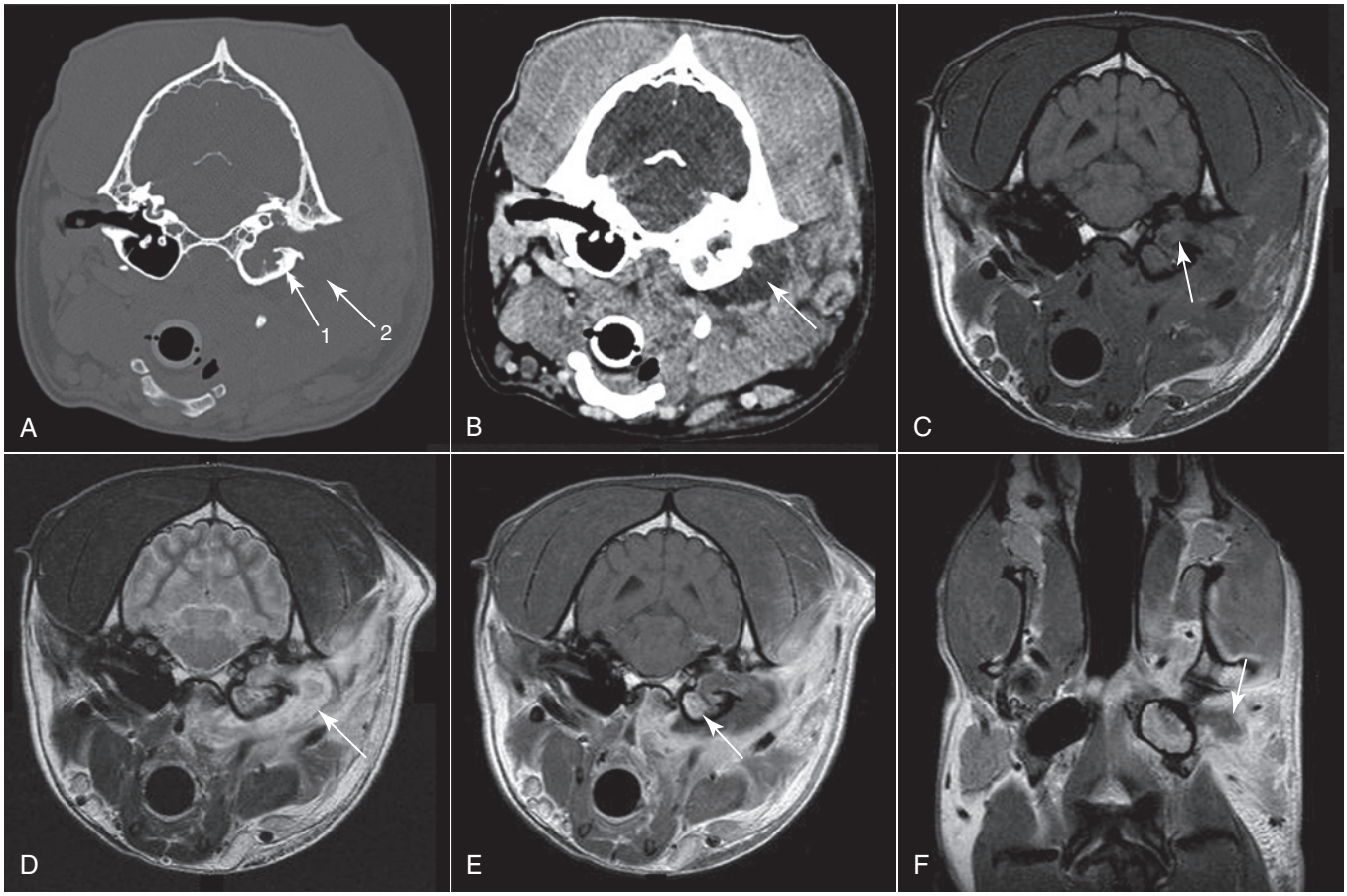


FIG. 14.12 (A) Computed tomography (CT) image set on a bone window (window width [WW] = 25,000; window level [WL] = 480). Note the osseous thickening of the left bulla *(arrow 1),* the soft tissue attenuating material within the bulla, and the more hypoattenuating material closely associated with the left bulla *(arrow 2).* (B) CT image set on a soft tissue window (WW = 300; WL = 30) following administration of contrast. Note the lack of enhancement of hypoattenuating material near the left bulla *(arrow),* but there is enhancement of the tissues surrounding this region. (C–D) Transverse T1- and T2-weighted images of the bulla. Note the improved soft tissue contrast within the left bulla *(arrow)* compared with the CT images. (D) Hyperintensity of the surrounding tissue is characteristic of increased fluid within these tissues *(arrow).* (E) A transverse T1 postcontrast image. Arrow shows contrast enhancement within the bulla. (F) A dorsal T1 postcontrast image at the level of the left bulla. The hyperattenuating tissue on these images correlates with increased vascularity of the tissues. Note how the contrast enhancement is similar to the CT images, especially in the abscessed, nonenhancing region adjacent to the left bulla *(arrow).* These findings are consistent with otitis media with regional abscess formation and cellulitis. The right bulla is unremarkable.

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sequences. On CT, the regional inflammation and abscessation can be identified from normal tissues (Fig. 14.13). The appearance of the foreign body depends on its composition. Acquiring images in every plane allows for accurate localization of the foreign body, aiding in the diagnosis and surgical planning.

Vascular diseases may also be assessed with these modalities. Congenital anomalies, such a portosystemic shunt, can be delineated with both CT and MR angiography (Fig. 14.14). Many of these techniques require precise timing to follow the bolus of intravenous contrast through the vessels of interest (Fig. 14.15). Digital subtraction is a technique where a noncontrast image is subtracted from a contrast image to maximize the visibility of the vascular structure. This is more commonly utilized to delineate smaller, less discrete vessels of interest. With MRI, there are several

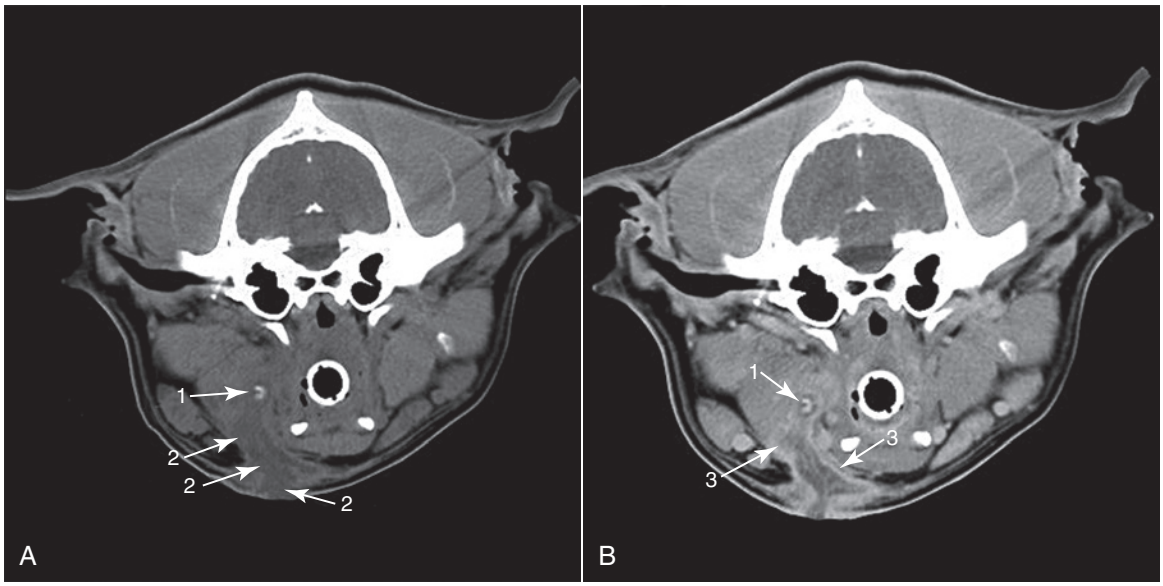


FIG. 14.13 Transverse computed tomography images at the level of the tympanic bulla, (A) before and (B) after administration of contrast, respectively (window width 241:window level 54). A hyperattenuating crescent-shaped foreign body is present in the soft tissues to the right of the endotracheal tube *(arrow 1)*. Heterogeneous soft tissue attenuating material is presently surrounding this structure and extending ventrally to the surface of the skin through the draining tract *(arrows 2)*. Note the contrast enhancement of the soft tissue surrounding the draining tract and foreign body *(arrows 3)*.

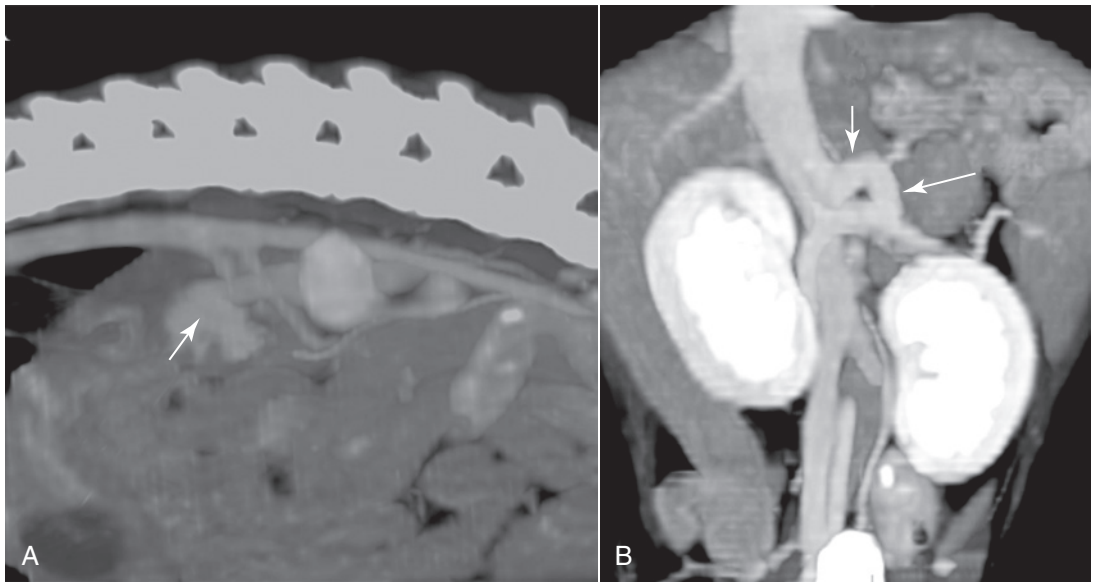


FIG. 14.14 Two-dimensional multiplanar (A) sagittal and (B) frontal reconstructed computed tomography images, centered at the level of a typical portocaval extrahepatic portosystemic shunt (PSS). The PSS is identified by the *arrows* and is an enlarged tortuous vessel connecting the portal vein to the caudal vena cava. (Courtesy Dr. Robert Bahr, Center for Veterinary Health Sciences, Boren Veterinary Medical Teaching Hospital, Stillwater, OK.)

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FIG. 14.15 Renal contrast-enhanced magnetic resonance angiography study. Aorta, renal arteries, and kidneys are clearly delineated.

imaging techniques that can be utilized to obtain an MR angiogram, many of which do not depend on the administration of intravenous contrast. Some of these techniques will likely be utilized in the future to assess the renal vasculature in feline kidney transplant procedures. Techniques such as TWIST (time-resolved angiography with interleaved stochastic trajectories) allow for a bimodal sampling of MRI K-space in such a way that the temporal resolution can be increased without substantially harming image quality. This means that multiple image frames can be taken quickly and stored so that the precise timing becomes less important. Because intravenous contrast is excreted through the kidneys, CT-based excretory urography is a commonly performed clinical procedure used to aid in the diagnosis of ectopic ureters.

CT is the modality of choice to assess for pulmonary thromboembolism because of its rapid acquisition time. This characteristic also makes it the most clinically applicable modality for the evaluation of other thoracic and thoracic wall diseases, such as lung lobe torsions; to assess for pulmonary metastatic disease when thoracic radiographs are inconclusive; to assess for causes of pleural effusion; and to evaluate the extent of regional neoplasia (Fig. 14.16). Motion causes significant artifacts with both CT and MRI. Motion artifact is more severe with MRI compared with CT; however, the rapid acquisition of modern CT allows for thoracic imaging without the use of gated software. The entire thorax can be imaged in a single breath hold. Any region of motion imaged with MRI requires the use of gated software.

A great example of gated MRI is cardiac MR studies. These studies provide high-resolution cine information that can be used to accurately measure left and right ventricular functional parameters such as ejection fraction and stroke volume. This information can be consistently relied on to provide an estimate of the state of disease in the cardiac muscle. This type of information is often acquired with ultrasound as well, but if performed with MRI, flow quantification of the aortic and pulmonary valves can also be performed. The extent of regurgitation through the valves can be precisely measured over the heart cycle by examining the velocity of the blood flow through the valve. The heart can also be evaluated with high-end modern multislice CT scanners; 128+ slice CTs are sufficiently fast to perform outstanding gated cardiac scans. By using contrast agents, the blood pool and heart muscle itself can be well separated and functional analysis performed. In this way, ejection fraction, stroke volume, and wall-thickening parameters can be computed with great precision. As fast as modern 128+ slice systems are, they do have limitations with high heart rate, namely blurring and edge artifacts. This can complicate the imaging of smaller animals. It is important to note that cardiac gating does not reduce the need for some kind of motion compensation for breathing, and a breath hold significantly reduces artifacts in this type of study. This is not normally a problem because a typical cardiac CT study on a 128-slice system only requires 5 seconds.

Soft tissue surgeons commonly utilize CT and MRI to aid in the planning of complex surgeries, often involving some type of invasive neoplasm. Not only can the tumor be delineated, the major vessels can also be identified. The multiplane reconstructed images, 3D images, and the ability to have plastic models constructed from the imaging data are extremely valuable for the preoperative assessment and planning of these patients. Computed tomographic biopsy procedures are also frequently performed, most commonly of the vertebral bodies and primary pulmonary nodules/masses, which cannot be visualized with ultrasonography (Fig. 14.17).

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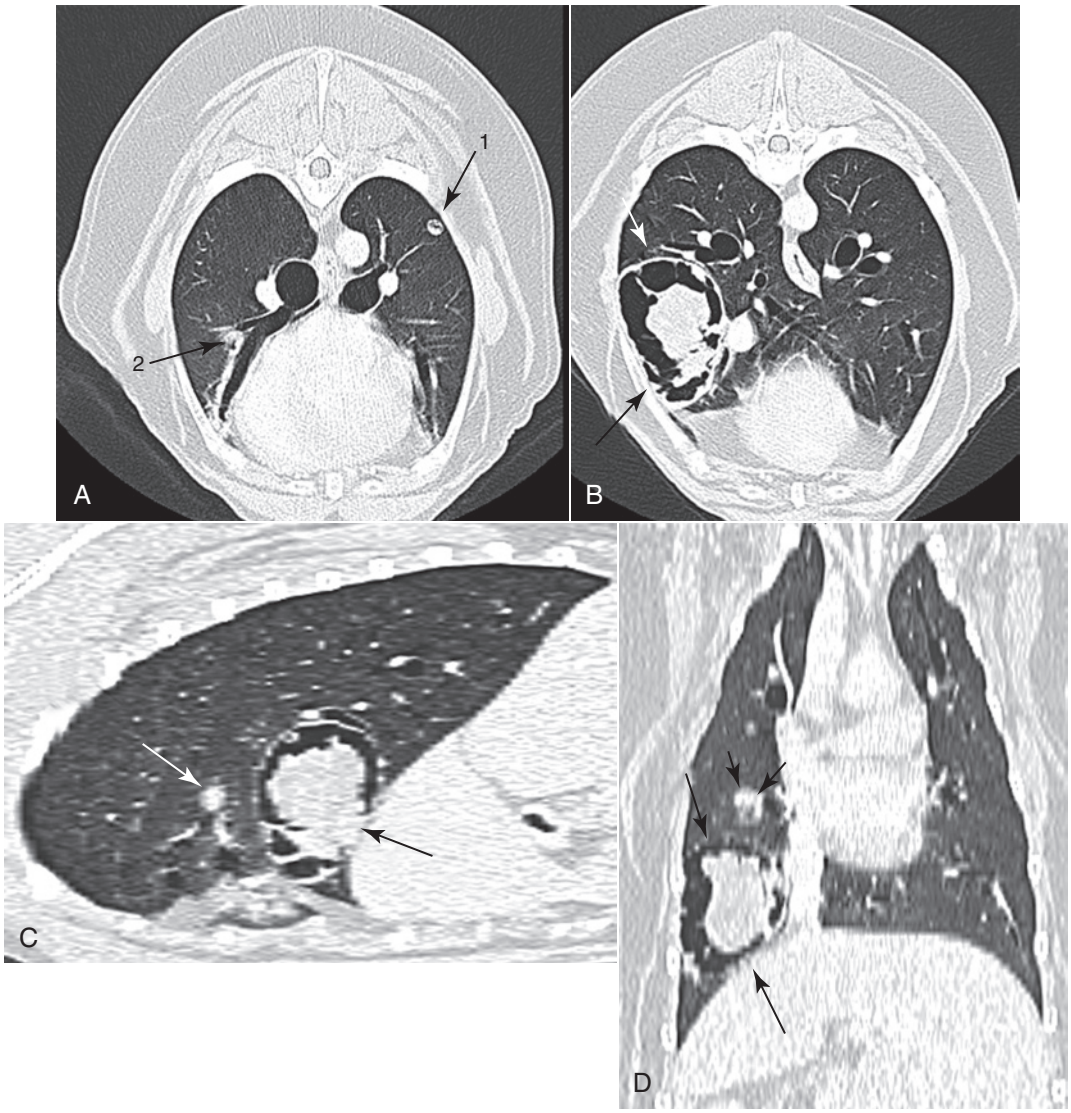


FIG. 14.16 Computed tomography images from a dog with pulmonary neoplasia. All images are post–contrast administration and are set on a lung window (window width = 1500; window level = 600). (A) Two cavitary metastatic lesions cranial to the large primary cavitary lung mass present in (B). One metastatic lesion is present in the peripheral aspect of the dorsal left cranial lung lobe *(arrow 1),* and the other is adjacent to the bronchus of the right middle lung lobe *(arrow 2).* (B) A large cavitary lung mass is present in the peripheral aspect of the right caudal lung lobe *(arrow).* (C) A reconstructed sagittal image of the right hemithorax, which shows the relationship between the metastatic lesion *(white arrow)* and the mass *(black arrow).* (D) A dorsal reconstructed image showing the same relationship as (C), the primary mass *(large arrows)* and metastatic lesions *(small arrows).* The other hyperattenuating structures in the images represent contrast in the pulmonary vessels.

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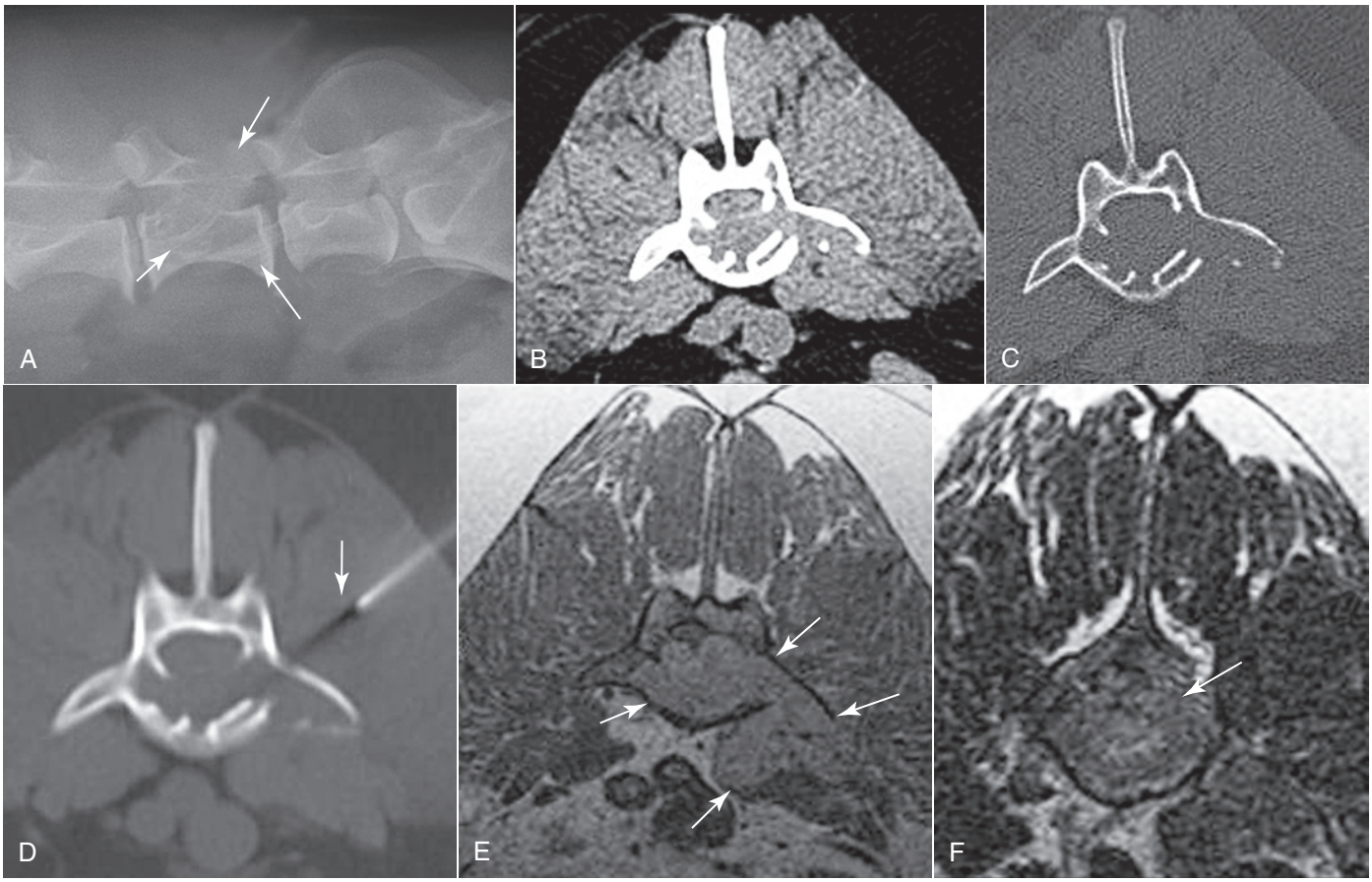


FIG. 14.17 Comparative images of a plasma cell tumor located at the sixth lumbar vertebral body. The computed tomography (CT) images demonstrate the osteolysis better than magnetic resonance imaging (MRI); however, the invasive soft tissue is much more clearly delineated on the MRI. (A) Lateral radiograph of the caudal lumbar region. (B–D) Transverse CT images of L6. (E–F) Transverse T1-weighted and T2-weighted MRI of L6, respectively. (A) Faint radiolucency associated with L6 *(arrows)* indicating the presence of a tumor. (B) Soft tissue window (window width [WW] = 350; window level [WL] = 30). (C) Bone window (WW = 2500; WL = 480). (D) CT biopsy procedure. The linear hyperattenuating structure is the biopsy needle. Immediately distal to the needle is a linear hypoattenuating artifact *(arrow)* caused by the needle. (E) Osteolysis of the lumbar vertebrae and the invasiveness of the tumor *(arrows)*. (F) Invasive nature of the tumor *(arrow)*.

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FIG. 14.18 Distal canine femoral osteosarcoma lesion. (A) T1-weighted, postcontrast, fat-saturated sagittal magnetic resonance (MR) image. (B) T2-weighted, fat-saturated cranial MR image. There is an extensive extramedullary component to this mass *(arrow 1)* and a long zone of transition through the femoral diaphysis. (A) demonstrates intramedullary contrast enhancement *(arrow 2);* (B) shows an intramedullary hyperintensity *(arrow 3),* both of which represent the neoplastic invasion of normal intramedullary fat. Normal suppressed bone marrow fat remains present in the most proximal aspect of the femoral diaphysis *(arrow 4).* (Courtesy Dr. Susan Kraft, Colorado State University.)

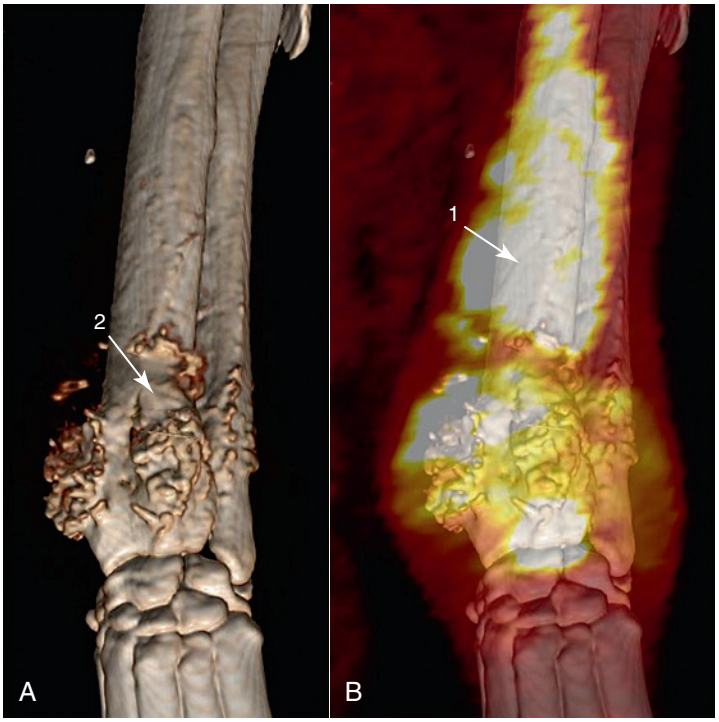


FIG. 14.19 (A) Computed tomography (CT) surface rendering of a canine distal radial osteosarcoma. (B) Positron emission tomography/CT fusion image of the same lesion. The white regions represent the highest uptake of fluorodeoxyglucose (^{18}FDG) (i.e., the highest amount of abnormal physiologic activity). Yellow represents increased cellular uptake of the ^{18}FDG, and red represents normal physiologic or background uptake of the ^{18}FDG. Note how much more extensive the intense physiologic activity *(arrow 1)* of the neoplasm is when compared with the osseous anatomic change associated with the neoplasm *(arrow 2).* This is the same dog as shown in Fig. 14.8, at a later stage of progression.

CT, MRI, PET, and PET/CT have obvious and profound clinical applicability for oncologic surgery. A complete list of clinical examples is beyond the scope of this text. Osteosarcoma is a common tumor in veterinary medicine, and the extent of this neoplasm and the clinical outcome of patients are studied by utilizing radiography, nuclear scintigraphy, and PET/CT. The true extent of this tumor near the cellular level cannot be determined with radiographs or CT. The physiologic changes within the affected tissue may not be significant enough to show an altered physical density that is detectable with CT. MRI delineates the soft tissue changes associated with the tumor and affected bone (Fig. 14.18). Specifically, MRI has been utilized to assess treatment options of osteosarcoma cases, including the use of limb-sparing surgical procedures. PET imaging reveals physiologic changes with great sensitivity and specificity. The fusion of ^{18}FDG PET with CT also shows the physiologic expansion of the osteosarcoma (Fig. 14.19).

In summary, these advanced imaging modalities are key to more fully assessing complex anatomy, tissue vascularity, aberrant vessel formation, and extent of a disease process. As a planning tool for complex surgical procedures, they allow for either anatomic imaging without the superimposition of other anatomic structures or an assessment of the functional physiologic extent of the disease process.

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15



Surgery of the Integumentary System

GENERAL PRINCIPLES AND TECHNIQUES

WOUND MANAGEMENT

SURGICAL ANATOMY

The skin is composed of the epidermis, dermis, and associated adnexa. The outermost layer *(epidermis)* is thin but protective; it is especially thin in areas with abundant hair and slightly thicker in areas without much hair. The thickest epidermis is on the nose and footpads, where it is keratinized. The epidermis is avascular, receiving nourishment from fluid penetrating the deeper layers and from dermal capillaries. The thicker, vascular *dermis* lies deep to the epidermis, which it nourishes and supports. The dermis is composed of collagenous, reticular, and elastic fibers surrounded by a mucopolysaccharide ground substance. Fibroblasts, macrophages, plasma cells, and mast cells are found throughout this layer. The dermis contains blood and lymph vessels, nerves, hair follicles, glands, ducts, and smooth muscle fibers. The hypodermis, or *subcutis*, lies below the dermis.

Musculocutaneous vessels are the primary vessels supplying skin in human beings, apes, and swine; however, dogs and other loose-skinned animals lack musculocutaneous vessels. Musculocutaneous vessels run perpendicular to the skin's surface, whereas vessels supplying canine and feline skin approach and travel parallel to the skin and are direct cutaneous vessels. For this reason, some human pedicle grafting techniques have limited application in dogs and cats. Terminal arteries and veins branch from direct cutaneous vessels and form subdermal (deep) plexus, cutaneous (middle) plexus, and subpapillary (superficial) plexus. The subdermal plexus supplies hair bulbs and follicles, tubular glands, the deeper portion of the gland ducts, and arrectores pilorum muscles. The cutaneous plexus supplies sebaceous glands and reinforces capillary networks around hair follicles, tubular gland ducts, and arrectores pili muscles. The subpapillary plexus lies on the outer layer of dermis, and capillary loops from this plexus project into and supply the epidermis. The capillary loop system is poorly developed in dogs and cats compared with human beings and swine, which is why canine skin does not usually blister with superficial burns.

The subdermal plexus is of major importance to skin viability. In areas where there is a panniculus muscle (cutaneous trunci, platysma, sphincter colli superficialis, sphincter colli profundus, preputialis, supramammarius muscles), the subdermal plexus lies both superficial and deep to it. Therefore surgeons must undermine the fascial plane beneath the cutaneous musculature to preserve the integrity of the subdermal plexus. Where the panniculus is absent, such as in the extremities, the subdermal plexus runs in the deep surface of the dermis, requiring that one undermine well below the dermal surface.

WOUND HEALING

Wound healing is a biologic process that restores tissue continuity after injury. It is a combination of physical, chemical, and cellular events that restore wounded tissue or replace it with collagen. Wound healing begins immediately after injury or incision. The four phases of wound healing are inflammation, debridement, repair, and maturation. Wound healing is dynamic; several phases occur simultaneously. The first 3 to 5 days are the lag phase of wound healing because inflammation and debridement predominate and wounds have not gained appreciable strength. Healing is influenced by host factors, wound characteristics, and other external factors.

Stages of Wound Healing

Inflammatory Phase

Inflammation is a protective tissue response initiated by damage. This phase is characterized by increased vascular permeability, chemotaxis of circulatory cells, release of cytokines and growth factors, and cell activation (macrophages, neutrophils, lymphocytes, and fibroblasts). Hemorrhage cleans and fills wounds immediately after injury. Blood vessels constrict for 5 to 10 minutes to limit hemorrhage, then dilate and leak fibrinogen and clotting elements into wounds. Vasoconstriction is mediated by catecholamines, serotonin, bradykinin, and histamine. The extrinsic coagulation mechanism is activated by thromboplastin released from injured cells. Platelet aggregation and blood coagulation form a clot that ensures hemostasis and provides a scaffold for cell migration. Platelets also release potent chemoattractants and growth factors (epidermal, platelet-derived, transforming growth factors α and β) that are necessary in later stages of wound healing (Box 15.1). Fibrin and plasma transudates fill wounds and plug lymphatics, localizing inflammation and "gluing" wound edges together. Fibronectin dimers within the clot become covalently crosslinked

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ABSTRACT

Many cutting-edge modalities are becoming readily available to the veterinary practitioner for management of complicated open wounds, including enzymatic debridement, low-level laser therapy, and negative pressure wound therapy. However, fundamental wound care principles allow for successful management in most wound cases. Good basic wound management allows for rapid healing or provides a healthy wound bed for surgical closure. Principles of open wound care include prevention of further contamination, wound lavage, debridement of devitalized tissue, and appropriate bandage placement and use of wound dressings.

KEYWORDS

wound management
wound healing
debridement
topical antimicrobials
cleansing solutions
skin viability
drains
bandaging
wound dressings
tension
skin flaps
skin graft
thermal injury
bite wound
tail amputation
digit amputation

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BOX 15.1 Growth Factors Important in Wound Healing

BFGF: basic fibroblast growth factor
EGF: epidermal growth factor
KGF: keratinocyte growth factor; also known as *growth factor 7*
PDGF: platelet-derived growth factor
TGF-α and TGF-β: transforming growth factor
VEGF: vascular endothelial cell growth factor

to fibrin and to themselves in the presence of activated factor XIII, forming a provisional extracellular matrix. This blood clot formation stabilizes the wound's edges and provides limited wound strength. It also provides an immediate barrier to infection and fluid loss and a substrate for early organization of the wound. Scabs form when the blood clot dries; they protect wounds, prevent further hemorrhage, and allow healing to progress beneath their surface. Inflammatory phase cells such as platelets, mast cells, and macrophages secrete growth factors or cytokines, which initiate and maintain the proliferative phase of healing. Inflammatory mediators (i.e., histamine, serotonin, proteolytic enzymes, kinins, prostaglandins, complement, lysosomal enzymes, thromboxane, and growth factors) cause inflammation that begins immediately after injury and lasts approximately 5 days. White blood cells leaking from blood vessels into wounds initiate the debridement phase.

Debridement Phase

An exudate composed of white blood cells, dead tissue, and wound fluid forms on wounds during the debridement phase. Chemoattractants encourage neutrophils and monocytes to appear in wounds (approximately 6 hours and 12 hours after injury, respectively) and initiate debridement. Neutrophils increase in number for 2 to 3 days. They prevent infection and phagocytize organisms and debris. Degenerating neutrophils release enzymes and toxic oxygen products that facilitate breakdown of bacteria, extracellular debris, and necrotic material, and they stimulate monocytes. Monocytes are essential for wound healing; neutrophils are not. Monocytes are major secretory cells synthesizing growth factors that participate in tissue formation and remodeling. Monocytes become macrophages in wounds at 24 to 48 hours. Macrophages secrete collagenases, which remove necrotic tissue, bacteria, and foreign material. They may coalesce and form multinucleated giant cells with phagocytic functions. Macrophages also secrete chemotactic and growth factors. Growth factors (e.g., platelet-derived growth factor, transforming growth factor-α, transforming growth factor-β, fibroblast growth factor, and interleukin-1) can initiate, maintain, and coordinate formation of granulation tissue. Chemotactic factors (e.g., complement, collagen fragments, bacterial endotoxins, and inflammatory cell products) direct macrophages to injured tissue. Macrophages also recruit mesenchymal cells, stimulate angiogenesis, and modulate matrix production in wounds. Platelets release growth factors important for fibroblastic activity. Lymphocytes appear later in the debridement phase than neutrophils and macrophages. They secrete soluble factors that may stimulate or inhibit migration and protein synthesis by other cells. However, they usually improve the rate and quality of tissue repair. Although healing is severely impaired when macrophage function is suppressed, neutropenia and lymphopenia do not inhibit healing or the development of wound tensile strength in sterile wounds.

Repair Phase

The repair phase usually begins 3 to 5 days after injury. Macrophages stimulate DNA and fibroblast proliferation. Cytokines, in concert with extracellular matrix molecules, stimulate fibroblasts in the surrounding tissue to proliferate, express appropriate integrin receptors, and migrate into wounds. Fibroblasts are stimulated by transforming growth factor-β to produce fibronectin, which facilitates cell binding and fibroblast movement. Platelet-derived growth factor and basic fibroblast growth factor are also involved. A tissue oxygen content of approximately 20 mm Hg and slight acidity also stimulate fibroblast proliferation and collagen synthesis. Fibroblasts originate from undifferentiated mesenchymal cells in surrounding connective tissue and migrate to wounds along fibrin strands in the fibrin clot. Fibroblasts migrate into wounds just ahead of new capillary buds as the inflammatory phase subsides (2–3 days). They invade wounds to synthesize and deposit collagen, elastin, and proteoglycans that mature into fibrous tissue. Orientation is initially haphazard, but after 5 days, tension on wounds causes fibroblasts, fibers, and capillaries to orient parallel to the incision or wound margin. Wound fibrin disappears as collagen is deposited. Collagen synthesis is associated with an early increase in wound tensile strength. As the wound matures, there is a notable increase in the ratio of type I (mature) to type III (immature) collagen. The amount of collagen reaches a maximum within 2 to 3 weeks after injury. As the collagen content of a wound increases, the number of fibroblasts and the rate of collagen synthesis decrease, marking the end of the repair stage. The fibroblastic interval of healing lasts 2 to 4 weeks, depending on the nature of the wound. Fibroblast migration and proliferation, collagen production, and capillary ingrowth are delayed if macrophages are absent.

Capillaries invade wounds behind migrating fibroblasts by the process of angiogenesis. Angiogenesis is complex, relying on interaction of extracellular matrix with cytokines that stimulate migration and proliferation of endothelial cells. Stimulus for angiogenesis probably includes macrophage production of mitogenic and chemotactic factors for endothelial cells, and low oxygen tension and increased lactic acid, which affect cytokine production. Basic fibroblast growth factor and vascular endothelial growth factor are specific angiogenic factors. Capillary buds originate from existing blood vessels with columns of capillary endothelial cells migrating toward the site of injury and uniting with other capillary buds or disrupted vessels. New capillaries increase oxygen tension in wounds, augmenting fibroplasia. Mitotic activity in adjacent mesenchymal cells increases as blood begins to flow in new capillaries. Lymphatic channels develop similar to capillary buds but more slowly. Lymphatic drainage of wounds is poor during early healing. The combination of new capillaries, fibroblasts, and fibrous tissue forms bright red, fleshy granulation tissue 3 to 5 days after injury.

Granulation tissue is formed at each wound edge at a rate of 0.4 to 1 mm/day. Unhealthy granulation tissue is white and has a high fibrous tissue content with few capillaries (Figs. 15.1 and 15.2). Granulation tissue fills defects and protects wounds. It provides a barrier to infection, a surface for epithelial migration, and a source of special fibroblasts (i.e., myofibroblasts), which are important in wound contraction. Myofibroblasts are believed to contain proteins (actin and myosin) that contribute to wound contraction. Myofibroblasts are not found in normal tissue, incised and coapted wounds, or tissue surrounding a contracting wound.

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FIG. 15.1 An open wound with unhealthy granulation over the right stifle in a 4-year-old Walker Hound.



FIG. 15.2 An open wound on the dorsum of a 9-year-old shih tzu demonstrating healthy granulation tissue and initial stages of epithelialization.

Epithelium is an important barrier to external infection and internal fluid loss. Epithelial repair involves mobilization, migration, proliferation, and differentiation of epithelial cells. *Epithelialization* begins almost immediately (24–48 hours) in sutured wounds with good edge-to-edge apposition because there is no defect for granulation tissue to fill (see Fig. 15.2). Epithelialization begins in open wounds when an adequate granulation bed has formed (usually in 4–5 days). In partial-thickness skin wounds, epidermal migration over the wound surface begins almost immediately from both the wound margins and epidermal appendages, such as hair follicles and sweat glands. Epidermal cells at the margin of the wound undergo a phenotypic alteration that includes retraction on intracellular monofilaments, formation of peripheral cytoplasmic actin filaments, and temporary dissolution of the desmosomes and hemidesmosomes, which release keratinocytes to migrate beneath the eschar at the junction between any remaining necrotic tissue and extracellular matrix of the viable connective tissue. The epidermal cell path of migration is determined by integrins expressed on the membranes of migrating epidermal cells. Chalone, a water-soluble glycoprotein found in the epidermis, inhibits epithelial mitosis in normal tissue but is diminished in wounds, which allows epithelial cells along wound margins to divide and migrate across the granulation tissue. Other growth factors secreted by platelets, macrophages, and fibroblasts may also be involved. Increased basal cell mitotic activity occurs as early as 24 to 48 hours after wounding.

Epithelial migration is random but guided by collagen fibers. Migrating epithelial cells enlarge, flatten, and mobilize, losing their attachments to the basement membrane and other epithelial cells. Basal cells at wound edges develop microvilli and extend broad, thin pseudopodia over the exposed surface of collagen bundles. They develop intracytoplasmic microfilaments and selectively fix antiactin and antimyosin antibodies. Epithelial cells in the layers behind these altered cells migrate over them until they contact the wound surface. Cells continue to slide forward until the wound surface is covered. The migrating cells move under scabs and produce collagenase, which dissolves the base of the scab so it can be shed. Contact on all sides with other epithelial cells inhibits further cell migration (contact inhibition). Initially, new epithelium is only one cell layer thick and fragile, but it gradually thickens as additional cell layers form. After a basement membrane has been established, epithelial cells become plump, develop mitoses, and proliferate, restoring the normal, stratified, squamous epithelium architecture. Some hair follicles and sweat glands may regenerate, depending on the depth of skin damage. Epithelial migration also occurs along suture tracts, which may lead to a foreign body reaction, sterile abscess, scarring, or all of these. Epithelialization of suture tracts can be minimized by early removal of sutures. New epithelium is usually visible 4 to 5 days after injury. Epithelialization occurs faster in a moist environment than in a dry one. It will not occur over nonviable tissue. Epithelial migration is energy dependent and related to oxygen tension. Anoxia prevents epithelial migration and mitosis, whereas hyperbaric oxygen therapy (HBOT) may enhance migration.

Wound contraction reduces the size of wounds subsequent to fibroblasts, reorganizing collagen in granulation tissue and myofibroblast contraction at the wound edge. Contraction occurs simultaneously with granulation and epithelialization but is independent of epithelialization. Wound contraction involves a complex interaction of cells, extracellular matrix, and cytokines. Significant fibroblastic invasion into the wound is necessary for contraction to begin. Centripetal, full-thickness skin edges are pulled inward by contraction, and wounds may be noticeably smaller by 5 to 9 days after injury. The surrounding skin stretches (intussusceptive growth) during wound contraction, and the wound takes on a stellate appearance. Contraction progresses at a rate of approximately 0.6 to 0.8 mm/day. Wound contraction stops when wound edges meet, when tension is excessive, or when myofibroblasts are inadequate. Wound contraction is limited if skin around wounds is fixed, inelastic, or under tension, and it is inhibited if myofibroblast development or function is impaired. Contraction can also be impaired by antiinflammatory steroids, antimicrotubular drugs, and local application of smooth muscle relaxants. If wound contraction stops before granulation tissue is covered, epithelialization may continue and cover the wound.

Maturation Phase

Wound strength increases to its maximum level because of changes in the scar during the maturation phase of wound healing. Wound maturation begins once collagen has been adequately deposited

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in wounds (17–20 days after injury) and may continue for years. The cellularity of granulation tissue is reduced as cells die. There is also a reduction in collagen content of the extracellular matrix. Collagen fibers remodel with alteration of their orientation and increased crosslinking, which improves wound strength. Fibers orient along lines of stress. Functionally oriented fibers become thicker. Type III collagen gradually decreases, and type I collagen increases. Nonfunctionally oriented collagen fibers are degraded by proteolytic enzymes (matrix metalloproteinases) secreted by macrophages, epithelial cells, endothelial cells, and fibroblasts within the extracellular matrix. The most rapid gain in wound strength occurs between 7 and 14 days after injury as collagen rapidly accumulates in the wound. Wounds gain only approximately 20% of their final strength in the first 3 weeks after injury. Slower increase in wound strength then occurs, but normal tissue strength is never regained in wounds; only 80% of original strength may be regained. As the number of capillaries in fibrous tissue declines, the scar becomes paler. Scars also become less cellular, flatten, and soften during maturation. Collagen synthesis and lysis occur at the same rate in maturing scars.

Moist Wound Healing

A moist wound environment allows optimal healing. Wound fluid is allowed to remain on the wound, keeping it moist. In a moist environment, debridement is hastened and selective, granulation tissue formation is promoted, and epithelialization is faster. Allowing wound fluid to remain in contact with wounds fosters autolytic debridement by endogenous enzymes that break down necrotic, but not healthy, tissue. Autolytic debridement occurs within 72 to 96 hours under an occlusive bandage. White blood cell phagocytosis decreases bacterial load and removes necrotic debris. White blood cells migrate more readily in a moist environment. Wound fluid also contains cytokines and growth factors that stimulate granulation tissue, angiogenesis, and reepithelialization. Chemotactic factors in wound fluid attract neutrophils and macrophages that secrete additional enzymes, cytokines, and growth factors. Moist wounds limit infections because more white blood cells are found in the wound, and there is improved phagocytosis and a lower pH. Scabs do not form with moist wound healing; therefore white blood cells are not trapped in the scab, and topical medications better penetrate the wound. If the animal is receiving systemic antibiotics, wound fluids may contain antibiotics, which help prevent or control infection. Low oxygen tension under an occlusive bandage stimulates macrophage activity, fibroblast proliferation, and capillary ingrowth. The rate of epithelialization is twice as fast for wounds kept moist with occlusive dressings than for air-exposed wounds. Epithelial cells travel faster and have a shorter distance for epithelialization to occur in moist environments; in air-exposed wounds, migrating epidermal cells must travel beneath a crusted scab and devitalized dermis to reach their destination. Hydrophilic, occlusive, or semiocclusive bandages help keep a wound warm and moist. Increased warmth enhances enzymatic activity. A moist wound is less painful and pruritic, tissues do not desiccate, and scar formation is less. Potential disadvantages of moist wound healing include bacterial colonization (not infection) of the wound surface, folliculitis, and maceration of the wound border.

Host Factors Affecting Wound Healing

Old animals tend to heal slowly, often because of concurrent disease or debilitation. Malnourished animals and those with serum protein concentrations below 1.5 to 2 g/dL may have delayed wound healing and diminished wound strength. Hepatic disease may cause clotting factor deficiencies. Hyperadrenocorticism delays wound healing because of excess circulating glucocorticoids. Animals with diabetes mellitus have delayed wound healing and a predisposition to wound infections. Uremia occurring within 5 days of injury impairs healing by altering enzyme systems, biochemical pathways, and cellular metabolism. Obesity is associated with a higher incidence of postoperative wound infections in human beings. The risk of postoperative wound infection in dogs and cats also increases as the duration of anesthesia increases.

Cutaneous wounds are slower to heal in cats than dogs. Sutured wounds in cats are only half as strong as similar wounds in dogs after 7 days of healing. Cat wounds that are allowed to heal by secondary intention heal slower, produce less granulation tissue (that is more peripherally located), and heal more by contraction of wound edges than dogs.

Wound Characteristics Affecting Wound Healing

Intact surfaces, such as periosteum, fascia, tendon, and nerve sheath, do not support granulation tissue; therefore these surfaces heal slowly when exposed. Fenestration or drilling holes in exposed cortical bone may improve granulation by releasing osteogenic or other factors. Foreign material in wounds (e.g., dirt, debris, sutures, surgical implants) can cause intense inflammatory reactions interfering with normal wound healing. Release of enzymes designed to degrade foreign bodies destroys wound matrix, prolongs inflammation, and delays the fibroblastic phase of tissue repair. Exposure of the wound to antiseptics delays healing and may predispose to infection. Warmth (30°C [86°F]) allows wounds to heal more quickly and with greater tensile strength than if they are at room temperature. A moist wound promotes recruitment of vital host defenses and cells, encouraging wound healing. Bandages help keep wounds warm and moist. Wounds (incisions) created with sharp surgical instruments heal faster and with less necrosis at the wound margin than those made with scissors, electrosurgery, or lasers. Wound infection interferes with the repair phase of healing. Contaminated tissues become infected if invasive bacteria multiply to 10^5 colony-forming units (CFU) per gram of tissue. Development of wound infection depends on the degree of tissue trauma, amount of foreign material present, delay between injury and treatment, and effectiveness of host defenses. Bacterial toxins and associated inflammatory infiltrates cause cell necrosis and vascular thrombosis. Wound exudates can separate tissue layers and further delay healing. Inflammation caused by infection further compromises vasculature, causing additional necrosis.

Healing depends on blood supply, which delivers oxygen and metabolic substrates to cells. Impairment of blood supply by trauma, tight bandages, or wound movement slows healing. Macrophages resist hypoxia, but epithelialization and fibroblastic protein synthesis are oxygen dependent. Collagen synthesis requires 20 mm Hg partial pressure of oxygen (pO_2). HBOT increases tissue oxygen and produces more rapid gains in wound strength. Accumulation of fluid in dead space delays healing because the hypoxic fluid environment of a seroma inhibits migration of reparative cells into wounds. Fluid mechanically prevents adhesion of flaps or grafts to the wound bed.

Recruitment, proliferation, and cellular function in wound healing are controlled by growth factors; proteins synthesized and released by cells are involved in wound healing. Numerous

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growth factors have been identified, including platelet-derived growth factor, epidermal growth factor, fibroblast growth factor, and type-transforming growth factor. Platelet-derived growth factors are found in granules, whereas macrophages must be stimulated to synthesize and release growth factors.

Fibronectins are glycoproteins critical to wound healing. They stimulate cell attachment and migration and are found in soluble form in plasma and in insoluble form in connective tissue matrix. Macrophages, endothelium, fibroblasts, and epithelium synthesize and release fibronectin. Fibronectin in the coagulum probably assists initial migration of cellular elements (macrophages and epithelium) into wounds. It binds bacterial cell wall components, collagen, actin, thrombospondin, heparan sulfate, hyaluronic acid, fibrin, cell surface receptors, and other fibronectin molecules. Fibronectin may also be important in providing an early wound-healing matrix and in interlinking cellular and matrix components during healing. Fibronectin in wounds declines as healing nears completion. Proteoglycans are also important in all phases of wound healing. The matrix during cell migration contains elevated concentrations of nonsulfated glycosaminoglycans (i.e., hyaluronate). As wound maturation progresses, more sulfated glycosaminoglycans (i.e., chondroitin sulfate and heparan sulfate) appear.

External Factors Affecting Wound Healing

Radiation therapy (see p. 242) and some drugs delay wound healing. Glucocorticoids depress all phases of wound healing and increase chances of infection. Vitamin A and anabolic steroids may reverse the effects of glucocorticoids on wound healing. Antiinflammatory drugs suppress inflammation but have little effect on wound strength. Aspirin may delay blood clotting. Most chemotherapeutic drugs (e.g., cyclophosphamide, methotrexate, and doxorubicin) inhibit wound healing. Radiation therapy can profoundly inhibit wound healing, depending on dose and time of exposure relative to the time of injury. It reduces the quantity of blood vessels, affects collagen maturation, and causes increased dermal fibrosis. Therefore chemotherapeutic drugs and radiation therapy should be avoided for 2 weeks after surgery. Vitamin A, vitamin E, and aloe vera may promote healing in irradiated wounds. Exposure to pico-tesla electromagnetic field treatment improves strength of sutured wounds and speeds contraction of open wounds in rats. HBOT increases dissolved oxygen in plasma, which stimulates growth of new capillaries; therefore it may be useful for treatment of ischemic wounds. Ultrasonography and phototherapy (low-powered laser) shorten the inflammatory phase of healing and enhance the release of factors that stimulate the proliferative stage of repair. Use of controlled subatmospheric pressure dressings helps remove interstitial fluid, which allows tissue decompression, helps remove tissue debris, and promotes wound healing (see p. 192).

MANAGEMENT OF OPEN OR SUPERFICIAL WOUNDS

Wounds should be covered with a clean, dry bandage immediately after injury or when the animal is brought for treatment to prevent further contamination and hemorrhage (Box 15.2). Life-threatening injuries should be treated and the animal's condition stabilized before further wound management is undertaken. When appropriate during stabilization, bandages should be removed and the wound assessed and classified as either contaminated or infected and as an abrasion, laceration, avulsion, puncture, crush, or burn wound. The "golden period" is the first 6 to 8 hours between wound contamination at injury and bacterial multiplication to greater than 10^5 CFU per gram of tissue. A wound is classified as infected rather than contaminated when bacterial numbers exceed 10^5 CFU per gram of tissue. Infected wounds are often dirty and covered with a thick, viscous exudate.

BOX 15.2 Fundamentals of Wound Management

- Temporarily cover the wound to prevent further trauma and contamination.
- Assess the traumatized animal and stabilize its condition.
- Clip and aseptically prepare the area around the wound.
- Culture the wound.
- Debride dead tissue and remove foreign debris from the wound.
- Lavage the wound thoroughly.
- Provide wound drainage.
- Promote healing by stabilizing and protecting the cleaned wound.
- Perform appropriate wound closure.

Abrasions are superficial and involve destruction of varying depths of skin by friction from blunt trauma or shearing forces. Abrasions are sensitive to pressure or touch and bleed minimally. A *laceration* is created by tearing, which damages skin and underlying tissue. Lacerations may be superficial or deep and have irregular edges. *Avulsion wounds* are characterized by the tearing of tissues from their attachments and the creation of skin flaps. Avulsion injuries on limbs with extensive skin loss are called *degloving injuries*. A *penetrating* or *puncture wound* is created by a missile or sharp object, such as a knife, pellet, or tooth that damages tissue. Wound depth and width vary depending on the velocity and mass of the object creating the wound. The extent of tissue damage is directly proportional to missile velocity. Pieces of hair, skin, and debris can be embedded in wounds. *Crush injuries* can be a combination of other types of wounds with extensive damage and contusions to skin and deeper tissue. *Burns* may be partial- or full-thickness skin injuries caused by heat or chemicals (see p. 237).

Wounds less than 6 to 8 hours old with minimal trauma and minimal contamination can be treated by lavage, debridement, and primary closure. In general, the sooner treatment begins, the better the prognosis. Penetrating wounds should not be primarily apposed without surgical exploration (see p. 243). Severely traumatized and contaminated wounds, wounds older than 6 to 8 hours, or infected wounds should be treated as open wounds to allow debridement and reduction of bacterial numbers. Most wounds are surgically apposed after infection has been controlled; however, some wounds heal by contraction and epithelialization (healing by secondary intention).

NOTE Penetrating wounds, particularly bite wounds, should not be closed primarily without surgical exploration and debridement.

Often sedation or anesthesia is required for initial wound inspection and care. The objective of open wound care is to convert the open, contaminated wound into a surgically clean wound that can be closed. Aseptic technique, gentle tissue handling, and hemostasis are essential. Severely contaminated or infected wounds should be cultured after initial inspection. The area

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surrounding the wound should be widely clipped and prepped. The wound may be protected from clipped hair and detergents by applying a sterile, water-soluble lubricant (K-Y Jelly) or by placing saline-soaked sponges in the wound and covering with a sterile pad or towel. As an alternative, the wound may be temporarily closed with sutures, towel clamps, staples, or Michel clips. Hair may be clipped from the wound margin with scissors dipped in mineral oil to prevent hair from falling into the wound. Povidone-iodine or chlorhexidine gluconate skin scrubs are used to prepare clipped skin. The detergents in antiseptic scrubs cause irritation, toxicity, and pain in exposed tissue and may potentiate wound infection. Alcohol is very damaging to exposed tissue and should be used only on intact skin.

Initial wound management begins with removal of gross contaminants and copious lavage using a warm, balanced electrolyte solution, sterile saline, or tap water (500–1000 mL) (Table 15.1). Sterile isotonic saline or a balanced electrolyte solution (lactated Ringer's solution) are the preferred lavage solutions. No one type of fluid has been shown to be preferred in preventing wound infection. Tap water is effective and less detrimental than distilled or sterile water, although it causes some mild hypotonic tissue damage (cellular and mitochondrial swelling). Wound lavage reduces bacterial numbers mechanically by loosening and flushing away bacteria and associated necrotic debris. Lavage may be facilitated by the use of noncytotoxic wound cleansers (e.g., Vetericyn Plus). In general, these cleansers are applied to loosen debris and soften necrotic tissue during bandage changes; they act as a surfactant, disrupting the ionic bonding of particles and organisms to the wound and allowing them to be easily rinsed off with saline or balanced electrolyte solutions. Lavage following application of these cleansers, however, is not necessary. Antibiotics or antiseptics (e.g., chlorhexidine or povidone-iodine; see p. 188) in the lavage solution reduce bacterial numbers; however, these agents may damage tissue. Antiseptics have little effect on bacteria in established infections. However, tris–ethylenediaminetetraacetic acid (tris-EDTA) may be added to lavage solutions to help lyse gram-negative bacteria, such as *Pseudomonas aeruginosa, Escherichia coli*, and *Proteus vulgaris*, and may have synergistic effects with certain systemic antibiotics. Lavaging is preferred to scrubbing the wound with sponges. Sponges inflict tissue damage that impairs the wound's ability to resist infection and allows residual bacteria to elicit an inflammatory response.

Bacteria are effectively removed from the wound surface by high-pressure lavage. Traditionally, a 35- or 60-mL syringe and an 18-gauge needle have been thought to generate approximately 7 to 8 psi of pressure; however, it has been shown that it generates pressures substantially higher than this (18.4 ± 9.8 psi).[1] The most consistent delivery method to generate 7 to 8 psi is a 1-L bag of fluid within a cuff pressurized to 300 mm Hg (Fig. 15.3). Higher pressure (70 psi), generated by pulsatile lavage instruments (i.e., Water Pik, Surgilav, or Pulsavac debridement system), is more effective in reducing bacterial numbers and removing foreign debris and necrotic tissue, but it may drive bacteria and debris into loose tissue planes, damage underlying tissue, and reduce resistance to infection. Bulb syringes or fluid bottles with

TABLE 15.1 Suggested Wound Cleansers

Cleanser	Advantages	Disadvantages
Commercial cleansers (e.g., Vetericyn Plus)	Surfactant breaks the bonds between foreign bodies and the wound surface Convenient	Most ionic surfactants and many nonionic surfactants have been shown to be toxic to cells, delay wound healing, and inhibit the wound's defense mechanisms Expense
Tap water	Availability Low cost Ease of application	Hypotonic Cytotoxic trace elements Not antimicrobial
Balanced electrolyte solution: LRS, Normosol	Isotonic Least cytotoxic	Not antimicrobial
Normal (0.9%) solution	Isotonic	Slightly more acidic than LRS Not antimicrobial
0.05% Chlorhexidine (1 part stock solution to 40 parts sterile water or LRS) or (~25 mL stock solution per liter)	Wide antimicrobial spectrum Good residual activity Not inactivated by organic matter	Precipitates in electrolyte solutions More concentrated solutions are cytotoxic and may slow granulation tissue formation *Proteus, Pseudomonas,* and *Candida* are resistant Corneal toxicity
0.05% Chlorhexidine with Tris-EDTA	Makes bacteria more susceptible to destruction by lysozymes, antiseptics, and antibiotics. Rapidly lyses *P. aeruginosa, E. coli,* and *Proteus vulgaris* Increases antimicrobial effectiveness approximately 1000-fold	Precipitates in electrolyte solutions More concentrated solutions are cytotoxic and may slow granulation tissue formation Corneal toxicity
0.1% povidone-iodine (1 part stock to 100 parts LRS) or (~10 mL stock to 100 mL LRS)	Wide antimicrobial spectrum	Inactivated by organic matter Limited residual activity Cytotoxic at concentrations >1% Contact hypersensitivity Thyroid disorders if absorbed

EDTA, Ethylenediaminetetraacetic acid; *LRS,* lactated Ringer's solution.

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FIG. 15.3 Wound lavage of the dog from Fig. 15.1 using 1 L of 0.9% saline in pressure bag, maintained at 300 mm Hg with extension tubing and an 18-gauge needle.

holes made in the cap do not generate enough pressure to remove bacteria and debris adequately.

Debridement

Healing is delayed if necrotic tissue is left in the wound. Devitalized tissue is removed from the wound by debridement. Debridement involves removal of dead or damaged tissue, foreign bodies, and microorganisms that compromise local defense mechanisms and delay healing. The goal of debridement is to obtain fresh clean wound margins and wound bed for primary or delayed closure. Devitalized tissue is removed by surgical excision, autolytic mechanisms, enzymes, wet-dry bandages (see p. 197), or biosurgical methods. The extent of devitalized tissue is usually obvious within 48 hours of injury.

Surgical Debridement

Devitalized tissue should be surgically excised in layers beginning at the surface and progressing to the depths of the wound. This can be done by sharp dissection, electrosurgery, or laser. Bones, tendons, nerves, and vessels must be preserved, but bone sequestra should be removed because they may prevent complete granulation of the wound (especially with metacarpal and metatarsal degloving injuries) and predispose the wound to infection. Muscle should be debrided until it bleeds and contracts with appropriate stimuli. Extensive debridement of subcutaneous tissue should be avoided as it may delay wound healing, particularly in cats, and may place wounds at a higher risk for infection. Contaminated fat should be liberally excised because it is easily devascularized and harbors bacteria, but cutaneous vessels must be spared to maintain the viability of overlying skin.

As an alternative, the entire wound can be excised en bloc if sufficient healthy tissue surrounds the wound and vital structures can be preserved. The danger of surgical debridement is removal of an excessive amount of possibly viable tissue. With penetrating wounds or punctures, it may be necessary to enlarge the wound to assess the extent of injury and allow debridement. Electrosurgery or a carbon dioxide laser can be as effective as sharp surgical debridement of devitalized tissue. They have the advantage of providing simultaneous hemostasis, which helps prevent debridement of normal tissue. Low-level laser therapy (LLLT) has been advocated to stimulate wound healing in chronic wounds by shortening the inflammatory phase and enhancing the release of factors that stimulate the proliferative stage of repair. Increased collagen deposition and endothelial cell, fibroblast, and myofibroblast proliferation are the most significant effects.

Surgical debridement of obviously devitalized tissue is often combined with autolytic debridement (discussed later) to remove surface contaminants and tissue of questionable viability. After surgical debridement, wounds are often treated as open wounds with hydrophilic dressings and bandages. Provision of adequate wound drainage and a viable vascular bed is important to wound healing. The wound should be closed when it appears healthy or when a bed of healthy granulation tissue has formed, unless wound closure by contraction and epithelialization is anticipated.

Autolytic Debridement

Autolytic debridement is accomplished through creation of a moist wound environment to allow endogenous enzymes to dissolve nonviable tissue. It is often preferred over surgical or bandage debridement in wounds with questionable tissue viability, as it is highly selective for devitalized tissue and markedly less painful than other methods of debridement; however, it is a much slower process. Autolytic debridement is accomplished with hydrophilic, occlusive, or semiocclusive bandages (see pp. 195–196), which allow wound fluid to remain in contact with nonviable tissue.

Bandage (Mechanical) Debridement

Dressings that are allowed to dry on the wound, such as wet-to-dry bandages or dry-to-dry bandages, adhere to the wound surface and pull the debris and strip the superficial layers off the wound bed when removed. In addition to mechanical debridement, wet-to-dry wound dressings provide adequate wound protection and coverage, maintain a moist wound environment, and absorb moderate amounts of wound exudates. These dressings are most effective in early stages of wound healing or in the management of wound infection. However, debridement is painful and nonselective, as healthy or healing tissue may be traumatized. Because of these disadvantages, as well as the availability of other methods of wound debridement, these bandages are not recommended by some. However, wet-to-dry bandages are a very economic and effective dressing in the first few days of open wound care.

Enzymatic Debridement

Enzymatic debriding agents are used as an adjunct to wound lavage and surgical debridement. They are beneficial in patients that are poor anesthetic risks or when surgical debridement may damage healthy tissue necessary for reconstruction. Enzymatic agents break down necrotic tissue and liquefy coagulum and bacterial biofilm, allowing better antibiotic contact with wounds and enhanced exposure for development of cellular and humoral immunity; they do not damage living tissue if used properly. Available enzymes do not digest burned skin, necrotic bone, and connective tissue. Enzymes must remain in contact with the wound for an adequate time to produce the desired effect; therefore these products are slow to work and can be expensive. Local tissue irritation may also occur.

Collagenase-based products (e.g., Santyl) are effective for enzymatic debridement and are used in human wounds, specifically decubital ulcers, pressure sores, and burn wounds. More

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familiar products to veterinary surgeons, such as Granulex and Accuzyme, among others, are no longer available due to safety concerns.

Biosurgical Debridement

Maggot therapy using greenbottle fly larvae (*Lucilia sericata*) debrides wounds as the maggots secrete proteolytic digestive enzymes into the wound. Sterile medicinal maggots are bred specifically for biosurgery. A single maggot may consume up to 75 mg of necrotic tissue each day. They require an optimal temperature, an oxygen supply, and a moist wound. Maggot therapy is best suited to necrotic, infected, or chronic nonhealing wounds. The maggots remove necrotic tissue, disinfect the wound, and promote granulation tissue formation. Medicinal maggots are applied to the wound at a density of five to eight per square centimeter. A hole is cut into a self-adhesive hydrocolloid dressing that matches the wound dimensions. This dressing is applied to the wound to prevent the maggots from crawling onto intact skin and to absorb wound secretions. The dressing is covered to trap the maggots in the wound, changing absorbent layers as necessary. Maggots are usually applied for two 48-hour cycles each week.

ANTIBIOTICS

Selective use of antibiotics may help prevent or control integument infections after injury or surgery. Minimally or moderately contaminated wounds less than 6 to 8 hours old may be cleaned and closed or treated without antibiotics. Severely contaminated, crushed, or infected wounds, or wounds older than 6 to 8 hours, typically benefit from antibiotic therapy. Contaminated wounds and those with established infection should be cultured before antibiotics are given, and antibiotic selection should ultimately be based on culture and susceptibility testing. Ideally, quantitative bacterial counts should be performed before grafts or flaps are placed over granulating wounds. Reconstruction should be delayed if bacterial counts are greater than 10^5 CFU per gram of tissue.

Systemic antibiotics should be given if there is a high risk of bacteremia or disseminated infection. A broad-spectrum antibiotic should be administered while awaiting culture and antimicrobial susceptibility results. Antibiotic blood levels should be present at the time of surgery when antibiotics are used prophylactically for clean or clean contaminated procedures. Prophylactic antibiotics are optimally given intravenously 30 minutes prior to the surgical incision being made (see Chapter 9). Contamination occurring during surgery is usually limited to the patient's skin flora; therefore drugs effective against gram-positive skin flora, especially *Staphylococci* spp. (see p. 80), should be selected (e.g., 22 mg/kg cefazolin given intravenously).

TOPICAL WOUND MEDICATIONS

Topical Antimicrobials and Antibiotics

Antimicrobial agents and antibiotics eliminate or reduce the number of microorganisms that destroy tissue in a wound. Topical rather than systemic antibiotics are preferred for open wounds. Mildly or moderately contaminated wounds do not benefit from combined topical and systemic antibiotic therapy; however, combined therapy is advantageous in heavily contaminated wounds. Antibiotics applied within 1 to 3 hours of contamination often prevent infection. Benefits of topical drugs should outweigh their cytotoxic effects. Antibiotics used effectively as topical ointments or added to lavage solutions are penicillin, ampicillin, carbenicillin, tetracycline, kanamycin, neomycin, bacitracin, polymyxin, and cephalosporins. Once infection is established, topical and systemic antibiotics have no beneficial effect in preventing suppuration of wounds undergoing closure. Wound coagulum prevents topical antibiotics from reaching effective levels in tissues deep in the wound and also prevents systemic antibiotics from reaching superficial bacteria. These wounds must be debrided to allow antimicrobial access to bacteria.

Advantages of topical antibiotics over antiseptics in wound management include selective bacterial toxicity, efficacy in the presence of organic material, and combined efficacy with systemic antibiotics. Disadvantages include expense, narrower antimicrobial spectrum, potential for bacterial resistance, creation of superinfections, systemic or local toxicity, hypersensitivity, and increased nosocomial infections. Antibiotic solutions are preferable to ointments and powders. Ointments liberate antibiotics slowly and may be occlusive, promoting growth of anaerobic bacteria. Powders act as foreign bodies and should not be used.

Triple Antibiotic Ointment

Triple antibiotic ointment (bacitracin, neomycin, polymyxin) is effective against a broad spectrum of pathogenic bacteria commonly infecting superficial skin wounds. However, its efficacy against pseudomonads is poor. Zinc bacitracin is responsible for enhancing reepithelialization of wounds but can retard wound contraction. Because these drugs are poorly absorbed, systemic toxicosis (nephrotoxicity, ototoxicity, neurotoxicity) is rare. The ointment is more effective for preventing infections and treating mildly infected wounds.

Silver Sulfadiazine

Silver sulfadiazine in a 1% water-miscible cream or ointment (Thermazene, Silvadene) is effective against most gram-positive and gram-negative bacteria and most fungi. It also serves as an antimicrobial barrier, can penetrate necrotic tissue, and enhances wound epithelialization. It is thought to be the drug of choice to treat burn wounds; however, in one study of partial-thickness burn wounds in people, honey dressings were found to be superior to silver sulfadiazine ointment. In vitro toxicity to human keratinocytes and fibroblasts and inhibition of polymorphonuclear cells and lymphocytes has been shown. These wound-retardant effects of silver sulfadiazine are reversed when it is combined with aloe vera. Silver-impregnated dressings are also available and preferred by some due to less discomfort and less frequent bandage changes (see p. 200). Ointments remain effective for up to 3 days, whereas dressings may be left in place for 7 days. Only a small amount of silver is released slowly from its molecular lattice over a sustained period, reducing cytotoxic effects of ionic silver and making it nonstaining, nonirritating, and nonsensitizing while maintaining its antimicrobial effects. These products are also hydrophilic, helping to maintain a moist wound environment and absorbing exudates.

Nitrofurazone

Nitrofurazone (Furacin) has broad-spectrum antibacterial and hydrophilic properties. It has little effect against *Pseudomonas* spp. Its polyethylene base gives it hydrophilic properties, enabling it to draw body fluid from wound tissue, which helps dilute tenacious exudates so they can be absorbed into bandages. Nitrofurazone delays wound epithelialization. It loses some of its antibacterial effects in the presence of organic matter.

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Gentamicin Sulfate

Gentamicin sulfate is available as a 1% ointment or powder (Garamycin), but solutions are preferred. Products with an oil-in-water cream base slow wound contraction and epithelialization. It is especially effective in controlling gram-negative bacterial growth (*Pseudomonas* spp., *Escherichia coli*, *Proteus* spp.). It is often used before and after grafting and for wounds that have not responded to triple antibiotic ointment. However, gentamicin in an isotonic solution does not inhibit contraction, and it promotes epithelialization.

Cefazolin

Cefazolin is an effective antimicrobial against gram-positive and some gram-negative organisms. Topical cefazolin (the combined systemic and topical dose should not exceed 22 mg/kg) provides high levels of antibiotic in wound fluid. The drug's minimum inhibitory concentration is prolonged in wounds when it is applied topically compared with systemic administration. Topically administered cefazolin is 95% bioavailable and rapidly absorbed; therefore systemic levels equal wound fluid levels within 1 hour.

Mafenide

Mafenide (hydrochloride or acetate) is a topical sulfa compound available as an aqueous spray most commonly used for burn wounds in humans. It has a broad spectrum against many gram-positive and gram-negative bacteria, including *Pseudomonas* spp., *Clostridium* spp., and methicillin-resistant *Staphylococcus aureus*; it is particularly useful on severely contaminated wounds.

Topical Wound-Healing Enhancers

Many topical agents have been used in the management of open wounds, some with documented benefit and others without. Unfortunately, there is a lack of well-controlled prospective studies in the veterinary literature to determine ideal agents to promote wound healing in dogs and cats.

Aloe Vera

Aloe vera gel is extracted from the aloe vera leaf and contains 75 potentially active constituents. Aloe vera has been used on burns because of its antibacterial activity against *Pseudomonas aeruginosa.* It also inhibits fungal growth. The antiprostaglandin and antithromboxane properties of aloe vera medications are beneficial in maintaining vascular patency and thus helping avert dermal ischemia. Aloe vera medications may also stimulate fibroblastic replication. Aloe vera has the ability to penetrate and anesthetize tissue. Acemannan, a component of aloe vera extract gel, promotes wound healing (see later discussion). It is also found in other preparations. Allantoin, another component of aloe vera extract gel, stimulates tissue repair in suppurating wounds and resistant ulcers by promoting epithelial growth. Use on full-thickness wounds is discouraged because of its antiinflammatory effects. Aloe vera counteracts the inhibitory effects of silver sulfadiazine when the two are combined.

Acemannan

Acemannan is available as a topical wound hydrogel or a freeze-dried gel form. It is indicated for managing superficial and deep partial-thickness burns, lacerations, dermal ulcers, abrasions, and nonhealing wounds. Acemannan is a β-(1,4) acetylated mannan derived from the aloe vera plant that enhances early stages of healing. Acemannan stimulates macrophages to secrete interleukin-1 (IL-1) and tumor necrosis factor alpha (TNF-α), which enhance fibroblast proliferation, neovascularization, epidermal growth and motility, and collagen deposition to form granulation tissue. Acemannan may also bind growth factors, prolonging their stimulating effect on formation of granulation tissue. The freeze-dried form enhances healing over exposed bone and has hydrophilic properties, which help cleanse the wound and reduce wound edema. The most effective time to begin topical application is in the early inflammatory stage of healing with daily application under a bandage, continuing into the repair stage of healing. The greatest effects are seen in the first 7 days of application. Excess granulation tissue can occur, especially with the freeze-dried form, which inhibits wound contraction.

Tripeptide-Copper Complex

Glycyl-L-histidyl-lysine tripeptide-copper complex (Iamin) stimulates wound healing and is a chemoattractant for mast cells, monocytes, and macrophages, which stimulate debridement, angiogenesis, collagen synthesis, and epithelialization. Enzymes involved in collagen crosslinking need copper. The best time to begin tripeptide-copper complex application is the late inflammatory and early repair phases, with treatment continuing into the later repair phase. It has been effective in accelerating wound healing in chronic, ischemic open wounds. Its greatest effect is in the first 7 days of its use. Exuberant granulation tissue may be a problem with this agent.

Maltodextrin

Maltodextrin (Multidex) is a D-glucose polysaccharide; it is available in a hydrophilic powder or gel form containing 1% ascorbic acid for use on contaminated and infected wounds as a wound healing stimulant. It is reported to stimulate healing by supplying glucose for cell metabolism via hydrolysis of its polysaccharide component. Its hydrophilic property draws fluid through the tissue, keeping it moist. Maltodextrin is chemotactic and pulls neutrophils, lymphocytes, and macrophages into the wound. Maltodextrin also has antibacterial and bacteriostatic properties. It reduces odor, exudates, swelling, and infection, and it may enhance early granulation tissue formation and epithelialization. After debridement and lavage, a 5- to 10-mm layer of maltodextrin is applied to the wound and covered with a bandage from the early inflammatory stage into the repair stage of healing. Daily bandage changes, lavage, and reapplication are recommended.

Honey

Honey is an old agent that has seen renewed interest. Proposed benefits include enhancing wound debridement, reducing edema and inflammation, promoting granulation tissue formation and epithelialization, and improving wound nutrition (Box 15.3). It

BOX 15.3 Benefits of Honey in Wound Healing

- Reduces edema
- Reduces inflammation
- Accelerates wound debridement
- Promotes granulation tissue formation
- Accelerates epithelization
- Antimicrobial

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has a wide antibacterial effect through its enzymatic production of hydrogen peroxide from glucose, hypertonicity, low pH, inhibin content, and other unidentified components. Honey increases collagen content, accelerates collagen maturation resulting from crosslinking, and maintains optimal pH conditions for fibroblast activity. Honey contains a wide range of amino acids, vitamins, and trace elements, in addition to readily assimilable sugars that stimulate tissue growth. Honey should be used early in the course of wound healing and discontinued once a healthy granulation bed is present. Honey can also be used for treating partial-thickness burns and may have greater wound healing properties than silver-based preparations.

Specific preparations of unpasteurized medicinal honey are recommended for wound care (e.g., Medihoney, Therahoney, Manuka Honey AD). Honey is applied by impregnating sterile gauze, which is then positioned on the wound and covered with a thick, absorbent bandage. Alternatively, it can be applied as a gel or paste and covered with a nonadherent dressing. The wound is rebandaged one to three times a day depending on the amount of strikethrough.

Sugar

Sugar has similar hypertonic and hyperosmolar effects to honey and also attracts macrophages, accelerates sloughing of devitalized tissue, provides a cellular energy source, and promotes formation of a healthy granulation bed. Granulated sugar is applied in a 1-cm-thick layer, and absorbent bandages are placed to absorb the excess wound fluid. The bandage is changed one to three times a day depending on the amount of strikethrough. Alternatively, a sugar paste made of castor sugar, icing sugar, glycerin, and hydrogen peroxide can be used instead of granulated sugar.

Growth Factors

Application of growth factors to stimulate more rapid healing has been investigated. Application of growth factors assumes the wound is deficient in specific growth factors. Knowing which factor is deficient in what amount at what time is nearly impossible in the complex healing process. Evidence indicates that applying single growth factors to wounds is not as effective as the combination of growth factors that the body produces. Allowing these factors to remain on the wound in the wound fluid under an occlusive or semiocclusive bandage is preferred to adding exogenous growth factors. A few growth factors are available commercially, including recombinant human-derived platelet-derived growth factor (Regranex), but efficacy studies in dogs and cats have yet to be performed.

Platelet-derived products. Platelet-rich plasma (PRP) contains a high concentration of platelets and is, therefore, rich in growth factors and cytokines and could be useful in improving wound healing (see Chapter 31). PRP has been evaluated in both equine and canine models with equivocal results. As a result, topical application of autologous PRP is thought to be best utilized in wounds with massive tissue loss or in chronic wounds for a fresh-source of mediators.

Hydrolyzed bovine collagen. Hydrolyzed bovine collagen (Woun'Dres Collagen Hydrogel, Collasate) has hydrophilic properties that help create a moist environment for autolytic debridement in early phases of wound healing and an optimal environment for epithelialization in latter stages. There is little histologic evidence of an inflammatory reaction in dogs. The collagen matrix provided serves as a lattice for ingrowth of fibroblasts, thus facilitating the repair phase of wound healing. It is probably most effective when used in the late inflammatory and early repair phases of healing to accelerate epithelialization.

Chitosan

Chitosan (HemoCon) is a polysaccharide derived from the chitin exoskeleton of shellfish. The active ingredient, glucosamine, is reported to enhance the function of inflammatory cells, growth factors, and fibroblasts to promote formation of granulation tissue and accelerate wound healing. These dressing are hemostatic and antibacterial. Information regarding the use of chitosan in veterinary medicine is limited.

WOUND CLEANSING SOLUTIONS

Wound cleansing solutions should have ideal antiseptic properties with minimal cytotoxicity. They are used primarily in the initial phases of wound management to decrease bacterial load and rid wounds of necrotic tissue and debris. Once the wound is clean, balanced electrolyte or physiologic saline solutions are ideal for irrigating it (see Table 15.1). Tap water (not distilled) is not an ideal wound cleanser, but is acceptable to initially remove dirt and debris when there is severe contamination. Tap water's hypotonicity causes cell swelling, which can cause significant cell destruction and delay wound healing with prolonged use. Antiseptic solutions are used early in wound management to reduce bacterial numbers and reduce chances of infection. They are contraindicated in clean wounds because all antiseptics have some cytotoxic effects and may be more damaging than beneficial to wound healing.

Commercial Wound Cleansers

Read the label carefully when selecting a commercial wound cleanser; many are a combination of agents that are contraindicated for use in wounds because of cytotoxic effects. Some ingredients to avoid include hydrogen peroxide, sodium hypochlorite, and hydrochlorous acid. The cleansing activity of many of the commercially available cleansers depends on a surfactant that breaks the bonds between foreign bodies and the wound surface. Most ionic surfactants and many nonionic surfactants are toxic to cells, delay wound healing, and inhibit the wound's defense mechanisms. An example of an available commercial wound cleanser is Vetericyn Plus.

Chlorhexidine Diacetate

The preferred antiseptic solution for wound lavage is 0.05% chlorhexidine diacetate because of its wide spectrum of antimicrobial activity and sustained residual activity. It has antibacterial activity in the presence of blood and other organic debris, has minimal systemic absorption and toxicity, and promotes rapid healing. A 0.05% solution is created by diluting one part of 2% stock solution with 40 parts of sterile water. Chlorhexidine forms heavy precipitates in electrolyte solutions, but this neither delays wound healing nor interferes with antibacterial activity. More potent solutions may slow the formation of granulation tissue with prolonged wound contact. Residual activity may last as long as 2 days, and effectiveness increases with repeat application. Potential drawbacks of chlorhexidine include resistance to *Proteus, Pseudomonas,* and *Candida,* as well as corneal toxicity.

Povidone-Iodine

A 1% or 0.1% povidone-iodine solution (10% stock solution diluted 1:10 or 1:100, respectively) is used frequently for wound lavage

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because of its wide spectrum of antimicrobial activity. Iodine compounds are active against vegetative and sporulated bacteria, fungi, viruses, protozoa, and yeasts. A 0.1% solution is recommended. This concentration kills bacteria within 15 seconds, and there is no known bacterial resistance. Povidone-iodine is a water-soluble, strongly acidic (pH 3.2) iodophor produced by combining molecular iodine with polyvinylpyrrolidone. Frequent reapplication (every 4–6 hours) is required when it is used as a wetting solution because residual activity lasts only 4 to 8 hours, and organic matter (i.e., blood and serous exudate) inactivates the free iodine in povidone-iodine. Iodine absorption through the skin and mucous membranes may cause excess systemic iodine concentrations and transient thyroid dysfunction. The low pH of povidone-iodine can cause or intensify metabolic acidosis when the solution is absorbed. Scrubbing wounds with povidone-iodine detergents damages tissue and potentiates infection. Contact hypersensitivities may occur in as many as 50% of dogs scrubbed with povidone-iodine compounds. Povidone-iodine at 0.5% is cytotoxic to fibroblasts.

Tris-EDTA

Tris-EDTA (disodium calcium salt of ethylenediamine tetraacetic acid buffered with tris [hydroxymethyl] aminomethane) added to lavage solutions increases permeability of gram-negative bacteria to extracellular solutes and leakage of intracellular solutes. Tris-EDTA solution is prepared by adding 1.2 g of EDTA and 6.05 g of tris to 1 L of sterile water. Sodium hydroxide is used to adjust the pH of the solution to 8, and the solution is mixed and autoclaved for 15 minutes. Treated bacteria are more susceptible to destruction by lysozymes, antiseptics, and antibiotics. Tris-EDTA in sterile water rapidly lyses *P. aeruginosa*, *E. coli*, and *Proteus vulgaris*. The addition of tris-EDTA to a 0.01% chlorhexidine gluconate solution increases antimicrobial effectiveness approximately 1000-fold. Antimicrobial synergism against *E. coli* occurs between tris-EDTA and penicillin, oxytetracycline, and chloramphenicol. Similarly, tris-EDTA and gentamicin, oxytetracycline, polymyxin B, nalidixic acid, and triple sulfonamide have synergistic activity against *P. vulgaris*.

Other Solutions

Acetic acid at 0.25% or 0.5% is occasionally used as a lavage solution. Its antibacterial effect is achieved by lowering wound pH. Wound acidification is beneficial in wounds that contain urea-splitting organisms (e.g., *Pseudomonas* spp.); however, resistance to acetic acid may develop. Acetic acid is more cytotoxic to fibroblasts than bacteria. Hydrogen peroxide and Dakin's solution should not be used as wound lavage solutions. Hydrogen peroxide, even in low concentrations, damages tissue and is a poor antiseptic. It is an effective sporicide; therefore it may be beneficial if clostridial spores are suspected. Hydrogen peroxide dislodges bacteria and debris from wounds by effervescent action. Dakin's solution is a 0.5% solution of sodium hypochlorite (1:10 dilution of laundry bleach). It releases free chlorine and oxygen into tissue, killing bacteria and liquefying necrotic tissue. However, even at half or quarter strength, Dakin's solution is detrimental to neutrophils, fibroblasts, and endothelial cells and should not be used as a wound lavage solution.

OTHER WOUND TREATMENT METHODS

Low-level Laser Therapy

Therapeutic LLLT is widely used on open wounds to potentially accelerate wound healing. However, there is potential for cellular damage and delay in wound healing if administered at higher doses or used inappropriately. The exact mechanism is unknown, but LLLT is thought to modulate processes at the cellular level leading to increased protein synthesis and stimulation of cellular growth and cellular differentiation. In a 2016 study in a canine wound model, LLLT increased keratinocyte cellular migration and proliferation at doses of 0.1, 0.2, and 1.2 J/cm^2, whereas exposure to 10 J/cm^2 had the opposite effect.[2] Protocols for the use of therapeutic laser vary widely in the literature (e.g., based on laser type, energy level, duration of exposure, or frequency intervals), making determination of the effectiveness of this modality challenging. For example, a 2015 study in veterinary medicine found no difference in wound healing between acute wounds in dogs treated with LLLT and those treated without.[3] The most consistent recommendation in the veterinary literature is 2 to 6 J/cm^2 once daily for 7 to 10 days for acute wounds and 2 to 8 J/cm^2 once daily for chronic wounds, using a laser with a wavelength range of 630 nm (visible light) to 904 nm (infrared), with between 1 and 15 W of power; however, an optimal delivery protocol has not been determined.[4]

Hyperbaric Oxygen Therapy

HBOT is the inhalation of pure oxygen at a pressure greater than 1 atmosphere, thereby significantly increasing tissue oxygenation. HBOT has been shown to be beneficial in the short-term treatment of chronic, hypoxic wounds in humans.[5] However, data from the use of HBOT in veterinary medicine are lacking.

Pulsed Electromagnetic Field

Experimentally, pulsed electromagnetic field treatment of open wounds enhances wound epithelialization and may promote early wound contraction without adverse effects on perfusion or tensiometric, histologic, clinicopathologic, or electroencephalographic parameters. A pulsed electromagnetic field generates complex multiform pulses of oscillating electromagnetic fields in the ultra-low frequency range (0.5–18 Hz).

ASSESSMENT OF SKIN VIABILITY

Skin circulation may deteriorate for 5 days after surgery because of edema and other factors. Skin viability is clinically assessed by color, warmth, pain sensation, and bleeding. Viability may also be assessed by dyes, transcutaneous oxygen or carbon dioxide, laser Doppler velocimetry, ultrasonic Doppler flow detection, and scintigraphy. Nonviable skin is black, bluish black, or white, and the area may be nonpliable, cool, and devoid of sensation. Normal skin is warm, pliable, and pink with normal capillary refill (difficult to assess) and pain sensation. Areas of questionable viability are often blue or purple, and capillary refill and sensation are poor.

Intravenous injection of vital stains fluorescein (10 mg/kg) or xylenol orange (90 mg/kg) has been used to assess vascular integrity of skin but has not been better than visual observation. Transcutaneous oxygen (pO_2) or carbon dioxide (pCO_2) monitoring allows immediate evaluation for ischemia but requires prolonged, quiet recumbency, and transcutaneous oxygen or carbon dioxide sensors left in place longer than 3 hours may cause superficial burns. Skin generally survives if a transcutaneous pO_2 value of approximately 60 mm Hg is maintained. Transcutaneous pO_2 values of 30 to 60 mm Hg may be associated with partial or complete survival. Transcutaneous pCO_2 values are

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lower at the base of skin flaps (approximately 53 mm Hg) than at the apex (approximately 106 mm Hg), where ischemia is most apt to occur. Laser Doppler velocimetry is an indicator of capillary blood flux that may give an accurate assessment of local circulation. Probes must be placed away from major vessels to monitor relative blood flow, volume, and velocity (factors that vary with species, site, and instrumentation). Ultrasonic Doppler flow detection is a noninvasive, inexpensive means of determining blood flow and predicting viability in an area. Two sounds are usually heard with each arterial pulse, but only one sound is heard with proximal occlusion or stenosis. Areas of nonviable tissue may also be identified by evaluating the area with scintigraphy after injection of technetium 99-m methylene diphosphate.

INTEGUMENTARY SURGERY

The fundamental surgical principles for reconstructive surgery are listed in Box 15.4. Incisions made with scalpel blades cause less tissue trauma than those made with scissors, electrosurgery, or lasers. The CO_2 laser is the best-suited laser for skin incisions; it creates wounds that hemorrhage minimally because vessels are sealed. Skin edges should be manipulated atraumatically using skin hooks or fine-tooth forceps. The deep or subdermal plexus must be preserved during dissection and excision to ensure skin survival. It is important to undermine at the level of the subcutaneous fat to prevent transection of the subdermal plexus. To prevent transecting the direct cutaneous arteries that supply the subdermal plexus, dissection should be performed under the cutaneous muscles (panniculus, preputialis, supramammarius, platysma, and sphincter colli muscles) or in the distal extremities in the deep dermal layer. Risk of infection after surgery is diminished by antimicrobial prophylaxis, but is increased by several factors (Box 15.5).

Sutures

Suture acts as a foreign body in wounds. A buried suture greatly reduces the critical number of bacteria required to cause infection because the suture causes direct irritation, harbors bacteria, and generates ischemic islands of tissue. The smallest and fewest sutures possible should be used to close a wound. Approximating sutures should be used to bring tissue edges into anatomic apposition. A 3-0 or 4-0 absorbable suture (e.g., polyglyconate, polydioxanone, poliglecaprone 25, glycomer 631, polyglactin 910) with a swaged taper point needle should be used to close subcutaneous and subcuticular tissue. A 3-0 or 4-0 monofilament, nonabsorbable suture (e.g., nylon, polypropylene, polybutester) with a reverse cutting needle is preferred for most skin sutures. Skin sutures should be placed at least 0.5 cm from the wound margins. Wounds closed with interrupted sutures have less edema, improved microcirculation, and higher initial tensile strength, whereas continuous suture patterns allow for faster wound closure, less use of suture material, and create a better seal across the incision line. Suture tension should just appose edges, as loosely approximated wounds are stronger during the first 21 days.

Staples

Skin staples used to appose skin edges are less time consuming to place than sutures; however, it is more difficult to correctly align skin edges, and staples are less secure than sutures. Additionally, reduced infection and inflammation have been associated with the use of suture material than with stainless steel staples for skin closure. Apply skin staples perpendicular to the incision after aligning and apposing the edges with thumb forceps; then apply moderate pressure before the trigger is compressed (Fig. 15.4). Space staples approximately 5 mm apart.

Absorbable staples composed of polyglycolic-polyglactic acid copolymers have been introduced for subcuticular closure and are meant to replace cutaneous closure by other methods. These staples are degraded by hydrolysis, losing 60% of their holding strength in 14 days and having a tissue half-life of 10 weeks. Subcuticular staples are applied after aligning the skin edges

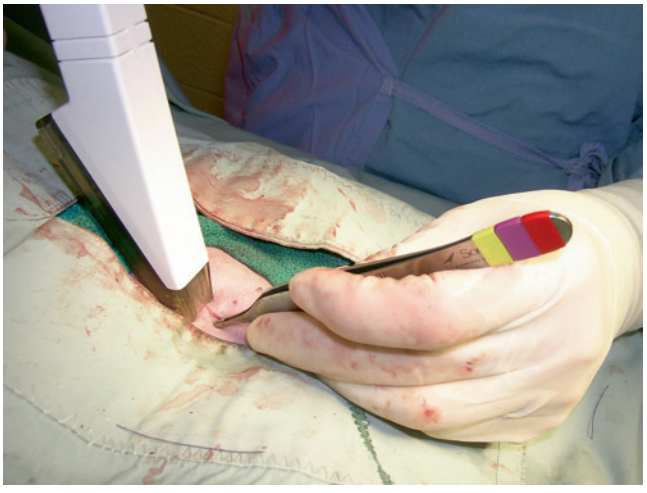


FIG. 15.4 Proper application of skin staples.

BOX 15.4 Fundamental Surgical Principles for Reconstructive Surgery

- Use strict asepsis in preparation of surgical team, room, and instruments and during surgery
- Handle tissue gently
- Preserve vascularity
- Remove necrotic tissue
- Maintain hemostasis
- Approximate tissue anatomically without tension
- Obliterate dead space
- Use appropriate suture materials and implants

BOX 15.5 Predictive Major Risk Factors for Postoperative Surgical Site Infection

- Increasing American Society of Anesthesiologists' preoperative assessment score ~3
- Increasing duration of anesthesia (~30% increase for each hour)
- Increasing duration of surgery (doubles every 70–90 minutes)
- Increasing number of persons in operating room (1.3 times higher/person)
- Dirty classification of wound site
- No preoperative or intraoperative antimicrobial prophylaxis (6–7 times more likely)
- Increasing duration of postoperative intensive care stay (1.16 times for each additional day)
- Wound drain (foreign materials reduce number of microorganisms required for infection by 10^4)
- Increasing patient weight

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with forceps and then firing the stapler in the subcuticular tissue; staples are spaced approximately 1 cm apart. Subcuticular staples incite less inflammation than subcuticular polyglactin 910 sutures or skin staples; therefore they have a less detrimental effect on wound healing.

Tissue Adhesives

Cyanoacrylate tissue adhesives (see also p. 191) may be used in selected procedures to facilitate skin closure or secure drains. They are often used to hold skin in apposition following onychectomy, tail docking, and ovariohysterectomy in population control programs. Adhesives allow a quick cosmetic closure with less risk of infection or scarring when used properly. Initial wound strength is less than sutured wounds (5-0 nylon), but strength at 5 to 7 days is equivalent or greater with adhesives. Sometimes skin sutures and adhesives are used together to reduce the number of sutures used. Of the nonabsorbable tissue adhesives, N-butyl or isobutyl-2 cyanoacrylates (e.g., Vetbond, LiquiVet, GLUture) are preferred over propyl or methyl cyanoacrylates as they are less toxic. These adhesives should not be placed within the wound or incision but rather over the apposed surface to prevent foreign body reactions. An absorbable, sterile, methoxypropyl cyanoacrylate tissue adhesive is available (Tissumend II Sterile). It may be used externally and internally, with trials having been performed on lung, liver, spleen, kidney, and cornea. It has hemostatic properties, enhances healing, is nonreactive, and absorbs via hydrolysis in 60 to 90 days.

Fibrin tissue glues (Tisseel) reduce hemorrhage when vessels are anastomosed, provide a barrier to microleakage, reduce suture line tension following intestinal anastomosis, and have an adhesive effect, improving tensile strength after skin edges are apposed with sutures. Two-component fibrin glues are mainly composed of bovine or human thrombin and concentrated fibrinogen, duplicating the final stages of the coagulation cascade.

Drains

Dead space allows seepage and accumulation of blood and serum in a warm, moist environment that is ideal for bacterial proliferation. Dead space may be eliminated by layered wound closure when adequate tissue is available, by suture obliteration, by compression bandages, or by drainage. Leaving a wound open provides optimal drainage for an animal's injury. Implanting drains allows evacuation of potentially harmful fluids (e.g., blood, pus, and serum) from wounds and helps eliminate dead space. Drains are often necessary for treatment of bite wounds, lacerations, skin avulsions or separations, mastectomies, seromas, and abscesses. Drains may help maintain contact between a flap or graft and its bed. Drains may be either passive or active. Passive drains (e.g., Penrose drains) depend on gravity for fluid evacuation, whereas active drains require a vacuum. Penrose drains are used most commonly to drain subcutaneous spaces. Active drains increase drain efficiency and reduce drain-related infection. They are especially useful in draining deep wounds and after grafting. Negative pressure can be applied to active drains intermittently or continuously; continuous suction reduces chances of drain occlusion with fibrin or blood clots and encourages tissue apposition. Active drains may be open, with an air vent into the wound, or closed. With vented drains (e.g., sump) there is a danger of retrograde contamination of particulate matter and bacteria passing through the air vent into the wound. Filtered vents reduce the risk of contamination. Closed active drains are preferable to Penrose drains (Fig. 15.5).



FIG. 15.5 A closed suction drain is being used to evacuate an abscess between the thoracic wall and scapula.



FIG. 15.6 For proper passive drain placement, a Penrose drain is anchored dorsally in the wound, exits in the most gravity-dependent area, and does not exit the primary incision. Cover exiting passive drains with a small tie-over bandage to absorb wound fluid and minimize ascending infection.

Penrose drains or passive tube drains should be used in clean wounds only if both the exposed end and the wound can be covered with a sterile compressive dressing or bandage. Superficial, passive drains should be secured to the skin at the dorsal aspect of the wound by direct visualization or blind suture placement (Video 15.1). They should be exited through a stab incision at least 1 cm from the primary incision and positioned such that they allow maximum flow by gravity (Fig. 15.6).

For effective drainage of small spaces, a closed suction drain can easily be made using a butterfly catheter and an evacuated tube or syringe (Video 15.2). The syringe adapter is removed from the plastic tubing, and the tube is fenestrated before the drain is placed in the wound (Fig. 15.7). After wound closure, the needle is inserted into the evacuated tube (5–10 mL) to apply suction. As an alternative, the needle can be removed, the tube fenestrated, and the adapter attached to a syringe with suction

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FIG. 15.7 A butterfly catheter and Vacutainer tube are used for active closed drainage of small spaces, such as the ear.

applied. Active collection systems should be emptied frequently to maintain constant negative pressure. The collection reservoir is replaced or emptied when it loses negative pressure or fills with fluid. Most collection systems lose pressure as they become more than half full. Initial collection may simply remove air from the wound as the vacuum is established, and fluid drainage may not occur until several reservoir changes. The volume of fluid collected should be measured and recorded.

The smallest diameter and fewest number of drains with the fewest number of exit holes should be used to prevent complications. Drains should not exit or lie directly under the primary incision. No part of the drain should be in contact with haired skin. Secure drains to the skin so they cannot be removed prematurely or allowed to retract into the wound. Drains should be protected with a bandage that is changed before "strikethrough" occurs. Strikethrough is the saturation of a bandage with fluid that wets both inner and outer surfaces. An Elizabethan collar or bucket can prevent self-inflicted drain or bandage damage. The animal should be kept in a clean, dry environment and have limited exercise. Tissue fragments, fibrin, or viscous exudates may cause drain malfunction. Drains are foreign bodies and cause drainage until removed. They should be removed when the discharge is serosanguinous and the volume has diminished to one-fourth or less of the original drainage. Most wound drains can be removed after 2 to 5 days. Closed suction drains placed under grafts are usually removed after 48 to 72 hours, when drainage has diminished. Be careful when removing drains to prevent disrupting the skin–wound bed interface. Apply a bandage after drain removal to absorb any residual drainage and stabilize the wound site.

BOX 15.6 Benefits of Negative Pressure Wound Therapy

- Increases blood flow
- Reduces edema
- Increases rate of granulation tissue formation
- More rapid reduction in numbers of microorganisms
- Removal of exudate
- Greater flap survival

NOTE All drains should be protected by a bandage.

The major disadvantage of drains is that they serve as retrograde conduits for skin contaminants to enter the wound. Drains also impair tissue resistance to infection and may disrupt graft adherence. Drains made from latex incite more inflammatory reaction than those of Silastic or silicone. To prevent incisional dehiscence and herniation, drains should not exit from primary incisions.

Negative-Pressure Wound Therapy (Video 15.3)

Negative-pressure wound therapy (NPWT) is an alternative method of wound drainage that uses open-cell foam and subatmospheric pressure (e.g., VAC, Curato). Medical-grade, open-cell, polyurethane ether foam dressing with pore sizes between 400 and 600 µm fitted with an evacuation tube is cut to the specific wound configuration and placed into the wound defect. Exposed viscera are covered with mesh or omentum, and major vessels with adjacent soft tissue, before the foam is positioned. The wound site is bandaged, and the tubing is connected to a collection reservoir. The reservoir is connected to a vacuum pump, and subatmospheric pressure (125 mm Hg) is applied continuously or intermittently for cycles of approximately 5 minutes on and 2 minutes off. Continuous suction may be less painful and is often applied during the initial 48 hours of use. The foam is changed every 48 hours (except initially after grafting) to prevent tissue ingrowth, and the bandage is changed as necessary. Fluid is pulled from the wound, creating a moist environment and reducing local tissue swelling.

The benefits of NPWT are increased blood flow, increased rate of granulation tissue formation, more rapid reduction in numbers of microorganisms, and greater flap survival following application of this vacuum system (Box 15.6). In addition, this system facilitates readhesion of degloved tissue and healing of chronic wounds, acute wounds, skin flaps, and skin grafts. Complications may include pain, wound margin dermatitis, excessive ingrowth of granulation tissue, and wound desiccation with vacuum loss. Use of NPWT has been shown to accelerate wound healing in both dogs and cats when compared to conventional foam dressings for open wound.[6,7]

Tourniquets

Tourniquets help control hemorrhage of distal extremities, improving visualization and reducing operating time. However, tourniquets should not be used on traumatized limbs or those

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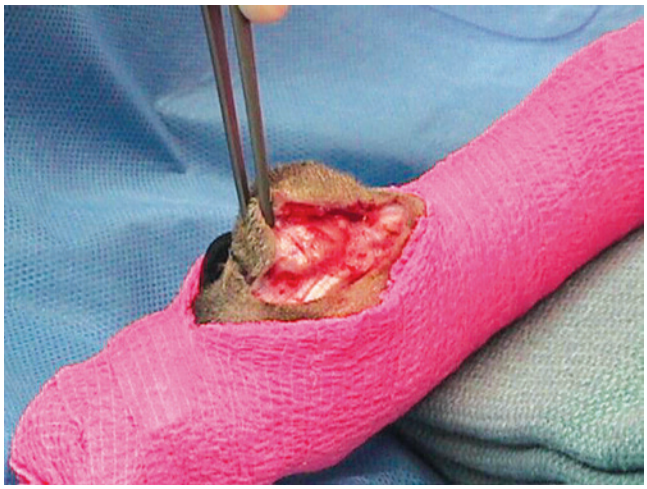


FIG. 15.8 Sterile elastic wrap may be used as a short-term tourniquet. An incision made in the elastic wrap allows access to the lesion for excision.

with vascular injury or circulatory compromise. Pneumatic tourniquets with pressures below 300 mm Hg for less than 3 hours should be used. The limb should be elevated for approximately 5 minutes or exsanguinated with a rubber bandage before the tourniquet is applied. Exsanguination is contraindicated with local suppuration, deep venous thrombosis, or neoplasia. Pressure is more evenly applied if two or three layers of orthopedic padding are placed under the tourniquet. The tourniquet cuff should be applied at a point of maximum circumference, where nerves and blood vessels are protected against direct compression. Although of uncertain value, releasing the tourniquet 10 minutes for every hour of inflation has been recommended. For a short-term tourniquet (as an alternative to or in combination with a pneumatic tourniquet), apply sterile elastic wrap (Vetrap). Apply it from the digits proximally without excess tension, and then incise it beginning at the toes to expose the surgical area (Fig. 15.8). Complications of tourniquet use are ischemia, hypoxia, or acidosis of local tissue; neurapraxia; and muscle damage.

WOUND CLOSURE

Wounds may be closed immediately (primary wound closure); within 1 to 3 days after injury, when they are free of infection but before granulation tissue has appeared (delayed primary wound closure); or after the formation of granulation tissue (secondary closure); or they may be allowed to contract and epithelialize (healing by secondary intention). Wounds closed in the presence of contamination, necrotic tissue, excessive tension, or dead space are apt to dehisce, often with the loss of additional tissue because of bacterial toxins and pressure necrosis. If there is any doubt whether a wound should be closed, it is best to leave it open. Factors that affect the decision not to close wounds include the following:

1. *Amount of time that has elapsed since injury.* Wounds older than 6 to 8 hours are initially treated with bandages.
2. *Degree of contamination.* Obviously contaminated wounds should be thoroughly cleansed and initially treated with bandages.
3. *Amount of tissue damage.* Wounds with substantial tissue damage have reduced host defenses and are more likely to become infected; therefore they initially should be treated with bandages.
4. *Completeness of debridement.* Wounds should remain open if the initial debridement was conservative and if further debridement is necessary.
5. *Status of the wound's blood supply.* A wound with questionable blood supply should be observed until the extent of nonviable tissue is determined.
6. *The animal's health.* Animals unable to tolerate prolonged anesthesia are best treated with bandages until their health improves.
7. *Extent of tension or dead space.* If excessive tension or dead space is present, wounds should be bandaged to prevent dehiscence, fluid accumulation, infection, and delayed wound healing.
8. *Location of the wound.* Large wounds in some areas (e.g., limbs) are not amenable to closure.

Primary healing occurs with clean, incised wounds that are held together. Healing is initiated by movement of epithelial cells from the two edges of the wound toward the center that usually meet within 4 to 7 days of the incision. Primary wound closure is infrequently indicated following trauma. It should only be performed if less than 6 to 8 hours (within the golden period) have elapsed since injury; there is minimal tissue contamination, loss, or trauma; the wound is suitably cleaned by lavage and debridement; there is good hemostasis; and there is no tension or dead space. Traumatic wounds contaminated by feces, saliva, purulent exudate, or soil should not be closed primarily. *Delayed primary closure* is indicated for mildly contaminated, minimally traumatized wounds that require some cleansing and debridement or when the wound is older than 6 to 8 hours. Wounds are first lavaged and debrided to control local contamination or infection. These wounds should be treated with bandages after injury and before closure. Delayed primary closure allows staged debridement and maximum wound drainage. The wound should be surgically closed after bandaging and debridement for 3 to 5 days when it appears clean.

Lavage the wound with a balanced electrolyte solution or sterile saline to remove debris and reduce bacterial numbers (see p. 184). Next, thoroughly explore the wound. Anastomose transected tendons in clean wounds (see p. 1283). Primarily appose large motor nerves that are transected sharply and cleanly; otherwise, delay nerve repair for 2 to 3 weeks until the wound has healed. If excessive dead space surrounds a wound, use passive or active (preferred) drains. Appose subcutaneous tissues with 3-0 or 4-0 buried interrupted or continuous approximating sutures (e.g., polydioxanone, polyglyconate, glycomer 631, poliglecaprone 25). Bring the skin edges into apposition with buried walking sutures (see p. 209) or subcuticular sutures (e.g., 3-0 or 4-0 polydioxanone, polyglyconate, poliglecaprone 25). Use approximating sutures in skin (e.g., 3-0 or 4-0 polypropylene, polybutester, or nylon).

Wounds with considerable tissue loss, contamination, or infection, or those older than 6 to 8 hours should be treated as open wounds. Initially, they should be lavaged, explored, and debrided. Tendons, ligaments, and vessels may be damaged beyond repair. Identifiable tendon ends should be tagged. Apply a hydrophilic bandage (see p. 195) that immobilizes the area and promotes formation of a healthy granulation bed. The wounds will initially begin healing by contraction and epithelialization and may heal completely *(healing by secondary intention)*. Healing by secondary intention is often less expensive and results in normal-appearing skin if contraction is complete. Body wounds

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are more apt than leg wounds to completely close by secondary intention. Disadvantages of healing by secondary intention include contracture with disfigurement, incomplete healing, and fragile epithelial scars with large wounds. Alternatively, healthy wounds may be repaired by *secondary closure* or by use of a flap or graft. Secondary closure occurs at least 3 to 5 days after injury, after a healthy granulation bed has formed. Granulation tissue helps control infection in the wound and fills tissue defects. Secondary closure is appropriate when the wound is severely contaminated or traumatized, when epithelialization and contraction will not completely close the wound, or when healing by secondary intention is undesirable. Secondary closure involves resecting the granulation bed and skin margins, lavaging the wound, and apposing skin edges. Secondary closure can also be accomplished by resecting skin margins, debriding the surface of the healthy granulation bed, and apposing skin edges over the granulation tissue. Wound closure in the presence of granulation tissue is somewhat difficult because the tissue is thickened and not pliable. Excising the entire granulation bed gives a more cosmetic closure. If secondary closure is not possible, a flap or graft can be applied over the defect. After wound closure, an absorbent, nonadherent bandage should be applied to support the wound and absorb exudate. Bandages should be changed once or twice daily if a passive drain is used; if little drainage is expected and if drains are not used, once every 3 to 4 days may be adequate.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative wound care should optimize healing and be tailored to the type of wound. Wounds should be evaluated frequently for infection, tension, fluid accumulation, dehiscence, and necrosis. They may be evaluated visually and with ultrasonography, which helps detect and localize fluid accumulation. An ultrasound scan allows monitoring of wound depth and width and identifies blood clots, edematous regions, granulation tissue, scar tissue, epidermis, and eschar. Wounds should be protected with clean, dry bandages. Bandages act as a barrier against exogenous bacteria and support the wound during the first few days after surgery. Healing, sutured wounds become increasingly resistant to bacterial penetration. The patient and its environment should be kept clean, and adequate nutrition should be provided. Analgesics (see Chapter 13) and antibiotics (see Chapter 9) should be used postoperatively as necessary.

Infection may be suspected if the patient has fever, leukocytosis, anorexia, and/or depression. Although these abnormalities can be a normal response to stress and surgery and do not indicate infection, they are generally more exaggerated with infection than with nonseptic inflammation. If infection is suspected, samples should be collected for culture and cytology. If a surgical wound becomes infected, the sutures should be removed and the incision treated as an open wound with appropriate bandages (see p. 201). Topical or systemic antibiotics (see previous discussion) are used to control infection based on the results of susceptibility testing. The wound may be closed after the infection has resolved.

Sutures should be removed from wounds in 7 to 14 days, even though the wound has regained only 20% to 30% of its original strength. Scarring and infection associated with sutures are greater when sutures are left for longer periods. Most complications are prevented by using good surgical and wound management techniques. Potential complications include inflammation, edema, seroma and hematoma formation, drainage, infection, dehiscence, necrosis, granulomas, contracture, and failure to heal.

Seromas are caused by excessive dead space or motion. They are prevented by closing dead space, using drains, and applying bandages to immobilize and support the wound. Seromas are treated by immobilizing the area and applying a pressure bandage. Large seromas may be drained, although this increases the risk of infection. Dehiscence may occur if tissue necrosis occurs, if sutures are placed too close to the margin, if tension exceeds suture strength, if sutures absorb too quickly, or if sutures strangulate and cut through tissue. Dehiscence may also occur secondary to self-trauma, infection, severe cough, hypoproteinemia, hypovolemia, or administration of drugs that interfere with healing. Wounds that dehisce may be allowed to heal by secondary intention or may be debrided and resutured. A *granuloma* is a chronic inflammatory process that may be caused by foreign material. Contracture or excess scar formation occurs most commonly at sites of motion (e.g., elbow or stifle) or body orifices. Contracture limits motion and may be disfiguring. Scar revision or excision is necessary when contracture interferes with activities. Wounds may fail to heal when the area is infected or has been irradiated or when the animal has received chemotherapy or is severely debilitated or malnourished.

BANDAGES

No single type of bandage provides the optimum environment for all wounds or for total healing of a particular wound. Bandages provide wound cleanliness, control the wound environment, reduce edema and hemorrhage, eliminate dead space, immobilize injured tissue, and minimize scar tissue. They also provide comfort, absorb and allow characterization of wound secretions, and give an aesthetic appearance. Bandages keep wounds warm, which improves wound healing and facilitates oxygen dissociation (Box 15.7). Covering wounds with a bandage promotes an acid environment at the wound surface by preventing carbon dioxide loss and absorbing ammonia produced by bacteria. An acid environment increases oxygen dissociation from hemoglobin and subsequently increases oxygen availability in wounds.

Bandages should be comfortable and clean. Uncomfortable bandages annoy patients, who may then mutilate the bandage or wound or both. Pressure should be applied over and distal to wounds, rather than proximal to them, to minimize venous or lymphatic compromise. When outer layers of a bandage become wet, bacteria readily pass from the outer surface and colonize wounds. Wounds bandaged with adherent or nonhydrophilic contact layers are generally unbandaged, inspected, and treated daily; however, wounds with excessive tissue damage, exudate, or established infection may require bandage changes twice or three times daily. Adherent bandages should be changed more frequently if the gauze is saturated with exudate and slides on

BOX 15.7 Desirable Characteristics of Wound Dressings

- Removes exudates and toxic components
- Maintains high humidity at the wound-dressing interface
- Allows gaseous exchange
- Provides thermal insulation
- Relieves pain
- Protects from secondary infection
- Protects from particulate or toxic contaminants
- Allows dressing removal without wound trauma

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BOX 15.8 Hydrophilic Contact Layers

- Hypertonic saline
- Calcium alginate
- Polyurethane foam
- Hydrogel
- Hydrocolloid
- Some topical medications

BOX 15.9 Factors in Contact Layer Selection

- Phase of wound healing
- Amount of exudate
- Wound location and depth
- Presence or absence of eschar
- Amount of necrosis
- Presence of infection

BOX 15.10 Factors That May Affect Wound Contraction

Stimulate or Enhance

Acemannan
Tripeptide copper complex
Occlusive hydrogel dressings
Equine amnion
Pulsed electromagnetic field radiation
Adjustable horizontal mattress sutures
Skin stretchers

Inhibitors

Corticosteroids
Silver sulfadiazine
Mafenide acetate
Hydrocolloid dressings
Porcine small intestine submucosa
Thick skin grafts or flaps

the wound during bandage changes. Bandages applied over wounds with healthy granulation tissue and support bandages used to immobilize fractures may need changing only once every 2 to 4 days. Hydrophilic bandages (Box 15.8) are designed to be left on the wound for 3 to 7 days depending on the characteristics of the contact layer and the amount of exudate being produced. Analgesia or anesthesia may be required for initial bandage changes.

WOUND BANDAGING MATERIALS

Bandages have three basic layers: the contact dressing (i.e., primary layer), the intermediate (i.e., secondary layer), and the outer (i.e., tertiary) layer.

Contact (Primary) Layer

The contact layer touches the wound surface and should remain in contact with it during movement. It is used to debride tissue, deliver medication, transmit wound exudate, or form an occlusive seal over the wound. No single dressing will produce the optimum microenvironment for all wounds or for all stages of wound healing on a single wound. The appropriate contact layer is selected based on the phase of wound healing, amount of exudate, wound location and depth, presence or absence of an eschar, and amount of necrosis or infection. The contact layer should also minimize pain and prevent excess loss of body fluids (Boxes 15.7–15.10 and Tables 15.2 and 15.3). It may be adherent or nonadherent, and occlusive or semiocclusive (Table 15.4). *Adherent* contact layers are used for mechanical debridement of necrotic tissue and debris, and for absorption of wound exudates. These dressings are economic and easy to place, but they should only be used on a short-term basis early in the course of wound healing as the wound debridement is nonselective, and normal healing tissue may be damaged when the bandage is removed. They cause pain on removal and serve little purpose in the absence of nonviable tissue. *Nonadherent* contact layers have traditionally been selected when granulation tissue has formed, but current wound care standards recommend use of hydrophilic nonadherent contact layers for all wounds. These products are very absorptive; they create a moist environment to facilitate healing and reduce the frequency of bandage changes (usually once every 3–7 days). Semiocclusive bandages allow air to penetrate and exudate to escape from the wound surface. They are the most commonly used bandages in veterinary medicine. Occlusive bandages are impermeable to air and fluid. They are used on less exudative wounds to keep tissue moist. Semiocclusive bandages are less likely to macerate adjacent normal tissue.

Wet Adherent

A wet adherent contact layer is used when the wound surface has necrotic tissue, foreign matter, or a viscous exudate. Sterile, wide-mesh gauze moistened, not soaked, with 0.9% saline is applied to the wound. Hypertonic saline (see p. 196) may be used as wetting solutions in grossly infected wounds. The wet dressing dilutes wound exudate so that the intermediate layer of the bandage can absorb it. Necrotic tissue and foreign material adhere to the gauze as it dries and are removed from the wound along with the bandage. Wet bandages absorb faster than dry bandages and are more comfortable. Potential disadvantages of a wet adherent contact layer are pain and tissue damage during bandage changes, bacterial proliferation, tissue maceration, and strikethrough (exudate absorption through outer bandage layer). Rewetting the dried dressing with warm saline facilitates removal and reduces pain during bandage changes; however, it also diminishes the mechanical debridement effect.

Dry Adherent

This type of contact layer is infrequently used because it is painful to remove, viable cells may be removed with necrotic debris, and the wound may desiccate. A dry adherent contact layer was primarily used when the wound surface had a large amount of low-viscosity exudate that does not aggregate. With this bandage an absorbent, wide-mesh gauze is used without a cotton filler. Dry gauze absorbs exudate and adheres to necrotic tissue and debris. The bandage is removed after the primary layer has absorbed fluid and debris and then dried.

Adherent Film or Skin Sealant

Transparent liquids are spread in a thin layer over the wound dry, creating a film or bandage that acts as a barrier between the skin or granulating wound and the external environment (e.g., Cavilon No Sting Barrier Film). They are made from natural ingredients, primarily hetastarches and water. These films are generally permeable to water vapor and occlusive to bacteria

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TABLE 15.2 Guide to Dressings Based on Purpose

Purpose	Suggested Products
Cleanse wound	Noncytotoxic commercial cleanser; balanced electrolyte or saline solution
Absorb exudate	Absorption beads, pastes, powders, and pads: Alginates Foams Hydrocolloids Hydrogels Composite dressings
Autolytic debridement: cover wound to allow endogenous enzymes in wound fluid to self-digest eschar and fibrinous slough	Same as above, plus transparent films
Chemically debride devitalized tissue	Enzymatic debridement agents: Granulex, live yeast Preparation-H
Add moisture to wound	Hydrogel Hypertonic saline dressing Medicinal honey
Maintain moist wound environment	Hydrophilic ointments Foam Hydrocolloids Hydrogels Transparent films
Fill dead space	Absorption beads, pastes, powders, and tapes: Alginates Hydrocolloid Hydrogel Foam
Reduce swelling to improve perfusion	Hypertonic saline
Prevent contamination	Biguanide-impregnated antimicrobial gauze Occlusive Semiocclusive
Reduce bacterial numbers	Biguanide-impregnated antimicrobial gauze Antibiotics
Cover and protect wound	Nonadherent, hydrophilic dressing with appropriate intermediate and outer bandage layers
Protect surrounding skin from moisture and trauma	Moisture barrier ointments Skin sealants Transparent film dressings Bandage
Reduce odor	Vapor-permeable film or polyurethane foam with activated charcoal

and water. They are nonirritating and do not stain. They prevent skin irritation from exudates, urine, or feces contamination. They may be used alone over wounds or with a bandage. Reapplication is necessary every 3 to 4 days.

Nonadherent

Nonadherent contact layers do not stick to the wound surface, and most are *semiocclusive*. They are used to promote moist wound healing and epithelialization and to prevent wound dehydration while allowing excess fluid to drain, which reduces tissue maceration. These dressings are composed of wide-mesh gauze impregnated with paraffin (Jelonet), petrolatum (Adaptic), petrolatum and 3% bismuth tribromophenate (Xeroform), or other petrolatum-based ointments (Aquaphor, Furacin). Nonadherent dressings are used when wounds have newly formed granulation tissue and some remaining exudate but are not epithelialized. Xeroform, in particular, allows egress of fluid and bacteria from wounds through the mesh. Fibrin from the wound bed causes temporary bonding of the dressing to wounds as the dressing dries. It is inexpensive and associated with low infection rates but is painful to remove. Epithelialization is rapid. Nonadherent dressings without petrolatum (Telfa or Dermacea) have less chance of adherence to the wound and are used once epithelialization begins.

Other types of nonadherent contact layers are *occlusive dressings*. Occlusive dressings are impermeable to air and are used on healthy wounds with minimal exudation during the repair phase of healing. Occlusive dressings accelerate wound epithelialization and collagen synthesis by maintaining a moist wound environment. They require less frequent changes than other types of bandages. Some types of occlusive dressings have a hydrocolloid, adhesive layer (e.g., DuoDerm) (see p. 199). Other occlusive, nonadherent contact layers include polyurethane films, hydrogels, and hydrophilic beads, flakes, powders, and pastes.

Interactive dressings are nonadherent contact layers that create and control a moist wound environment and modulate cellular activity and growth factor release within the wound to facilitate moist wound healing through all phases of healing. These dressings are designed to meet the different environmental stages found in the different phases of wound healing and include, but are not limited to, hydrocolloid, alginate, antimicrobial, and biologic dressings.

Moist wound healing is promoted by incorporating hydrophilic materials that are placed on the wound bed or in the wound cavity. Hydrophilic dressings include hypertonic saline, calcium alginate, polyurethane foam, hydrogel, hydrocolloid, and some topical medications. Hydrophilic materials should not extend onto normal skin to prevent maceration of tissue adjacent to the wound. Barrier ointments or skin sealant adhesive films may be applied adjacent to the wound to prevent maceration if necessary. Some products with adhesive borders designed for people do not adhere around animal wounds well because of higher skin lipid concentrations and rapid hair regrowth. Most nonadherent, hydrophilic contact layers are highly absorbent, requiring bandage changing only every 3 to 7 days. Product costs are higher, but with fewer bandage changes the overall cost is comparable to more frequently changed adherent bandages.

NOTE No single dressing will produce the optimum microenvironment for all wounds or for all stages of wound healing of a single wound.

Hypertonic Saline

Hypertonic saline dressings (Curasalt, Curity) are indicated for infected, necrotic, and heavily exudative wounds and eschars. These dressings are loosely woven gauze impregnated with 20% sodium chloride and should be used in the inflammatory stages of healing during the first few days of wound care. Osmotic action desiccates bacteria and necrotic tissue, and reduces edema,

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TABLE 15.3 Guide to Dressings Based on Wound Characteristics

Wound	Objective	Wound Characteristics	Dressing to Consider
Acute mild contamination and tissue damage	Hemostasis, debride, reduce contamination, provide moist environment	Hemorrhaging <6–8 hours old	Calcium alginate Hydrogel, hydrocolloid
Acute moderate to severe contamination and tissue injury	Reduce contamination, debride Provide moist environment	Gross dirt and debris in wound; contusions, avulsions, ischemia, and <6–8 hours old	Hydrogel, hydrocolloid
Exposed muscle, fascia, subcutaneous tissue	Provide moist environment Encourage granulation	Clean wound	Calcium alginate, hydrogel
Necrotic	Promote debridement Provide moist environment Absorb exudates	Dry eschar or slough Excessive exudate and surface devitalized	Hypertonic saline or hydrogel Hydrogel or calcium alginate
Granular	Provide moist environment Encourage granulation	Irregular or incomplete granulation, minimal to moderate exudate	Calcium alginate, hydrogel
Needs epithelialization	Provide moist environment to promote and protect resurfacing	Healthy granulation tissue: pink, smooth Dry, moist	Hydrogel sheet, hydrocolloid Foam

TABLE 15.4 Contact Layer Wound Dressings

Type	Trade Name	Indication	Action
Adherent Dressings			
Wet-dry	—	Inflammatory phase, high-viscosity exudate	Absorbs, debrides, hydrates, disrupts granulation tissue Change daily to 3 times daily
Dry-dry	—	Inflammatory phase, low-viscosity exudate	Absorbs, debrides, disrupts granulation tissue Change daily to 3 times daily
Antimicrobial sponge or roll gauze	Kerlix AMD	All phases	Kills surface bacteria, prevents bacterial penetration of bandage
Hypertonic saline	Curasalt; Curity	Inflammatory and debridement phases Necrotic, infected, heavy exudates	Osmotic action desiccates bacteria and necrotic tissue Nonselective debridement Decreases edema, resulting in increased perfusion Change every 1–3 days, apply only once or twice
Adhesive Dressings			
Semipermeable films	OpSite, Tegaderm, Bioclusive, Bioguard	Low- and high-exudate wounds with granulation tissue Covering for sutured wounds	Adhesive sticks to skin around wound; may have a thin nonadherent contact layer; creates a moist environment Accelerates epithelialization; permeable to water vapor but occlusive to bacteria and water Change every 1–3 days
Skin protectant films	Cavilon No Sting Barrier Film	Skin irritation, chronic wounds, burns, hot spots	Forms a film over surface; protects skin from urine, feces, and tape; allows rapid healing Reapply every 3–4 days
Semiocclusive to Occlusive Nonadherent Dressings			
Calcium/calcium-sodium alginate (pad, ribbon, fiber)	Curasorb, Sorbalgon	Transition from inflammatory to repair phase, heavy exudate; burns; lacerations; incisions; biopsy	Pad forms a gel when absorbing exudate, which cleans the wound; it also is hemostatic and encourages epithelialization and granulation Do not use overexposed tendon, bone, or necrotic tissue Cover with semiocclusive pad Change every 5–7 days
Paraffin or petrolatum impregnated:	Jelonet, Adaptic	Early repair phase, viscous to sanguineous exudate	Increases wound contraction, absorbs bacteria and exudate, delays epithelialization Change every 1–3 days
With 3% bismuth tribromophenate	Xeroform		
Rayon/polyethylene glycol (sheets, gel) or cotton (pad) Perforated polyester film with cotton	Telfa Pads, Dermacea	Early to midrepair phase when epithelialization begins, sanguineous exudate to dry; sutured wounds; minor lacerations	Increases wound contraction, absorbs bacteria and exudate; may promote exuberant granulation Change every 1–3 days

Continued

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TABLE 15.4 Contact Layer Wound Dressings—cont'd

Type	Trade Name	Indication	Action
Polyurethane film	Tegaderm, OpSite, Bioguard, Bioclusive	All healing phases. Partial thickness injuries; wounds with granulation tissue and minimal exudate; may use as covering for hydrogel or hydrophilic pastes or powders	Increases epithelialization; may cause tissue maceration and bacterial proliferation Change every 1–3 days
Hydrocolloid (sheet, paste, powder; some are semiocclusive)	Flexicol, Duoderm	Early repair phase; healthy granulation tissue bed with advanced wound contraction and little to moderate exudate; pressure sores; burns; cavity wounds; degloving	Increases epithelialization and comfort; adhesiveness may reduce contraction; use until granulation tissue fills wound; promotes granulation and may cause hypergranulation; gas permeable but has water-impermeable coating or is totally impermeable; if infection develops under dressing, discontinue Change every 2–3 days
Hydrogel (mesh, paste, gel, sachets)	Hydrasorb, Curafil, Curagel, Intrasite Gel, BioDres, Geliperm, Carravet, Tegagel, Nu-gel, Solugel	Inflammatory, debridement, and repair phases; dry, sloughing, or necrotic wounds; abrasions; blisters; superficial wounds Use with minimal exudates	Absorbs fluid; keeps wound moist; permits autolytic débridement; increases collagenase activity in burns Discontinue when granulation tissue fills the wound; may promote exuberant granulation Promotes contraction Can use to deliver topicals: acemannan (Carravet), hyaluronic acid, chondroitin sulfate (Tegaderm), silver, and metronidazole Change bandage every 4–7 days
Foam			
Silicone Foam Also gel dressing	Cavi-Care, CicaCare, Silastic gel	Same as for polyurethane foam	Same as for polyurethane foam May prevent or reduce formation of exuberant granulation tissue
Biologic Dressings			
Collagen	Helix3 CM	Inflammatory to repair phase	Reduces pain, heat, water loss, and contamination; stimulates epithelialization, collagen synthesis, and granulation; increases contraction (amnion); reduces bacterial numbers; controls exudates Provides scaffold Experimental results are mixed
Chitosan	Hemocon, ExcelArrest XT	Hemostasis	Provides a matrix for hemostasis that is also antibacterial
Porcine small intestinal submucosa (SIS)	Oasis Wound Matrix	Repair phase	Absorbable scaffold for tissue ingrowth
Porcine bladder extracellular matrix	ACell Vet, Scaffold	Repair phase	Absorbable scaffold for tissue ingrowth
Silicone membrane and porcine collagen	Biobrane	Early to mid-repair phase when epithelialization begins, sanguineous exudate to dry; sutured wounds; minor lacerations	Increases wound contraction, absorbs bacteria and exudate; transparent and conforms, but may adhere
Miscellaneous Dressings			
Activated charcoal	Actisorb	Debridement to repair phase with severe infection	Adsorbs bacteria and reduces odor Provides moist wound healing Prevents formation of exuberant granulation
Silver impregnated	Acticoat, Silvercel, Silvasorb, Actisorb, Algidex AG, Aquacel	Inflammatory to repair phase with high bacterial load; burns	Reduces bacteria; strips facilitate drainage Change every 3–4 days
Maltodextrin: powder or gel	Multidex, Algidex AG	Debridement; slow-healing wounds	Cleanses and promotes healing in contaminated and infected wounds; hydrophilic; chemotactic to white blood cells; yields glucose to promote healing; antibacterial and bacteriostatic

resulting in improved perfusion. These dressings quickly convert necrotic wounds to moderately exudating and granulating wounds. Debridement is nonselective, so use is limited to one or two applications; these dressings are usually followed with placement of alginate, hydrogel, or foam dressings once granulation tissue has begun to form. Hypertonic saline bandages should be changed at least every 24 hours to prevent saline dilution and wound desiccation.

Hydrogel

Hydrogels, or water polymer gels (e.g., Hydrosorb), are modified crosslinked polysaccharide formulations (gelatin or polysaccharide)

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available as gels, dry or hydrated sheets, or impregnated gauze. They are hydrophilic and swell when interacting with aqueous solutions; they contain approximately 90% to 95% water when hydrated. Gels are moisture retentive, nonadherent, nonocclusive, and highly comfortable, providing a cool, soothing effect and some pain relief. These dressings are highly absorptive, creating a moist wound environment that promotes debridement, granulation, and epithelialization. They are used to treat dry, sloughing, or necrotic wounds and abrasions, lacerations, burns, and minor skin irritations. Hydrogels are very effective in hydrating a wound, and they increase collagenase activity to facilitate autolytic debridement and promote granulation. They may be used as a vehicle for antibiotics or other antimicrobials, including metronidazole, silver sulfadiazine, and acemannan. Dressings should be cut to the size of the wound to prevent maceration of adjacent tissue. An outer bandage is used to secure the dressing to the wound surface. Bandages are changed every 4 to 7 days.

Hydrocolloids

Hydrocolloids (e.g., Flexicol) are biocompatible hydrophilic polymers, such as sodium carboxymethylcellulose or hydroxyethylcellulose with pectins and gelatin or embedded in an elastic mesh. They may be occlusive or semiocclusive adhesive pads (not to wound bed), pastes, or powders. Pads consist of an inner (often adhesive) layer of thick absorbing hydrocolloid and an outer, thin, water-resistant, bacterial-impervious, polyurethane film. They are flexible and highly absorbent and adhere to both wet and dry tissue. Wound exudates are absorbed, and the hydrocolloid swells, forming a gel on the wound surface. The gel expands, filling the wound cavity, keeping it moist. Hydrocolloids maintain a moist wound environment, promote autolytic debridement, and insulate the wound bed. They increase epithelialization and comfort, but adhesiveness to surrounding tissue may reduce contraction and cause hypergranulation. Dressings are useful on partial- or full-thickness wounds with clean or necrotic bases, including pressure sores, minor burns, or granulating wounds with necrotic tissue, but they are not intended for heavily exudative or infected wounds. Hydrocolloid pads should extend 2 cm beyond the wound onto normal skin that may be protected with a skin sealant to prevent maceration. Cavitary wounds should be loosely packed with a filler agent to fill dead space before applying a hydrocolloid pad. The hydrocolloid dissolves as it absorbs fluid, forming a yellow-colored fluid. Bandage changes are typically needed every 3 to 5 days, when exudates begin to leak or the dressing loosens. Wound inspection is difficult as the pads are not transparent. Use of these dressings should be discontinued if infection develops under them.

Some hydrocolloid dressings have an adhesive layer and are occlusive (e.g., DuoDerm). The hydrocolloid adheres to skin around the wound, and the dressing over the wound interacts with the wound fluid to create a nonadherent, occlusive hydrocolloid gel. These dressings are used on wounds with an established granulation tissue bed, advanced contraction, minimal fluid production, and initial epithelialization. The wound should first be lavaged and dried. The hydrocolloid dressing then is warmed between the palms to make it soft and pliable. The dressing is cut to an appropriate size and shape to cover the wound, and the protective film is removed. The dressing is applied with light pressure until it adheres to the skin. Adherence is a problem in dogs and cats but may be improved by clipping the hair adjacent to the wound. It may be necessary to apply a light intermediate and outer bandage layer to hold the hydrocolloid dressing in place over mobile areas. The dressing is removed after 2 to 3 days when the outer surface of the hydrocolloid feels like a fluid-filled blister or if leakage occurs. The gel is lavaged or wiped from the wound, and a new dressing is applied. Wounds dressed with hydrocolloid contact layers are less painful and epithelialize more rapidly than wounds covered by semiocclusive, nonadherent dressings; however, wound contraction is reduced. The dressings are not transparent, which makes wound monitoring difficult.

Calcium Alginate

Calcium alginates (e.g., Curasorb, Sorbalgon) are nonocclusive, nonadhesive, hydrophilic, moisture-retaining dressings. They are classified as fibrous dextranomers. Calcium alginate products are derived from salts of alginic acid obtained from the algae *Phaeophyceae*, found in seaweed, forming pads, ropes, ribbons, or packing whose fibers are either calcium sodium alginate or pure calcium alginate. Calcium alginate is used as a hemostatic agent for lacerations, postoperative wounds, donor sites, nasal sinuses, or dental alveoli; a formulation in combination with zinc improves this ability. Calcium alginate is used as a wound filler or dressing that stimulates granulation in wounds after they have been debrided, and increases epithelialization. These dressings also absorb moderate to large amounts of exudate (20–30 times its weight in fluid) and are appropriate during the inflammatory or repair phases of healing for wounds. Ion exchange with the exudates causes a biodegradable viscous hydrogel to form on the surface of the wound. The released calcium ions and a phospholipid surface promote the activation of prothrombin in the clotting cascade. Placement of calcium alginate dressings overexposed muscle, tendon, bone, or dry necrotic tissue is not recommended. Bandages are changed every 1 to 5 days depending on the amount of wound exudates.

Foams

Polyurethane foam dressings (e.g., Copa, Permafoam) are highly absorbent, hydrophilic dressings with a nonadherent contact surface. Some have adhesive borders to adhere to normal adjacent skin. They are highly conforming, vapor permeable, and an effective barrier to bacterial penetration. These dressings are designed to protect and cushion a variety of wounds, especially those in difficult areas. Products vary in conformability, permeability, and absorbency. They maintain a moist wound environment, support autolytic debridement, and promote epithelialization; therefore they are appropriate for partial- or full-thickness wounds with small to moderate amounts of exudates, moist necrotic wounds, or clean granulating wounds. The foam pads are easy to apply and can be cut to fit the wound dimensions. The foam should be wetted with saline or medications if the wound is mildly exudative, or it can be used with another contact layer to prevent adherence. Dressing changes are recommended every 1 to 5 days depending on wound exudation.

Antimicrobial (Polyhexamethyl Biguanide) Gauze Dressings

Sterile gauze sponges, roll gauze, or nonadherent pads impregnated with polyhexamethyl biguanide (Kerlix AMD sponge or roll, Telfa AMD nonadherent dressing) may be used alone or on top of another contact layer. Polyhexamethyl biguanide is a polymeric biguanide with antimicrobial action against gram-positive and gram-negative bacteria. Susceptibility to biguanides varies among bacterial species, with greater efficacy noted against gram-positive bacteria. Polyhexamethyl biguanide is rapidly bactericidal at high concentrations, causing disruption of bacterial

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cytoplasmic membranes with leakage and precipitation of cell contents. It is tissue compatible and does not have any apparent negative effects on wound healing. It is similar to chlorhexidine and is used in contact lens solutions, disinfectants, antiseptics, and other products to control bacterial growth. Dressings impregnated with polyhexamethyl biguanide resist bacterial colonization on wounds and have prolonged local activity. Environmental bacteria are inhibited from penetrating the bandage to gain access to the wound, and bacteria on the wound are inhibited from contaminating the environment. Both gauze sponges and rolls are available. Premoisten the gauze if it is used as a packing material.

Other Antimicrobial Dressings

Dressings impregnated with iodine, chlorhexidine acetate, activated charcoal, antibiotics, and silver are also available to aid in the prevention and control of infection. Iodine is found in crosslinked polymerized dextran dressings. As the dressing hydrates in the moist wound environment, elemental iodine is released to exert an antibacterial effect and to interact with macrophages to produce TNF-α and IL-6. The slow release is designed to maintain adequate levels of active iodine for approximately 48 hours.

Silver-impregnated dressings (e.g., Acticoat) release silver from the dressing over time to kill bacteria. Bacterial susceptibility includes *E. coli, Klebsiella pneumoniae, P. aeruginosa, Streptococcus* spp., and *Staphylococcus* spp., including methicillin-resistant *Staphylococcus aureus*. These dressings are most effective during the inflammatory and repair phases of wound healing, and they are of great benefit in the management of burn wounds (see p. 237). Numerous forms of silver-impregnated dressings are available, as silver is often combined with other agents (e.g., alginate [Silvercel], activated charcoal [Actisorb], hydrogel [Silvasorb], maltodextrin [Algidex AG], and foam [Aquacel Ag Foam]).

Activated charcoal dressings (Activate, Actisorb) provide a moist wound healing environment, absorb bacteria, prevent exuberant granulation tissue formation, and may reduce wound odor. They are intended for heavily infected wounds during the debridement phase to the repair phase of wound healing.

Antibiotic-impregnated collagen sponges are used in human surgery to treat or prevent surgical site infections. CollaRx is made from denatured type 1 bovine collagen impregnated with gentamicin. In addition to its antibacterial effects, these sponges exert a hemostatic effect by causing the adhesion and aggregation of platelets and bridge proteins. The fully biodegradable material releases high levels of antibiotic at the implant site, whereas serum levels remain below toxic levels.

Bioactive Dressings

Bioactive dressings are composed of materials that originate from living tissue. Bioactive dressings include those derived from fibroblasts by tissue cell cultures, artificial skin derived from keratinocytes, and extracellular matrix dressing derived from the submucosal layers of the porcine small intestine or urinary bladder, as well as biologic dressings such as skin grafts, amniotic membrane, and peritoneum. Biologic dressings may serve as contact layers on wounds with minimal exudate.

Collagen dressings (Helix3 CM) are derived from bovine or swine material and rendered nonantigenic as a result of enzymatic purification. They are available in sheets, particles, pastes, or gels. They are believed to accelerate wound repair by the provision of a matrix for cellular migration. They are used most often on large avulsion wounds as a scaffold for tissue ingrowth. Extracellular matrix scaffolds have an angiogenic effect and recruit stem cells to migrate into the acellular scaffold to reconstruct and remodel severely damaged or missing tissue.

Porcine small intestinal or urinary submucosa (Oasis Wound Matrix, ACell Vet Scaffold) can be used as a wound dressing to provide a biocompatible, absorbable scaffold for tissue ingrowth.

Chitosan dressings (Hemcon Patch or Bandage, ExcelArrest XT) are derived from the chitin, a natural polysaccharide from the exoskeleton of shellfish. This biodegradable dressing provides an antibacterial barrier; however, its primary use is for hemostasis.

Biobrane is a temporary wound dressing composed of an ultrathin, semipermeable silicone membrane bonded to a flexible, knitted nylon fabric with pores. The two layers are covalently bonded to porcine collagen peptides to increase wound adherence. Biobrane is flexible and stretches to conform to the area. It is incorporated into the wound and is therefore comfortable. The wound can be seen through the dressing, and minimal fluid collects. The dressing is removed when epithelialization is complete or infection is detected. This dressing's primary use is for superficial and partial-thickness wounds.

Transparent Vapor-Permeable Films

Transparent film dressings (e.g., OpSite, Tegaderm, Bioguard, Bioclusive) are thin, transparent, moisture-retentive dressings made of polyurethane. They are comfortable, water resistant, and allow wound inspection without dressing removal. They are adhesive to dry skin but nonadhesive to wounds. They are semipermeable, transmitting oxygen, water vapor, and carbon dioxide, but occlusive to water and bacteria (e.g., *Pseudomonas, Staphylococcus,* and *E. coli*). Drainage collects under the membrane. The moist environment accelerates healing. Uses include superficial or partial-thickness wounds with few or no exudates and dry, necrotic eschars to provide a moist autolytic debridement. They are not used on infected or exudative wounds or thin, fragile skin. They may be used during the entire healing period. They are applied so they extend at least 2 cm beyond the wound margins. Applying a skin sealant on intact surrounding skin before dressing application helps prevent maceration and skin stripping. They do not adhere well to areas with skin folds or unshaved hair and are better suited to people than animals. Adherence can be improved around the perimeter with vapor-permeable film spray.

Intermediate (Secondary) Layer

The intermediate layer of a bandage is an absorbent layer that removes and stores deleterious agents (e.g., blood, serum, exudate, debris, bacteria, and enzymes) away from the wound surface. Bacterial growth is retarded if the bandage allows fluid evaporation and exudate becomes concentrated. The intermediate layer should have capillarity for absorption and should be thick enough to collect fluid. The intermediate layer also pads the wound from trauma, splints the wound to prevent movement, and holds the contact layer against the wound. Absorbent cotton, combine roll, or cast padding may be used. Enough pressure must be applied during application of this layer to eliminate spaces between the wound and contact layer and between the contact layer and intermediate layer. Such spaces allow fluid to accumulate, which promotes tissue maceration; however, excessive compression impairs absorption and interferes with blood supply and wound contraction. The outer layers of the intermediate layer can be

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made nonabsorptive by applying petrolatum, which inhibits environmental fluid from reaching the wound.

Outer (Tertiary) Layer

The tertiary layer holds the other bandage layers in place and protects them from external contamination. Roll gauze or surgical adhesive tape are used for the outer bandage layer. Porous tape allows fluid evaporation and promotes dryness, but it allows surface bacteria to contaminate the wound when it becomes wet. Conversely, wound bacteria can migrate through taped bandages and contaminate the environment. Using antimicrobial roll gauze (Kerlix AMD sponge or roll) to compress the intermediate bandage layer helps control bacteria on the wound surface and helps prevent environmental organisms from migrating through the bandage to the wound. Waterproof tape protects the wound from environmental fluids but creates an occlusive bandage that may lead to tissue maceration. It should be used only in areas predisposed to getting wet (e.g., the feet), and it often is used in combination with porous tape. Elastic adhesive tapes (e.g., Elastikon) apply pressure, conform to the area, and immobilize it. Support rods or splints may be incorporated into the outer layer of the bandage if additional immobilization is required. Custom-made or modified garments (e.g., VetGood Protective Medical Pet Suit, Suitical Recovery Suit, Surgi Snuggly, Surgi-Sox, DogLeggs) can be used to protect or replace the tertiary layer of bandages applied to the body or head. Elizabethan collars, side or body bars, and tape hobbles are commonly used to prevent the patient from chewing on the bandage.

TYPES OF BANDAGES

When applying bandages, appropriate materials of adequate width should be used to prevent a tourniquet effect. Porous materials allow air to circulate and moisture to escape. All bandages should be applied as smoothly as possible to prevent ridges and lumps, which may cause irritation and skin necrosis. Each turn of the bandage should overlap the previous turn by 50%. It is sometimes advantageous to unroll elastic tapes and allow them to relax before encircling a body part to prevent application of excessive pressure. Owners must be instructed on proper bandage care. Bandages should be evaluated frequently for signs of slippage or strikethrough. The surface of all bandages should be kept clean and dry. Patients should be observed for discomfort, swelling, hypothermia, skin discoloration, dryness, or odor, which may indicate that the area has been bandaged improperly. Bandages applied too tightly impair circulation and damage soft tissue. Digits should be exposed when extremity bandages are applied to allow sensation and circulation to be monitored. Loose bandages cause pressure sores or slippage. Patients should be restrained from chewing at the bandage, and exercise should be limited to short leash walks. When the patient is outside, bandages should be covered with a plastic bag or other waterproof material to protect them from dirt and moisture. The waterproof material should be removed within 30 minutes to prevent excess accumulation of moisture under the bandage.

Signs of pain (i.e., excessive licking or chewing at the bandage) and swelling may indicate an ill-fitting bandage, which should be replaced to prevent serious complications. Bandages that apply too much or uneven pressure, especially over bony prominences, may lead to ischemia and pressure necrosis. If deep tissues are affected, amputation of a limb may be necessary. Skin grafting or flaps may be necessary to reconstruct more superficial injuries.

Tie-Over Bandages

The contact and absorbent layers of a bandage can be held in place with a tie-over bandage when the wound is in an area inaccessible to standard bandaging techniques (e.g., hip, shoulder, axilla, or perineum) (Fig. 15.9).

Place several sutures (e.g., 2-0 nylon, 0 nylon, or polypropylene) in the skin surrounding the wound, tying them with a loose loop. Apply an appropriate contact layer and an intermediate bandage layer on the wound. Hold these layers in place by lacing sterile gauze or umbilical tape through the loose skin sutures. As an alternative, staple long strands of suture 2 to 3 cm from the wound edges around the periphery of the wound, then tie or clamp these sutures over the bandage to hold it in place. Cover the area with an outer bandage layer if possible (Fig. 15.10).

Stabilizing Bandages

Stabilizing bandages help immobilize fractures to minimize further tissue damage during transport for definitive fracture fixation. These bandages are heavily padded and are often referred



FIG. 15.9 (A) A large open wound on the lateral thigh of a 6-year-old Greyhound. (B) A tie-over bandage is used to manage the wound.

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FIG. 15.10 Apply several sutures or skin staples with loose loops around the periphery of wounds to create a tie-over bandage in areas inaccessible to standard bandaging techniques. Apply the primary and secondary bandage layers, then hold the tertiary layer in position by lacing umbilical tape or heavy suture through the loose skin sutures or staples.

to as Robert Jones bandages (see p. 981 for application technique). After fracture fixation with splints, external fixators, or internal fixation, stabilizing bandages may be used to support injured tissue, reduce swelling, and treat open wounds. The type of wound and condition of the tissue determine the type of bandage applied to the wound.

Postoperative or Closed Wound Bandages

Bandages may be applied to areas without an open wound to absorb fluid from a drain or incision line, to support the incision, to compress dead space, to apply pressure, or to prevent trauma or contamination. These bandages improve the patient's comfort by supporting wounds.

Place a nonadherent, absorbent dressing over the incision line and several layers of wide-mesh, absorbent gauze over drains. Determine the thickness of the intermediate layer based on the amount of drainage expected. Be sure to use adequate padding over the end of the drain to prevent strikethrough. Assess the character and amount of drainage with each bandage change.

Incisions expected to have minimal drainage may be protected from environmental contaminants with a nonadherent absorptive pad, which is incorporated into an adhesive polyurethane film dressing.

Pressure Bandages

Pressure bandages facilitate control of minor hemorrhage, edema, and excess granulation tissue. Direct application of a glucocorticoid ointment to the wound may help control excess granulation tissue. The more convex the surface, the greater the pressure exerted by the dressing on the tissue.

Apply an absorbent, nonadherent contact layer over the area of hemorrhage or excess granulation tissue. Use a thick, absorbent intermediate layer and elastic adhesive tape for the outer bandage layer. Wrap the elastic tape carefully to prevent excess pressure, which can impair arterial, venous, and lymphatic circulation and cause tissue necrosis or nerve damage. Check for discomfort, swelling, hypothermia, dryness, or odor, which may indicate that the area has been bandaged too tightly. Remove the bandage within 24 hours if it was applied to control hemorrhage.



FIG. 15.11 Pressure relief bandage applied over the greater trochanter in a dog. The bandage is made from a firmly rolled towel that is cut, taped, and applied over the bony prominence.

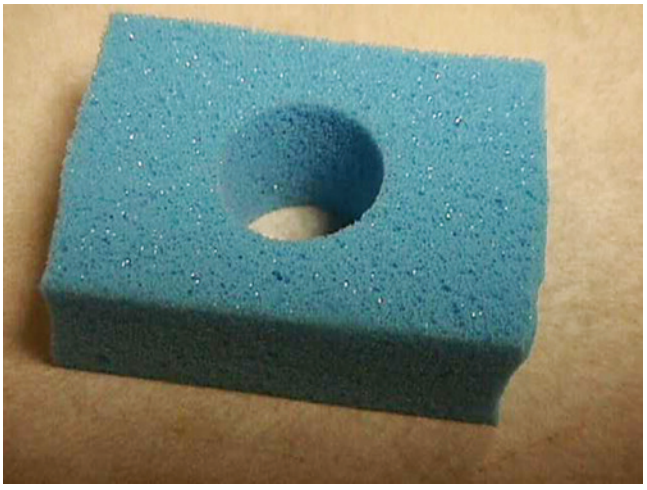


FIG. 15.12 A hole has been cut through this foam sponge to distribute pressure around a pressure sore or wound.

Pressure Relief Bandages

Bandages designed to prevent pressure over an area (usually a bony prominence) are used to treat or prevent pressure sores (see p. 246). Preventing pressure encourages healing over bony prominences. Most pressure relief bandages use a doughnut-shaped bandage, foam, or pipe insulation (Fig. 15.11) to distribute pressure around rather than over the wound. The bandage should be large and thick enough to prevent pressure over the bony prominence. A circular opening may be created in the bandage and used to treat the wound without removing the entire bandage.

A doughnut-shaped bandage may be created from cast padding, medium-density foam sponge, or a small towel (Figs. 15.11 and 15.12). *Create a doughnut-shaped bandage by rolling a towel or cloth into a tight cylinder, securely taping it to maintain the roll, and forming it into an appropriately sized circle. Create a doughnut-shaped bandage from cast padding by folding several layers together and cutting a hole in the center to accommodate the bony prominence or to surround the wound. Create a doughnut opening in thick foam by cutting a hole in the center to accommodate the bony*

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prominence or to surround the wound. Center the doughnut-shaped bandage over the lesion or bony prominence and secure it to the skin with tape so that it does not slip.

These bandages may be difficult to maintain in position, and taping directly to the skin may cause skin irritation. Pipe insulation bandages are usually used to protect the olecranon. *Create bandages from foam rubber pipe insulation tubes by splitting the tube and cutting a hole where the bony prominence will lie. If necessary, use two or three thicknesses of pipe. Stack and tape the pieces of pipe together. When using the bandage over the olecranon, first pad the cranial surface of the radiohumeral joint with cast padding to prevent joint flexion and to keep the dog from lying in sternal recumbency. Then tape the cast padding and pipe insulation in place. Use a spica type of bandage (see the discussion on bandaging extremities below) if necessary to hold the bandage in position. When using the doughnut to redistribute pressure from a wound, first apply the contact and intermediate layers of the bandage, then incorporate the doughnut in the outer bandage layer.*

BANDAGING TECHNIQUES

Bandaging the Thorax and Abdomen

The thorax and abdomen are often bandaged to cover wounds, surgical incisions, or drainage devices. These bandages should be applied firmly, but without constricting the chest or abdomen. Abdominal pressure bandages are occasionally used when abdominal hemorrhage is suspected. Their effectiveness lasts only 1 to 2 hours, and they should be removed within 4 hours. When placing an abdominal pressure bandage, bandage layers should be applied firmly. A rolled towel can be placed along the midline to reinforce the bandage before tape is applied.

Apply a nonadherent contact layer over the incision or wound. Hold the contact layer in position with combine rolls, cast padding, or cotton. Use padding, gauze, and tape rolls 3 to 6 inches (7–15 cm) wide and wrap the padding circumferentially around the torso with slight pressure. Overlap each wrap by approximately one-half to one-third the width of the roll. Increase the thickness of the intermediate layer with increasing amounts of expected drainage. Reduce rostral or caudal slipping of the bandage by wrapping the intermediate and outer bandage layers between the legs and over the shoulders or hips in a crisscross fashion. Encircle the torso with one wrap of bandage material, then direct the bandage from the right inguinal area (axillary area) to the left perineal area (shoulder area). Encircle the torso again and continue across the right perineal area (shoulder area), through the left inguinal area (axillary area), to the left flank (thorax). Repeat the crisscross pattern several times. Also reduce slippage by adhering 0.5 to 1 inch (1–3 cm) of tape to the hair. Do not wrap the bandage so tightly that thoracic expansion is inhibited. Hold the intermediate layer in place with elastic tape gauze or stockinette. Cut a length of stockinette (3 inches [7.5 cm] for cats and small dogs; 4–6 inches [10–15 cm] for medium and large dogs) slightly longer than the length of the body from head to rump. Cut small holes in the stockinette to accommodate the legs. Place the stockinette over the head, and pull the front legs through the leg holes before rolling the stockinette caudally. Pull the hind legs through the leg holes. Secure the bandage with tape. In male dogs, cut a hole in the bandage to accommodate the prepuce or divert urine with a catheter to keep the bandage dry.

Bandaging the Head

Most head bandages are placed to protect an ear that has been traumatized or has surgical incisions (Fig. 15.13). Similar bandages can be used to cover the eye. Head bandages may interfere with breathing if they are applied too snugly or with neck flexion. A properly applied bandage should allow insertion of the fingers between the bandage and the chin to allow room for neck flexion without airway obstruction. If the bandage is too tight, an incision can be made partway across the bandage under the chin. Leaving one ear out of the bandage helps to keep the bandage from sliding. Extreme caution should be used when removing the bandage to prevent laceration or amputation of the pinna. Commercial products are available (e.g., Surgi-Sox Aural Compression Bandage, DogLeggs).



FIG. 15.13 A head bandage is being used to manage multiple head and neck bite wounds in a 5-month-old German shepherd. The tape muzzle was placed to manage maxillary and mandibular fractures.

Apply 1-inch (3 cm) porous tape directly to the edge of the pinna to form a stirrup (see Fig. 17.23). Fold the ear over an absorbent pad or gauze sponges onto the dorsum of the head, and wrap the tape around the head to secure the ear in position. Using a similar technique, pad and place the opposite pinna over the first pinna if indicated. Place a nonadherent contact layer over an incision or place gauze sponges over the end of a passive drain. Hold the pinna and contact layers in position with 2- to 3-inch (5–7.5 cm) cast padding or cotton roll. Encircle the head, passing the rolls of bandage material cranial and caudal to the opposite ear unless both ears are immobilized. Starting under the chin, wrap loosely and overlap each wrap by approximately one-third the width of the roll. Cover this intermediate bandage layer with overlapping wraps of elastic gauze or stockinette. To prevent slippage, secure the bandage in position with elastic tape attached to the skin and hair at the cranial and caudal edges of the bandage. If it will be necessary to medicate the ear, cut holes in the bandaging over the external acoustic meatus.

Bandaging the Extremities

A soft, padded extremity bandage is used to cover abrasions, lacerations, or incisions and can be modified to accommodate splints for joint or bone immobilization. Modifications of the basic padded bandage may be necessary to allow immobilization, prevent slippage, or protect digits. Immobilization is accomplished by placing a spoon splint, molded thermoplastic splint, fiberglass splint, or aluminum rods between the intermediate and outer bandage layers. These materials may sometimes replace the outer layer. It is important to ensure adequate padding at the ends of the splint material to

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prevent skin irritation. Placing additional padding over possible pressure points under a splint or bandage increases the pressure on these points when compressed; therefore it is better to use a doughnut pad to redistribute the pressure to a larger area surrounding the pressure point (see Figs. 15.11 and 15.12).

Begin by applying a 1-inch (2.5 cm) porous tape stirrup to the dorsal and ventral or medial and lateral surfaces of the paw (Fig. 15.14A). Extend stirrups 3 to 8 inches (7.5–20 cm) beyond the digits to help prevent the bandage from slipping distally. If necessary, use a loose layer of elastic gauze to help secure the stirrups. Insert small pledgets of cotton or other absorbent material between the digits and the metatarsal-metacarpal pads and digital pads. Apply an appropriate contact layer over the wound (see p. 195). Snugly apply cast padding around the paw beginning at the level of the second and fifth digital pads. Wrap obliquely so that the third and fourth digits protrude slightly beyond the bandage (see Fig. 15.14B). Overlap the cast padding (2- to 3-inch width [5–7.5 cm]) one-half to two-thirds of its width as it is advanced up the leg. Continue the bandage to the proximal radius and ulna (tibia and fibula) or above the elbow (stifle), depending on the site of the injury. Use enough padding to create the bulkiness necessary for protection. Snugly wrap elastic gauze (2- to 3-inch width [5–7.5 cm]) over the cast padding to conform the padding to the limb, overlapping each turn by one-half the width of the material. Separate the tape stirrups and attach them to their respective sides of the bandage (see Fig. 15.14C). Apply an outer layer of elastic tape (2- to 3-inch width), overlapping one half the width with each turn (see Fig. 15.14D). Avoid overstretching the tape to prevent compromising limb circulation. Check exposed digits three and four frequently for swelling, coolness, and discomfort; remove the bandage and evaluate the limb if these signs are observed.

Eliminate pressure from digital pad injuries by using a triangular piece of medium-density foam cut to the size of the carpal or tarsal pad or a medium-density foam doughnut to protect carpal or tarsal pad injuries (see Fig. 15.12). Placing a solid foam pad outside the intermediate bandage layer before the outer tape layer is placed helps to relieve pressure from the digits. Similarly, place the doughnut before applying the outer bandage layer.

Slippage can be prevented by extending the bandage to encircle the shoulder and thorax (hip and caudal abdomen), creating a spica-type of bandage (Fig. 15.15). This bandage immobilizes the shoulder or hip in addition to the more distal joints and often incorporates splint material. The intermediate and outer bandage layers crisscross cranial and caudal to the affected limb and caudal and cranial to the contralateral limb (as described on p. 206) for abdominal and thoracic bandages. The bandage is reinforced with splint rod, fiberglass casting tape, or thermoplastic splint material if fractures are to be temporarily stabilized or if additional wound immobilization is desired.

Temporary immobilization of injuries below the elbow or stifle can also be accomplished by applying a Robert Jones bandage or a modified Robert Jones bandage. A Robert Jones bandage is a large, bulky bandage that provides stabilization by applying compression to a thick cotton layer (see p. 981). A modified or light Robert Jones bandage has much less cotton padding, making it less bulky. Modified Robert Jones bandages are used to reduce limb edema after surgery.

Onychectomy, digit amputation, or pad reconstruction may benefit from a bandage to protect the digits and reduce hemorrhage. In these cases, stirrups should be applied laterally and the digits covered with gauze sponges or a nonadherent contact layer.

Reflect layers of 2-inch (5 cm) cast padding from dorsal to ventral and then ventral to dorsal over the end of the paw (Fig. 15.16A). Extend the cast padding in a spiral pattern to the midradius and midulna (tibia and fibula). Leave the proximal ends of the tape stirrup exposed to aid bandage removal. Cover the cast padding with elastic gauze. Fold the tape stirrups to their respective sides. Cover the bandage with tape from the distal extremity to the proximal hair (see Fig. 15.16B).

PRINCIPLES OF PLASTIC AND RECONSTRUCTIVE SURGERY

SKIN TENSION AND ELASTICITY

Reconstructive surgery is commonly performed to close defects that occur secondary to trauma, to correct or improve congenital abnormalities, or after removal of neoplasms. A variety of reconstructive procedures are available; it is important to select the appropriate technique or techniques to prevent complications and avoid unnecessary cost. Although large lesions, particularly those on the trunk, often heal by contraction and epithelialization (see p. 181), wound closure may be preferable. Large or irregular defects can sometimes be closed using relaxing incisions or "plasty" techniques (e.g., V-to-Y plasty, Z-plasty). Large defects or those on the extremities may require that tissue be mobilized from other sites. Pedicle flaps are tissues that are partly detached from the donor site and mobilized to cover a defect (see p. 217); grafts involve the transfer of a segment of skin to a distant (recipient) site (see p. 232). Careful planning and meticulous, atraumatic surgical techniques are necessary to prevent excessive tension, kinking, and circulatory compromise. The amount of skin available for transfer varies between sites on the same animal and between breeds. Little skin can be mobilized in the extremities, whereas advancing adjacent tissue can often close large defects over the trunk. The character of the recipient bed influences the choice of reconstructive technique. Properly developed and transferred local flaps can survive on avascular beds, whereas grafts and distant flap transfers require vascular beds (i.e., healthy granulation tissue, muscle, periosteum, and paratenon).

Hirudiniasis, or the attachment of leeches to skin, can help in reconstructive and microvascular surgery. Leeches are recommended only for tissues with impaired venous circulation. The medicinal leech is *Hirudo medicinalis*. After a blood meal, a leech can go for months without eating. Leeches produce a small bleeding wound that mimics venous outflow. The leech eats an average of 5 mL of blood, but blood oozes from the wound for 24 to 48 hours after the leech detaches because of anticoagulants and vasodilator substances introduced into the wound. There is a significant risk of infection with *Aeromonas hydrophila* when leeches are used.

TENSION LINES AND TENSION RELIEF

The location of the wound, elasticity of surrounding tissue, regional blood supply, and character of the wound bed should be considered when planning reconstructive surgery. Grasping and lifting the skin in the proposed flap or graft area and allowing it to retract spontaneously assesses skin tension and elasticity. Evaluating the amount of tension that can be tolerated by tissue is subjective. Apposing incision edges under too much tension causes incisional discomfort and pressure necrosis, resulting in sutures "cutting out" and partial or complete incisional dehiscence.

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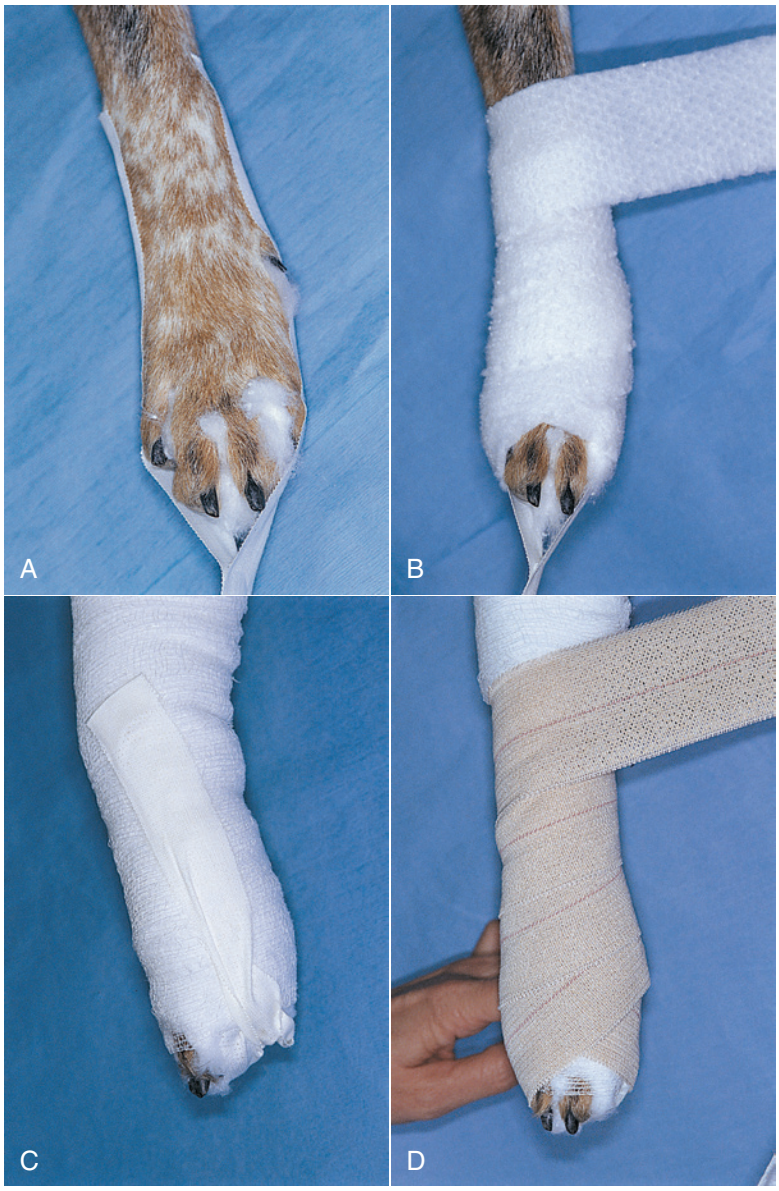


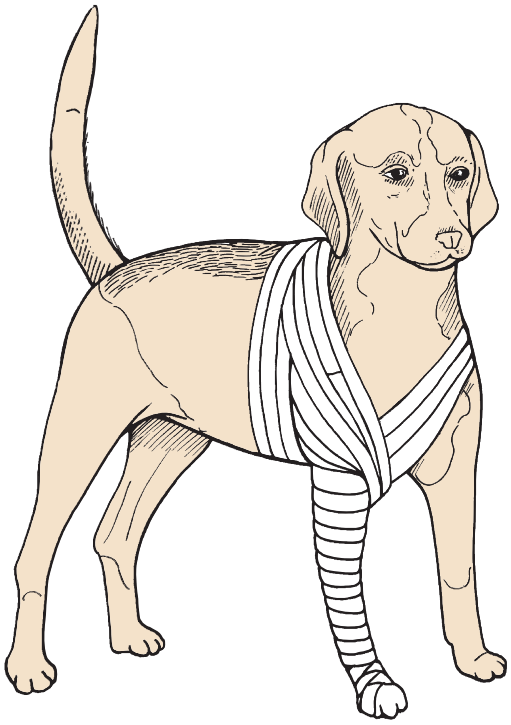
FIG. 15.14 (A) For a leg bandage, apply a 1-inch (2.5 cm) porous tape stirrup to the dorsal and ventral or medial and lateral surfaces of the paw. Insert an absorbent material between the digits and the metacarpal or metatarsal and digital pads. (B) Apply cast padding or cotton over an appropriate contact layer, overlapping wraps by one-half to two-thirds the width of the roll. Keep the third and fourth digits exposed. Conform the padding to the limb by applying elastic gauze. Apply a splint for greater immobilization between the padding and elastic gauze (optional). (C) Fold the tape stirrups over the gauze. (D) Apply an outer layer of 2- to 3-inch-wide (5–7.5 cm) elastic tape, overlapping one-half of the width of the tape with each turn.

Methods of reducing tension include undermining wound edges, selecting appropriate suture patterns, and using relief incisions, skin stretching, and tissue expansion. In addition, the animal is always positioned for surgery, such that mobile skin is not pinned against the table; using pads and flexing the appropriate joints accomplish this. If these methods do not allow primary apposition, wounds may be allowed to heal by secondary intention or may be reconstructed with flaps or grafts.

Tension Lines

Tension lines are formed by the predominant pull of fibrous tissue within the skin. General lines of tension have been mapped in animals, but breed, conformation, gender, and age variations occur (Fig. 15.17). Tension causes incised skin edges to separate and widens linear scars (Fig. 15.18). Incisions should be made parallel to tension lines. Incisions and wounds along tension lines heal better, faster, and with more aesthetic results, whereas those made across tension lines tend to gape. Incisions made at an angle to tension lines take a curvilinear shape. Incisions made across tension lines require more sutures for closure and are more likely to dehisce than those made parallel to tension lines. Traumatic wounds should be closed in the direction that prevents or minimizes tension. Wound edges should be manipulated before closure to determine which direction the suture line should run

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FIG, 15.15 For proximal extremity lesions, continue the bandage up the leg, around the chest or abdomen, and between the legs to create a spica-type bandage.

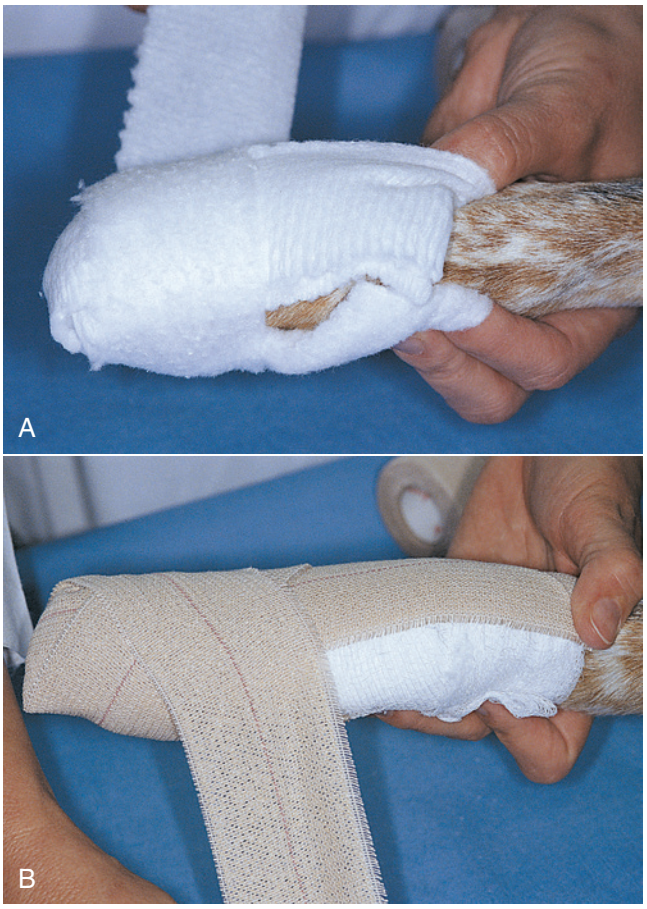


FIG. 15.16 A paw bandage is placed in a manner similar to a leg bandage, except that the digits are covered. (A) After placing stirrups and the contact layer, reflect cast padding over the digits from dorsal to ventral and then ventral to dorsal. Then wrap the padding around the distal limb. (B) Conform the bandage to the limb with elastic gauze and secure the bandage with elastic tape in a similar fashion.

to minimize tension (Fig. 15.19). If tension is minimal, a wound should be closed in the direction of its long axis. The direction of closure should prevent or minimize the creation of "dog ears," or puckers, at the ends of suture lines.

Tension Relief

Skin is undermined by using scissors to separate the skin or panniculus muscle (or both) from underlying tissue. Undermining skin adjacent to a wound is the simplest tension-relieving procedure. It releases skin from underlying attachments so that its full elastic potential can be used as it is stretched over the wound. Skin should be undermined deep to the panniculus muscle layer to preserve subdermal plexus and direct cutaneous vessels that run parallel to the skin surface (Fig. 15.20). Where there is no panniculus muscle layer (middle and distal portion of the extremities), the skin should be undermined in the loose areolar fascia deep to the dermis to preserve the subdermal plexus. Elevated skin should include a portion of the superficial fascia with the dermis to preserve the direct cutaneous arteries. In areas where skin is closely associated with an underlying muscle, a portion of the outer muscle fascia should be elevated with the dermis rather than dissecting these structures. Prevent subdermal plexus injury by using an atraumatic surgical technique, including cutting skin with a sharp scalpel blade instead of scissors and avoiding crushing instruments (e.g., Allis tissue forceps). Brown-Adson thumb forceps, skin hooks, or stay sutures should be used to manipulate skin. Tissue layers are separated by repeatedly inserting Metzenbaum scissors with the blades closed, opening the blades, and then withdrawing the scissors in an open position. Tissue is snipped with the scissors as necessary. As an alternative, the partly opened scissors can be advanced along the cleavage plane without snipping. While undermining, determine if tension relief is adequate by periodically attempting to approximate the skin edges.

Bleeding is usually insignificant during undermining. Excessive bleeding may be controlled with electrocoagulation or ligation; however, skin tension and bandaging usually control hemorrhage and prevent seromas. Undermining areas near wound margins associated with delayed wound closure requires that the epithelialized skin edge be separated from the granulation tissue. The skin should be excised with a scalpel blade at the junction of normal skin and new epithelium. The incision should be continued through the granulation tissue at the normal cleavage line of subcutaneous fascia, deep to the subdermal plexus. Wound closure under excessive tension, rough surgical technique, and division of direct cutaneous arteries interfere with cutaneous circulation and may cause skin necrosis, wound dehiscence, or infection. Surgical manipulation of recently traumatized skin should be minimized until circulation improves. Resolution of contusions, edema, and infection indicates improved skin circulation.

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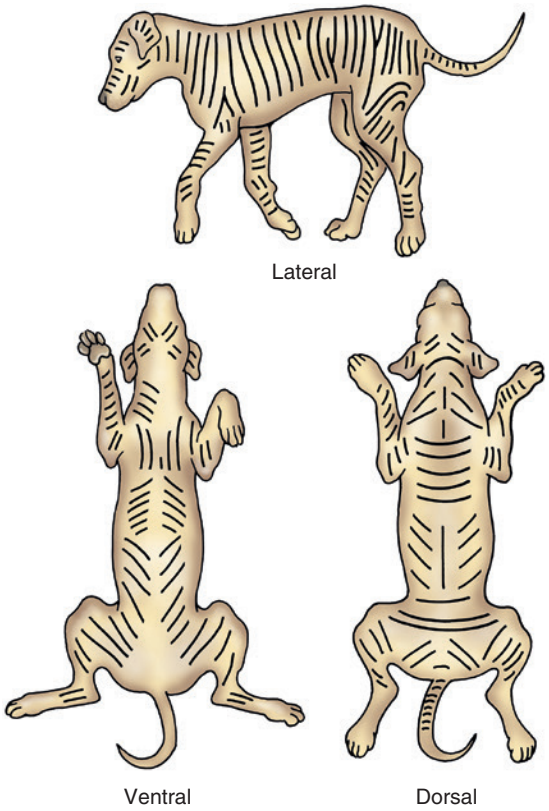


FIG. 15.17 Approximate skin tension lines in dogs. (Modified from Irwin DHG. Tension lines in the skin of the dog. *J Small Anim Pract*. 1966;7:595.)

Skin Stretching and Expansion

Skin stretching and expansion is a technique used in reconstructive surgery that takes advantage of the skin's ability to stretch beyond its natural or inherent elasticity, by the processes of mechanical creep and stress relaxation, when continuous tension is applied. During this process, dermal collagen fibers are stretched, and tissue fluid is slowly displaced from around collagen fibers, which are straightening and compacting longitudinally in the direction of the stretching force. Skin can be prestretched hours to days before surgery to allow closure with less tension at the time of the procedure. Presuturing, adjustable sutures, skin stretchers, and skin expanders are used in this technique (Fig. 15.21). Presuturing is performed 24 hours before surgery to prestretch the skin. Lidocaine, sedation, or general anesthesia is administered

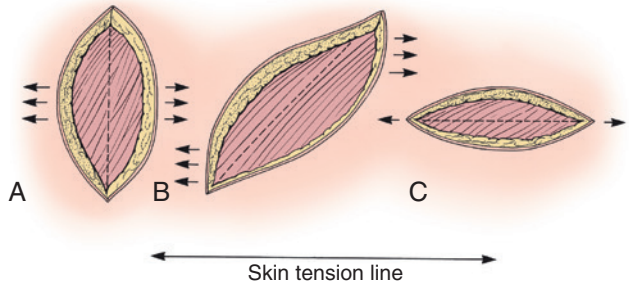


FIG. 15.18 Incisions made across tension lines. (A) Perpendicular or (B) oblique incisions gape and require more sutures for closure than incisions that (C) parallel skin tension lines.

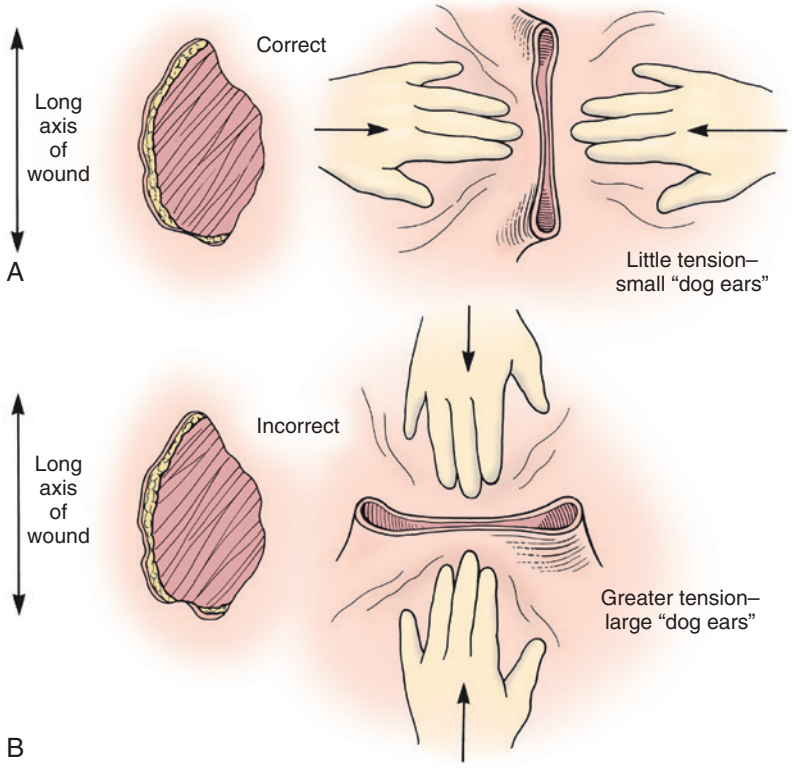


FIG. 15.19 Wound edges should be manipulated to determine the direction of least tension and minimal "dog ear" formation. (A) Wound edges showing little tension and small dog ears. (B) Wound edges showing greater tension and large dog ears.

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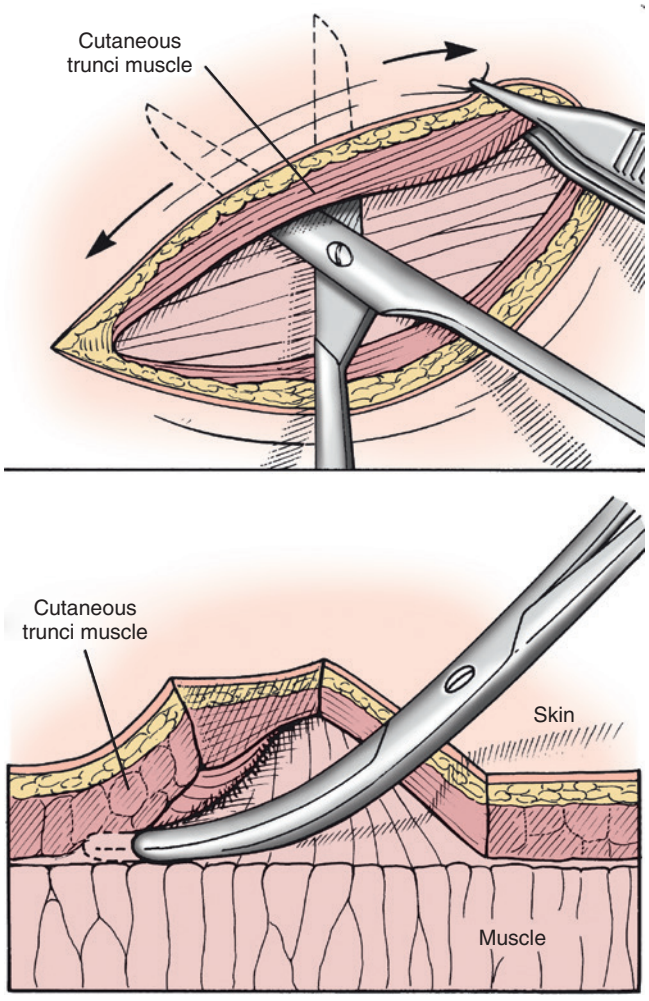


FIG. 15.20 Before wound closure, use scissors to undermine skin and subcutaneous tissue or skin and panniculus muscle and to separate them from the underlying tissue.

before suture placement to relieve discomfort. Tension sutures (interrupted Lembert or vertical mattress sutures of 2-0 or 0 monofilament, nonabsorbable suture material) are placed to imbricate skin on apposing sides of the lesion (see Fig. 15.21). Sutures are placed 3 to 5 cm from the proposed incision site. The sutures exert their effect primarily on healthy skin immediately adjacent to the proposed surgical site, and they are not adjustable. Presuturing is effective only in areas where elastic skin is limited (i.e., extremities). An adjustable horizontal suture can be used to stretch skin over a wound gradually. This is a continuous intradermal suture (2-0 to 3-0 nylon, polypropylene, or polybutester) anchored at one or both ends with a button secured on the skin surface with a split shot fishing weight. On succeeding days, traction on the suture advances the wound edges over the wound, and new weights are applied to maintain tension. These sutures are used primarily on limb wounds in which the wound edges cannot initially be apposed.

Skin stretchers (e.g., X-Banders) are a noninvasive device capable of stretching skin both adjacent to and distant from the surgical site. More skin can be stretched or recruited using this technique than by presuturing or tissue expanders. Skin stretchers are most effective on the neck and trunk. Self-adherent skin pads with a thin coat of additional cyanoacrylate adhesive are applied to clipped, clean, dry skin adjacent to and distant from the surgical site, depending on the amount of stretching needed. Pads are placed 1 to 2 cm from the wound margin with their long axis perpendicular to the direction of skin tension. Elastic connecting cables are attached to pads on one side of the wound and stretched before they are attached to the pads on the opposite side of the wound. An additional row or tier of pads and cables can be placed more distant from the wound if further skin recruitment is required. The cables are adjusted every 6 to 8 hours to generate the optimal high-tension load to accelerate skin stretching or deformation. Sufficient skin may be recruited within 24 to 48 hours, although 96 hours may be required. Pads are peeled from the skin or removed with glue solvent before surgery. Little or no undermining is generally required after skin stretching. Skin stretchers are also used before removal of a mass or after surgery when incisions are closed under excessive tension. Application of a skin stretcher helps alleviate tension and prevent dehiscence (see Fig. 15.21). Pads are generally removed 3 to 5 days after surgery.

Tissue expanders are inflated in subcutaneous tissue to stretch overlying skin, allowing creation of larger flaps for closing defects. They are beneficial in overcoming tissue shortage and obtaining skin with desirable qualities. Tissue expanders reduce dermal thickness and temporarily increase epidermal mitosis. Subcutaneous fat and muscle adjacent to the tissue expander atrophy, and neurapraxia may occur. Tissue expanders have an inflatable bag and reservoir made of silicone elastomer. Although expensive, they are available in various sizes and shapes (e.g., Integra). Careful planning is required for optimal expansion and reconstruction. The base of the expander should approximate the size of the donor site. The incision for insertion of the expander should be made parallel to tension lines at the leading edge of the future flap, or skin adjacent to the site will be inappropriately stretched. The device is placed subcutaneously and inflated with saline. Rapid expansion requires intermittent intraoperative short-term inflation of the expander. It involves inflating the expander for 2 to 3 minutes, deflating and letting the tissue rest for 3 to 4 minutes, and then repeating the cycle two or three times before creating a flap. Gradual expansion involves injecting to a given pressure or volume at intervals spanning days to weeks (usually every 2–7 days). Inflation at injection is continued until the skin feels tense or looks blanched or until discomfort is perceived. When the tissue is sufficiently stretched to allow reconstruction, the device is removed and a skin flap is created to close the defect. Complications of tissue expanders include pain, seroma formation, scar widening, infection, dehiscence, skin necrosis, and implant failure, and they should not be used in previously irradiated tissue. Axial pattern flaps (see p. 222) are preferable to tissue expanders for large wound reconstruction.

SUTURE PATTERNS

Subdermal Sutures

Subdermal fascia is strong and tolerates tension better than subcutaneous tissue or skin. Sutures placed in subdermal or subcuticular tissue reduce tension on skin sutures and bring skin edges into apposition. These sutures also reduce scarring. For subdermal and subcuticular sutures, 3-0 or 4-0 polydioxanone, poliglecaprone 25, or polyglyconate suture on a cutting needle with a buried knot is used.

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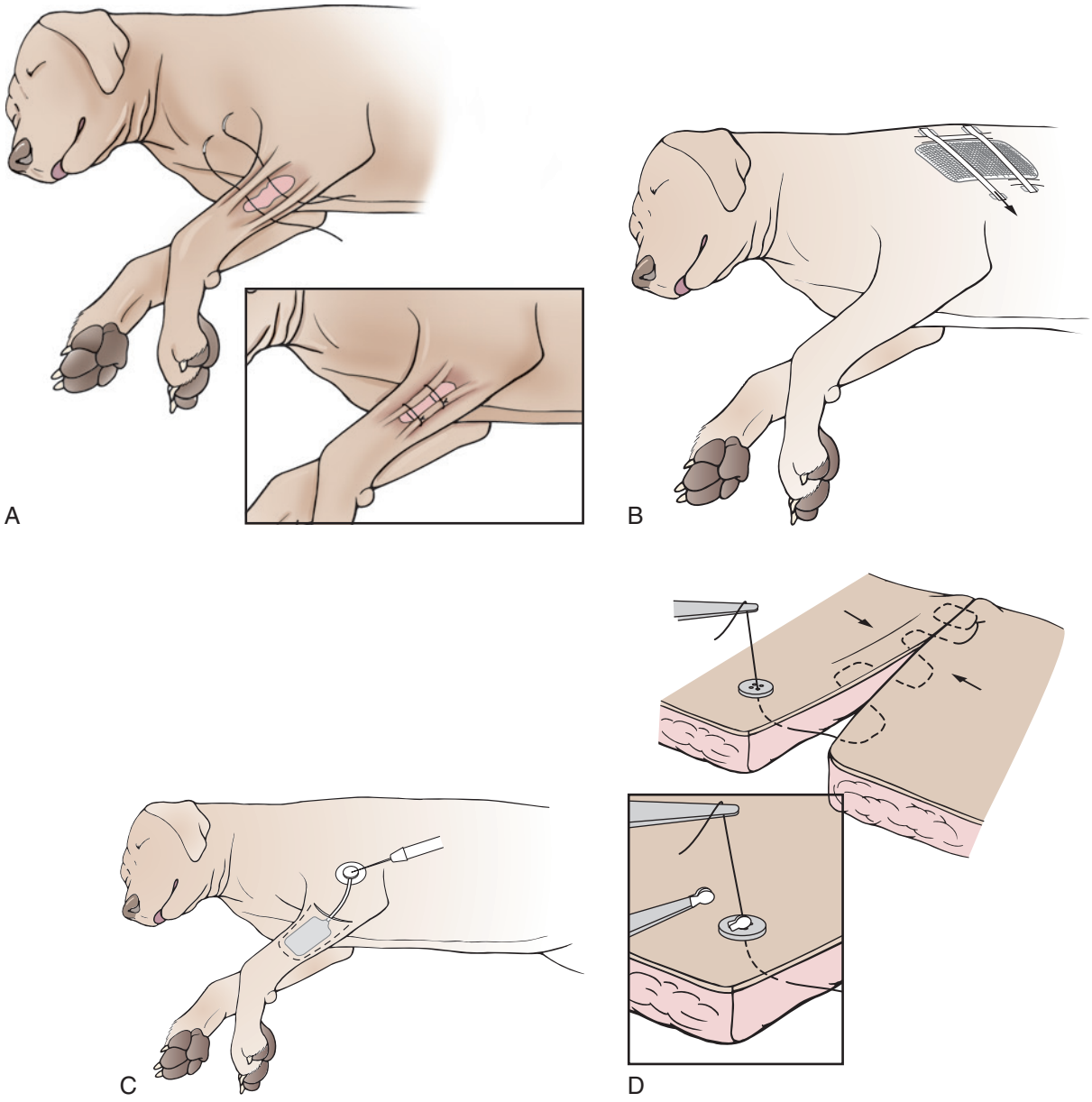


FIG. 15.21 Methods for recruiting skin to close wounds under tension include (A) presuturing, (B) skin stretchers, (C) inflatable tissue expanders, and (D) adjustable sutures.

Walking Sutures

Walking sutures move skin across a defect, obliterate dead space, and distribute tension over the wound surface. Skin is advanced toward the center of the wound by placing rows of interrupted, subdermal sutures beginning at the depths of the wound. The suture (e.g., 2-0 or 3-0 polydioxanone, poliglecaprone 25, or polyglyconate) should be placed through fascia of the body wall at a distance closer to the center of the wound than the bite through the subdermal fascia or deep dermis (Fig. 15.22). Walking sutures do not penetrate the skin surface. Tying the suture advances skin toward the wound center. Walking sutures are placed no closer than 2 to 3 cm apart. Successive rows of walking sutures further advance the skin toward the center of the wound. Sutures are placed on both sides of the defect to advance undermined skin toward the center. The number of walking sutures should be minimized to prevent creating subcutaneous loculi or compromising circulation. Subdermal and skin sutures are used to complete wound closure.

External Tension-Relieving Sutures

External tension-relieving sutures help prevent sutures from cutting out, which occurs when pressure on skin within the suture loop exceeds the pressure that allows blood flow. Pressure is reduced by spreading it over a larger area of skin. Placing sutures farther from the skin edge or using mattress or cruciate sutures helps disperse pressure. Other suture patterns that help relieve tension include alternating wide and narrow bites using simple interrupted sutures or placing pulley sutures, such as "far-near-near-far" or "far-far-near-near" patterns. The standard tension-relieving suture for the skin is called the *vertical mattress*

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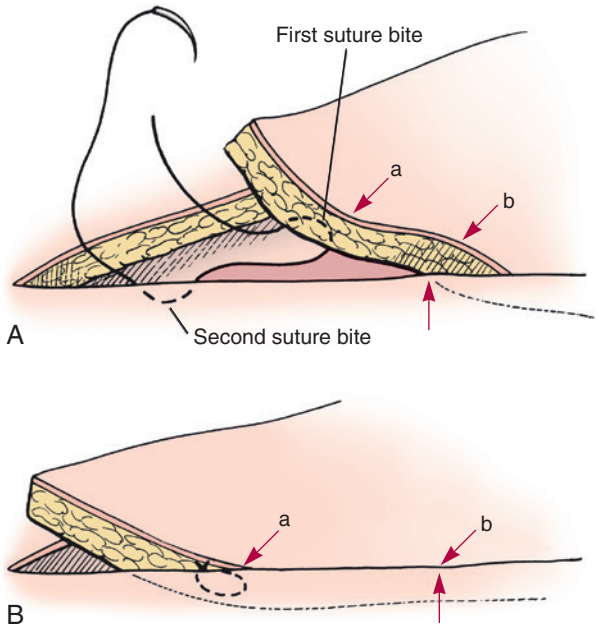


FIG. 15.22 Use walking sutures to advance skin toward the center of the wound. (A) Place the suture through the fascia of the body wall at a distance closer to the center of the wound than the bite through the subdermal fascia or deep dermis. (B) Note that the distance from *a* to *b* increases because of skin stretching when the suture is tied.

suture. A tension-relieving row of vertical mattress sutures should be placed 1 to 2 cm away from the primary row of sutures apposing the skin edges. The vertical mattress sutures (2-0 to 0 polypropylene, polybutester, or nylon) are placed while the skin is approximated with towel clamps or skin hooks before apposition of the skin edges with approximating sutures (e.g., 3-0 or 2-0 polypropylene, polybutester, or nylon). Tension-relieving vertical mattress sutures can usually be removed by the third day after surgery, when collagen fibers have reorganized and fibrin has stabilized the wound edges. Horizontal mattress sutures with or without rubber tubing stents may be used; however, they have a greater potential for impairing local cutaneous blood flow to the skin edges.

PREVENTION OF "DOG EARS"

"Dog ears," or puckers, may be prevented or corrected at the end of a suture line by unequal suture spacing or by resecting a small ellipse or triangle of skin. Placing sutures close together on the convex side of the defect and farther apart on the concave side of the wound may prevent dog ears (Fig. 15.23A). Dog ears may be corrected by outlining with an elliptic incision, removing redundant skin, and apposing the skin edges in a linear or curvilinear fashion (see Fig. 15.23B). As an alternative, the dog ear may be incised in the center to form two triangles; one triangle should be excised and the other used to fill the resultant defect (see Fig. 15.23C–D), or both triangles may be excised and the edges apposed, creating a linear suture line (see Fig. 15.23E). Thin, elastic skin is less prone to the formation of dog ears than thick skin. Many dog ears flatten without excision.

RELAXING INCISIONS

Relaxing incisions, or incisions made near a defect to allow apposition of skin edges, are beneficial in allowing skin closure around fibrotic wounds or over important structures, or before radiation therapy after extensive tumor excision. They are rarely indicated except on distal extremities, around the eyes and anus, or to cover tendons, ligaments, nerves, vessels, or implants.

Simple Relaxing Incisions

Relief incisions heal by contraction and epithelialization in 25 to 30 days. Some relaxing incisions surrounded by loose elastic tissue can be closed primarily after the wound is approximated.

Begin undermining at the edge of the defect at the point of maximum skin tension and continue until the edges can be apposed under tension. Close the wound and make a relaxing incision at the point where undermining stopped or where tension lines are observed (Fig. 15.24A). Begin the incision at the point of maximum skin tension and extend it as necessary to relieve excess tension. If necessary, make the relief incision before skin closure. Place a nonadherent pad over the relief incisions and suture line, followed by a padded bandage. Initially, change the bandage every 1 or 2 days.

Multiple Punctate Relaxing Incisions

Multiple punctate relaxing incisions are small, parallel, staggered incisions made in skin adjacent to a wound to allow closure with reduced tension (see Fig. 15.24B). *Undermine skin around the wound, and place a continuous subdermal suture pattern. Tighten the suture, starting at one end of the incision and working toward the other end. If the skin edges do not appose in an area, make a small incision approximately 1 cm long in adjacent skin on either side of the wound, approximately 1 cm from the wound. If excessive tension persists, make a second row of incisions 0.5 to 2 cm lateral to the first row. Tighten the suture to appose the wound edges; continue the procedure along the length of the wound. Place skin sutures to appose the original wound, then place a nonadherent bandage. Change the bandage daily during the early stages of healing and less often as healing progresses.*

Tension-relieving incisions heal by secondary intention. Multiple punctate relaxing incisions are more cosmetic than single relaxing incisions but provide less relaxation and have a higher risk of causing significant circulatory compromise.

V-to-Y Plasty

V-to-Y plasty is a type of relaxing incision that provides an advancement flap to cover a wound. It is used to close chronic, inelastic wounds or wounds that would distort adjacent structures if closed under tension. It is commonly used in eyelid surgery. A V-shaped incision is made approximately 3 cm from the wound (see Fig. 15.24C). The original wound is closed after undermining skin (see Fig. 15.24D). The V relief incision is closed in the shape of a Y. Closure is begun at the ends of the V until tension develops (see Fig. 15.24E). The remainder of the relief incision is closed as the stem of the Y (see Fig. 15.24F).

Z-Plasty

Z-plasty is a technique that lengthens or relaxes an incision. The Z may be incorporated into the wound, or a separate Z may be made adjacent to the wound to facilitate closure with less tension. The central limb of the Z is the wound or primary incision. The

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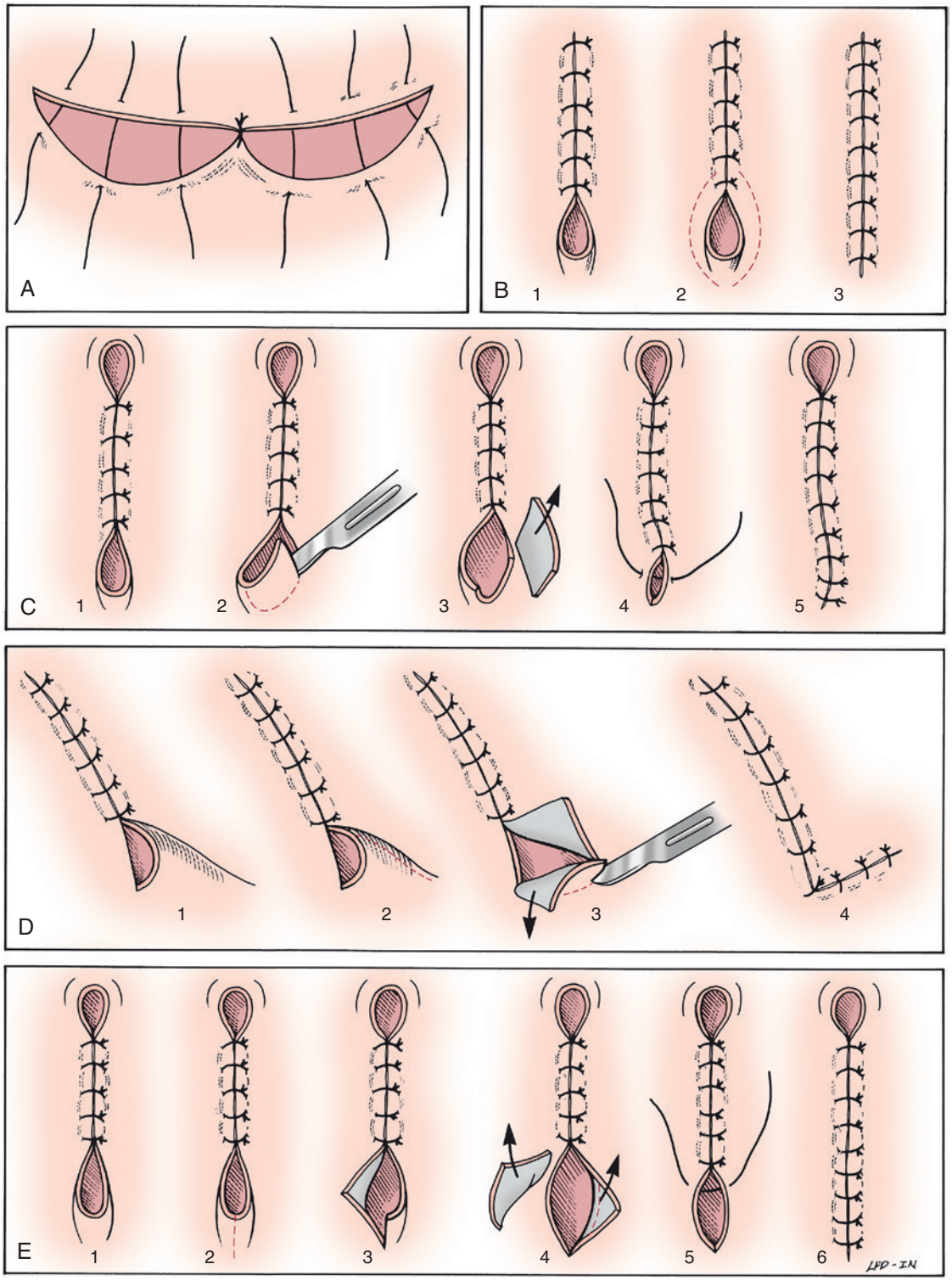


FIG. 15.23 Prevent or correct "dog ears" or puckers at the end of suture lines by (A) using unequal suture spacing or by (B) resecting an elliptic segment of skin, or (C–D) one large triangle, or (E) two smaller triangles of skin.

two arms of the Z are made the same length as the central limb (see Fig. 15.24G). The angles of the Z can vary between 30 degrees and 90 degrees, but 60 degrees is advised. Larger angles give more length gain (45 degrees gives approximately a 50% increase; 60 degrees approximately a 75% increase). Length is gained along the original central limb of the Z when the flaps of the Z are transposed (see Fig. 15.24H). A Z-shaped incision is made with the central arm of the Z parallel to the direction length as needed. The flaps are undermined before transposition and suturing (see Fig. 15.24, *inset*).

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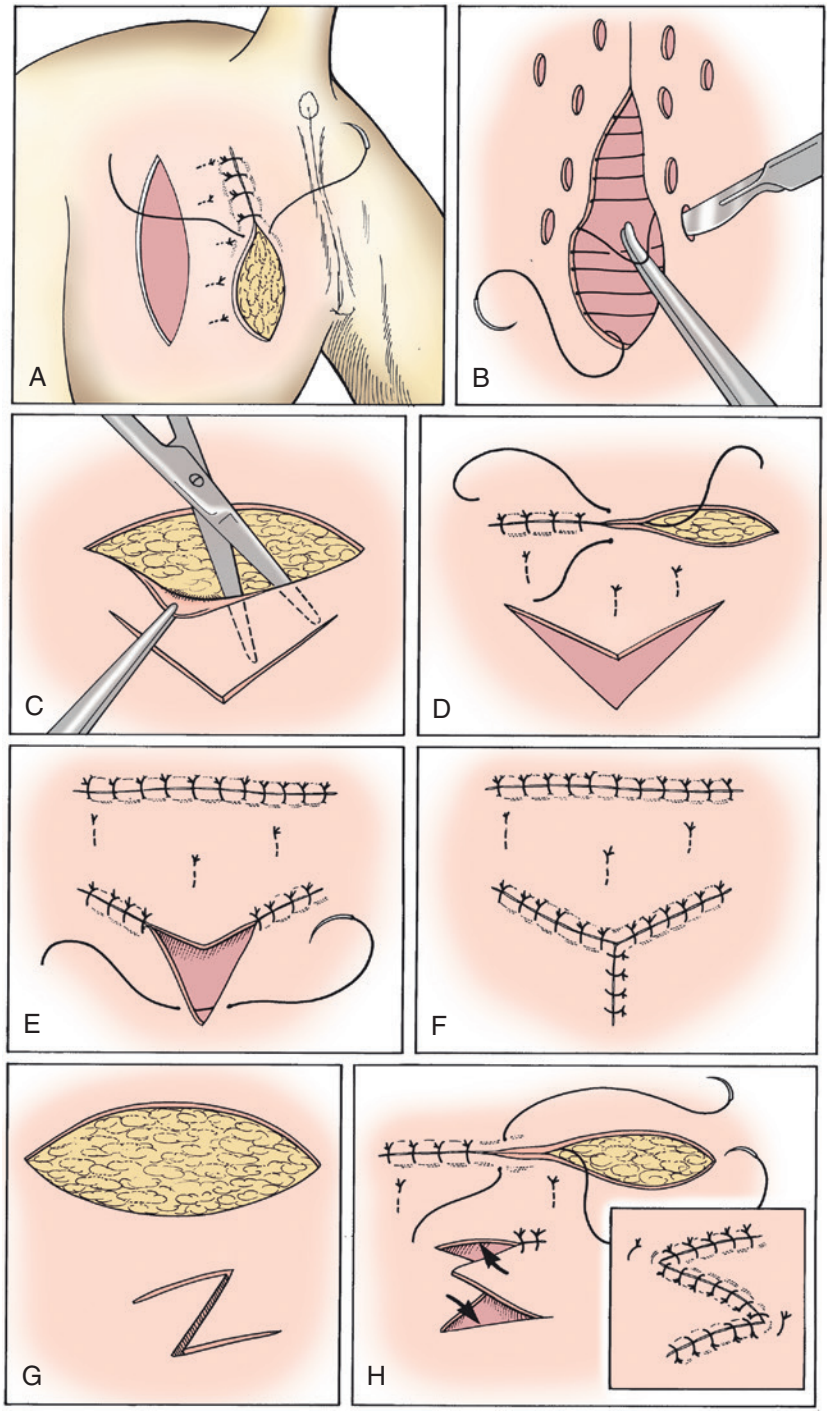


FIG. 15.24 Make relaxing incisions near the defect to allow skin apposition. (A) After undermining the skin, unilateral or bilateral simple relaxing incisions are made adjacent to the wound. (B) After preplacing a continuous subcuticular suture pattern, multiple punctate incisions are made parallel to the wound. (C–F), V-to-Y plasty provides an advancement flap to cover the wound. (G) and (H), A Z-plasty can be made adjacent to or involving the wound to allow wound closure.

SKIN BIOPSY

Skin biopsies are required to diagnosis some dermatologic problems, skin infections, and tumors. With chronic dermatitis, biopsy is sometimes useful only to rule out other disorders such as neoplasia. Secondary skin infections should be eliminated before biopsy, if possible. Biopsies of chronic skin abnormalities should include multiple samples of representative lesions. Usually, biopsy of abnormal skin is sufficient, but samples of normal skin are helpful for comparison when evaluating scaling disorders, primary seborrhea, depigmenting or hyperpigmenting lesions, and alopecia.

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Clip the hair, carefully leaving approximately 0.25 inch (5–6 mm), and avoid inflicting skin trauma. When the primary problem is dermatologic, and not infectious or neoplastic, do not scrub or disinfect biopsy sites because this may adversely affect the pathologist's interpretation. Scrubbing may interfere with identification of the type of keratin and presence of surface crust, scales, microorganisms, or parasites. *Mark the lesions by circling or drawing a 2- to 3-cm line in the direction of the hair growth with an indelible marker. Perform the biopsy with a sharp 6- or 8-mm biopsy punch and a small, sharp-sharp scissors or scalpel blade for cutting the specimen from the subcutaneous tissue. Hold the skin taut around the biopsy site with the thumb and forefinger. Place the punch perpendicular to the skin surface with the lesion in the center. Rotate the punch in one direction while applying moderate pressure until a pop is felt or to the hub of the punch. Apply gentle pressure for hemostasis. Grasp and gently retract the specimen with small tissue forceps; then transect the attached subcutaneous tissue. Blot blood from the specimen, and place it on a tongue depressor or cardboard marked with the direction of hair growth to prevent curling. Appose skin edges (3-0 or 4-0 monofilament nonabsorbable suture) with a single suture.*

Alternatively for bullae, nodules, or deep lesions, make a wedge or elliptic-shaped incision around the lesion or at the junction of normal and abnormal tissue with a scalpel blade. Laser biopsies are not advised as laser-induced artifacts may make small specimens nondiagnostic.

Specimens of tumors can be obtained by incisional, excisional, or needle core biopsies. *Unlike specimens collected for dermatologic disorders, clip the hair and prepare it for aseptic surgery. Perform incisional biopsies such as skin biopsies using a punch or elliptic incision through both normal and abnormal tissue. Perform needle core biopsies using a TruCut biopsy instrument with samples taken from peripheral and central areas of the mass.*

Excisional biopsies are described later and should include all previous biopsy incisions, punctures, and needle tracts. *Immediately after collection, place samples in fixative (10% neutral buffered formalin) to preserve their integrity. Make sure the pathologist can orient the specimen correctly and if necessary mark with ink or sutures. Provide a gross description of the lesion because evidence of erythema is lost in the fixative.* It is usually wise to also submit tissue for bacterial and/or fungal culture.

REMOVAL OF SKIN TUMORS

Before a tumor is removed, skin tension and elasticity should be assessed, but excessive tumor manipulation should be avoided. The direction of skin tension lines, shape of the excision, and method of closure should be planned before surgery. A large area should be clipped and aseptically prepared for surgery, especially if there is a chance that skin flaps may be needed for closure. Excision of skin tumors should include the tumor, previous biopsy sites, and wide margins of normal tissue in three dimensions (i.e., length, width, and depth). For benign tumors, remove the tumor and 1 cm of normal tissue; for malignant tumors, a margin of more than 2 to 3 cm may be necessary for complete local excision (Fig. 15.25). These margins are taken in all dimensions, including the deep margin if feasible. Induration of the periphery of the lesion resulting from a fibroplastic host response may help identify the gross limits of the tumor. The margin distance should be greater for aggressive, infiltrative tumors (i.e., mast cell tumors, melanomas, squamous cell carcinomas, soft tissue sarcomas, feline mammary adenocarcinomas, hemangiopericytomas, infiltrating lipomas). Tumor invasion is affected by the type of surrounding tissue. Tissue easily infiltrated by tumor cells (i.e., fat, subcutaneous tissue, muscle, and parenchyma) should be resected with the tumor. Cartilage, tendon, ligaments, fascia, and other collagen-dense, vascular-poor tissues are resistant to neoplastic invasion and therefore are often spared during resection. Excision of infiltrative or aggressive tumors should extend at least one fascial layer below the detectable tumor margins. Radical tumor excision (i.e., removal of an entire compartment or structure, amputation, or lobectomy) is indicated for poorly localized tumors or those with high-grade malignancy.

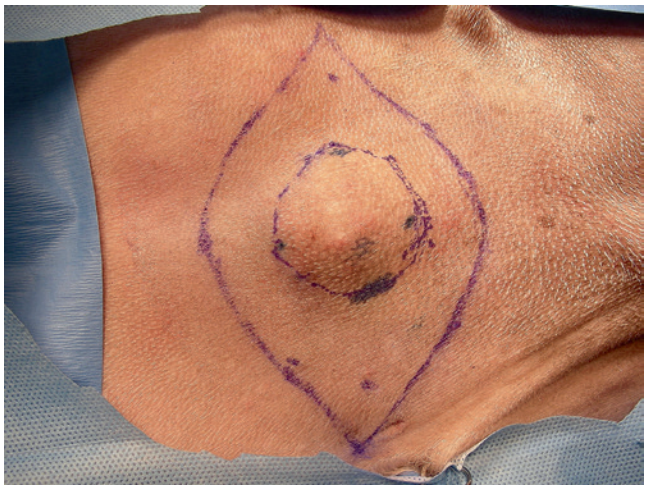


FIG. 15.25 A surgical marker was used to delineate a 2- to 3-cm margin for excision of a malignant tumor involving the skin and subcutaneous tissues.

NOTE Excision of infiltrative or aggressive tumors should include greater than 2 to 3 cm of "normal" tissue around the lesion. Extend the dissection at least one fascial layer below the detectable tumor margins.

Perform resections as atraumatically as possible to protect adjacent tissue and to prevent tumor seeding. Use a length-to-width ratio of 4:1 when making elliptic incisions around a tumor to minimize dog ear formation. As the ellipse is started through all layers (skin, subcutaneous tissue, and fascia or muscle), place a suture through them to maintain alignment and prevent retraction of one layer from the others. When removing multiple masses from the same animal, first remove lesions believed benign and change instruments and gloves (and drapes if necessary) after removal of each mass to prevent spreading tumor cells from one site to another. Ligate the blood supply as early as possible to prevent systemic spread of tumor cells or substances (e.g., histamine and heparin). Irrigate the wound bed after tumor incision. Aspirate or biopsy regional lymph nodes to stage the disease. Replace tumor-contaminated (or possibly contaminated) instruments, drapes, and gloves before wound closure. If radiation therapy is anticipated, mark resection margins within the wound using metallic staples to facilitate planning radiation therapy. Mark tumor margins with sutures or dyes and submit all resected specimens for histologic evaluation. Place the sample in approximately 10 parts formalin to 1 part tissue.

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Adequate fixation with 10% neutral, buffered formalin requires that specimens be less than 5 to 10 mm wide. Identification of the tumor type is imperative in determining the appropriate postoperative therapy and the prognosis.

NOTE Local tumors most often recur because the surgical margins for the original tumor were inadequate; be sure to mark tumor borders.

CLOSURE OF IRREGULAR SKIN DEFECTS

Although it is advisable to remove skin lesions with an elliptic skin incision to facilitate closure, some lesions cause irregularly shaped defects because of their size or location. Skin elasticity and tension lines should be assessed before excision and closure.

Circular Defects

Circular excision of lesions saves the most normal skin compared with other excision patterns. Skin tension lines may convert defects of other shapes into circular defects. Circular defects are difficult to close because dog ears tend to develop. They may be closed using a variety of techniques. The linear, combined V, and bow tie techniques are preferred. Conversion of a circular defect by a fusiform or elliptic excision with a 4:1 length-to-width ratio resects more skin than may be necessary (156%). The linear technique may be used for small defects when skin edges can be apposed without the formation of large dog ears. Sutures are placed parallel to the direction of skin tension lines, beginning at the center of the defect. The dog ears at each end of the suture line are excised and the remaining defects apposed (Fig. 15.26A).

The combined V technique is used when skin apposition causes dog ears and limited skin is available for reconstruction. This technique does not remove additional normal skin. Two equilateral triangles are designed on opposite sides of the circular defect with the central axis 45 degrees from the long axis (tension lines) of the defect. The sides of each triangle are incised such that the vertex of the V points to the longitudinal axis of the defect. The skin flaps are rotated and sutured to convert the

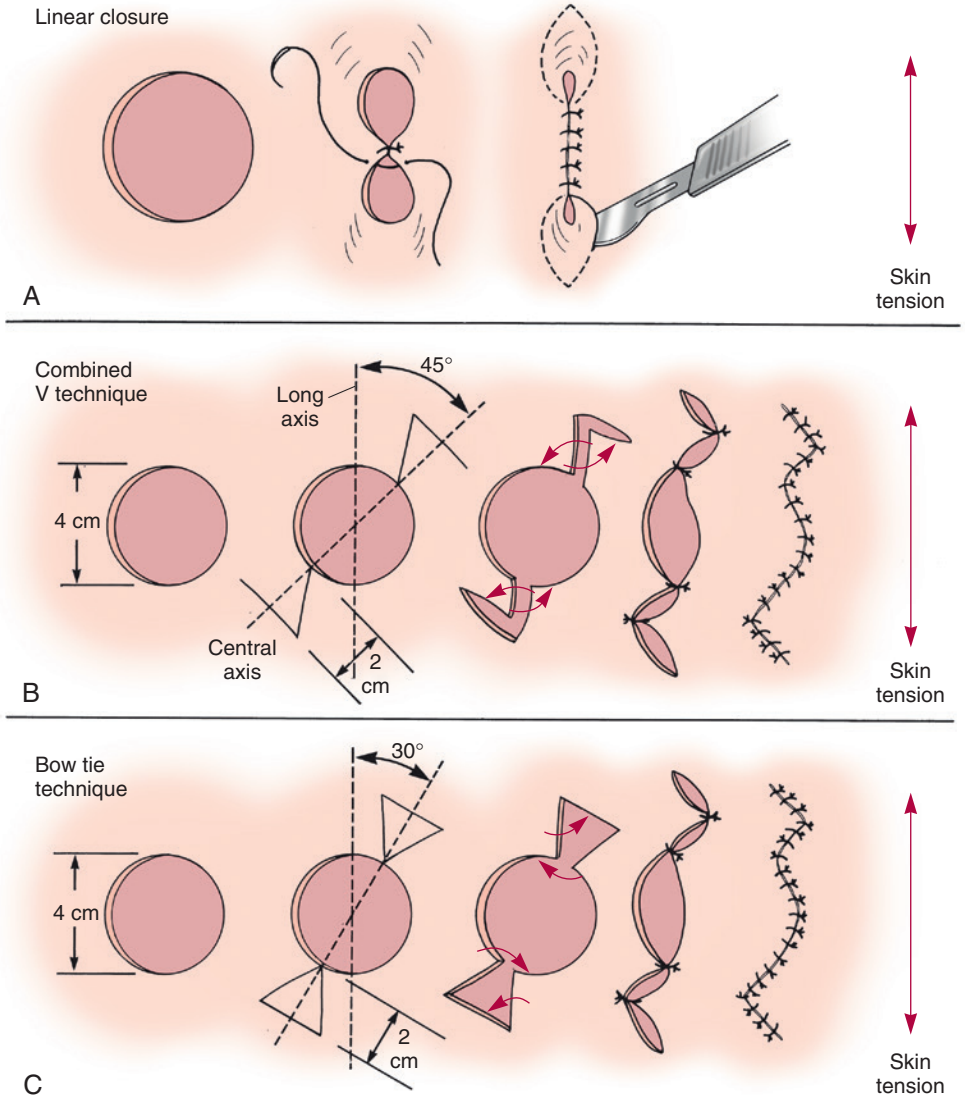


FIG. 15.26 Closure of small circular defects. (A) Use a linear closure if the skin edges can be apposed without creating large "dog ears." (B) Use a combined V technique when limited skin is available for reconstruction. (C) Use a bow tie technique when abundant skin is available.

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circular defect into a smaller, irregular fusiform defect. Edges of the converted defect are apposed with approximating sutures (see Fig. 15.26B). The bow tie technique is used when skin apposition results in large dog ears and abundant skin surrounds the defect. This technique removes 36% additional skin. Two equilateral triangles are removed from opposite sides of the circular defect, with the central axis of each triangle 30 degrees from the long axis of the skin tension lines. The flaps are transposed and sutured into their new positions to shorten the sides of the original circle and transform the shape of the defect (see Fig. 15.26C).

Triangular Defects

Triangular lesions may be closed by shifting local tissue or rotating flaps. A simple closure technique is to begin at each point of the triangle and suture toward the center of the defect to create a Y-shaped suture line (Fig. 15.27A). A rotational flap is a semicircular or three-quarter circular flap of skin that is rotated about a pivot point into the defect (see Fig. 15.27B). Rotational flaps are used when skin is available on only one side of the defect or when moving skin from one side of the defect results in distortion of adjacent structures (i.e., near the eye or anus). Bilateral rotational flaps are used when little movable skin is available, but it is movable on two sides of the defect. Flaps should be sufficiently large (approximately 4:1 length-to-width ratio) to prevent tension on surrounding tissue. If tension is noted, a backcut into the base of the flap may ease tension and allow the flap to move by a combination of rotation and transposition (see Fig. 15.27C). Tension may also be relieved by removing a small triangle of skin (Burow's triangle) at the end of the semicircle opposite the defect.

Square and Rectangular Defects

Square or rectangular defects may be closed using centripetal, unilateral, or bilateral advancement flaps or rotational flaps. Centripetal closure begins with suture closure at each corner of the defect and advances toward the center to form an X suture line (Fig. 15.28A). This technique should be used when skin is available on all four sides of the defect. A unilateral or single-pedicle advancement flap should be used to close defects with mobile skin on only one side and in the same plane as the defect. Parallel incisions are made from two corners of the defect at least as long as the width of the defect, and skin is undermined and advanced over the defect (see Fig. 15.28B). If necessary, relaxing incisions are made at the base of the flap. An H-plasty or a double-pedicle advancement flap is used to close large defects that have mobile skin available on two sides of the defect (see Fig. 15.28C). A rotational or transposition flap is used to cover defects that have mobile skin on only one side of the defect and available skin in a plane different from that of the defect (see Fig. 15.28D). These flaps become effectively shorter with increasing rotation. They should be longer than the defect to achieve adequate coverage without tension. The width of the flap base should at least equal the width of the defect. The diagonal from the pivot point of the flap to the farthest corner of the defect should be equal to the diagonal from the flap's pivot point across the flap. A dog ear forms at the base of the flap opposite the pivot point.

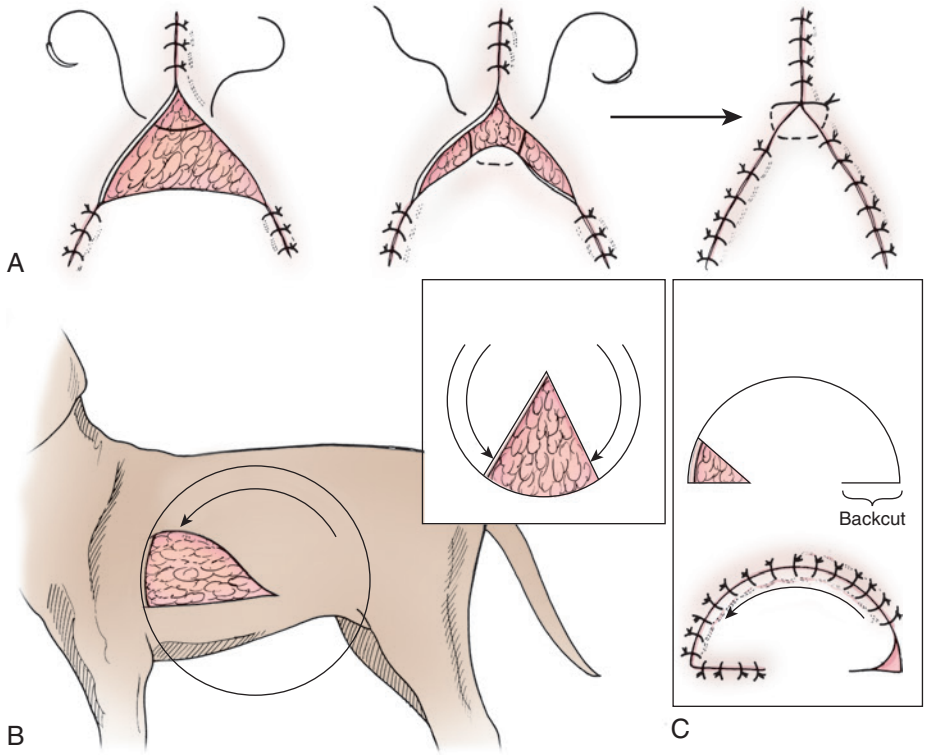


FIG. 15.27 Repair of triangular defects. (A) Close the defect as a Y by beginning at each point and suturing toward the center. (B) Create one or two rotational flaps at the defect edge. (C) A backcut or excision of Burow's triangle may be necessary to relieve tension at the base of the flap.

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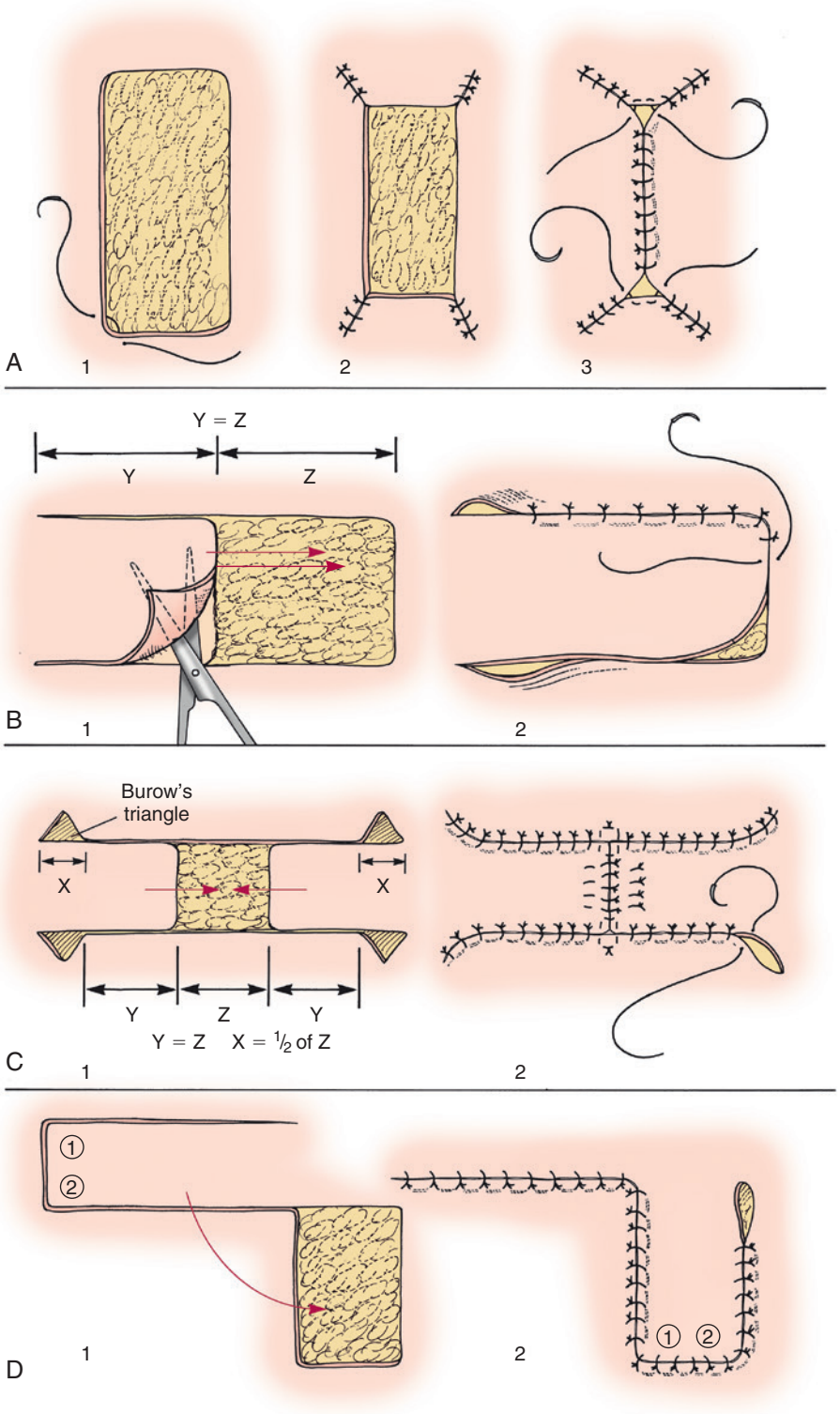


FIG. 15.28 Repair of square or rectangular defects. (A) Close from the corners and advance to the center to form an X suture line. Use a (B) unilateral or (C) bilateral advancement flap to close defects with mobile skin on only one or two sides of the defect. (D) Use a rotational or transposition flap to cover defects with mobile skin in a plane different from the defect.

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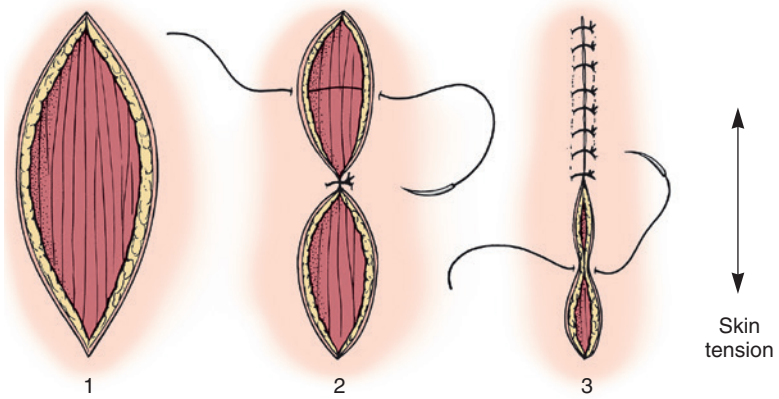


FIG. 15.29 Close fusiform defects *(1)* by placing the first suture across the widest part of the wound. Continue to divide each segment of the defect in half with subsequent sutures *(2* and *3).*

Fusiform Defects

Fusiform, or elliptic, defects are closed by first placing a suture across the widest part of the defect. Continue to divide each remaining segment in half with subsequent sutures to achieve a linear closure without dog ears (Fig. 15.29).

Crescentic Defects

In crescentic defects, one side is longer than the other. These defects are closed beginning at the midpoint. Each remaining segment is divided in half with subsequent sutures, and sutures on the convex side are spaced closer together than sutures on the concave side. Dog ears are removed as necessary (Fig. 15.30).

PEDICLE FLAPS

Pedicle flaps are "tongues" of epidermis and dermis that are partly detached from donor sites and used to cover defects. The base, or pedicle, of the flap contains the blood supply essential for flap survival. Pedicle flaps often allow immediate coverage of a wound bed and prevent the prolonged healing, excessive scarring, and contracture associated with healing by secondary intention. They can be classified in various ways based on the location, blood supply, and tissue formation. A specific flap may be classified in more than one way. Most flaps are called *subdermal plexus flaps*; however, those with direct cutaneous vessels are called *axial pattern flaps.*

Flaps that remain attached to the donor bed by only the direct cutaneous vessels and subcutaneous tissue are called *island flaps.* Flaps created adjacent to the defect in loose elastic skin are termed *local flaps. Interpolation flaps* are rectangular flaps that are rotated into a nearby rather than adjacent defect. Those created at a distance from the defect are *distant flaps,* which usually require multiple-stage reconstruction. Flaps that include tissue other than skin and subcutaneous tissue are called *compound* or *composite flaps* and may include muscle (myocutaneous), cartilage, or bone.

Increasing the width of a pedicle flap does not increase the surviving length of the flap. However, narrowing the base of the pedicle by backcut techniques increases the possibility of necrosis. The base of flaps should be slightly wider than the width of the flap body. Multiple small flaps may be preferable to a large flap if circulation is questionable. Delaying flap transfer 18 to 21 days after initial creation may improve circulation and survival in ischemic flaps (delay phenomenon). Flaps must be fixed to the edges of the recipient bed with no tension to allow revascularization and healing. Donor sites should have enough skin to permit primary closure and skin transfer to the recipient site. Donor sites with excessive motion and stress should be avoided.

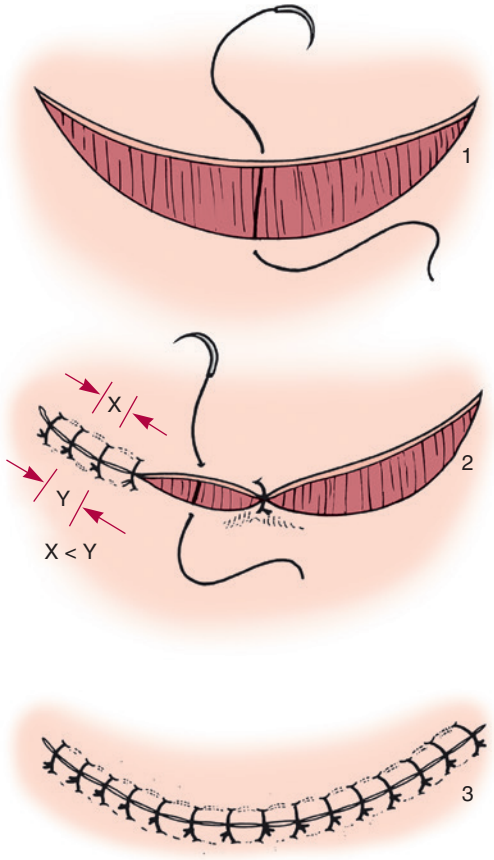


FIG. 15.30 Close crescent defects by *(1)* beginning at the midpoint and dividing each segment of the defect in half with subsequent sutures *(2* and *3).* Space sutures closer on the convex than the concave aspect of the defect.

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Reconstruction should be planned so that the color and direction of hair growth after transfer of flaps or grafts to the recipient site are similar to those of the donor site.

Hyperbaric oxygen treatment or NPWT may improve survival of the flap or graft. Hyperbaric oxygen treatment consists of breathing 100% oxygen in a chamber where pressure is maintained at greater than 1 atmosphere absolute or greater than sea level pressure. HBOT hyperoxygenates hypoxic tissue, stimulates fibroblasts, and enhances tissue revascularization. NPWT also increases blood flow and is associated with greater flap survival (see Box 15.6).

Venous congestion in a flap may be suspected if the flap becomes dusky or bluish in color, if capillary return is quicker than normal, and if rapid or dark bleeding occurs in response to a needle prick. Venous congestion may lead to failure of the flap or graft.

ADVANCEMENT FLAPS

Advancement flaps are local subdermal plexus flaps. They include single-pedicle, bipedicle, H-plasty, and V-Y advancement flaps (see Figs. 15.24, 15.27, and 15.28). Flaps are formed in adjacent, loose, elastic skin that can be slid over the defect. An advancement flap is developed parallel to lines of least tension to facilitate its forward stretch over a wound. It does not bring additional loose skin to the wound. Advancement flap stretching is opposed by retractive forces that may lead to dehiscence.

ROTATIONAL FLAPS

Rotational flaps are local flaps that are pivoted over a defect with which they share a common border. They are semicircular and may be paired or single. They may be used to close triangular defects without creating a secondary defect. A curved incision is created, and the skin is undermined in a stepwise fashion until it covers the defect without tension (see Fig. 15.27).

TRANSPOSITION FLAPS

Transposition flaps are rectangular, local flaps that bring additional skin when rotated into defects. A Z-plasty is a modified transposition flap (see Fig. 15.24G and H). Ninety-degree transposition flaps are aligned parallel to the lines of greatest tension to obtain the bulk of the flap required to cover the defect. The donor site is easily closed because minimal tension lines are perpendicular to the suture line. The width of the flap equals the width of the defect (see Fig. 15.28D). The length of the flap is determined by measuring from the pivot point of the flap to the most distant point of the defect; the length decreases as the arc of rotation increases past 90 degrees because of kinking and skin folding. Dog ears occur but flatten with time.

Other useful transposition flaps include the forelimb fold and flank fold flaps (Figs. 15.31–15.34). The size and length of skin fold flaps vary with body conformation and which three of the four flap attachments are severed. The four attachments are medial and lateral attachments to the upper limb and dorsal and ventral attachments to the trunk. Often a margin of the defect to be closed serves as one of the incisions. Skin fold flaps can be harvested bilaterally to close large axillary and sternal or inguinal wounds. Creation of these flaps begins by grasping the loose skin extending from the elbow to the body wall or flank to determine the amount of skin that can be harvested as a flap. Symmetric lateral and medial incisions are first outlined and then made. These incisions are connected with a crescentic incision made proximal to the elbow (stifle). The flap is elevated from the triceps or quadriceps, transposed, and sutured to the prepared wound bed. The donor site is apposed after flap transposition. Other elbow or flank fold flap configurations can be created for coverage of defects involving the lateral thorax, abdomen, hip, stifle, shoulder, or elbow by severing either the dorsal or ventral body wall attachments (see Figs. 15.32 and 15.34). The flank fold flap supplied by the lower branches of the ventral branch of the deep circumflex iliac artery can be considered an axial pattern flap. Similarly, if the elbow fold flap includes the lateral thoracic artery, it should be considered an axial pattern flap as well. A novel transposition flap can be created from scrotal skin after prescrotal castration and positioned in the perineum and thigh.

INTERPOLATION FLAPS

A variation of the transposition flap, the interpolation flap differs in that it lacks a common border with the wound; this leaves an area of interposed skin between the donor bed and the recipient wound. The flap is created in the same way as a transposition flap except that the length of the interpolation flap must include the length of the intervening skin segment (see Fig. 15.28). The subcutaneous tissue on the segment of flap overlying the intervening skin is left exposed. After approximately 14 days, this redundant segment of the flap is resected and the incised edges are sutured. As an alternative, a bridge incision can be made connecting the donor and recipient beds to facilitate flap transfer, which eliminates the need for a second surgical procedure.

POUCH AND HINGE FLAPS

Pouch flaps (bipedicle flaps) and hinge flaps (single-pedicle flaps) are direct, distant flaps useful for reconstructing lower extremity skin defects, although axial pattern or mesh grafts are more commonly used. Reconstruction using the pouch or hinge flap requires three stages: (1) debridement and granulation, (2) flap creation and healing, and (3) flap release. After a healthy bed of granulation tissue has formed, the skin on the limb and ipsilateral thoracoabdominal area is prepared for aseptic surgery. The limb is positioned along the animal's side, and two parallel dorsoventral incisions are made at locations that allow complete coverage of the defect (Fig. 15.35A).

Create a flap 1 to 2 cm wider than the defect to accommodate elastic contraction and stretching of the flap. Undermine the flap beneath the cutaneous trunci muscle, and place the foot within the pouch. Appose skin at the wound edges with the edges of the flap using interrupted approximating sutures (e.g., 3-0 or 4-0 polypropylene, polybutester, or nylon). Place three to four interrupted sutures through the skin of the flap into the granulation tissue to immobilize the flap over the defect. Place two or three retaining or tension-relieving sutures through the skin adjacent to the flap on the limb and body wall to help prevent the limb from shifting ventrally and placing tension on the flap. Bandage the limb against the body for 14 days, and change the bandage every 3 to 4 days. As an alternative, cut an access window in the bandage over the flap, allowing evaluation and wound care without removal of the entire bandage. Place a patch bandage over the access window between treatments. Release the limb from the pouch by making two horizontal incisions (dorsal and ventral) an appropriate distance from the paw to allow coverage

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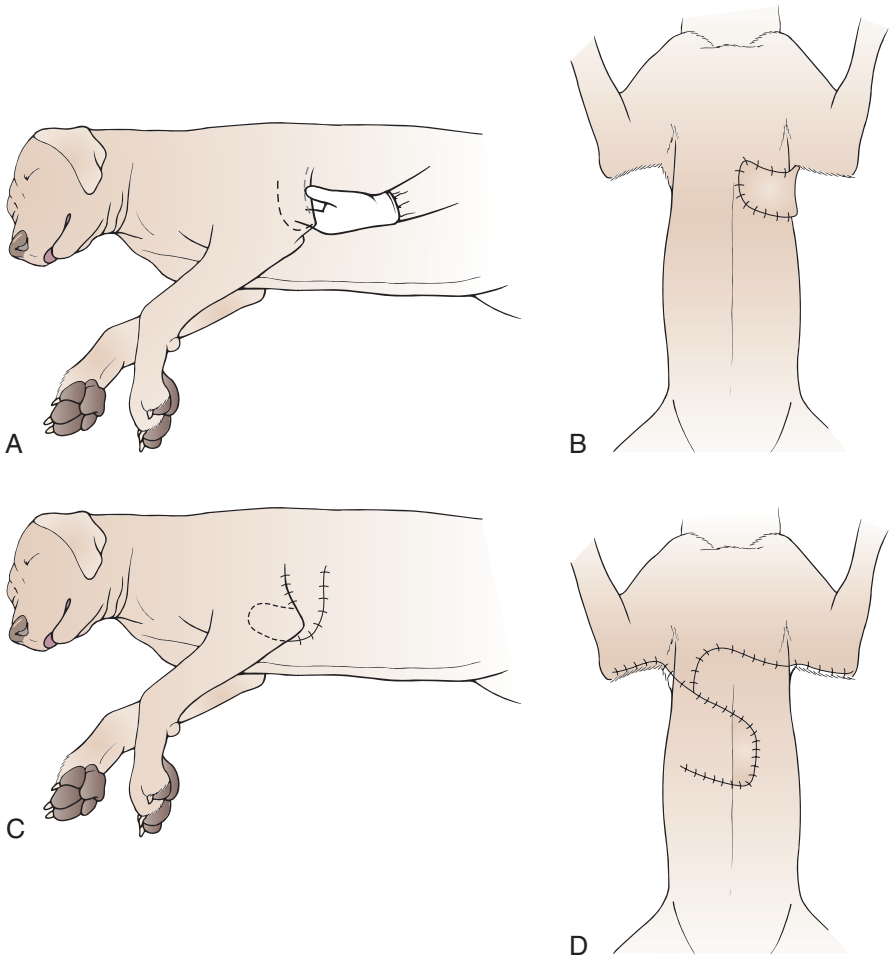


FIG. 15.31 The forelimb skin fold is harvested to close axillary or sternal wounds. (A) Grasp loose skin from the elbow to the body wall to determine the amount of skin that can be harvested. Dashed lines indicate incisions to create lateral and medial skin incisions to define the width of the flap, then connect the incisions with a crescent-shaped incision proximal to the elbow. (B–C) Elevate, transpose, and suture the flap into the wound, then close the donor site. (D) Create bilateral flaps to close larger wounds.

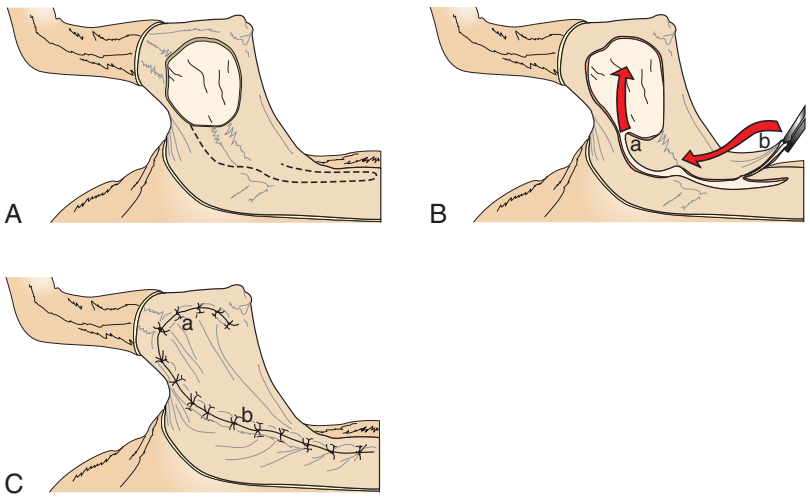


FIG. 15.32 This elbow fold flap is created to close a defect on the medial aspect of the proximal forelimb. (A) The dashed line indicates the incision of the ventral body wall attachment of the fold. (B) The flap is elevated and prepared for transposition. (C) The flap is sutured into position, and the donor site is closed.

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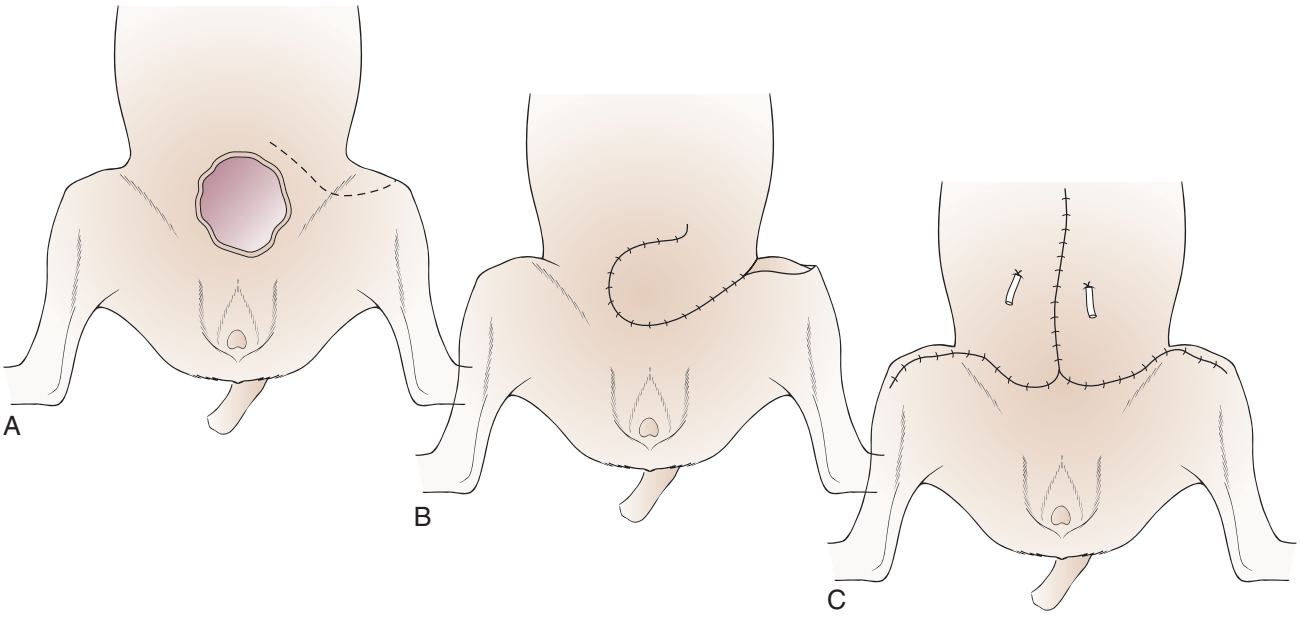


FIG. 15.33 The flank skin fold is harvested to close inguinal wounds. (A) Loose skin from the flank is incised to create a flap *(dashed line).* (B) Create lateral and medial skin incisions to define the width of the flap; then connect these incisions with a crescent-shaped incision proximal to the stifle; transpose and suture to defect. (C) Create bilateral flaps to close larger wounds.

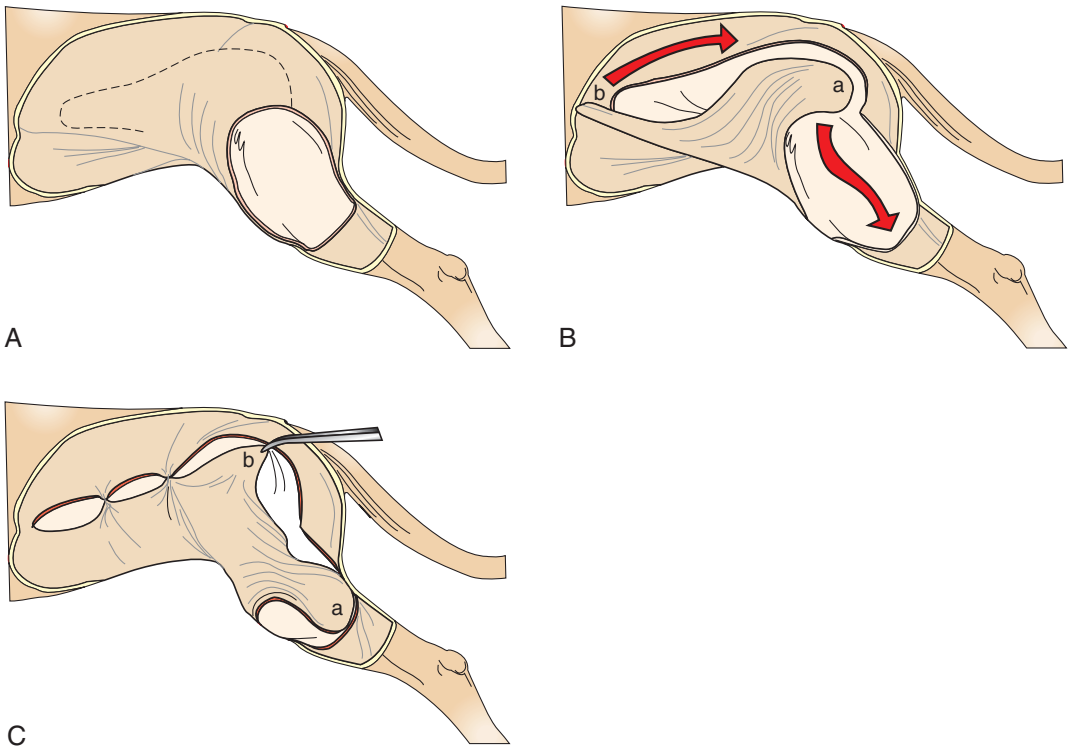


FIG. 15.34 A flank fold flap is used to close a defect on the lateral thigh. (A) The dashed line indicates the incision of the dorsal body wall attachment of the flank fold. (B) After creating the flap, the most dorsal aspect is transposed to the most distal aspect of the defect. (C) The flap and defect are closed.

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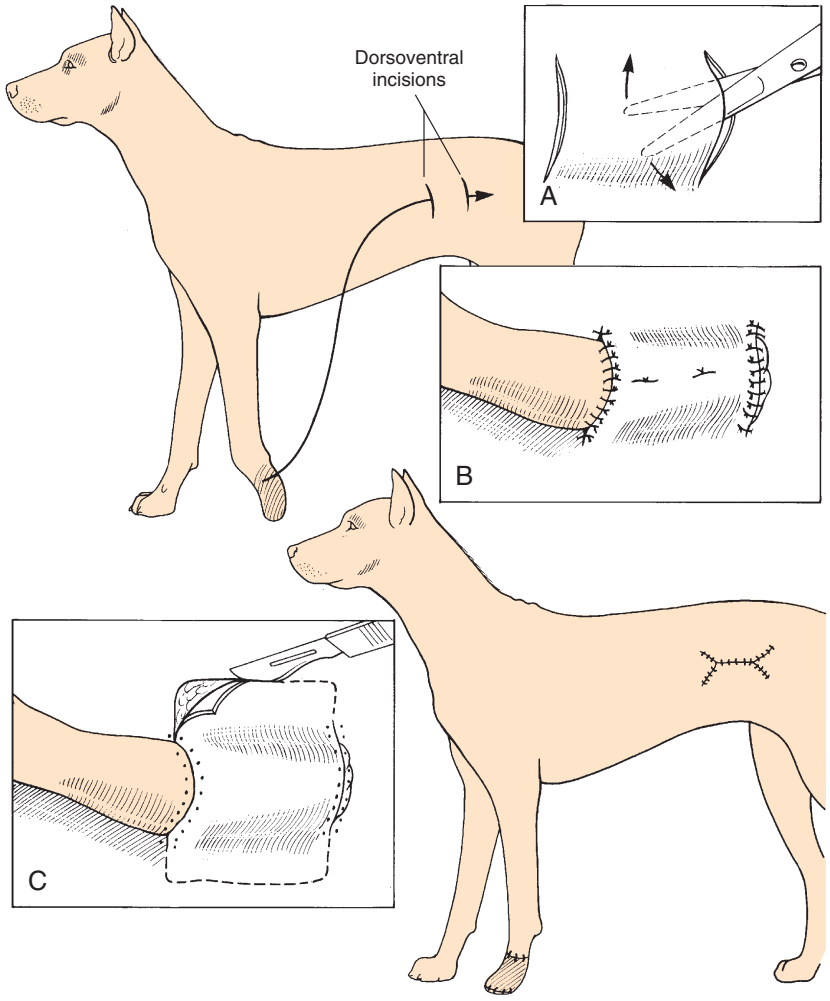


FIG. 15.35 Pouch flap. (A) Make two parallel dorsoventral incisions and undermine skin to create a pouch. (B) Position the limb inside the pouch and suture the edges of the defect to the flap. (C) After 2 to 3 weeks, release the limb and cover the remainder of the defect. Make two horizontal incisions to free the flap, then suture it to the remaining edges of the defect. Close the donor site.

of the palmar aspect of the defect (see Fig. 15.35B). Delayed release of the flap by dividing the pedicles in stages encourages flap survival.

Incise half of the lower pedicle, followed by release of the remaining half in 2 to 3 days. Two days later, begin delayed division of the upper pedicle in a similar fashion. Lavage the medial aspect of the paw to remove exudate and debride if necessary. Trim freed flap edges and suture to the opposing wound border with each division. Lavage the donor site and close with interrupted approximating sutures (e.g., 3-0 to 4-0 polypropylene or nylon) (see Fig. 15.35C).

Although this technique is successful in covering distal extremity skin defects, some animals may not tolerate the limb being positioned against the body wall, and temporary joint stiffness and muscle atrophy may occur.

TUBED PEDICLE FLAPS

A tubed pedicle flap uses a multistage procedure to "walk" an indirect, distant flap to a recipient site. The tube is made wider and longer (2–3 cm) than the recipient bed because these tubes contract as a result of decreased elasticity and fibrosis before transfer.

To create the tube, make two parallel incisions through the skin in an area where remaining skin can be reapposed without excess tension (Fig. 15.36A). Undermine skin between the two incisions. Suture incised edges of the flap together with approximating sutures (e.g., 3-0 or 4-0 polypropylene or nylon), creating a tube attached at both ends. Appose the edges of the donor site with approximating sutures (e.g., 3-0 or 4-0 nylon or polypropylene). After 18 to 21 days, transect one end of the tube and transpose it to the recipient bed. Transection can also be done in stages; half of the tube is incised and resutured in place, then 2 days later the remainder of the tube is transected and then transposed.

As an alternative, transpose the end of the tube nearer the donor site, and transect the other end of the tube and transpose it over the defect (see Fig. 15.36B) after an additional 18 to 21 days. Incise the tube and unroll it as needed to cover the defect, and suture the edges of the tube to the edges of the defect (see Fig. 15.36C). Appose skin edges at the tube's origin. Transect the remaining end of the

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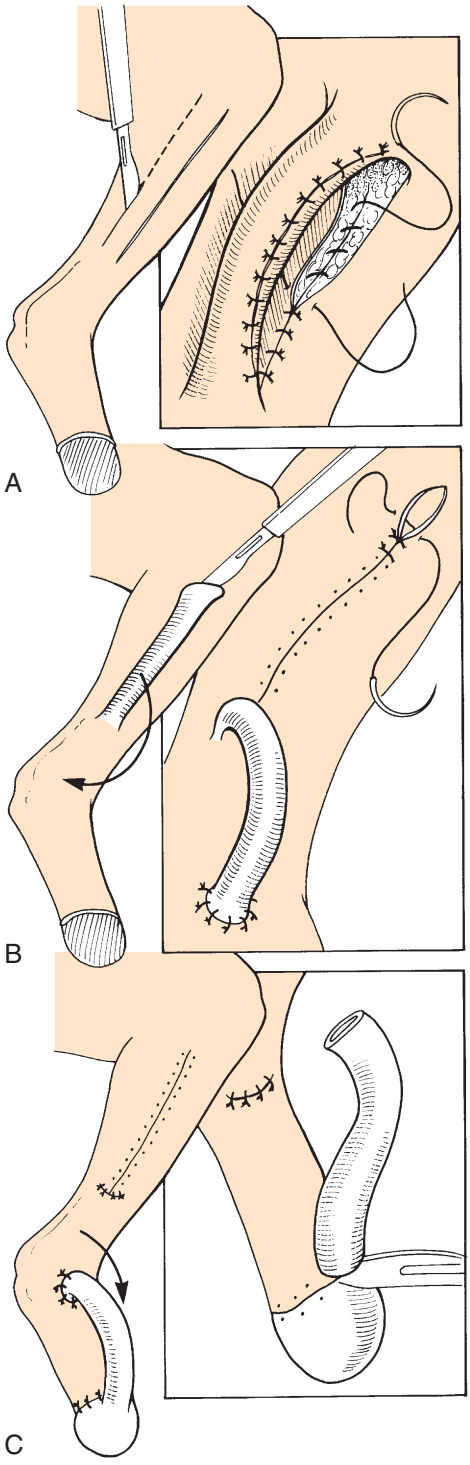


FIG. 15.36 Tubed pedicle flap. (A) Make two parallel incisions in mobile skin. Create a tube by suturing the edges together and appose the donor site. Note: The tube may need to be created more proximally on the limb than illustrated so that the skin is sufficiently mobile to close the donor site defect. (B) Approximately 3 weeks later, advance the tube toward the defect by severing one end of the tube and suturing it nearer the defect. (C) After another 3 weeks, sever the other end of the tube and use it to cover the defect or advance it closer to the defect.

tube after 18 to 21 days to complete coverage of the defect if necessary. The disadvantage of this technique is the number of stages and time required to accomplish wound closure.

AXIAL PATTERN FLAPS

Axial pattern flaps are pedicle flaps that include a direct cutaneous artery and vein at the base of the flap. The terminal branches of these vessels supply the subdermal plexus. They have better perfusion than pedicle flaps with a circulation from the subdermal plexus alone. Axial pattern flaps are elevated and transferred to cutaneous defects within their radius. They are usually rectangular or L-shaped flaps. Axial pattern flaps have been described using the caudal auricular artery branches, the superficial temporal artery, the omocervical artery (superficial cervical), the thoracodorsal artery, the lateral thoracic artery, the superficial brachial artery, the cranial superficial epigastric and caudal superficial epigastric arteries, the deep circumflex iliac artery, the genicular artery, and the lateral caudal arteries as direct cutaneous arteries in dogs (Fig. 15.37). Although similar flaps can be created in cats, only the thoracodorsal, caudal superficial epigastric, caudal auricular, superficial cervical, superficial temporal artery axial pattern, and reverse saphenous conduit flaps have been evaluated. Axial pattern flaps require careful planning, measuring, and mapping on the skin surface to minimize errors. Positioning is important to ensure that skin and underlying landmarks are in normal anatomic position. Limbs are placed in relaxed extension, and thoracoabdominal skin is grasped, lifted, and allowed to retract spontaneously to normal position before the flaps are outlined. Axial pattern flaps can be modified to create island arterial flaps by severing the cutaneous pedicle but preserving the direct cutaneous artery and vein. Island flaps have the potential for use as a free flap for transfer and microvascular anastomosis.

Axial pattern flaps are used most commonly to facilitate wound closure after tumor resection or trauma. The survival rate for axial pattern flaps is approximately twice that for subdermal plexus flaps of comparable size. Axial pattern flaps also provide durable, full-thickness skin that can be transposed primarily without the need for a vascular bed or postoperative immobilization. Complications include wound drainage, partial dehiscence, distal flap necrosis, infections, and seroma formation. The cosmetic results are good. Although historically the outcome following wound closure using axial pattern flaps has been reported as good to excellent, a 2015 retrospective study of 73 dogs reported good to excellent outcomes in 64%, with complications occurring in 89%.[8]

Caudal Auricular Axial Pattern Flap

The sternocleidomastoideus branches of the caudal auricular artery and vein may be used to reconstruct ipsilateral or contralateral defects involving the head and neck. The sternocleidomastoideus branches are located between the lateral aspect of the wing of the atlas and the vertical ear canal and are directed caudodorsally. The caudal auricular artery is located approximately 1 cm caudal to the base of the scutiform cartilage of the pinna (a palpable depression at the midpoint between the base of the ear and the wing of the atlas).

Position the forelimb in relaxed extension so that the scapula is perpendicular to the trunk. Outline the flap with the base centered over the lateral aspect of the wing of the atlas (see Fig. 15.37). Draw a caudal incision line parallel to the base at a point rostral

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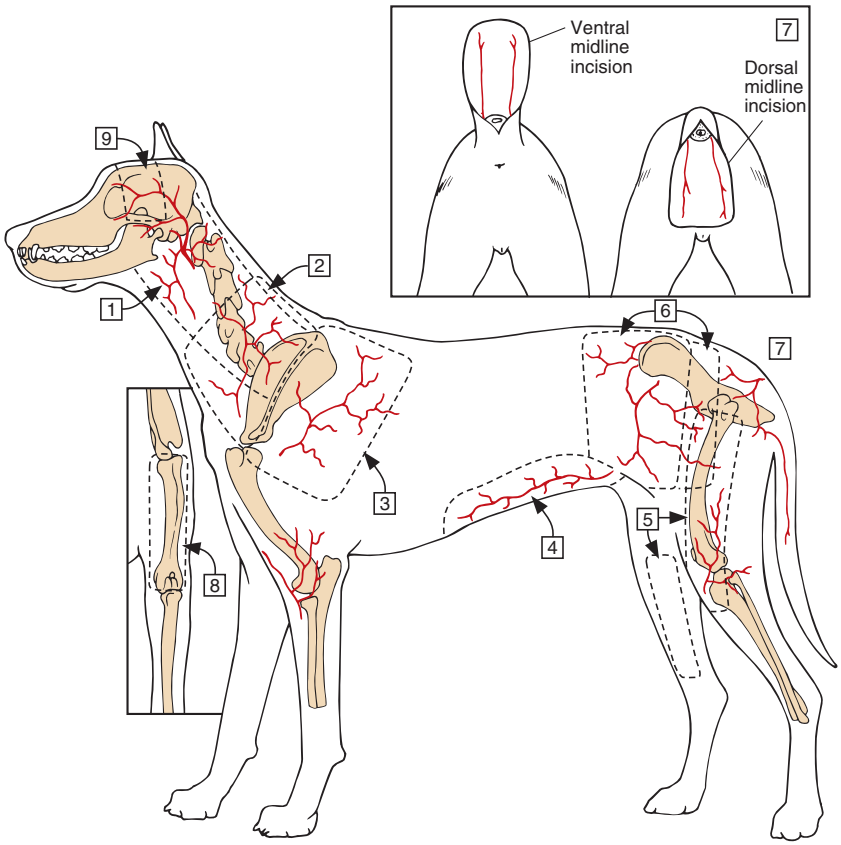


FIG. 15.37 Direct cutaneous vessels used in axial pattern flaps. *1,* Caudal auricular; *2,* omocervical; *3,* thoracodorsal; *4,* caudal superficial epigastric; *5,* medial genicular; *6,* deep circumflex iliac; *7,* superficial lateral caudal *(inset); 8,* superficial brachial *(inset)*; *9,* superficial temporal. Dashed lines outline anticipated flaps corresponding with each direct cutaneous vessel (neither a cranial superficial epigastric flap nor a reverse saphenous conduit flap is shown).

to the scapular spine, which will provide a flap long enough to cover the defect. Then draw dorsal and ventral lines that connect the base and the caudal incision line at a width that allows closure of the donor site. In cats, the dorsal border is nearer the dorsal midline. The width of the flap approximates that of the central third of the lateral aspect of the cervical area.

Incise the dorsal, ventral, and caudal lines and elevate the flap deep to the platysma (sphincter colli superficialis) muscle until the sternocleidomastoideus branches of the caudal auricular artery are identified. Rotate the flap into the defect, place drains, and appose skin edges. If there is interposing skin between the donor and recipient sites, make a bridge incision to connect the sites or partly tube the flap to span the interposing skin.

Superficial Temporal Artery Axial Pattern Flap

A cutaneous branch of the superficial temporal artery allows formation of an axial pattern flap that can be used to cover defects involving the face and head, especially the maxillofacial area. This flap is also used in oral reconstruction after partial maxillectomy. The superficial temporal artery lies in a subcutaneous position at the base of the zygomatic arch and extends rostrally along the zygomatic arch.

Position the animal in ventral recumbency. Mark the base of the flap caudally at the caudal aspect of the zygomatic arch and rostrally at the lateral orbital rim. Outline the flap by making two parallel lines; extend one line from each of these points dorsally and laterally to the middle of the dorsal orbital rim of the contralateral eye. Limit the width of the flap to the orbits and ears. Connect the parallel lines (see Fig. 15.37). Incise the outlined flap and elevate it with the frontalis muscle, which is a thin muscle overlying the temporalis muscle. Transpose the flap. Eliminate dead space with Penrose or closed suction drains and close the defects.

Omocervical Axial Pattern Flap (Superficial Cervical Axial Pattern Flap)

Omocervical axial pattern flaps are used for defects involving the face, head, ear, shoulder, neck, and axilla. By making a bridging incision between the angle of the jaw and the cranioventral edge of the donor site and creating a parapharyngeal tunnel, a staged, extended flap can be passed into the mouth to reconstruct oronasal defects caudal to the third premolar. They incorporate the superficial cervical branch of the omocervical artery and associated vein. Vessels originate adjacent to the prescapular lymph node at a site corresponding to the cranial shoulder depression and course dorsally just cranial to the scapula.

Position the patient in lateral recumbency with the forelimb in relaxed extension perpendicular to the trunk. Draw a line over the scapular spine to identify the caudal incision. Draw the cranial incision line parallel to the scapular spine at a site equal to the distance between the scapular spine and the cranial shoulder depression at the cranial edge of the scapula. Extend the lines to and continue along the dorsal midline. Extend the flap to the

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FIG. 15.38 A thoracodorsal axial pattern flap has been tubed and applied over an elbow wound.

contralateral scapulohumeral joint if necessary. As an alternative, create a right-angle design incorporating the skin over the dorsal aspect of the opposite scapula. Large omocervical flaps may require ligation of the opposite omocervical direct cutaneous artery and vein. Incise the outlined flap and undermine deep to the sphincter colli superficialis muscle. Transpose the flap. Eliminate dead space with Penrose or closed suction drains and close the defects.

Thoracodorsal Axial Pattern Flap

Thoracodorsal axial pattern flaps are preferable to omocervical flaps because they are more robust. They are used to cover defects involving the shoulder, forelimb, elbow, axilla, and thorax (Fig. 15.38). In cats, the thoracodorsal flap extends to the carpus. In dogs, distal limb coverage depends on the body conformation and limb length. The flap is based on a cutaneous branch of the thoracodorsal artery and associated vein located at the caudal shoulder depression at a level parallel to the dorsal border of the acromion.

Position the patient in lateral recumbency with the forelimb in relaxed extension perpendicular to the trunk. Outline the flap by drawing a line over the scapular spine to mark the cranial incision (see Fig. 15.37). Draw the caudal incision line parallel to the scapular spine at a site approximately twice the distance from the acromion to the caudal shoulder depression. Extend the lines to and continue along the dorsal midline. Create an L flap for extended coverage by extending the dorsal incision line by approximately 50% and creating a parallel incision line beginning at the approximate midpoint of the caudal incision line. Incise the outlined flap and undermine deep to the cutaneous trunci muscle. Transpose the flap. Create a tube or make a bridge incision as needed for distant transposition. Eliminate dead space with Penrose or closed suction drains and close the defects.

NOTE Flaps extended ventral to the contralateral scapulohumeral joint usually survive. Development of long thoracodorsal axial pattern flaps may require division of the opposite cutaneous branches of the thoracodorsal artery and vein.

Lateral Thoracic Axial Pattern Flap

The lateral thoracic axial pattern flap is similar to the thoracodorsal flap, but smaller with a less extensive approach. It is used to cover the elbow. The lateral thoracic artery extends from the caudal aspect of the shoulder joint running horizontally and slightly ventrally on the lateral thoracic wall ventral to the thoracodorsal artery. It arises from the axillary artery close to the caudal border of the first rib. Deep branches supply the axillary lymph node, the ventral portion of the latissimus dorsi muscle, and part of the deep pectoral muscles. More superficial cranial branches to the elbow skin fold supply this axial pattern flap. The distribution of this vessel varies slightly between breeds.

Position the patient in lateral recumbency with the forelimb in relaxed extension perpendicular to the trunk. Palpate the caudal aspect of the shoulder joint and identify the origin of the thoracodorsal artery (see the previous discussion) to estimate the location of the lateral thoracic artery in a more ventral location. Draw the flap with the ventral border along the dorsal border of the deep pectoral muscle and the dorsal border parallel to this line with the artery in the center. Incise along these lines extending toward but not including the second mamma. Connect the parallel lines caudally, undermine the flap deep to the cutaneous trunci, and transpose up to 90 degrees to cover the defect. Place a closed suction drain, secure the flap to the defect, and appose the donor site.

Superficial Brachial Axial Pattern Flap

Superficial brachial axial pattern flaps are used to cover defects involving the antebrachium and elbow. These flaps depend on a small branch of the brachial artery located 3 cm proximal to the elbow (superficial brachial artery).

Position the patient in dorsal recumbency with the leg suspended in an elevated position. Outline the flap by drawing parallel to the humeral shaft two lines that extend dorsally and gradually converge at or below the greater tubercle. Center the base of the flap over the anterior third of the flexor surface of the elbow. Elevate the flap to the base, being especially careful to preserve the subdermal plexus, superficial brachial vessels, and cephalic vein. Rotate the flap into the defect, place drains, and appose skin edges. Flap length and survival preclude use of this flap to cover wounds in the carpal area.

Caudal Superficial Epigastric Axial Pattern Flap

The caudal superficial epigastric axial pattern flap is a versatile flap that is used to cover defects involving the caudal abdomen, flank, prepuce, perineum, thigh, and hind leg (Fig. 15.39). In cats, the flap extends over the metatarsal area. In dogs with long bodies and short limbs, it may extend to or below the level of the tibiotarsal joint. The flap includes the three to four caudal mammary glands and is supplied by the caudal superficial epigastric artery and associated vein that pass through the inguinal ring.

Position the patient in dorsal recumbency. Outline the flap with the ventral midline as the location of the medial incision. In male dogs, incorporate the base of the prepuce. Mark a parallel lateral incision at a distance equal to the distance from the mammary

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FIG. 15.39 A caudal superficial epigastric axial pattern flap has been harvested and applied over a distal limb wound.

teats to the midline. Determine the number of mammary glands to include in the flap based on the size of the defect. Create the flap by connecting the two parallel lines between the first and second or second and third glands with a crescent-shaped incision. Undermine the flap at the level of the external abdominal oblique aponeurosis, deep to the supramammarius muscle. Make the flap wider as needed to cover the defect if abundant loose, elastic skin is available for closure. Transpose the flap, place drains, and appose skin edges.

NOTE Concurrent ovariohysterectomy is recommended as transposed glands remain functional. Mammae may be resected later if their appearance is objectionable.

Cranial Superficial Epigastric Axial Pattern Flap

The cranial superficial epigastric axial pattern flap is smaller and less versatile than the caudal superficial epigastric axial pattern flap; however, it can be quite useful for closure of large skin defects overlying the sternum. The flap is kept small because these vessels are short, and some flap necrosis is expected. The flap may include mammary glands three, four, and possibly five. In males, the flap ends cranial to the prepuce. Although there is some variability, the cranial superficial artery penetrates the medial aspect of the rectus abdominis muscle at the junction between the second and third mammary glands. Cranial and caudal superficial epigastric artery anastomoses occur between or near the third and fourth mammary glands. The positioning and creation of the flap are similar to those for the caudal superficial epigastric flap. The base of the flap is located in the hypogastric region, where the cranial epigastric vessel enters the skin lateral to the abdominal midline, and a few centimeters caudal to the cartilaginous border of the ventral thorax (xiphoid process).

Outline the flap with the ventral midline as the location of the medial incision. Mark a parallel lateral incision at a distance equal to the distance from the mammary teats to the midline. Determine the number of mammary glands to include in the flap based on the size of the defect. Create the flap by connecting the two parallel lines between the fourth and fifth glands or caudal to the fifth gland with a crescent-shaped incision. Undermine the flap at the level of the external abdominal oblique aponeurosis, deep to the supramammarius muscle. Ligate branches of the caudal superficial epigastric artery as necessary. Transpose the flap, place drains, and appose skin edges. Create an island flap by making a crescent-shaped incision between the second and third mammary glands. Use caution during dissection and manipulation to prevent trauma, kinking, or stretching of the cranial superficial epigastric vessels.

Deep Circumflex Iliac Axial Pattern Flap

The dorsal branch of the deep circumflex iliac vessel is used in flaps to cover defects involving the caudal thorax, lateral abdominal wall, ipsilateral flank, lateral lumbar area, medial or lateral thigh, greater trochanter, and pelvic area. The dorsal and ventral branches of the deep circumflex iliac artery originate at a point cranioventral to the wing of the ilium.

Position the patient in lateral recumbency with the hind limb in relaxed extension perpendicular to the body. Outline the flap by first drawing a line midway between the cranial border of the wing of the ilium and the greater trochanter. For the cranial incision, draw a second line parallel to the first line and equal to the distance from the iliac border to the caudal line Extend the lines to the dorsal midline and create an L extension, if needed, to cover the defect (see Fig. 15.37). Incise the outlined flap. Elevate the flap below the level of the cutaneous trunci muscle. Transpose the flap, place drains, and appose skin edges. The ventral branch of the deep circumflex iliac artery is used in flaps to cover defects of the lateral abdominal wall and as an island flap for pelvic and sacral defects.

Make the reference lines as for the previous flap. Draw the caudal incision line extending distally cranial to the border of the femoral shaft. Extend the cranial incision line down the flank and thigh region parallel to the caudal flap border. Connect the two lines above the patella. Elevate the flap below the level of the cutaneous trunci muscle. Incise the outlined flap. Transpose the flap, place drains, and appose skin edges. The flank fold flap is a variation of the ventral deep circumflex iliac axial pattern flap (see p. 218) developed for transposition into inguinal defects.

Genicular Axial Pattern Flap

Genicular axial pattern flaps are used to cover defects involving the lateral and medial tibia and potentially the tibiotarsal joint. These flaps are dependent on the short genicular branch of the saphenous artery and medial saphenous vein.

Position the patient in lateral recumbency. Mark a point 1 cm proximal to the patella and 1.5 cm distal to the tibial tuberosity (see Fig. 15.37). Extend these two points dorsally parallel to the femoral shaft, ending at the base of the greater trochanter. Connect the parallel lines dorsally. Incise the outlined flap. Elevate the flap and rotate it to cover the defect. Place drains and appose skin edges. Although circulation is usually sufficient, this is not a robust flap.

Reverse Saphenous Conduit Flap

Reverse saphenous conduit flaps are used for defects at or below the tarsus (Fig. 15.40). They are created by ligating and dividing the vascular connection between the femoral artery and vein and the saphenous artery and medial saphenous vein. Reverse blood flow occurs because of anastomoses between the cranial branch of the saphenous artery and the perforating metatarsal artery (via medial and lateral plantar arteries), the cranial branch of the lateral saphenous vein, and other venous connections with the cranial and caudal branches of the medial saphenous veins distal to the tibiotarsal joint. Preoperative angiography ensures the presence and function of the saphenous artery, medial saphenous vein, and femoral artery and vein.

Position the patient in lateral recumbency with the affected limb down. Roughly outline the flap by marking a line across the central

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FIG. 15.40 (A) A reverse saphenous conduit flap is being harvested and (B) applied over a wound on the distal limb.

third of the inner thigh at or slightly above the level of the patella. Make parallel lines 0.5 to 1 cm cranial and caudal to the branches of the saphenous artery and medial saphenous vein. Make the transverse incision as marked to expose the saphenous vessels and nerve. Ligate and transect the saphenous artery and medial saphenous vein at their junction with the femoral artery and vein. Extend the incisions distally as marked in a slightly converging fashion. Undermine deep to the saphenous vessels by elevating a portion of the medial gastrocnemius muscle fascia with the flap. Ligate and divide the peroneal (fibular) artery and vein. Do not elevate the flap beyond the anastomosis between the cranial branch of the medial saphenous vein and the cranial branch of the lateral saphenous vein. Rotate or partly tube the pedicle transfer to the defect. As an alternative, make a bridge incision between the donor site and the wound. Place drains and appose the defects.

Lateral Caudal Axial Pattern Flap

The lateral caudal arteries of the tail may be used to reconstruct areas involving perineum and caudodorsal trunk defects. The largest source of skin is from the proximal third of the tail. The tail skin may also be used as a tube flap to cover defects on the hind leg. The lateral caudal vessels are bilateral and are located in the subcutaneous tissue of the tail. The lateral caudal arteries arise from the caudal gluteal arteries and have several anastomotic branches with the median caudal artery. Use of this flap requires tail amputation. In a retrospective study of 13 dogs, all flaps ultimately healed with no long-term complications.[9] Four dogs had wound complications, but only one of these dogs required surgical revision.

Make a dorsal midline incision along the length of the tail to cover dorsocaudal defects (see Fig. 15.37). Make a ventral midline skin incision to cover defects on the hind leg. Dissect the subcutaneous tissue from the deep caudal fascia, preserving the right and left lateral caudal arteries and veins. Amputate the tail at the third or fourth caudal intervertebral space (see p. 253). Transpose the skin flap over the defect, place drains, and appose skin edges.

COMPOSITE FLAPS

Flaps composed of skin with muscle, bone, or cartilage are termed *composite flaps*. The pinna has been used as a composite flap to cover maxillofacial defects. Numerous myocutaneous flaps, with or without attached bone segments, have been used to facilitate reconstructive surgery.

Myocutaneous and Muscle Flaps

Muscle flaps with overlying skin (myocutaneous flaps) or without skin (muscle flaps) may be created to facilitate herniorrhaphy, to cover soft tissue defects, to contribute circulation to fractures, and to combat infection. They should be used only when reconstruction with local flaps (see p. 217), axial pattern flaps (see p. 222), or free grafts (see p. 232) is not feasible. These flaps must be sufficiently large to cover the defect and have an easily accessible and constant dominant vascular supply. Donor sites should be easily closed. Muscles in dogs and cats that may be sacrificed without loss of function include the cutaneous trunci, gracilis, trapezius, sternohyoid, sternothyroid, deep pectoral, anconeus, ulnaris lateralis, humeral head of the flexor carpi ulnaris, sartorius, semitendinosus, rectus femoris, cranial tibial, long digital extensor, and portions of the latissimus dorsi.

Myocutaneous Flaps

Myocutaneous flaps described in the veterinary literature include latissimus dorsi, cutaneous trunci, gracilis, semitendinosus, and trapezius muscles. These muscles are superficial, allowing easy access and elevation, and have direct cutaneous arteries exiting the muscle surface to supply overlying skin. A vascular pedicle sufficient to maintain circulation is required to facilitate flap rotation into defects. Increased rotation may impair circulation and require that the flap length be reduced. Distant transfer of gracilis, latissimus dorsi, transverse abdominis, and some trapezius flaps is possible with microvascular anastomosis.

NOTE Development of myocutaneous flaps requires the presence of direct cutaneous arteries exiting the muscle surface to supply the overlying skin.

Platysma Myocutaneous Flap

The platysma muscle is a well-developed muscle originating from the middorsal tendinous raphe of the neck and skin. It courses longitudinally toward the mouth over the parotid and masseter to the lips and ventrally to the midline. The platysma myocutaneous flap used to cover defects involving the head and neck is identical to the caudal auricular axial pattern flap (see p. 222).

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Latissimus Dorsi Myocutaneous Flap

The latissimus dorsi muscle is a flat, triangular muscle overlying the dorsal half of the lateral thoracic wall. It originates from thoracolumbar fascia of the thoracic and lumbar spinous processes and from muscular attachments to the last two or three ribs. The aponeurosis of the latissimus dorsi inserts on the major teres tuberosity of the humerus. The muscle flexes the shoulder, drawing the limb caudally. The ventral portion of the muscle is supplied by branches of the thoracodorsal artery (dorsal and lateral thoracic arteries), which penetrate the muscle and supply the cutaneous trunci muscle and skin. Intercostal arteries supply segmental branches to the dorsal portion of the latissimus dorsi muscle and the overlying cutaneous trunci muscle. Latissimus dorsi myocutaneous flaps are bulky because they contain the cutaneous trunci muscle and skin, subcutaneous fat, and latissimus dorsi muscle. They are best suited for thoracic defects, although they may be used for forelimb defects. Anatomic landmarks are the ventral border of the acromion, adjacent caudal border of the triceps muscle, head of the last rib, and distal third of the humerus that corresponds to the axillary skin fold (Fig. 15.41).

With the patient in lateral recumbency and the forelimb in relaxed extension perpendicular to the trunk, plan and outline the flap with a marking pen.

Draw a line from the caudal border of the triceps muscle to the vertebral attachment of the last rib. Draw a parallel line caudodorsally from the axillary skin fold and connect them to outline the flap (see Fig. 15.41). Incise skin and extend the incision through the underlying latissimus dorsi muscle. The muscle flap equals the size of the skin flap. Elevate the latissimus dorsi and skin as a unit. Isolate, ligate, and divide the lateral intercostal vessels deep to the latissimus muscle. Identify and preserve the thoracodorsal artery and vein. Transpose the flap to the desired location without occluding the thoracodorsal vessels. If necessary, make a bridge incision or partial tubing of the flap for transposition. Place a closed suction drain at the donor site and beneath the flap at the recipient site. Secure the flap in position and close the donor site.

The latissimus dorsi may be used alone as a muscle flap; it is harvested in a similar manner without the skin for this purpose.

Cutaneous Trunci Myocutaneous Flap

The cutaneous trunci muscle arises from the pectoralis profundus and forms a thin leaf covering most of the dorsal, lateral, and ventral walls of the abdomen (see Fig. 15.41). It is more closely associated with skin than underlying structures. Blood supply is from small muscular branches and direct cutaneous arteries supplying the overlying skin. The cutaneous trunci overlying the latissimus dorsi muscle receives two to four short, direct cutaneous branches of the thoracodorsal artery caudal to the border of the triceps muscle. Elevating the cutaneous trunci muscle with skin helps preserve the subdermal plexus. Cutaneous trunci myocutaneous flaps are more pliable and elastic than latissimus dorsi myocutaneous flaps and are preferred for forelimb flaps.

Plan and outline the flap in the same way as for a latissimus dorsi myocutaneous flap. Incise the skin as outlined, but do not extend the incision beyond subcutaneous tissue between the cutaneous trunci and latissimus dorsi. Elevate the cutaneous trunci by dissecting the loose subcutaneous tissue. Ligate and divide branches of the proximal lateral intercostal direct cutaneous vessels. Transpose the flap to its desired location without occluding the thoracodorsal vessels. If necessary, make a bridge incision or partial tubing of the flap for transposition. Place a closed suction drain beneath the flap at recipient and donor sites. Secure the flap in position and close the donor site.

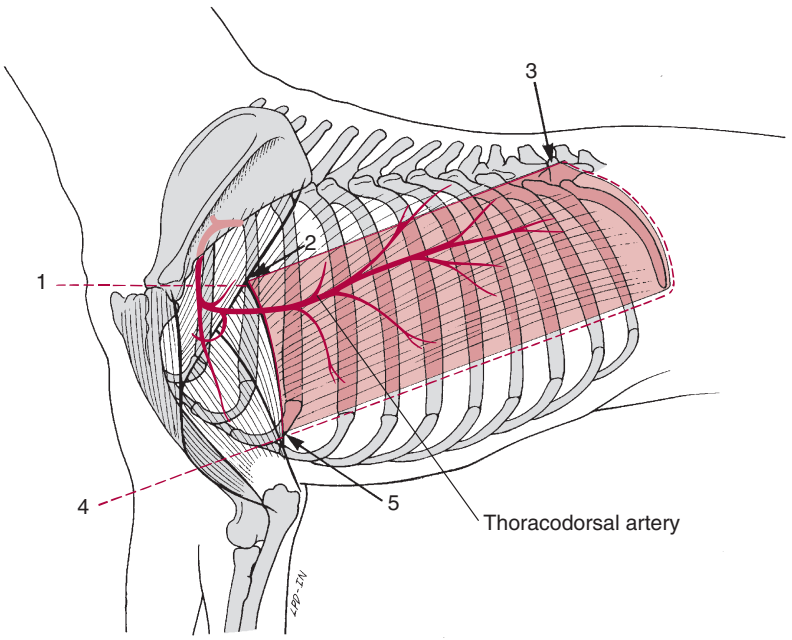


FIG. 15.41 Landmarks for the latissimus dorsi and cutaneous trunci myocutaneous flaps. *1,* Ventral border of the acromion; *2,* adjacent caudal border of the triceps muscle; *3,* vertebral attachment of the last rib; *4,* distal third of the humerus; and *5,* axillary skin fold. To construct the flaps, draw a line from *2* to *3* and a second parallel line from *5.* Incise and connect the two parallel lines dorsally.

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Trapezius Osteomyocutaneous Flap

Trapezius osteomyocutaneous flaps are generally used for defects in the neck, cranial thorax, or proximal thoracic limb; however, they may be transferred to distant sites with microvascular anastomosis. The trapezius is a thin, triangular muscle divided into cervical and thoracic parts. The cervical part of the trapezius is overlapped by the cleidocervicalis muscle and the thoracic part by the latissimus dorsi muscle. The muscle originates from the median raphe of the neck and supraspinous ligament from the level of the third cervical vertebra to the ninth thoracic vertebra and inserts on the scapular spine. It acts to elevate and abduct the forelimb. Lameness and scapular fractures may occur when this flap is used. Only the scapular spine, and not the scapular body, remains viable. The bone in this flap is weak and should be used as a source of osteogenesis rather than support. Dorsal, caudal, and cranioventral borders of the flap are 2 cm ventral to the dorsal midline, 2 cm caudal to the scapular spine, and a line between the acromion and transverse process of the third cervical vertebra, respectively.

Make a triangular skin incision over the cervical part of the trapezius muscle (Fig. 15.42). Incise the origin of the cervical part of the trapezius on the dorsal midline. Dissect the incised trapezius from the cleidocervicalis and omotransversarius muscles, preserving the attachment to the scapular spine and the prescapular branch of the superficial cervical vascular pedicle. Dissect the caudal half of the supraspinatus muscle from its attachment on the scapular spine and body. Incise the deltoideus and thoracic part of the trapezius attachments to the scapular spine. Dissect the cranial half of the infraspinatus from the scapular spine and body. Create a bone flap using an air-powered burr or saw. Dissect the medial attachments of the subscapularis and serratus ventralis muscles from the bone flap. Elevate the osteomusculocutaneous flap and transfer it to the recipient site, preserving the prescapular branch of the superficial cervical vascular pedicle. Place a closed suction drain at the donor site and close the defect with approximating sutures (for muscle, use 2-0 or 3-0 polydioxanone or polyglyconate suture; for subcutaneous tissue, use 3-0 or 4-0 polydioxanone or polyglyconate suture; for skin, use 3-0 or 4-0 nylon or polypropylene suture). Apply a bandage over the donor and recipient sites for support and fluid absorption.

Muscle Flaps

Muscle flaps may be transposed beneath skin to fill defects, repair hernias, and treat paralysis. Many muscles have been used to facilitate adjacent visceral repair and to fill defects. The use of muscle in reconstruction is limited by the tissue available and the surgeon's imagination. Muscles are capable of contributing circulation to areas of ischemia caused by trauma or radiation therapy. They may also provide support, facilitate return of function, improve cosmesis, and reduce wound contamination and sepsis. The latissimus dorsi may be used with or without a cutaneous flap to cover thoracic wall defects (see previous discussion). The muscle flap may be used with mesh or other implants to give support and is sutured to adjacent muscle or fascial planes. The transversus abdominis muscle has been used to facilitate

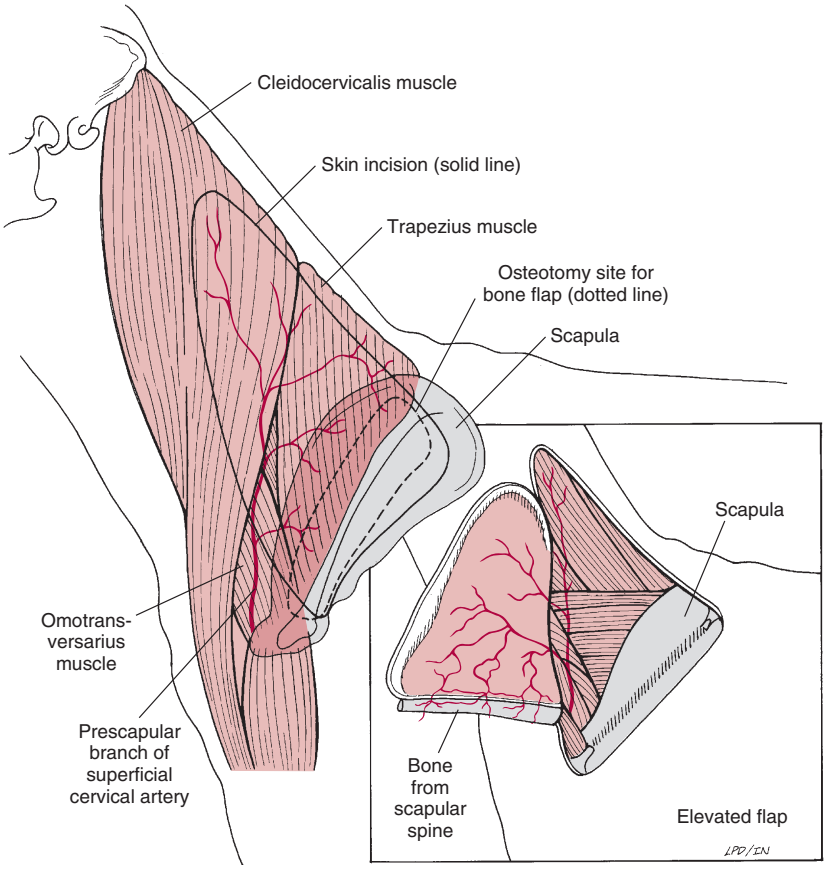


FIG. 15.42 Outlined trapezius osteomyocutaneous flap for regional reconstruction. The inset shows the flap ready for relocation.

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repair of the diaphragmatic hernia. The internal obturator, superficial gluteal, and semitendinosus muscles have been used in perineal hernia repair (see p. 496). Caudal abdominal hernias or defects can be reinforced with pectineus or sartorius muscle flaps. Esophageal repair may be facilitated by use of intercostal, diaphragm, sternocephalicus, or sternothyroideus muscles (see p. 374). The sternohyoideus and sternothyroideus muscles may be used to cover laryngotracheal defects. Biceps femoris or deep gluteal muscle flaps are sometimes used to cushion the femoral head and neck ostectomy site (see p. 1213). Reconstruction of distal antebrachial, carpal, and metacarpal injuries can be assisted by transposition of the humeral head of the flexor carpi ulnaris. The semitendinosus has been used to reconstruct tibial defects.

External Abdominal Oblique Muscle Flap

The external abdominal oblique muscle is elastic and mobile and may be used to facilitate repair of defects in the abdominal wall or caudal thoracic wall. This flap may be used to fill defects larger than 10 × 10 cm in medium-size dogs. The external abdominal oblique muscle is a long, flat muscle covering the ventral half of the lateral thoracic wall and lateral abdominal wall. Its fibers are directed caudoventrally. It is divided into a costal part arising from the fifth to thirteenth ribs and a lumbar part arising from the last rib and the thoracolumbar fascia. It has a wide aponeurosis that inserts on the linea alba and cranial pubic ligament and contributes to the external rectus fascia, external inguinal ring, and prepubic tendon. The cranial branch of the cranial abdominal artery supplies the middle zone of the lateral abdominal wall and is accompanied by the cranial hypogastric nerve and satellite vein. The deep branch of the deep circumflex artery anastomoses with the cranial and caudal abdominal arteries and is the main supply to the caudodorsal fourth of the abdominal wall. It is accomplished by a satellite vein and joined by the lateral cutaneous femoral nerve.

Make a paracostal skin incision from the level of the epaxial muscles to the ventral midline, beginning 5 cm caudal to the 13th rib. Identify and divide the lumbar fascial edge of the external abdominal oblique muscle, leaving a 0.5- to 1-cm margin of fascia along the muscular edge (Fig. 15.43). Undermine the lumbar external abdominal oblique muscle. Identify and preserve the neurovascular pedicle (branches of the cranial abdominal artery and cranial hypogastric nerve and satellite vein) in a craniodorsal location caudal to the 13th rib. Divide the dorsal fascial attachment and sever the lumbar external abdominal oblique muscle at the level of the 13th rib. Transpose the flap to an adjacent defect. Overlap the defect with the flap, and suture the inner fascial surface with 2-0 polydioxanone or polyglyconate using a simple interrupted pattern. Place a closed suction drain and appose the defect edges.

Cranial Sartorius Muscle Flap

Cranial sartorius muscle flaps are used to repair prepubic tendon ruptures or femoral hernias when tissue trauma, retraction, and fibrosis preclude adequate anatomic reapposition. This type of flap may also be used to cover femoral trochanteric ulcers. In dogs, the sartorius muscle consists of two long, flat, straplike muscles on the craniomedial surface of the thigh. In cats, the sartorius muscle has only one belly and an origin and insertion similar to that of the dog. The feline sartorius muscle has one

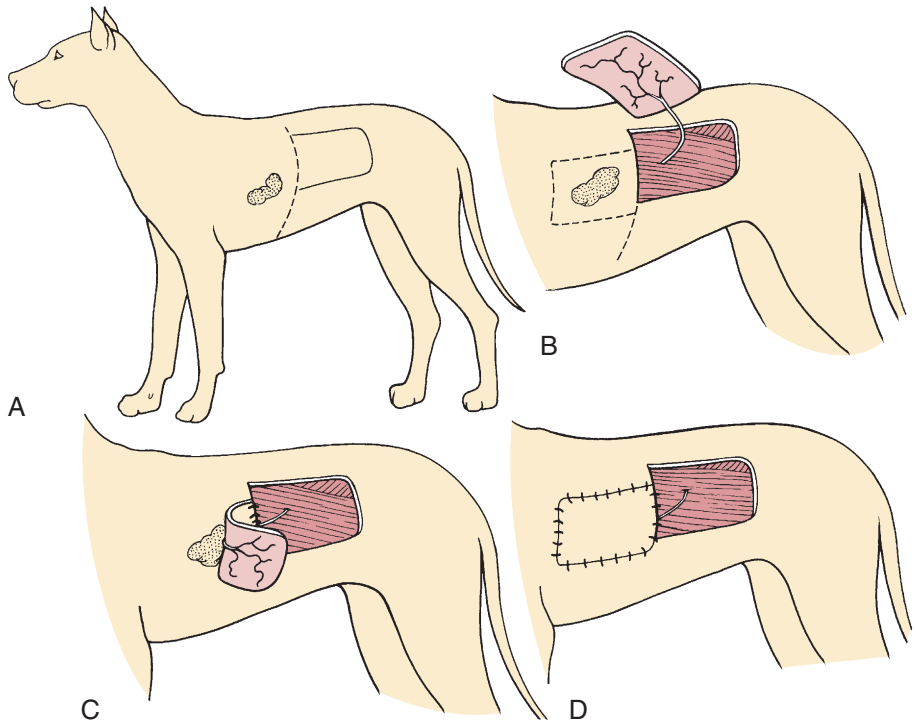


FIG. 15.43 An external abdominal oblique flap is created to reconstruct defects involving the caudal thorax or abdominal wall. (A) Make a paracostal incision from the epaxial muscles toward the ventral midline, beginning 5 cm caudal to the 13th rib. (B) Incise the lumbar fascial edge of the external abdominal oblique muscle and undermine it, preserving the neurovascular pedicle. (C–D) Transpose and suture the muscle over an adjacent defect.

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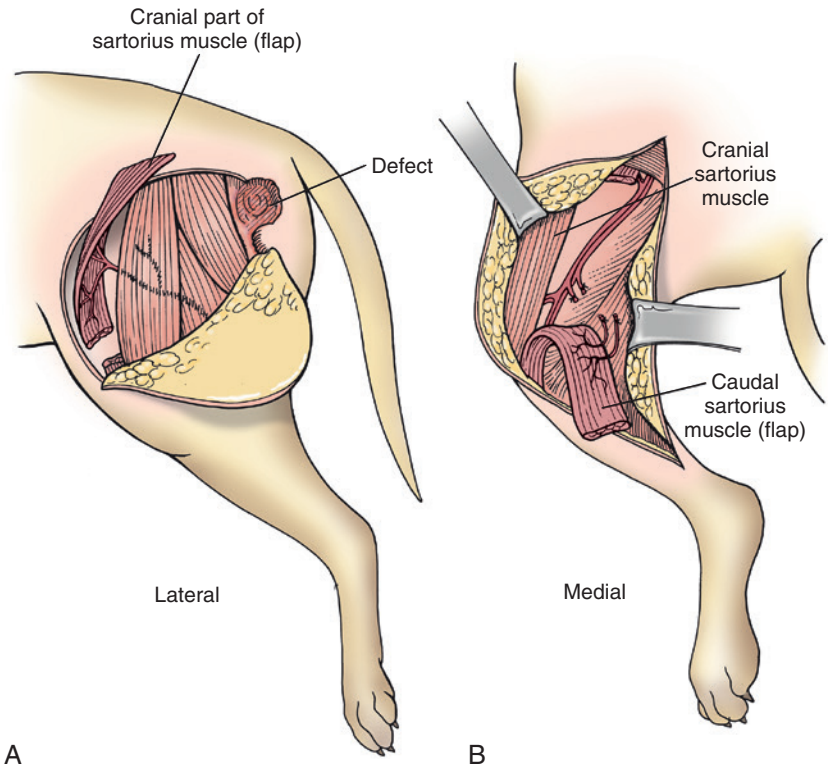


FIG. 15.44 (A) Use a cranial sartorius muscle flap to reconstruct prepubic tendon ruptures, femoral hernias, or femoral trochanteric ulcers. (B) Elevate a caudal sartorius muscle flap to cover defects over the tibia or metatarsus.

or more large vascular pedicles entering either the origin or insertion of the muscle and other smaller pedicles. In dogs, the origin of the cranial part of the sartorius is the crest of the ilium and the thoracolumbar fascia. The cranial sartorius inserts on the patella with the rectus femoris of the quadriceps. A single major vascular pedicle, branches of the femoral artery and vein, enters the proximal one-third of the muscle caudally. The muscle acts to extend the stifle and flex the hip.

Incise the skin of the medial thigh over the cranial sartorius extending to the inguinal region, and dissect subcutaneous tissue to expose the muscle (Fig. 15.44A). Isolate the muscle by blunt and sharp dissection from the caudal sartorius and quadriceps femoris muscles and other tissue. Transect the muscle distally at its insertion on the tibia and elevate it to its vascular pedicle, which enters the proximal one-third of the muscle caudally. Rotate the flap up to 180 degrees into adjacent defects. As an alternative, create an island muscle flap by subperiosteal elevation of the muscle's ilial origin. Suture muscle borders to adjacent fascial planes to cover the defect with absorbable suture material (e.g., 2-0 or 3-0 polydioxanone, poliglecaprone 25, or polyglyconate). Place a closed suction drain in the donor and recipient sites, and close the defect or defects.

Caudal Sartorius Muscle Flap

Caudal sartorius muscle flaps can be rotated distally to cover defects over the tibial or metatarsal area. These flaps may facilitate fracture repair when healing is impaired by osteomyelitis or poor circulation. Preoperative angiography ensures that the saphenous artery is not the primary source of circulation to the traumatized area and distal extremity. The caudal sartorius muscle originates from the cranial ventral iliac spine and adjacent ventral border of the ilium. It inserts on the cranial border of the tibia in common with the gracilis muscle. The caudal sartorius muscle has a segmental blood supply with a dominant vascular pedicle off the saphenous artery and medial saphenous vein at the distal third of the muscle belly. It acts to flex the hip and stifle.

Make a skin incision on the medial aspect of the thigh along the length of the caudal sartorius, and dissect subcutaneous tissue to expose the muscle (see Fig. 15.44B). Transect the caudal sartorius muscle approximately 4 cm distal to its origin on the ilium. Double ligate and transect the saphenous artery and medial saphenous vein where they join the femoral artery and vein. Prevent traumatizing the saphenous vessels in the medial tibial region and along the caudal border of the muscle. Transect the caudal sartorius near the tibial crest for complete mobilization. This creates an island muscle flap dependent on the saphenous artery and medial saphenous vein. *Extend the skin incision and further mobilize the vascular pedicle as needed. Transfer the flap to the desired location and secure. Place a closed suction drain and close the defects at the donor recipient sites.*

Flexor Arpi Lnaris Muscle Flap

The humeral head of the flexor carpi ulnaris muscle is useful for reconstructing injuries involving the tissues of the antebrachial, carpal, or metacarpal areas. The humeral head of the flexor carpi ulnaris lies cranial to the ulnar head of the flexor carpi ulnaris except distally, where the tendon lies caudally. The origin of the humeral head is the medial epicondyle of the humerus; its insertion is the accessory carpal bone. The blood supply to the proximal portion of the muscle is from varying pedicles of the recurrent ulnar, ulnar, and deep antebrachial arteries. A vascular

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pedicle from the caudal interosseous artery enters the distal end of the humeral head on its deep face near the accessory carpal bone. There are intramuscular anastomoses between the proximally coursing branches of the caudal interosseous artery and the descending branches of the ulnar and deep antebrachial arteries. The action of the flexor carpi ulnaris is to flex the carpus.

Incise along the caudolateral aspect of the antebrachium from just below the elbow extending 1 to 2 cm distal to the accessory carpal bone to expose the muscle. Incise the antebrachial and carpal fascia to identify the humeral head lying between the ulnar head of the flexor carpi ulnaris caudally and the ulnaris lateralis laterally. Transect the distal tendon of the ulnar head to completely expose the humeral head. Dissect the fascial attachments of the humeral head and transect at the junction of the proximal and middle third of the muscle (Fig. 15.45). Make a bridge incision to the wound and rotate the muscle medially or laterally into the wound. Appose the muscle to the surrounding viable tissue, and place drains as needed. Apply a moist, nonadherent bandage over the exposed muscle if the wound is not immediately covered with skin.

Temporalis Muscle Flap

Temporalis muscle flaps are used to close orbitonasal defects or to improve cosmesis after orbital exenteration. The temporalis muscle is fan shaped and arises from the temporal fossa, inserting on the mandibular coronoid process. The temporal and masseter muscles fuse between the zygomatic arch and the coronoid process. The temporal branches of the superficial temporal artery, cranial deep temporal artery, and caudal deep temporal artery supply the temporalis muscle. The blood supply enters the muscle near its narrow insertion on the mandible and runs in a ventrodorsal direction, paralleling the muscle fibers. The muscle closes the mandible in conjunction with the masseter and medial pterygoid muscles.

Make a cranial to caudal incision centered over the orbit to expose the temporalis muscle (Fig. 15.46). Preserve the superficial temporal artery. Dissect and transect the temporalis fascia from the zygomatic arch. Incise and subperiosteally elevate the desired portion of temporalis muscle. Rotate the flap around its insertion to the recipient site, resecting the zygomatic arch and lateral orbital ligament as needed. Secure the flap over the defect with approximating sutures (e.g., 2-0 or 3-0 polydioxanone, poliglecaprone 25, or polyglyconate). Obliterate dead space with a closed suction drain and close cutaneous defects routinely.

OMENTAL FLAPS

Omental flaps may be used to cover soft tissue defects, contribute to circulation and drainage, enhance healing, control adhesion, and combat infection, similar to muscle flaps. Although less durable than muscle flaps, they stimulate formation of granulation

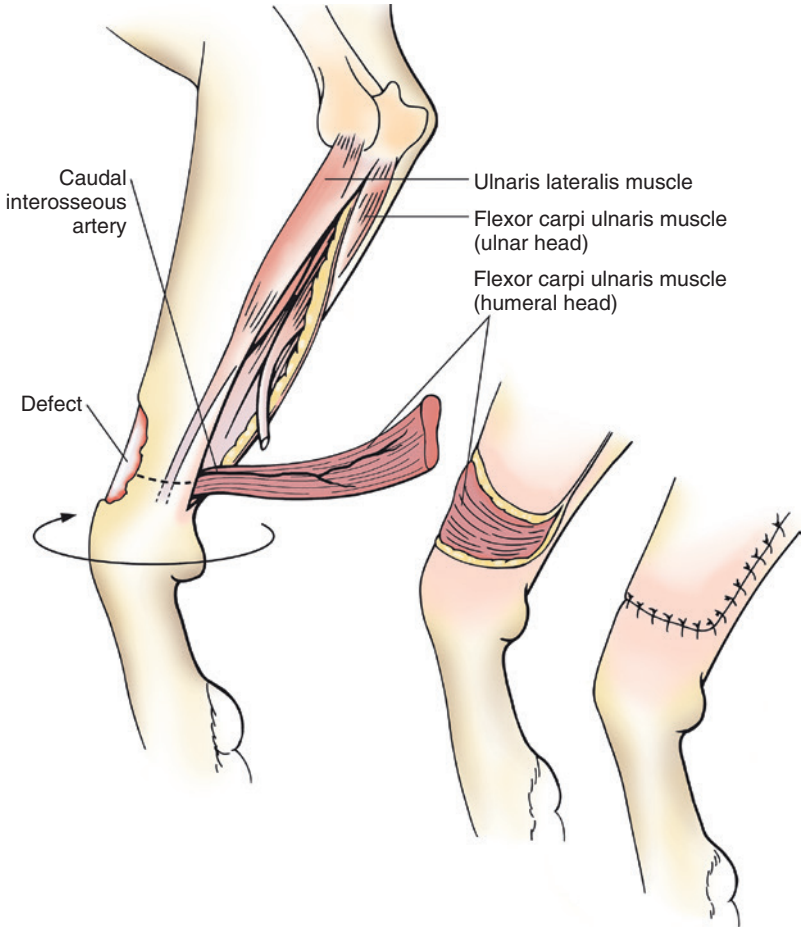


FIG. 15.45 Use a flexor carpi ulnaris flap with the caudal interosseous vascular pedicle entering the muscle on the deep surface near the tendon of insertion to cover wounds in the antebrachial, carpal, or metacarpal areas.

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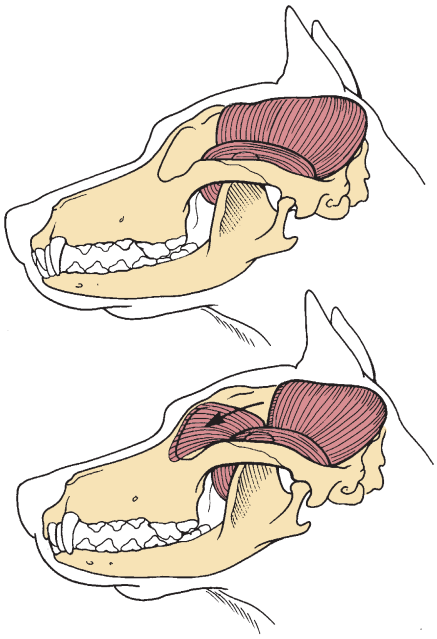


FIG. 15.46 Use a temporalis muscle flap to close orbitonasal defects.

tissue to allow earlier wound closure with skin flaps or grafts. They are especially useful for chronic nonhealing wounds involving the thorax, abdomen, and inguinal and axillary areas and can be used for facial and distal extremity wounds if extended omental lengthening or microvascular transfer is used. In dogs and cats, the omentum is a thin, double sheet of mesothelium that folds on itself in the caudal abdomen. It attaches ventrally to the greater curvature of the stomach and dorsally to the pancreas and spleen. The omental blood supply is from peripheral vessels of the right and left gastroepiploic arteries.

Two methods may be used to mobilize the omentum. One involves creating a vascular pedicle involving either the right or left gastroepiploic artery; the other involves releasing the omentum from the pancreas and then lengthening it with an inverted L-shaped incision. Both methods require a ventral midline celiotomy. The multiple vessel ligations required for the former technique increase the risk of hematoma formation, which may affect omental flap viability.

Create a vascular pedicle based on either the right or left gastroepiploic artery by ligating the segmental gastric arteries as they leave the gastroepiploic artery and enter the greater curvature of the stomach. Ligate either the right or left gastroepiploic artery, depending on which side of the body the omentum is needed. Incise additional attachments to the pancreas and spleen, and ligate vessels as necessary to harvest an appropriate length of omentum for transposition.

*To perform the other lengthening technique, retract the dorsal omental leaf cranially and exteriorize the spleen. Release the dorsal leaf from the pancreas using sharp dissection, and ligate or cauterize vessels as encountered. Ligate and transect the one or two vessels originating from the splenic artery close to the spleen. Extend the dorsal leaf caudally, unfolding the omentum. Begin the inverted L-shaped incision on the left side just caudal to the gastrosplenic ligament (*Fig. 15.47*). Double ligate and transect omental vessels when encountered as you extend the incision across one-half to two-thirds of the omentum's width. Continue the incision caudally, parallel to the remaining omental vessels, along two-thirds the length of the omentum. Ligate or cauterize vascular branches as encountered. Rotate the omentum caudally to fully extend the pedicle.*

After the omentum has been mobilized, make a small incision (2–3 cm) through the lateral abdominal wall near the wound or several centimeters from the celiotomy for distant wounds. Create a subcutaneous tunnel to the wound, transpose the omentum through this tunnel, and secure it to the wound with interrupted absorbable sutures. Take care when handling and transposing the omentum to keep it warm and moist and to prevent occlusion of remaining omental vessels to maintain its viability.

Possible complications after omental transposition include seroma formation, herniation through the omental exit hole, and flap necrosis.

MICROVASCULAR FLAP TRANSFER

Specialized training and equipment are necessary to successfully perform microvascular transfers. The required equipment includes an operating microscope with good optics, 9-0 to 11-0 suture material, approximating and atraumatic vascular clamps, microscissors, jeweler's forceps, ophthalmic needle holders, vessel dilators, and coupling devices. Most axial pattern skin flaps and muscle flaps can be used for microvascular transfer if vessels in the flap are large enough and if vessels of similar size are available near the wound bed. Peripheral angiography of the recipient site is advised to ensure that recipient vessels are present and functioning. Recipient vessels that have been used clinically for free flap transfer include the ulnar artery and cephalic vein in the forelimb; the cranial tibial artery and the dorsal branch of the medial saphenous vein or the plantar branch of the medial saphenous artery and its vena comitans in the hind leg; and the left infraorbital and superior labial arteries for palatal reconstruction. Anastomosis of recipient vessels should occur outside the area of trauma.

Once the location of the vascular pedicle has been identified during creation of the flap, magnifying loupes and the operating microscope are used to identify and isolate the vessels. Vessel clamps are applied, and the vessels are ligated and transected proximal to the clamps. At the recipient bed, the wound undergoes final debridement, and the vessels are identified and isolated in a similar manner. End-to-end anastomosis is preferred to end-to-side anastomosis because it is technically easier. Vessel ends are irrigated with heparinized saline, and excessive periadventitial tissue is removed. Arteries and then vein are anastomosed. Failure of flap transfer occurs if vessels are kinked or if they thrombose.

SKIN GRAFTS

Skin grafts are the transfer of a segment of free dermis and epidermis to a distant recipient site. They may be full thickness (epidermis and entire dermis) or partial thickness (epidermis and a variable portion of the dermis). They are used for defects that cannot be reconstructed by direct apposition or skin flaps (usually limb and large trunk defects). Skin graft survival depends on absorption of tissue fluid and revascularization. *Autografts* (grafts from the same animal) are most useful; however, *allografts* (grafts from the same species, also called *homografts*) and *xenografts* (grafts from different species, or heterografts) that

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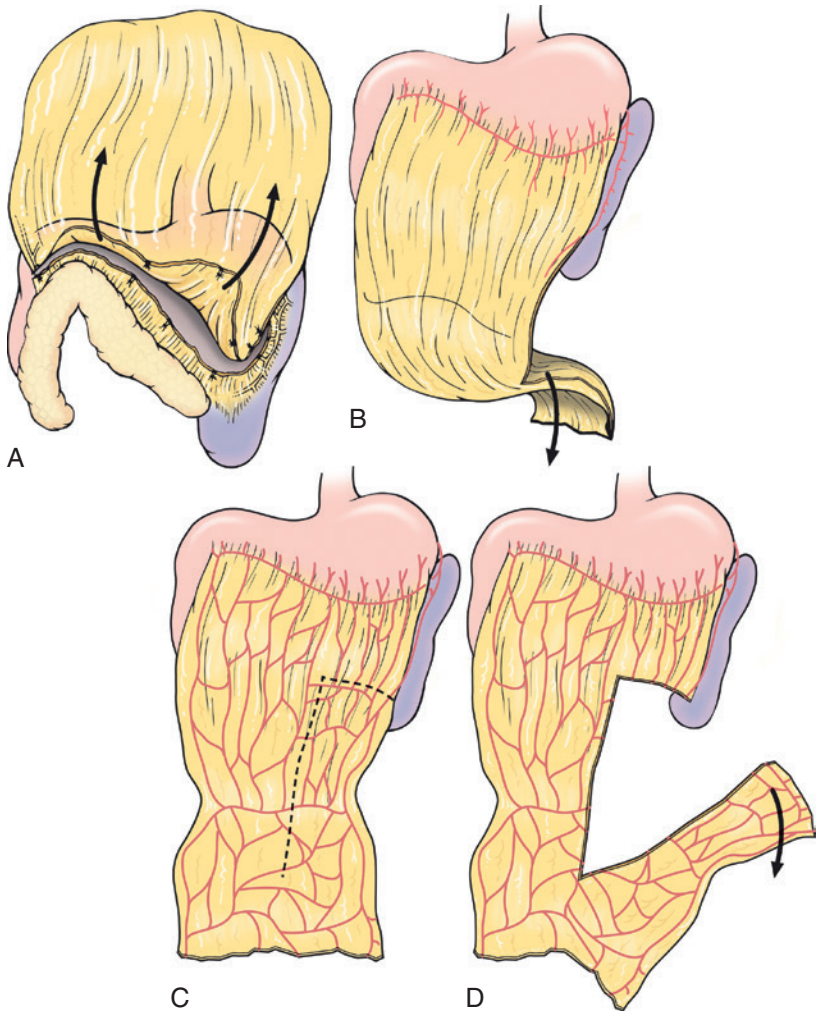


FIG. 15.47 Omental extension. (A) Exteriorize the omentum and spleen, then retract the dorsal leaf cranially and free it from its pancreatic attachments. (B) Extend the dorsal leaf of the omentum caudally. (C) Make an inverted L-shaped incision just caudal to the gastrosplenic ligament. (D) Rotate the left side caudally to achieve full extension.

are eventually rejected may be used to temporarily cover and protect large burned or denuded areas. Making templates of the defect and graft and drawing reference lines on the skin are helpful in planning reconstruction.

Successful graft healing, or graft "take," is dependent on the establishment of arterial connections and adequate drainage. This must occur by the seventh or eighth postoperative day, or the graft will die. The graft bed must supply adequate vasculature for the graft. Healthy granulation tissue or a fresh, clean wound free of infection and debris may serve as the graft bed. Healthy muscle, periosteum, and peritenon can support a skin graft. Bone, cartilage, tendon, and nerve that are denuded of their overlying connective tissue do not support grafts. Poor graft take occurs over avascular fat, crushing injuries, infected tissue, irradiated tissue, old or hypertrophic granulation tissue, and chronic ulcers. Chronic granulation tissue should be excised to allow new granulation tissue to form before grafting (approximately 4–5 days). The surface of healthy granulation tissue may be debrided by excising a thin layer (0.5–2 mm) with a blade or by rubbing with a gauze sponge before grafting. If bleeding persists, hemorrhage may be controlled with pressure or pinpoint electrocoagulation. The graft bed should be covered with moistened sponges while preparing the graft. A graft adheres to its bed by fibrin contraction soon after being placed. Fibrous tissue forms after fibroblasts, leukocytes, and phagocytes invade the area. The strength of graft adherence increases as fibrous tissue forms; by the tenth postoperative day, a firm union has occurred. Good graft contact with the graft bed is essential to adherence and graft take. To achieve good contact, the graft bed must be free of debris and irregularities. Immobilizing the graft with sutures and bandages minimizes graft movement over the wound and facilitates adhesion. Use of subatmospheric pressure drainage (see p. 192), which improves graft contact with the recipient bed and drainage, is being advocated to facilitate graft survival. Improperly applied or wrinkled bandages or those that apply excess pressure or abrade the graft can result in graft necrosis. Bandages should be well padded and bulky to restrict limb motion.

Plasmatic imbibition initially nourishes the graft and keeps the graft vessels dilated until the graft revascularizes. Capillary action pulls the fibrinogen-free, serum-like fluid and cells from the graft bed into the dilated vessels of the graft. Absorption of hemoglobin products gives the graft a bluish black color. The absorbed fluid

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diffuses into the interstitial tissue of the graft, causing edema; edema reaches its maximum at 48 to 72 hours after grafting. As venous and lymphatic drainage improves, fluid is taken away from the graft, and the edema regresses. Anastomosis of graft vessels with graft bed vessels of similar sizes *(inosculation)* may begin within 1 day of grafting. Vascular buds from the graft bed follow the fibrin scaffold to meet preexisting severed graft vessels. Vascular anastomoses form, and blood flow to the graft begins. Initially blood flow is sluggish and disorganized, but it improves and approaches normal by the fifth or sixth day. Fluid accumulations (i.e., seroma or hematoma) inhibit inosculation. Grafts may also be revascularized by the ingrowth of new vessels from the bed into the graft. New vessels form by endothelial sprouting and anastomosis with another sprout or formed vessel. Vascular sprouts may be found within the lower layers of the graft in 48 to 72 hours. New vascular connections remodel, differentiate, and mature until a system of arterioles, venules, and capillaries forms. New lymphatic vessels develop in the graft and establish lymphatic drainage by the fourth or fifth postoperative day.

Fluid accumulation within or under the graft and movement of the graft prevent good vascular connections from developing between the graft and the bed. A nonadherent, hydrophilic bandage should be placed immediately after grafting and left undisturbed for 24 to 72 hours to facilitate graft immobilization, fluid absorption, and graft adhesion, and to protect the graft from trauma. The frequency of bandage changes depends on the wound and varies from daily to every 3 to 4 days for at least 3 weeks. Grafts are pale when initially placed in a wound. They appear black and blue during the next 48 hours. The dark colors fade and a light reddish tinge appears by 72 to 96 hours. The entire graft should be red 7 to 8 days after surgery if graft survival is complete. Normal color gradually returns by the fourteenth day. Persistently pale areas are avascular and will undergo necrosis and slough. Black coloration indicates dry ischemic necrosis. Do not resect areas of questionable viability.

The most common causes of graft failure are separation from the graft bed, infection, and movement. They cause graft failure by disrupting the delicate fibrin bonds that bind the graft to the bed. Without adherence, revascularization and organization are impossible. Hematoma or seroma formation under the graft is a common cause of graft failure. Fluid mechanically separates the graft from its bed, impairing nutrition and revascularization. Meticulous hemostasis during graft bed preparation helps prevent hematomas and seromas. Nonexpanded mesh grafts, closed suction drainage, and vacuum-assisted drainage (see pp. 191 and 236) are the best methods of facilitating drainage. Mesh grafts have an advantage of not requiring placement of a tube that may disrupt graft adhesion and healing. Vacuum-assisted drainage has an advantage of promoting good contact between the graft and the recipient bed, and continuous drainage. The initial bandage change is recommended 24 to 72 hours after grafting to detect and drain fluid accumulation under the graft. The danger of fluid accumulation is greater than the risk of moving the graft during bandage manipulations. Infection is detrimental to graft survival because bacteria may cause dissolution of fibrin attachments or produce sufficient exudate to lift a graft from the recipient bed. Plasminogen activators and proteolytic enzymes released by bacteria disrupt the fibrin seal. β-Hemolytic streptococci and pseudomonads produce large amounts of plasmin and proteolytic enzymes. *Pseudomonas* spp. also produce elastase, which breaks down elastin; elastin adheres to fibrin, facilitating graft adhesion.

Donor site skin should have hair the same color, texture, length, and thickness as the hair surrounding the recipient site. The donor site should have enough skin to allow closure without tension after graft removal. Hair follicles may be damaged during graft harvesting and preparation or by poor graft revascularization. Removal of subcutaneous tissue may damage the base of hair follicles and reduce hair regrowth. Hair regrowth is usually noticed within 2 to 3 weeks after grafting; however, hair color may be altered after grafting. Split-thickness grafts result in sparse hair regrowth. Hair regrowth with strip, punch, and expanded mesh grafts is patchy. Full-thickness sheet grafts and nonexpanded mesh grafts result in the best hair regrowth and cosmetic appearance.

Reinnervation of grafts depends on the type and thickness of the graft, the amount of scar tissue formation, and the innervation of surrounding tissue. Return of sensation is greatest in flaps, less in full-thickness grafts, and least in split-thickness grafts. Reinnervation occurs from the margins of the graft. Pain is the first sensation to return, followed by touch, and last, temperature discrimination.

Full-Thickness Skin Grafts

Full-thickness skin grafts include the epidermis and entire dermis. They are indicated to cover large defects on flexor surfaces, thus preventing contracture and distal extremity defects (Fig. 15.48). After healing, full-thickness grafts resemble normal skin in hair growth, color, texture, and elasticity. They become pliable, movable, and durable. Full-thickness grafts take to the wound bed equally as well as split-thickness grafts. The disadvantages of full-thickness grafts include planning, tedious removal of subcutaneous tissue, and areas of nonviability. Full-thickness grafting techniques include meshes, plugs, strips, and sheets of skin.

Sheet Grafts

Sheet grafts are indicated to prevent contracture of defects on the distal aspect of the limbs and over flexor surfaces. They should be used only over uninfected granulation beds and when minimal fluid production is expected because fluid accumulation or drains prevent graft adhesion. Sheet grafts are less flexible, less expansive, and less conforming than mesh grafts. The donor site is the skin of the lateral thoracic wall, back, shoulder, or other areas with abundant skin. The lateral thoracic wall is the preferred donor site because the skin is relatively thin and well haired.

Aseptically prepare the surgical sites. Debride, lavage, and control hemorrhage in the graft bed before placing the graft. Make a pattern of the defect using a sterile towel or paper template. Using the pattern of the defect as a guide, harvest a segment of skin approximately 1 cm larger than the pattern from the donor site with the hair oriented in the proper direction. Excise all subcutaneous tissue from the graft with a scalpel blade during harvest or it will interfere with revascularization (Fig. 15.49). As an alternative, excise the graft, stretch it, and fix it to a piece of stiff cardboard or drape it over the index finger, and then remove subcutaneous tissue with a Metzenbaum scissors. Keep grafts moist by periodically submerging them in a bowl of saline or lactated Ringer's solution; this also rinses off fat fragments and helps identify remaining subcutaneous tissue. Keep the donor site moist with saline-soaked sponges while placing the graft. Place the graft in the defect with the hair properly oriented and with uniform contact with the recipient bed. Overlap the skin edges with the graft to ensure complete wound coverage and to prevent the skin edges from curling underneath. Tack the

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FIG. 15.48 (A) Injuries involving the distal extremities commonly require grafting. Grafts should be applied over healthy granulation tissue. (B) A full-thickness sheet graft has been applied over the wound.

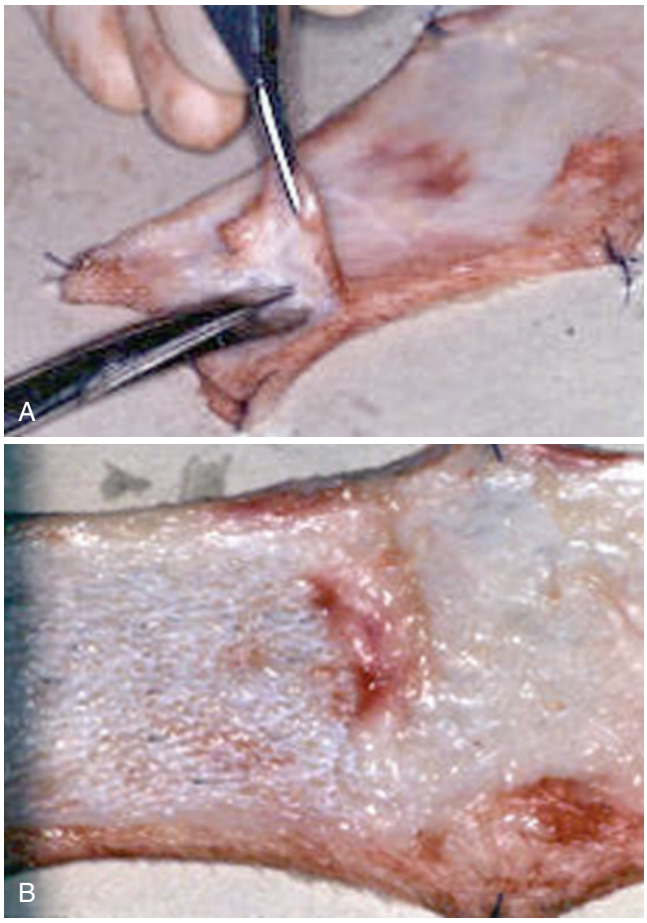


FIG. 15.49 (A) Subcutaneous tissue being removed from a full-thickness skin graft. (B) Close-up view of graft after subcutaneous tissue has been removed to expose hair follicles.

graft in position with interrupted sutures. Place a closed suction drain beneath the graft or make one or more stab incisions to promote drainage. Appose the edges of the graft and wound with staples or simple interrupted or continuous sutures (e.g., 3-0 or 4-0 nylon or polypropylene) placed 3 to 4 mm apart. Close the donor site by undermining and apposing wound edges or using a pedicle flap. Bandage the graft site with a nonadherent, hydrophilic, absorbent bandage that restricts the patient's motion. Change the evacuation tube as needed. Change the bandage, evaluate the graft 24 to 48 hours after surgery, and continue rebandaging as needed for 21 days. Where the graft overlaps skin edges, it will necrose and peel off when sutures are removed at 7 to 10 days.

Plug, Punch, or Seed Grafts and Strip Grafts

Plug, punch, or seed grafts and strip grafts are placed in a prepared granulation tissue bed. They are indicated for limb wounds and wounds with low-grade infection or irregular surfaces. They are best used on smaller wounds in areas not subjected to excessive wear or external trauma. These grafts are difficult to immobilize after implantation. The initial bandage change after surgery is delayed for 3 to 5 days to prevent displacing the grafts. Wounds that are parallel to the long axis of the limb lend themselves to strip grafting. Plugs and strip grafting are easy to perform and require no special equipment. However, excessive bleeding from the graft bed may float plugs out of the recipient site or delay revascularization. The cosmetic appearance is poor because of epithelial scarring and sparse hair growth, which result because the wounds heal by epithelialization from each graft and wound edge.

Prepare the graft bed by debriding and treating it as an open wound for several days. Harvest plugs of skin from the donor site with a 5- or 6-mm biopsy punch or tent the skin with a bent hypodermic needle or curved suture needle and resect a small piece of tissue. Harvest 5-mm-wide strips of skin freehand for strip grafting. Remove subcutaneous tissue from the dermis (see Fig. 15.49). For plugs, make small, slitlike pockets in the granulation tissue (2–4 mm deep, 5–7 mm apart), almost parallel to the wound surface. Insert a plug in each pocket after controlling hemorrhage, holding it in position with gentle pressure for 1 to 2 minutes. As an alternative, cut holes in the granulation tissue with a 4-mm skin biopsy punch (approximately 2 mm smaller than the punch used for harvesting the graft), approximately 1 to 2 cm apart, and insert the skin plugs into these holes. Make grooves 2 mm wide and 3 to 5 mm apart for strip grafting. After hemorrhage has been controlled, lay a skin strip in each groove and anchor it with an interrupted suture at each end. Bandage and splint the graft site with nonadherent, hydrophilic, absorbent materials. Excise and reappose the donor site or treat it as an open wound with bandages. Change the graft bandage 3 to 4 days after surgery, taking care not to dislodge any of the grafts. Rebandage the area as needed until healing is complete. Bandages should be bulky and restrict motion.

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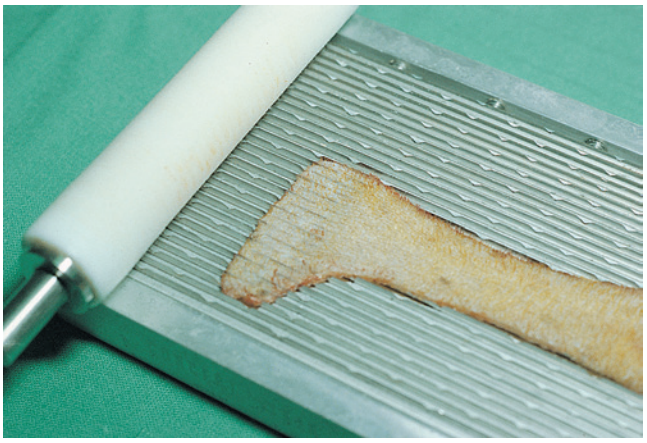


FIG. 15.50 After a skin graft has been harvested, it can be meshed (dermal side down) using a mesh graft expansion unit.



FIG. 15.51 Skin grafts may be meshed by making small, full-thickness incisions through the graft. The incisions are aligned in parallel rows.

Mesh Grafts

Mesh grafts may be either full-thickness or split-thickness grafts in which parallel rows of staggered slits have been cut. Meshing a sheet graft allows drainage, flexibility, conformity, and expansion. The degree of expansion is directly related to the length of the slits; longer slits equal greater expansion. As the graft is expanded, it shortens in the perpendicular plane. A sheet graft is meshed with a special mesh graft expansion unit (Fig. 15.50) or freehand (Fig. 15.51). Freehand slits are made in a sheet graft when it is fixed to cardboard after subcutaneous tissue has been removed. Slits are made with a No. 11 or No. 15 scalpel blade and should be approximately 5 to 15 mm long and 2 to 6 mm apart and oriented in staggered rows. Mechanically meshed grafts are more expansible because they expand in more than one direction; those meshed with freehand slits expand in only one direction.

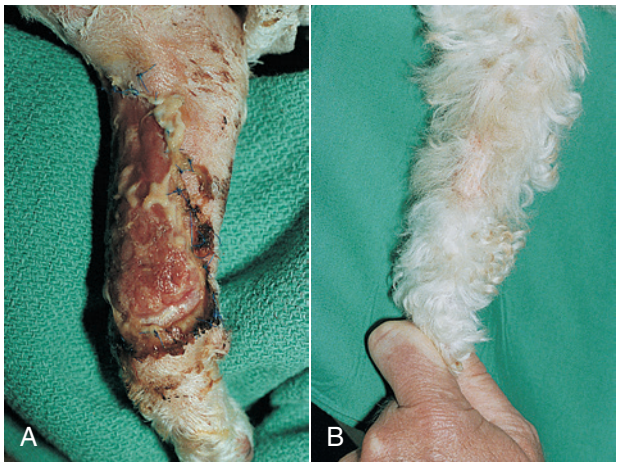


FIG. 15.52 (A) Skin graft in Fig. 15.40 at 6 days; note partial graft loss. (B) Cosmetic appearance after 100 days is good.

Cosmesis is improved if the slits are placed parallel to the skin tension lines.

A nonexpanded, full-thickness mesh graft is recommended for most grafting needs as it may be used under a wide range of circumstances, has a high success rate, and has a good cosmetic appearance. Meshing allows a graft to conform and adhere to irregular surfaces and allows placement on graft beds with exudation or blood. Meshing allows drainage, which facilitates graft adherence. The cosmetic appearance is as good as that with sheet grafts (Fig. 15.52). Survival is 90% to 100% when grafts are applied on healthy granulation beds and managed properly. Expanded mesh grafts are indicated when donor sites are limited and defects are large. They should be cut longer than the defect to account for shortening with expansion. A diamond-shaped pattern with tufts of hair between epithelial scars results when a mesh graft is expanded. This may not be cosmetically acceptable.

NPWT has been used in both clinical and experimental studies in dogs and cats with increased graft uptake and better overall outcomes.[6,10–12]

Split-Thickness Skin Grafts

Split-thickness skin grafts are composed of epidermis and a variable thickness of dermis. Feline skin is too thin for split-thickness grafting. Graft taken with split-thickness grafts is similar to that with full-thickness grafts. Split-thickness grafts are less durable and more subject to trauma than full-thickness grafts, and hair growth may be absent or sparse. In addition, the graft may appear scaly. Hair regrowth at the donor site of thick split-thickness grafts may also be sparse if the site is allowed to heal as an open wound rather than being resected. Skin of the lateral thoracic wall, back, shoulder, or another area with abundant skin may be used as the donor site. However, the flexibility of the abdominal wall and the irregularity of the thoracic wall make graft harvesting difficult. The relatively firm, flat surface over the epaxial muscles lateral to the dorsal spinous processes, lateral thigh, and arm muscles are better sites for harvesting skin.

Aseptically prepare the surgical sites. Debride, lavage, and control hemorrhage in the graft bed before placing the graft. Harvest the split-thickness graft with a dermatome or freehand. Inject sterile saline subcutaneously under the donor site to elevate the skin, and

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reduce contour irregularities. If using a dermatome, lubricate the skin surface with sterile mineral oil or water-soluble gel. Pull the skin in opposite directions over the donor site to make it taut. Harvest the graft. Using a scalpel, make a partial-thickness incision perpendicular to the skin surface. Then, holding a modified safety razor almost parallel to the skin surface, begin cutting. Place stay sutures in the cut edge of the graft to apply traction while cutting. Change blades as they become dull. Place the graft on the bed with hair growth oriented in the proper direction. Overlap the wound edges with the graft by 2 to 4 mm. Anchor the graft in place with interrupted sutures or skin staples. Irrigate under the graft with saline or thrombin. Apply a nonadherent, absorbent bandage with a splint to immobilize the area. Use a tie-over bandage if necessary. Holes inadvertently perforating the graft will allow drainage and eventually heal. *After harvest and grafting, excise the donor site and close it or manage it as an open wound with bandages. Perform the first bandage change 24 to 48 hours after surgery. Drain the area if a seroma or hematoma has formed by making a small incision in the graft and aspirating. Expect the graft overlapping the skin to necrose. Rebandage the area and change the bandage only as needed because movement of the graft bed interferes with revascularization.*

NOTE Increased pain is expected when the graft site is managed as an open wound; however, the wound will reepithelialize in approximately 3 weeks and additional split-thickness grafts can be harvested from the same site if necessary.

Stamp Grafts

Stamp grafts are square patches of split-thickness skin applied to a granulating wound. The graft bed and graft are prepared and managed in the same manner as for other split-thickness grafts. The graft is cut into patches ranging from 5 mm^2 to 25 mm^2. Patches are spaced 1 to 10 mm apart in graft bed depressions. These grafts are particularly susceptible to movement and easily displaced by bandages.

SURGICAL MANAGEMENT OF SPECIFIC SKIN DISORDERS

BURNS AND OTHER THERMAL INJURIES

Burns occur when heat energy is applied at a faster rate than tissue can absorb and dissipate it. Fires, electric heating pads, hair dryers, scalding water, steam, hot cooking oil, exhaust systems, and hot pipes are common sources of thermal burns in domestic animals (Fig. 15.53). The extent of injury is influenced by the temperature of the heat source, duration of contact, and tissue conductance. In humans, temperatures above 45°C (113°F) can cause coagulation necrosis and irreversible skin damage; a temperature of 70°C (158°F) for only 1 second causes a full-thickness burn. A transition zone separates completely devitalized tissue from uninjured tissue. The area in direct contact with the heat coagulates; cellular proteins denature and blood vessels coagulate. The transition zone is characterized by reduced blood flow, intravascular sludging, and potentially reversible tissue damage. Progressive dermal ischemia may occur in this area because of release of vasoactive substances (e.g., thromboxane A_2, histamine, leukotrienes, prostaglandins, oxygen free radicals), tissue edema, desiccation, and bacterial invasion. The transition zone is surrounded by an area of hyperemia where minimal damage occurs and healing is complete. It can be difficult to determine the burn depth and area of involvement as the depth of injury is not uniform and the skin surface is often leathery and covered by dry coagulum. Eschar is the residue of skin elements that have been coagulated by the heat. It is composed almost entirely of tough, denatured collagen fibers. Scabs contain dead cells and flimsy fibrin and, unlike eschar, are not a strong protective covering.



FIG. 15.53 A 360-degree thermal burn of the distal tibia in a 2-year-old cat. The limb was trapped in a double-pipe radiator. Note the pale appearance of the skin and distinct lines of demarcation proximally and distally.

Superficial *(first-degree)* burns affect only the epidermis. The area is painful, thickened, erythematous, and desquamated. Healing occurs rapidly (within 3–6 days) by epithelialization from stratum germinativum or adnexal dermal structures. Unlike human skin, canine skin does not act as an organ for heat dissemination; therefore dogs do not have the rich superficial vascular plexus that humans do. For this reason, dogs show less erythema with superficial burns than humans. Superficial partial-thickness burns are moist, blanch with pressure, and are sensitive to pain. They usually heal within 3 weeks because of epithelialization from deeper portions of the skin appendages. Healing is usually complete and occurs without grafting.

Deep partial-thickness *(second-degree)* burns cause major destruction of the dermis. The only remaining adnexal epithelium is in the upper layers of the subcutaneous fat. Subcutaneous edema and notable inflammation occur, and the hair does not epilate easily. Progressive damage during the first 24 hours results from the heat of injury and the release of proteolytic enzymes, prostaglandins, and vasoactive substances. Although these burns frequently heal without grafting, healing takes months, and scarring may be extensive. Healing occurs by reepithelialization from deep adnexa and wound margins. The burn must be protected against trauma and contamination while healing. Ineffective therapy may allow a second-degree burn to progress to a third-degree burn, especially if bacterial infection occurs.

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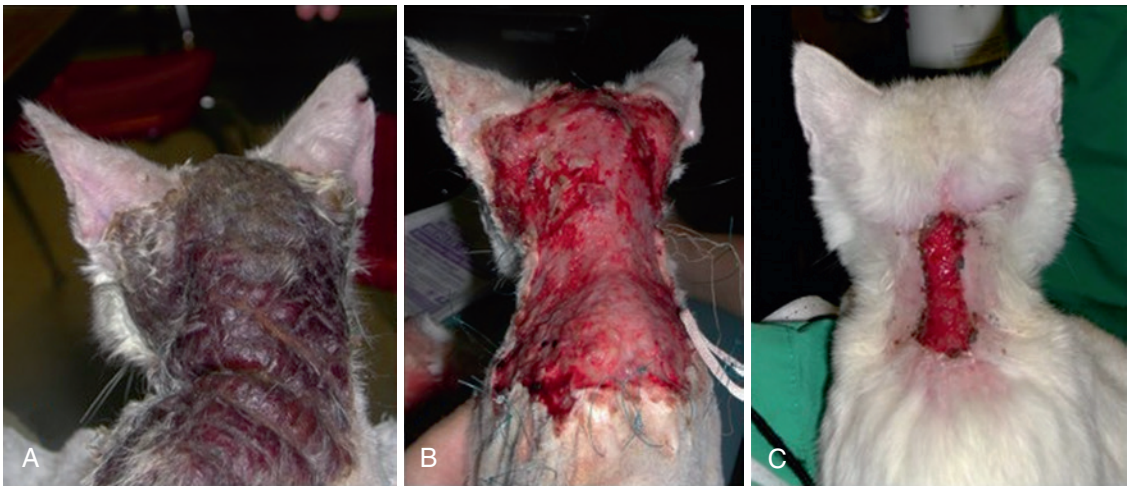


FIG. 15.54 (A) This 6-month-old cat has a thick eschar after receiving a burn of unknown etiology or duration. (B) The eschar was surgically excised and the wound debrided to fascial tissue. (C) The wound healed by second intention to a wide scar on the back of the neck.

Full-thickness *(third-degree)* burns form a dark brown, insensitive, leathery eschar (Fig. 15.54A). All skin structures are destroyed, and hair epilates easily. Third-degree burns are less painful than first- or second-degree burns as nerves have been destroyed. Superficial vascular thrombosis and deep vascular permeability cause subcutaneous edema and necrosis. Healing occurs by contraction and reepithelialization unless the wound is reconstructed. Some indication of injury depth may be obtained by elevating the eschar. First- and second-degree burn eschars split when elevated and bent to reveal underlying epidermis or dermis; third-degree burns may not split, or the split may extend to subcutaneous tissue. Early eschar removal is important as a necrotic eschar quickly becomes colonized on its deep surface and serves as a nidus of infection.

Burns that extend beyond the dermis are sometimes classified as *fourth-degree burns.* They have the same characteristics as third-degree burns but with additional tissue damage extending into the muscle and bone. Healing by secondary intention or reconstruction is usually required.

Burn wounds are sterile or colonized only by superficial bacteria during the first 24 hours. The large volume of dead tissue provides an excellent medium for bacterial growth, and occlusion of local blood supply impairs delivery of humeral and cellular defense mechanisms and systemic drugs to the wound. Superficial bacteria proliferate and invade the deeper tissue under the eschar within 4 to 5 days of injury. Initially, most organisms are gram-positive cocci, but by 3 to 5 days, the wound is colonized with gram-negative bacteria, typically *Pseudomonas* spp. Early removal of eschar and application of topical antibiotics are necessary to minimize the progression of damage.

Burns frequently cause shock and multiple organ failure due to fluid loss, fluid shifts, electrolyte imbalances, protein losses, myocardial depression, increased peripheral vascular resistance, and increased blood viscosity. Cardiac abnormalities, immunosuppression, anemia, renal failure, hepatic failure, and disseminated intravascular coagulation sometimes occur. More severe systemic signs are associated with large burn surface areas. Respiratory distress may occur from smoke inhalation (corrosive gases and chemical irritants), thermal burns of the upper airway, and carbon monoxide and cyanide poisoning. Smoke inhalation causes pulmonary edema with vascular congestion, interstitial edema, and atelectasis. Pneumonia often occurs several days after smoke inhalation.

Burns from contact rather than fire may not be immediately recognized. Moisture and flattening of the hair coat may be noted a few days after injury. This is rapidly followed by hair and skin loss, which makes demarcation of the burn area obvious. Burns may be prevented during surgery by using circulating warm water pads or bags inflated with circulating warm air (42°C [107.6°F] or lower) rather than electrical wire element pads. Thermal burns may also be caused by gloves or bottles filled with hot water. Anesthetized or hypothermic animals are particularly susceptible to burns from hot water bottles because of reduced circulation associated with vascular constriction. The longer the exposure, the greater the risk of burns. Burns may also be prevented by properly grounding patients when using electrosurgical units.

Treatment

The first priority in treating burns is to minimize tissue loss by administering first aid and preventing shock. Prevention of septic complications by good wound management is the next priority. Meticulous care immediately after injury to ensure adequate perfusion, hydration, and wound protection from trauma and infection may prevent progression of tissue damage and allow salvage of injured tissue. Early wound debridement and reconstruction are important to minimize morbidity. Cooling affected areas immediately after thermal injury (within 2 hours) may limit extension of tissue destruction. The area should be lavaged with cold water, or cold packs should be applied to the wound; however, it is important to prevent systemic hypothermia. Analgesics should be given as necessary to alleviate pain (see Chapter 13). Vital signs, mental status, hematocrit, total protein, urine output, central venous pressure, electrolytes, blood gases, and daily body weight should be monitored.

The size of the burn area can be estimated by measuring the area of burned skin with a metric ruler, dividing that area by the animal's total surface area (Table 15.5), and multiplying by 100. As an alternative, a rough estimate can be gained using the rule of nine: each forelimb of the animal represents approximately

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TABLE 15.5 Burns: Calculation of Total Body Surface Area

CONVERSION CHART BODY WEIGHT (KG) TO TOTAL BODY SURFACE AREA (M^2)

kg	m^2	kg	m^2
1.0	0.10	26.0	0.88
2.0	0.15	27.0	0.90
3.0	0.20	28.0	0.92
4.0	0.25	29.0	0.94
5.0	0.29	30.0	0.96
6.0	0.33	31.0	0.99
7.0	0.36	32.0	1.01
8.0	0.40	33.0	1.03
9.0	0.43	34.0	1.05
10.0	0.46	35.0	1.07
11.0	0.49	36.0	1.09
12.0	0.52	37.0	1.11
13.0	0.55	38.0	1.13
14.0	0.58	39.0	1.15
15.0	0.60	40.0	1.17
16.0	0.63	41.0	1.19
17.0	0.66	42.0	1.21
18.0	0.69	43.0	1.23
19.0	0.71	44.0	1.25
20.0	0.74	45.0	1.26
21.0	0.76	46.0	1.28
22.0	0.78	47.0	1.30
23.0	0.81	48.0	1.32
24.0	0.83	49.0	1.34
25.0	0.85	50.0	1.36

Total body surface area = $weight^{0.425} \times height^{0.725} \times 0.007184$ ($m^2 = kg^{0.425} \times cm^{0.725} \times 0.007184$) or Total body surface area = $0.1 \times weight\ (kg)^{2/3}$.
From Swaim SF. *Surgery of traumatized skin: management and reconstruction in the dog and cat.* Philadelphia: WB Saunders; 1980.

9% of the total body surface area (TBSA); each rear limb is 18% (two nines); and the dorsal and ventral thorax and abdomen are each 18%. Animals with partial-thickness burns involving less than 15% TBSA require minimal supportive therapy, whereas those with burns involving more than 15% TBSA require emergency supportive care. Euthanasia should be considered for those with burns involving more than 50% TBSA. Shock doses of lactated Ringer's solution or hypertonic saline solution should be administered to minimize and reverse signs of shock. The amount of isotonic fluid required during the first 24 hours may be estimated using the formula 3 to 4 mL/kg per percentage TBSA burned. Hypertonic saline solutions are beneficial in reducing total fluid requirements, limiting edema, and increasing cardiac output. Hypertonic saline (4 mL/kg bolus) plus lactated Ringer's solution (1 mL/kg per percentage TBSA burned) may be administered. When giving hypertonic saline, the serum sodium concentration should not exceed 160 mEq/L. Nonprotein colloid solutions (i.e., dextran 70, hetastarch) given in the early postburn period (16–24 hours after injury) may improve survival and reduce edema formation (see p. 33). Administration of protein colloids (i.e., fresh-frozen plasma or albumin) to hypoproteinemic patients should be delayed for 8 to 12 hours to allow the stabilization of membrane permeability and increased lymph return that reduces protein loss. Protein colloids given within the first 8 to 12 hours are lost into the burn wound and worsen edema formation. Dogs with partial-thickness burns involving 20% TBSA may lose 28% of their plasma volume during the first 6 hours. Transfusions (i.e., whole blood and packed red blood cells) may be necessary in anemic patients.

Respiratory distress should be treated by giving oxygen (mask, nasal insufflation, tracheostomy tube) and bronchodilators. The half-life of carbon monoxide is reduced with oxygen therapy. Continuous positive pressure ventilation may be necessary in some animals. Tracheostomy (see p. 842) and mechanical ventilation are indicated in patients with upper airway swelling or severe tracheobronchial secretions. The trachea and bronchi should be suctioned if necessary. Systemic antibiotics should be administered if bronchopneumonia occurs (see p. 888).

Aggressive nutritional support counters the increased metabolic demand and protein losses that occur in burn patients (see Chapter 10). Animals with moderate to severe wounds should be fed a high-protein, high-calorie diet. Early enteral feeding is important in preventing gastroduodenal ulceration. Proton pump inhibitors should be given if gastroduodenal ulceration is suspected (see p. 429).

Burn Wound Management

Removal of dead tissue is essential to the control of sepsis and promotion of a viable vascular bed suitable for surgical closure. Necrotic tissue may be debrided from burn wounds with dissection, autolytic, bandage, enzyme, or biosurgical techniques (see p. 185). Autolytic, enzymatic, and biosurgical debridement spare viable tissue that may be removed by surgical excision or bandages, but results vary; the best results are obtained with endogenous or exogenous enzymes on a moist, pliable eschar. Loose and obviously devitalized tissue in partial-thickness burns may be removed with scissors, hydrotherapy, or gauze sponge abrasion. With full-thickness burns, sharp excision to muscle fascia is necessary (see Fig. 15.54B). Early burn excision is recommended to minimize secondary infections and systemic effects (e.g., endotoxins and blood loss). Application of NPWT may prevent progression of partial-thickness burns by reducing edema and improving circulation.

Small burn wounds can be excised and closed primarily (Fig. 15.55). Closure is achieved by skin advancement or skin flaps. Larger wounds may be allowed to heal by contraction and epithelialization, or they may be grafted. Healing by secondary intention may take months or may be incomplete, and the resulting scar may be cosmetically unacceptable (see Fig. 15.54C). For these reasons, many large burns are debrided, allowed to form a healthy granulation bed, and then reconstructed using rotating skin flaps, axial pattern flaps, tissue expansion, or grafts. Early wound closure reduces wound management and secondary infection, and it shortens hospitalization. Scars are fragile and may erode and bleed easily. Squamous cell carcinomas occasionally occur in burn scars.

Numerous wound dressings have been evaluated specifically for burn care, and although most dressings have a beneficial effect on wounds, silver sulfadiazine delivers the best protection against wound colonization and infection. Silver sulfadiazine is bactericidal with activity against gram-positive and gram-negative bacteria and *Candida* spp. However, aloe vera is associated with

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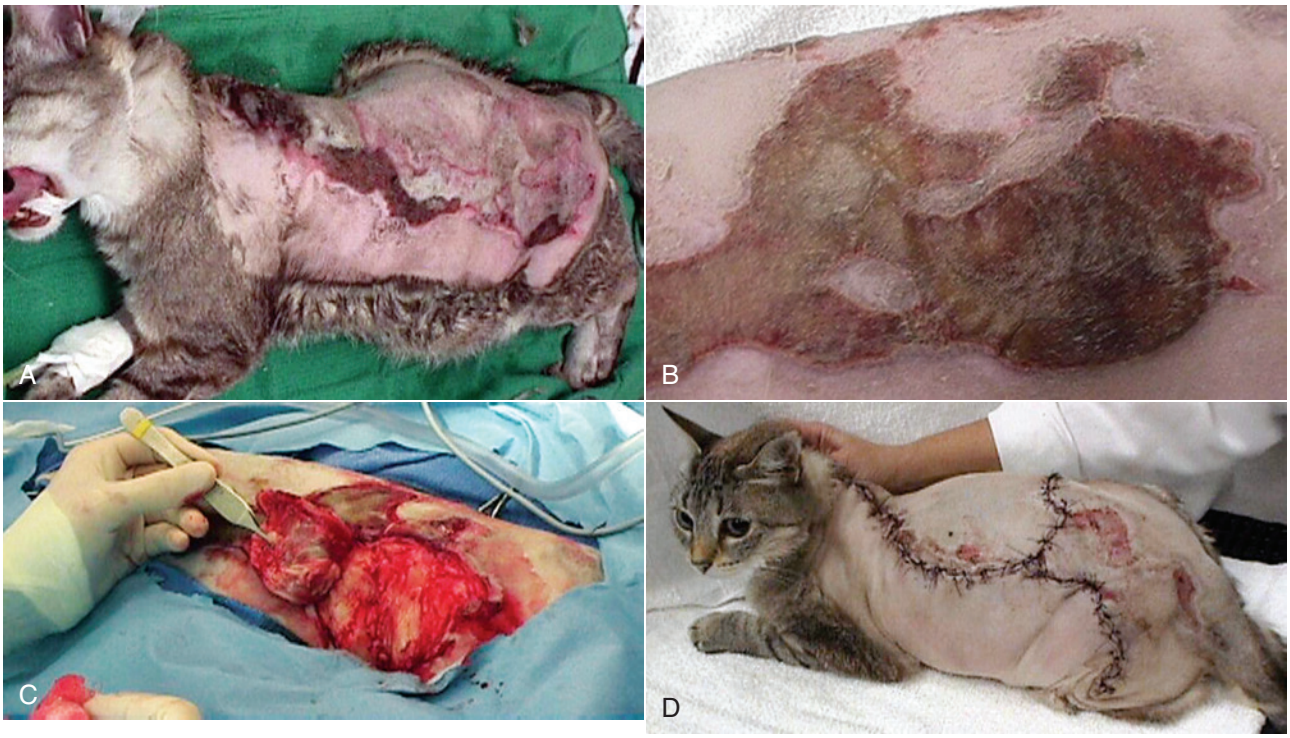


FIG. 15.55 (A) This cat received a thermal burn several days before presentation. (B) Close-up appearance of an area of the burn showing varying degrees of discoloration and a distinct line of demarcation. (C) The eschar is being surgically removed, exposing the subcutaneous tissue. (D) Postoperative appearance after excision and flap advancement to close the defect.

faster rates of reepithelization in partial-thickness burns. Paraffin gauze dressings have been shown to be associated with short healing times in superficial burns, and silver-based dressings are of most benefit in deeper burns. Alginate silver (e.g., Silvercel, Algidex AG) is an advanced wound dressing that combines the antimicrobial properties of silver with the absorptive wound management properties of calcium alginate and polyurethane foam; application to partial-thickness burns is associated with decreased pain scores and overall good wound outcome. Nanocrystalline silver dressings (Acticoat) have stronger antibacterial activity and are associated with less pain and reduced frequency of bandage changes.

Estimate the burn depth and calculate the size of the burn in relationship to the TBSA obtained from a weight conversion chart (see previous discussion and Table 15.5). Clip the wound and surrounding hair before gently lavaging with an antiseptic solution (e.g., 0.05% chlorhexidine diacetate). Cover the wound with a topical aloe vera compound or silver sulfadiazine (see p. 186), and then apply a hydrophilic bandage. After the first 24 hours, apply water-soluble, 1% silver sulfadiazine cream (Thermazene, Silvadene) to the wound once or twice daily or a slow-release silver sulfadiazine dressing (SilvaSorb) once every 3 to 7 days (see p. 200). Alternatively, apply medicinal honey to the wound (see p. 187). Bandage the wound, and aseptically manage it during subsequent bandage changes performed at intervals appropriate for the contact layer and amount of exudation (see Boxes 15.7–15.10 and Tables 15.2, 15.3, and 15.6). Medicinal honey bandages may require bandage changes several times daily. Remove the proteinaceous gel from the surface of the wound during bandage changes and before reapplication of topicals. Use gentle hydrotherapy to remove debris and clean the wound.

TABLE 15.6 Suggested Burn Wound Bandage Contact Layer

Eschar present—needs softening and debridement	Hypertonic saline or hydrogel dressing with silver sulfadiazine and biguanide-impregnated gauze
Eschar gone—needs further debridement	Hydrogel or hydrocolloid dressing and biguanide-impregnated gauze
Eschar gone—needs granulation	Hydrogel or calcium alginate and biguanide-impregnated gauze
Needs epithelialization	Polyurethane foam pad and biguanide-impregnated gauze

ELECTRICAL INJURIES

Electrical burns occur when current touches one point on the body, with or without an exit point. Chewing on electrical cords is the most common cause of electrical injury in small animals. Resistance to electrical current flow is greatest in bone and least in nerves (from greatest to least resistance, the order is bones, fat, tendon, skin, muscle, blood, and nerve). Low-voltage electrical current (<1000 V) follows the path of least resistance, which is usually along blood vessels. Low-voltage burns char tissue at the initial site, which decreases conductivity and limits further current flow, minimizing further injury. The initial contact point with high-voltage currents (>1000 V) negligibly decreases conductivity; therefore injury is extensive. Tissue necrosis occurs from vascular thrombosis and release of vasoactive substances. Tissue damage may be massive because of deep extension of the generated heat.

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Immediate death can result from respiratory paralysis or ventricular fibrillation, and tetany can result in spinal fractures.

Animals are often found collapsed in a tonic state with an electrical cord in the mouth. The body stiffens from contraction of striated muscles while receiving the electric current. Generalized tonoclonic activity with vomiting and defecation can also occur. If the animal survives, the tonic state resolves when the cord is removed from the mouth, although the animal may be weak and ataxic for a short period. Burns primarily occur on the lips, gums, palate, or tongue. These areas initially may appear charred, pale gray, or tan. Edema develops after 1 to 2 days. The extent of injury may not be apparent for 2 to 3 weeks. Pulmonary edema frequently occurs shortly after electrocution, causing dyspnea and moist rales.

Treatment

Affected patients should be examined frequently for pulmonary edema. Pulmonary edema may be treated with oxygen therapy, diuretics (i.e., furosemide, 2.5–5 mg/kg IV or IM every 1–4 hours as needed, or as a constant-rate infusion [CRI] at 0.66–1 mg/kg per hour), terbutaline, vasodilators (e.g., nitroprusside or nitroglycerine: these are potentially dangerous drugs; the reader is referred to a medical text for more information in using them), and aminophylline (5–10 mg/kg given intramuscularly or intravenously three times a day: IV administration should involve diluted drug given over 30 minutes). Terbutaline is generally preferred over aminophylline because the latter may cause vomiting, diarrhea, anxiety, or nervousness. IV administration in particular can cause tachycardia, arrhythmias, and central nervous system excitement and/or seizures. Caution should be used when giving aminophylline to dogs with seizure disorders because antiseizure medications (see Chapter 39) can lessen its effectiveness. It should be used cautiously in animals with hypothyroidism, liver or kidney disease, or congestive heart failure. Ventilatory support is needed if there is no response to medication.

Repair of damaged tissue should be delayed until the full extent of the injury is known. Technetium scans of muscle are helpful in determining viability. Minor burns may be allowed to heal by secondary intention. Oronasal fistulae must be repaired (see p. 346). Large lip wounds should be repaired to prevent oral drying and improve cosmesis.

FROSTBITE

Severe or prolonged cold may cause necrosis of exposed tissue. The extremities (i.e., ear, tail, scrotum, mammary glands, digits, and flank folds) are most commonly affected because of the sparse hair coat and poor peripheral circulation in these areas. Frozen tissue is pale, hypoesthetic, and cool. Thawed viable tissue is hyperemic, painful, and scaly. Nonviable tissue undergoes dry gangrene or mummification and sloughs. Superficial injuries involve skin and subcutaneous tissue, whereas deep injuries extend beyond the subcutaneous tissues. Ice crystals form in intracellular and extracellular spaces, causing cell damage and death.

Treatment

The affected body parts should be rapidly rewarmed in warm water (39°C–42°C [102°F–107.6°F]) for approximately 20 minutes to improve circulation. Affected areas become erythematous and edematous, form large vesicles, and are often painful, necessitating analgesics (see Chapter 13). Topical aloe vera or silver sulfadiazine should be applied to the affected areas. Bandages are used to prevent self-trauma. Conservative therapy should be continued until viable tissue can be distinguished from nonviable tissue (i.e., 3–6 weeks). Necrotic tissue should then be debrided and the area reconstructed if necessary. Healing may be complete beneath the mummified tissue.



FIG. 15.56 A chemical burn in a 6-year-old Border collie.

CHEMICAL INJURIES

Chemical burns from strong acids or alkalis or special chemicals destroy tissue by denaturing proteins or interfering with cell metabolism. The mechanism of injury is usually a direct chemical reaction rather than actual thermal injury, although thermal damage can result from an exothermic reaction (Fig. 15.56). Mechanisms of injury include oxidation, reduction, corrosion, dehydration, denaturation, and vesication. The severity of injury depends on the type of chemical and its strength; the volume involved, the contact time, and the depth of penetration; the chemical's mechanism of action; and the total area of contact. Corrosives (i.e., sodium-containing drain and oven cleaners, and phenol disinfectants) denature proteins, causing erosion and ulceration. Dehydrating chemicals (e.g., sulfuric and hydrochloric acids) desiccate tissue, and oxidizing agents (e.g., chromic acid, hypochlorite, and potassium permanganate) coagulate protein. Denaturizing agents (e.g., picric acid, tannic acid, acetic acid, formic acid, hydrofluoric acid) fix or stabilize tissue by the formation of salts. Vesicants (e.g., dimethyl sulfoxide, cantharides, halogenated hydrocarbons, gasoline) liberate tissue amines (histamine, serotonin) causing blisters.

Treatment

Resultant burns should be flushed with large volumes of water immediately after chemical exposure to remove the chemical and prevent further injury. Dry chemicals should be dusted off before irrigation. Flushing dilutes the chemical and dissipates the heat of chemical reactions. Do not immerse the area in water as this may spread chemical damage. After flushing with water, neutralizing agents, if used, are applied with gauze sponges, which are loosely wrapped over the area for 20 minutes. Attempting to neutralize alkaline injuries with acidic solutions (and vice versa) is dangerous and may lead to thermal injury from the resulting exothermic reaction. The animal should be prevented

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from licking the wound to prevent chemical burns of the tongue, oropharynx, and esophagus. Antimicrobials and bandages should be applied as described for thermal burns (see p. 237). Early debridement may prevent further chemical penetration and tissue destruction, but excessive tissue removal should be avoided.

Alkali

Lye (sodium hydroxide), cement (calcium, sodium, and potassium hydroxide), and many detergents produce saponification of fat and liquefaction necrosis of tissues, leaving them with a soapy feel. Irrigation should continue until the soapy texture is gone. Some alkalis (e.g., the common fertilizer anhydrous ammonia) penetrate the skin quickly and may require extensive irrigation for many hours.

Acid

Acids cause destruction by cell dehydration and production of acid albumins. Irrigate exposed skin with large volumes of water. Muriatic acid and sulfuric acid should be neutralized with soap before flushing. Hydrofluoric acid (risk in petroleum factories, glass etching, air condition cleaning, and chemical manufacturing) must be neutralized to stop penetration; irrigate with aqueous benzalkonium chloride or apply calcium gluconate gel, which precipitates residual fluoride ion. If pain persists, 10% calcium gluconate should then be injected into and around the lesion (0.5 mL/cm^2). Early local excision and grafting is indicated if pain continues or to prevent continued tissue damage. Calcium ions trapped as calcium fluoride may lead to hypocalcemia if hydrofluoric acid burns are extensive.

Special chemicals include phenol, phosphorus, and petroleum products such as gasoline and tar. Phenol has an anesthetic effect on the skin and is not water soluble. It is rapidly absorbed through the skin and may cause central nervous system depression, hypothermia, hypotension, intravascular hemolysis, and death. A lipophilic solvent (e.g., polyethylene glycol, propylene glycol, glycerol, vegetable oil) is used to cleanse the area. White phosphorus is used in insecticides, fertilizers, and incendiaries. It is lipophilic and is converted to phosphoric acid on contact with tissue. Its high penetrability may lead to systemic toxicity, causing damage to the liver and kidneys and depressed calcium levels. Phosphorus particles can also ignite when in contact with the air. Therefore copious lavage followed by wet soaks to prevent ignition is recommended. Rinsing the area with dilute 0.5% to 1% solution of copper sulfate results in the formation of a blue-gray cupric phosphide coating over the residual phosphorus particles to help prevent ignition and identify particles. An ultraviolet light can also be used to identify particles for removal. Gasoline's hydrocarbons act as a fat solvent and can damage skin, leading to absorption and systemic toxicity, causing multiorgan injury. Tar products cause thermal injuries. Cold water is used to cool the material, which is then dissolved and removed with a petrolatum-based ointment. If a petrolatum solvent is used, the burn may absorb it, leading to toxicity.

SKIN REACTIONS TO RADIATION THERAPY

Radiant energy from roentgen ray tubes, radioactive elements, luminous bodies, fluorescent substances, or the sun may cause injury. In animals, radiation injury is usually a consequence of external beam radiation therapy to cure or control various tumor types (Fig. 15.57). Effects of radiation therapy on normal tissue are related to the energy of the ionizing radiation, number of fractions given, total dose, and time elapsed following radiation. There are four degrees of radiation injury: (1) cutaneous erythema, (2) superficial epidermal (dry) desquamation, (3) moist desquamation from loss of basal layers of epidermis, and (4) necrosis with dermal destruction and irreversible ulceration. Cutaneous erythema occurs during the first week of therapy as a result of dilation of capillaries and increased vascular permeability. Superficial epidermal or dry desquamation occurs with doses less than 30 Gy and is characterized by pruritus, scaling, and increased melanin pigmentation. Moist desquamation occurs with doses greater than 40 Gy, being characterized by discomfort, bullous formation, epidermal sheds, edema, and fibrin exudates. Reepithelialization will occur within 10 days with moist desquamation if the wound does not become infected. Ulcers associated with moist desquamation often heal after therapy but recur. Higher doses of radiation lead to dermal destruction and irreversible ulceration.

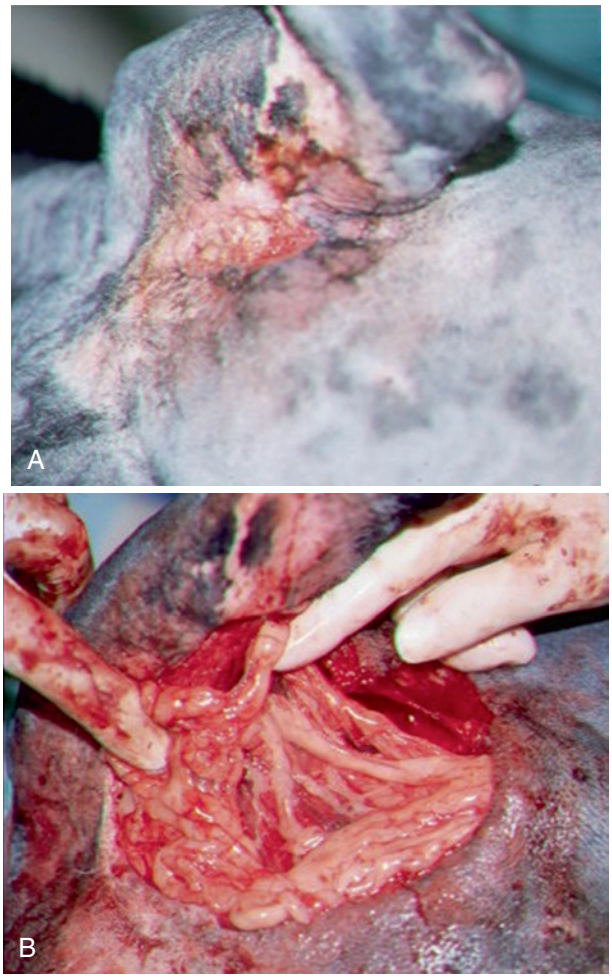


FIG. 15.57 (A) Acute tissue effects in the axilla of a 10-year-old mixed breed dog following full course radiation therapy. (B) Omentum was utilized to aid reconstruction and healing of the injury.

Radiation injury may also be classified as early (acute) or late (chronic) changes. Early (i.e., acute) injury is characterized by the development of edema, erythema, tenderness, and desquamation in the irradiated area within 1 to 4 weeks of radiation therapy. The skin may be either moist or dry. Acute injuries are due to

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depletion of rapidly dividing cells, occurring in renewing tissues, such as mucosa, epidermis, hair follicles, and sebaceous glands. Late (i.e., chronic) injury occurs in nonrenewing tissue, such as bone, nerves, and sometimes skin. Late injury is marked by dryness, pigmentation, induration, loss of pliability, atrophy or thinning, telangiectasia, keratosis, a decrease in adnexal structures, ulceration, progressive fibrosis, and a progressive decrease in circulation. Capillaries dilate and decrease in number and arteries sclerose as fibrous tissue increases and skin elasticity is lost. These changes are slow and progressive. Late injury may become evident weeks to years after radiation therapy. Radiation impairs soft tissue healing by (1) microvascular obliteration, (2) excessive fibrosis, and (3) disruption of cellular proliferation. Proliferating microvasculature, epithelial cells, fibroblasts, and myofibroblasts are damaged. After 1 year, the epidermis is thin, dry, and semi-translucent; hair follicles and sebaceous glands are usually absent. Much of the collagen and subcutaneous adipose tissue are later replaced by atypical fibroblasts and dense fibrous tissue that may cause induration of skin and limit movement. Eccentric myo-intimal proliferation of small arteries and arterioles may progress to thrombosis or complete obstruction. Ischemic ulcers are often a consequence of these vascular changes. Chronic changes to skin make it thin, hypovascular, extremely painful, and easily injured by slight trauma or infection. Irradiated tissue cannot tolerate the same degree of bacterial contamination as normal tissue, and antibiotics are not delivered in effective concentrations because of compromised microcirculation. The goal of treatment is to transform the chronic wound into an acute wound state.

NOTE When excising tissue that has been irradiated, be sure to submit all excised tissue for histologic examination.

Incision into previously radiated tissue should be avoided because healing is inhibited. Avoid or minimize surgical manipulations that may further compromise tissue. If surgery is to follow radiation therapy, it should be performed 2 to 8 weeks after therapy when radiation inflammation is subsiding and tissue circulation is still sufficient for healing.

Treatment

For acute radiation effects in the skin, treatment involves keeping the area clean, applying topical medications (hydrogel containing acemannan, silver sulfadiazine, or aloe vera cream), and preventing self-mutilation. *Cleanse the area with normal saline or a mild soap solution and rinse thoroughly to prevent irritation. Apply hydrophilic topicals that absorb water and act as mild lubricants.* Sometimes protective ointments or gels are applied to protect dry lesions. *Reduce itching with colloidal oatmeal bath (Aveeno Bath), cornstarch, or topical corticosteroid creams.* Moist desquamation injuries may also benefit from astringent soaks, antibiotics, and moisturizers. *Avoid any products with alcohol or menthol because they remove natural lipid and worsen the skin's reaction.* Experimentally, growth factors, orgotein, topical vitamin C, nonsteroidal antiinflammatory drugs, and helium-neon lasers have been used to treat acute radiation injuries. *Apply hydrophilic bandages to provide a moist wound-healing environment (see Box 15.8).* Most acute injuries heal within 10 to 14 days after conclusion of radiation therapy.

For chronic radiation injury, debridement of ulcers should be conservative, removing only necrotic tissue. Evaluating bleeding edges is not a reliable method of determining viability. Continual assessment and debridement are necessary to avoid removing tissue that will heal. Use topical antibiotics on irradiated wound beds after debridement because this tissue may have decreased resistance to infection and poor regional vascularity, leading to unreliable delivery of systemic antibiotics to the wound. Complete excision of all irradiated tissue may be necessary with chronic radiation wounds. Submit all excised tissue to determine if neoplasia is present. Wound closure is most successful when vascularized myocutaneous, cutaneous, muscle, or omental flaps are used (see Fig. 15.57B). Flaps are created outside the irradiated area and transposed over the wound. Healing by secondary intention and free grafts are apt to fail because of impaired vascularity. HBOT may improve healing by increasing the oxygen gradient in the wound or damaged tissue. Consider using NPWT devices (see p. 192), and apply hydrophilic bandages to encourage optimal healing (see Box 15.8).

ANIMAL BITE WOUNDS AND ABCESSES

Bites inflicted on one animal by another may be provoked or unprovoked. Bites often cause crushing, tearing, and avulsion injury that is more severe to the underlying tissue than is apparent from the often minor-appearing skin laceration or puncture (Fig. 15.58). This is because canine and feline skin is freely movable, and the bite pressure is high (150–450 psi). The most commonly affected sites are the neck, limbs, head, chest, shoulder, abdomen, and perineal regions. Multiple wounds are common. Mortality associated with bite wounds is less than 10% and is more common with thoracic bite wounds. Mortality is generally due to either infection or concurrent trauma; the wound is contaminated with the attacker's oral flora, as well as bacteria, hair, and other debris from the skin and environment. Devitalized tissue, dead space, compromised blood supply, and serum accumulate, creating a prime environment for bacterial growth. This type of environment is synergistic to anaerobic bacterial growth. Wound infection is common if treatment is delayed. Cat bites are more likely to become infected than dog bites.



FIG. 15.58 Multiple bite wounds on the antebrachium of a 1-year-old Staffordshire terrier.

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BOX 15.11 Normal Oral Flora of Dogs and Cats

Gram-Negative	Gram-Positive
Pasteurella multocida	*Bacillus* spp.
Enterobacter aerogenes	*Corynebacterium* spp.
Escherichia coli	*Staphylococcus aureus*
Pseudomonas fluorescens	*Staphylococcus epidermidis*
Acinetobacter calcoaceticus	*Staphylococcus saprophyticus*
Caryophanon spp.	*Streptococcus* spp.
Neisseria spp.	*Actinomyces* spp. (anaerobic)
Moraxella spp.	
Mycoplasma spp.	

Common aerobic isolates from bite wounds include *Pasteurella multocida*, *Staphylococcus* spp., *Enterococcus* spp., *Bacillus* spp., and *E. coli*; common anaerobic isolates include *Clostridium* spp. and *Corynebacterium* spp. (Box 15.11). Severe infections may occur after inoculation with *Mycobacterium* or *Sporotrichosis* organisms.

Culture all wounds and identify organisms present and their antimicrobial susceptibility. Initial antibiotic therapy before susceptibility data are available is based on whether organisms identified are gram-positive or gram-negative. Cephalosporins and penicillins are appropriate for many wounds as they have a good spectrum of activity and enter wound sites quickly when given intravenously. A single subcutaneous injection of cefovecin (8 mg/kg) repeated at 14-day intervals as necessary can be effective in naturally occurring wounds and abscesses in dogs and cats. Consider fluoroquinolones if gram-negative organisms are identified. Metronidazole or clindamycin can be administered if an anaerobic infection is suspected.

Manage bite wounds by clipping and scrubbing a wide area around the wound followed by copious lavage and cleaning of the wound (see p. 184). Explore full-thickness punctures and lacerations to remove debris and devitalized tissue and to determine the extent of the injury.

Penetration of the abdominal or thoracic cavity necessitates exploration of the cavity to identify and treat concurrent visceral injury. Ultrasonography and radiographs may help evaluate the wounds, but they may severely underestimate the amount of body wall or internal trauma.

Most external wounds should be managed with bandages (see p. 201) to optimize drainage and allow autolytic debridement. After 3 to 5 days, most acute wounds will be healthy enough for delayed primary closure with or without placement of passive or closed suction drains. More chronic bite wounds may require bandaging for a longer period. These wounds are closed secondarily (after the appearance of granulation tissue) or allowed to heal by secondary intention.

Closure of severely contaminated, infected, or traumatized wounds and sealing or healing of punctures often cause abscessation. *Treat abscesses by first clipping and aseptically preparing a wide area around the swelling or draining tract. Then, using aseptic technique, open the abscess by lancing or enlarging the tract's opening. Obtain cultures. Thoroughly lavage the abscess cavity, and debride necrotic tissue. Place a drain or leave the wound open to facilitate drainage and apply a bandage.*

NOTE Treat bite wounds with hydrophilic bandages for 3 to 5 days to minimize wound complications.

BOX 15.12 Snakebites: Key Points

Pit Vipers: Copperhead, Cottonmouth Water Moccasin, Rattlesnake

- Characteristics: Triangular heads with facial pits between eyes and nostrils, elliptic pupils, hollow fangs
- Usually bitten during spring and summer months
- Most commonly bitten on head; sometimes neck and legs
  - Verify bite by identifying two fang marks
  - May be bitten more than once
- Signs: Swelling, depression, tachypnea, bleeding punctures, petechial/ecchymotic hemorrhages, cardiac arrhythmias
- Expect: Leukocytosis, hemolytic anemia, thrombopenia, echinocytosis, abnormal coagulation, elevated fibrin split products

Treatment

- Supportive care: Fluids and analgesics; ± antiinflammatories, antihistamines, antibiotics ± oxygen, blood products, hetastarch, antiarrhythmic drugs
- Antivenom: Expensive and of uncertain value
- Wound: Mobilize; clip, aseptically prepare, and lavage wound; debride necrotic tissue

Do not use tourniquets, suction, or an incision to remove venom!

SNAKEBITE

Snakebites can cause severe local tissue damage and systemic effects. Snake venom is an extremely complex mixture of enzymes, proteins, and peptides; venom composition varies between species, individual snakes, and the bite itself. Venomous snakes in the United States include the Crotalidae (pit vipers) and Elapidae (coral snakes) subfamilies. The Crotalidae subfamily includes the copperhead, cottonmouth water moccasin, and rattlesnake. Pit vipers have a triangular head with facial pits between the nostrils and eyes, and vertically elliptic pupils (Box 15.12). Their fangs are hollow, retractable teeth (hinged) near the rostral maxilla. Coral snakes, along with many nonpoisonous snakes, have round heads, no pits, and round pupils. Coral snakes have small fangs that are fixed in the cranial maxilla. Their short fangs allow them to hang from the animals they bite. Nonvenomous snakes have teeth but no fangs.

Coral snake venom is primarily neurotoxic and hemolytic, causing moderate tissue reaction and pain at the puncture sites. There is a delay of several hours before the onset of systemic signs, which worsen gradually over 18 hours. The effects may last 7 to 10 days. Neurotoxicity caused by coral snake venom is characterized by central nervous system depression, vasomotor instability, and muscle paralysis. Coral snake envenomation may cause lethargy, tremors, ptosis, dysphonia, incoordination, and hematuria. Larger doses may cause vomiting, salivation, defecation, and generalized parasympathetic stimulation, followed by paralysis and death. The primary cause of death is respiratory paralysis.

Crotalid (pit viper) venoms are primarily enzymatic in activity (e.g., phospholipase A, phosphatases, exopeptidase, hyaluronidase, L-amino acid oxidase, proteases, and endopeptidase). These venoms are hematotoxic, vasculotoxic, and necrogenic. They alter the resistance and integrity of blood vessels, cause hypotension and bleeding, affect cardiac dynamics and nervous system function, and produce respiratory depression and myonecrosis. Bites most commonly occur on the face and legs. Bites correlate with the snakes' nonhibernating periods and times of day when owners are most likely to be walking with the pet. Signs of envenomation

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vary and depend on the species of snake, the volume of venom, and the size of the victim. The bite of a pit viper usually produces two puncture marks surrounded by edema. The fang marks may bleed. Erythema, edema, and pain are immediate local effects. Envenomation has not occurred if these signs are not seen within 20 minutes of the bite; envenomation does not occur in approximately 20% to 25% of bites. Progressive swelling and sometimes local hemorrhage occur with moderate to severe envenomation. Tissue discoloration caused by petechiae and ecchymosis occurs, followed by local tissue necrosis. Tissue damage varies with the depth of the bite and the amount of venom injected. Systemic signs of crotalid envenomation are usually lethargy, vomiting, diarrhea, hypotension, and shock. Other signs may include anorexia, salivation, thirst, lymph node pain, weakness, bradycardia or tachycardia, generalized tremors, coma, tachypnea, pulmonary edema, urinary and fecal incontinence, paralysis, convulsions, and hemorrhage. Venom-induced coagulation defects may be severe (activated clotting time >120 seconds). Other common laboratory abnormalities include echinocytosis (burr cells), leukocytosis, and thrombocytopenia. Serious effects of envenomation, such as respiratory paralysis and acute renal failure, may not develop for several hours; therefore animals should be monitored for a minimum of 24 hours after being bitten. A snake bite severity score (SSS; Box 15.13) can be used to help assess how significantly an animal is injured, as well as guide therapy, including need for antivenom administration.[13]

NOTE A canine vaccine (Red Rock Biologics) is available for Western Diamondback Rattlesnake bites. It may also protect against bites from the Western rattlesnake, sidewinder, timber rattlesnake, massasauga, and Copperhead. Partial protection may be obtained against Eastern Diamondback Rattlesnake bites. It does not protect against venom from the water moccasin (cottonmouth), Mohave rattlesnake, or coral snake.

Treatment

Treatment goals include neutralizing the venom, treating systemic effects and wounds, and reconstructing tissue defects (see Box 15.12). The animal should be supported with IV fluids. Glucocorticoids may help treat shock and reduce edema and pain. However, glucocorticoid use is controversial because it may enhance venom toxicity. Antihistamines are often given, and broad-spectrum antibiotics are used to inhibit wound infection. However, recent studies show little to no benefit to the administration of glucocorticoids, diphenhydramine, or prophylactic antibiotics.[14,15] Additional supportive care may include analgesics, sedatives, transfusions, and oxygen supplementation.

The bite wound should be immobilized and excitement or exertion avoided. The wound should be lavaged and cleaned with antiseptics or germicidal soaps. The use of tourniquets or incision, suction, or manipulation of the bitten area is rarely helpful and must be done immediately after the bite to have any beneficial effect. These techniques can also easily cause additional tissue damage. A tourniquet, if applied, should be placed 10 cm proximal to the fang marks, should be lightly constrictive, and should be released for 60 to 90 seconds every 30 minutes. However, tourniquets can prevent dilution of the venom and reduce tissue perfusion, thereby promoting ischemia and tissue necrosis.

The site should be clipped to facilitate examination for fang marks. Hospitalization and observation for systemic signs are indicated. Hemograms, coagulation profiles, urinalysis, and serum biochemical analyses should be monitored every 6 to 12 hours, depending on the severity of the envenomation. Persistent decreased platelet counts and prolonged clotting times suggest progressive venom activity. Myoglobinuria indicates rhabdomyolysis, and hemoglobinuria indicates hemolysis. The electrocardiogram should be monitored. The circumference of the edematous area around the bite should be measured and recorded to monitor progression.

Antivenom (antivenin) (Antivenin Cortalidae Polyvalent [ACP], Crotalidae Polyvalent Immune Fab [Crofab]) should be administered only if a snakebite is known to have occurred, but may not be indicated in every case of envenomation. One study

BOX 15.13 Snakebite Severity Score[13]

Pulmonary System	
Signs within normal limits	0
Minimal: slight tachypnea	1
Moderate: respiratory compromise, tachypnea, use of accessory muscles	2
Severe: cyanosis, dyspnea, extreme tachypnea, respiratory insufficiency, respiratory arrest of any cause	3
Cardiovascular System	
Signs within normal limits	0
Minimal: tachycardia, general weakness, benign dysrhythmia, hypertension	1
Moderate: tachycardia, hypotension (but tarsal pulse still palpable)	2
Severe: marked tachycardia, hypotension (nonpalpable tarsal pulse or systolic blood pressure <80 mm Hg), malignant dysrhythmia, or cardiac arrest	3
Local Wound	
Signs within normal limits	0
Minimal: pain, swelling, ecchymosis, erythema limited to bite site	1
Moderate: pain, swelling, ecchymosis, erythema involves less than half of extremity and may be spreading slowly	2
Severe: pain, swelling, ecchymosis, erythema involves most or all of one extremity and is spreading rapidly	3
Very severe: pain, swelling, ecchymosis, erythema extends beyond affected extremity, or significant tissue slough	4
Gastrointestinal System	
Signs within normal limits	0
Minimal: abdominal pain, tenesmus	1
Moderate: vomiting, diarrhea	2
Severe: repetitive vomiting, diarrhea, or hematemesis	3
Hematologic System	
Within normal limits	0
Minimal: coagulation parameters slightly abnormal, PT <20 s, PTT <50 s, platelets 100,000–150,000/mm^3	1
Moderate: coagulation parameters abnormal, PT 20–50 s, 50–75 s, platelets 50,000–100,000/mm^3	2
Severe: coagulation parameters abnormal, PT 50–100 s, PTT 75–100 s, platelets 20,000–50,000/mm^3	3
Very severe: coagulation parameters markedly abnormal with bleeding present or the threat of spontaneous bleeding, including PT unmeasurable, PTT unmeasurable, platelets <20,000/mm^3	4
Central Nervous System	
Signs within normal limits	0
Minimal: apprehension	1
Moderate: chills, weakness, faintness, ataxia	2
Severe: lethargy, seizures, coma	3
Total score (0–20)	

PT, Prothrombin time; *PTT,* partial thromboplastin time.

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found no difference in outcome in dogs receiving supportive care versus those receiving supportive care plus antivenom.[16] However, in an uncontrolled separate study, antivenom given to dogs with worsening envenomation syndrome had marked improvement in clinical signs.[13] When antivenom is given, it should be administered as soon as possible to limit tissue necrosis and prevent systemic reactions. Pretreating with antihistamines before intravenous administration may prevent anaphylactic reactions, although the incidence of adverse effects is low (~6%).[13] Recommended antivenom doses range from one to five vials. The number of vials used is directly related to the clinical signs, the animal's body fluid volume, and the location of the bite. Snakebites on digits or in small animals may require 50% more antivenom than those found elsewhere on the body or in larger animals. Although it is not known how long antivenom can be given after envenomation and still be effective, administration after 8 hours is considered of questionable value. In human beings, antivenom is given until pain associated with the bite is relieved. This end point is difficult to determine in animals; therefore antivenom should be given until serial examinations and coagulation profiles suggest that the animal's condition is stable. Administration of more vials of antivenom in crotalid envenomation is potentially associated with negative outcome in a large number of cases. Necrotic tissue should be treated as an open, infected wound (see p. 183). Bandages that support moist wound healing and debridement (see p. 195) should be applied until healthy granulation tissue has formed. Then nonadherent absorbent bandages (see p. 196) should be used and the wound allowed to heal by secondary intention or reconstructed with flaps or grafts.

PRESSURE SORES

Animals incapable of or unwilling to change positions are prone to pressure sores because pressure is exerted on bony prominences when animals lie down (Table 15.7). Pressure sores form when animals (especially large dogs) are recumbent for long periods because of paralysis, fractures, injuries, or illness. Pressure sores may also develop under improperly fitted or padded casts and bandages. Pressure on the wound pushes wound edges apart, complicating healing. They can be prevented by providing well-padded bedding (i.e., water mattress, air mattress, egg-crate type of foam-rubber mat, artificial sheepskin mat, and fleece), by repositioning the animal frequently, and by keeping the animal clean and dry. Bony prominences should be checked daily for signs of an impending ulcer (i.e., hyperemia, moisture, and easily epilated hair) (Box 15.14). The lateral humeral epicondyle, tuber calcaneus, greater femoral trochanter, tuber coxa, and ischiatic tuberosity are most susceptible to pressure sores (Box 15.15). Less common sites are the acromion of the scapula, lateral tibial condyle, lateral malleolus, olecranon, and sternum. Soft tissues, including skin, loose connective tissue, fat, deep fascia, and periosteum, cover these prominences. All intervening tissues are compressed when pressure is exerted on a bony prominence. Typically, repeated trauma and inflammation are mild, and protective callus develops. Prolonged or severe compression leads to soft tissue ischemia and cell death. Biochemical changes (oxygen free radicals, thromboxane) occur in ischemic tissue and contribute to necrosis. Elevated tissue thromboxane levels cause vasoconstriction, platelet aggregation, and ischemia to exacerbate already compromised tissue circulation. Persistent neutrophilia causes an ongoing inflammation, leading to a chronic wound.

As pressure sores begin developing, blood vessels dilate and inflammatory edema of skin and subcutaneous tissue occurs. If trauma persists, tissues break down, causing a hematoma or an open sore. Untreated hematomas are not absorbed because the surrounding tissues have been damaged. The fluid is mucinous and yellow to red. Tissues thicken around the hematoma, forming a false bursa (hygroma). The wall of the hygroma is thick and tough, composed of granulation tissue and collagen. The lining of this sac is pale and smooth or rough with irregular, villus-like projections extending into the lumen. Open sores may involve the epidermis and dermis or may extend through subcutaneous tissue and fascia to bone. Osteomyelitis or septic arthritis may develop. Diagnostic imaging may be helpful in determining the extent of soft tissue injury (ultrasound or computed tomography [CT]) and the presence of osteomyelitis or skeletal abnormalities (radiographs, CT, magnetic resonance imaging [MRI]).

Treatment

Treatment and prevention are similar (see Box 15.14). Early pressure sores are treated by padded bedding (water mattress, air mattress, egg-crate type of foam-rubber mat, artificial sheepskin mat, and fleece), bandaging the limb or using slings or side bars to eliminate pressure over the bony prominence, and changing the recumbent animal's position every 1 to 5 hours. A well-padded, doughnut-type of bandage or pipe insulation bandage should be applied to prevent trauma (see p. 201). In addition, the skin must be kept clean and dry. Treatment for open or chronic pressure sores is similar, but the response is poorer (see Table 15.7). Open wounds should be treated with topical agents and bandages that promote moist wound healing,

TABLE 15.7 Pressure Sore Classification and Treatment

Classification	Description	Treatment
Grade I	Dark red erythema with superficial to partial-thickness skin loss	Wound cleansing, debridement, bandaging, healing by secondary intention
Grade II	Full-thickness skin loss (ulcer) extending into the subcutis	Wound cleansing, surgical or bandage debridement, bandaging, and healing by secondary intention or wound excision and primary closure, delayed primary closure, or secondary closure
Grade III	Ulcer extends through subcutis to deep fascia overlying bony prominences; edges may be undermined	Treatment as for grade II, but drains may be necessary with undermined edges; muscle flaps or myocutaneous flaps and partial excision of the bony prominence may be necessary
Grade IV	Ulcer extends to bone; osteomyelitis or septic joint (or both) may be present	Treatment as for grade III, but sinus tracts and pockets may need to be excised or opened

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BOX 15.14 Prevention and Treatment of Pressure Sores

Prevention

- Keep pressure off vulnerable sites
- Bedding: Vinyl covered, thick egg-crate type of foam rubber with artificial sheepskin mat; water mattress or air mattress with fleece
- Keep clean and dry to prevent urine scalds
- Change animal's position every 1 to 5 hours
  - Rotate from right lateral, to sternal, to left lateral
  - Place in a sling for 2 to 4 hours daily
  - Support pelvic area in wheeled carts
- Apply properly padded and fitted bandages, splints, and casts
  - Do not apply extra padding over pressure points (actually increases pressure)
  - Use doughnut type of pad (see p. 202)
  - Reduce weight bearing: Foam pad, clamshell splint, or extension splints
- Apply skin toughening agents (e.g., Pad Toughening Compound)
- Provide good hygiene
  - Inspect skin daily
  - Reduce local skin moisture
  - Whirlpool baths
  - Clip perineal area if incontinent
- Physical therapy: Passive range of motion, massage, hydrotherapy, local hyperthermia and hypothermia, ultrasonography, neuromuscular stimulation (see Chapter 11)
- Provide good nutrition: High-protein, high-carbohydrate diet with vitamin supplements
- Surgery: Amputation, wedge osteotomy, arthrodesis or phalangeal fillet techniques for nerve or musculoskeletal abnormalities

Treatment

- Follow the prevention guidelines above
- Keep pressure off the wound
- Cleanse wound
- Debride and drain wound as necessary
- Apply appropriate topical medications (see p. 186)
  - Antibiotics
  - Acemannan products: Stimulate macrophages to produce cytokines and growth factors
  - D-glucose polysaccharide: antibacterial, bacteriostatic, and provides glucose for cell metabolism
  - Tripeptide-copper complex: Attracts mast cells and macrophages to stimulate healing
- Apply bandages that promote moist wound healing (see p. 195)
- Allow healing by secondary intention or transpose flaps to cover wound

BOX 15.15 Common Pressure Sore Sites

Most Common	Less Common
Lateral humeral epicondyle	Acromion of scapula
Tuber calcaneus	Lateral tibial condyle
Greater femoral trochanter	Lateral malleolus
Tuber coxae	Olecranon
Ischiatic tuberosity	Sternum

which encourages debridement and granulation. When deep ulcers are present, dead space should be drained and infected tissue and bone should be surgically debrided.

Small superficial wounds may heal by secondary intention. However, healing by secondary intention may not provide a surface durable enough to prevent reinjury. Secondary closure is performed after healthy granulation tissue has formed in large, deep wounds. Skin edges should be undermined and apposed if possible. Skin flaps (e.g., local advancement, transposition, axial pattern, or musculocutaneous [see pp. 217–228]) and grafts (see p. 232) may be necessary for a tension-free closure. Suture lines should not be positioned over bony prominences. A well-padded bandage and a soft padded bed should be used to protect the site during healing. Ulcers may recur if the cause goes uncorrected.

ELBOW HYGROMA

A hygroma is any chronic tissue swelling that contains serous fluid that develops as a result of chronic, repetitive compressive trauma to soft tissues over a bony prominence. An elbow hygroma (elbow seroma, olecranon bursitis) is the most common type of hygroma; it is a fluid-filled cavity surrounded by dense fibrous connective tissue that occurs over the lateral aspect of the olecranon (Fig. 15.59). Elbow hygromas often occur bilaterally as painless swellings. Most occur in young dogs (6–18 months old) of large and giant breeds before a protective callus forms over the bony prominence; however, they may occur in older animals with neuromuscular disease. Some dogs with thin skin and sparse subcutaneous fat are predisposed to hygromas. Others with hip dysplasia or pain from other orthopedic disease may exert excess pressure on the elbows while positioning themselves in sternal recumbency. Elbow hygromas vary in size, becoming larger and thicker with repeated trauma. They are usually sterile initially, but bacteria may be introduced during aspiration. Infected hygromas are painful. Small, painless hygromas are cosmetic problems that persist if not treated. Hygromas may also occur over other bony prominences (e.g., tuber calcaneus, greater trochanter, tuber coxa, tuber ischium, external occipital protuberance, and thoracic vertebral dorsal spinous processes).

Treatment

The primary treatment for elbow hygromas is elimination of repeated elbow trauma. Affected animals should be housed on yielding surfaces with soft, thick padding (see Box 15.14). External coaptation is applied to the elbow with a spica bandage (see p. 1166), elbow splint, or adjustable compressive bandage (e.g., DogLeggs). Most acute lesions regress slowly if repeated trauma is eliminated. The hygroma retains its ability to produce a fluid transudate until all granulation tissue and connective tissue

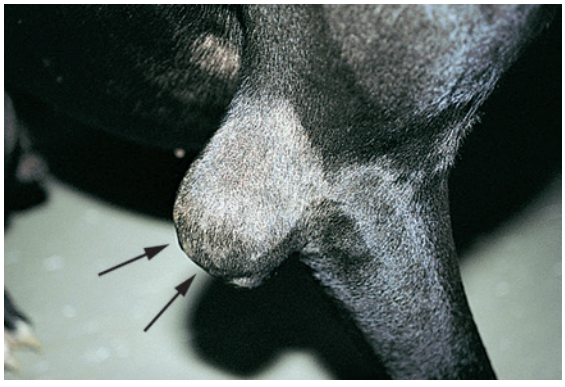


FIG. 15.59 Elbow hygroma in a dog. Note the fluid-filled swelling over the olecranon *(arrows)*.

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undergo fibrosis. Aspiration of the hygroma is of little benefit and may introduce bacteria. Although surgery should be avoided if possible as it often results in significant complications, development of infection or a chronic, fibrous capsule may require it. Infection requires drainage and administration of appropriate antibiotics. Prolonged drainage may be obtained by placing closed suction (preferred) or Penrose drains in the hygroma. Nonhealing wounds and infection are potential complications of drains. The advantage of this technique is that the protective callus is preserved; however, drains should not be used on ulcerated hygromas.

For nonulcerated (infected or sterile) hygromas, prepare the limb for aseptic surgery and make several dorsal and ventral stab wounds into the hygroma cavity. Probe the cavity, breaking down fibrous septa, and lavage it. Place one or more fenestrated closed suction drains in the hygroma cavity and secure them. Apply a nonadherent, absorbent bandage with a "doughnut" (see p. 202) to relieve pressure, absorb drainage, and prevent trauma. Change the bandage daily. Remove the drains when drainage becomes minimal and scar tissue adheres (i.e., 2–3 weeks). Continue to bandage the elbow for at least 1 week after removing the drains or until healing is complete.

When all conservative treatment attempts have failed, hygromas are occasionally surgically excised if fibrous tissue, fistulae, or infection develops without a large, fluid-filled cavity. The naturally protective callus is removed during excision, and postoperative management is often complicated. Incisions may dehisce and ulcerate, bandages are difficult to maintain, and recurrence is common. Wounds that dehisce may not heal. Small hygromas can be excised and the defect closed by undermining and advancing local tissue until skin edges can be approximated with interrupted sutures. Complete excision of fibrous tissue is ideal; however, when this is not possible because of the size of the lesion, the cavity lining should be debrided and lavaged, and a closed suction drain should be placed before closure. Incision and suture lines are positioned medial or lateral to the olecranon and other bony prominences if possible. It is difficult to excise large hygromas and close the wound without using skin flaps (i.e., axial pattern [see p. 222], pedicle [see p. 217], or myocutaneous [see p. 228]).

NOTE Surgical treatment of elbow hygromas may result in significant complications; however, surgery may be necessary in recurrent and complicated hygromas.

LICK GRANULOMAS

Lick granulomas (acral lick dermatitis, acral pruritic nodule, acropruritic granuloma, psychogenic dermatoses, and neurodermatitis) are self-induced by continuous licking or chewing. They are usually single and unilateral and may occur anywhere, although the cranial and medial aspects of the carpus-metacarpus and the cranial and lateral aspects of the tarsus-metatarsus are most commonly affected. Less frequent sites include the stifle, tibia, flank, and tail base. They usually occur in older, male, large breed dogs, especially Labrador retrievers, golden retrievers, German shepherds, German shorthair pointers, Great Danes, Saint Bernards, and Staffordshire terriers. Although wounds, foreign bodies, infections, and musculoskeletal pain may be initiating factors, most lick granulomas are believed to be psychogenic (obsessive-compulsive disorder) and associated with boredom, inactivity, or environmental change. A cycle of obsessive licking and secondary pruritus and infection develops that is difficult to interrupt. The lesion is sparsely haired, thickened, firm, ulcerated, erythematous, and surrounded by a hyperpigmented halo (Figs. 15.60 and 15.61). Secondary furunculosis and apocrine hidradenitis contribute to enlargement and inflammation of the lesion. Superficial tissue may erode and expose bone. Lameness sometimes results from the mechanical presence of the mass or from underlying periostitis. Dermatologic examination, skin scraping, fungal culture, biopsy, radiographs, electrodiagnostics, allergy testing, thyroid function testing, and/or hypoallergenic diet trials may be necessary. Diagnostic imaging may reveal periosteal proliferation secondary to soft tissue inflammation or associated arthritis, osteomyelitis, osteosarcoma, or foreign bodies. Biopsy to rule out neoplasia and deep cultures to determine bacterial involvement and antimicrobial sensitivity are recommended. Expected histopathology results include cellulitis, dermal fibroplasias, epidermal hyperplasia with compact hyperkeratosis, furunculosis, and vertical collagen streaking.



FIG. 15.60 Lick granulomas often occur in the carpal-metacarpal or tarsal-metatarsal area. They are sparsely haired, thickened, firm, ulcerated, erythematous, and surrounded by a hyperpigmented halo.



FIG. 15.61 Close-up view of a chronic lick granuloma.

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Treatment

Previously recommended treatments have included activity or environmental modification, bandaging, collars, muzzles, topical antichew agents, glucocorticoids, orgotein, radiation therapy, cryosurgery, surgical excision, behavior modifying drugs (phenobarbital, diazepam, hydroxyzine, naltrexone, hydrocodone), cobra venom, acupuncture, and other medications (i.e., fluocinolone acetonide, dimethyl sulfoxide, proteolytic enzymes). The results have been inconsistent, and recurrence is common.

Treatment should be individualized and initiated before the lesion becomes chronic and unresponsive. Initially, administer long-term antibiotic therapy (45–90 days) based on culture and susceptibility results; also use bandages and restraint devices. Periodic CO_2 laser crosshatching of the granuloma surface may help to eliminate hair embedded in the granulation tissue and facilitate control of infection. If an underlying cause is identified, it should be treated or eliminated (especially allergies), and behavior should be modified.

Treatment investigations involving behavior modification have shown beneficial results using serotonin reuptake-blocking drugs: clomipramine (1–3 mg/kg PO every 12–24 hours [dog] or 0.5 mg/kg PO per day [cat]), fluoxetine hydrochloride (1–3 mg/kg PO per day [dog] or 0.5–1 mg/kg PO per day [cat]), sertraline hydrochloride (0.5–1 mg/kg per day; compulsive disorder dose: 1–3 mg/kg per day [dog] or 0.5–1 mg/kg per day [cats]); amitriptyline (1–4 mg/kg PO every 12–24 hours [dog] or 0.5–1 mg/kg PO every 12 to 24 hours [cat]); or imipramine (2.2–4.4 mg/kg [dog] or 0.5–1 mg/kg PO every 12–24 hours [cat]); and electronic stimulation. Acupuncture may also be useful in treating lick granulomas. Although not commonly performed, surgical excision of a lick granuloma followed by reconstruction using direct apposition, flaps, or grafts is possible. The surgical site should be protected with a bandage until suture removal. However, the lesion usually recurs at the same or a different site unless the causative factor or factors are eliminated.

DERMOID SINUS (PILONDIAL SINUS)

A dermoid sinus or cyst is a tubular skin indentation that extends ventrally as a blind sac from the dorsal midline. It is a neural tube defect caused by incomplete separation of skin and the neural tube during embryonic development. Sinus depth varies; some are superficial, whereas others extend to the supraspinous ligament or dura mater (Table 15.8). Multiple or single draining tracts may be identified, especially in the cervical region (Fig. 15.62). Cervical sinuses are generally attached to the dorsal spinous process of the second cervical vertebra. They are most common in Rhodesian ridgebacks, occurring along the dorsal midline cranial and caudal to the midline ridge. Isolated cases have also been reported in many other breeds. Because the lesion is believed to be hereditary (complex dihybrid mode of inheritance) in Rhodesian ridgebacks, affected animals should be neutered.

Lesions can be recognized at a young age as openings on the dorsal midline with protruding hair or a whorl of hair. A tube or cord is palpated in the subcutaneous tissue when a skin fold is elevated in the area of the sinus. The cord is 1 to 5 mm in diameter and courses toward the spine. Cystlike subcutaneous swellings may also be palpated. The lumen of the sinus is filled with inspissated sebum, exfoliated keratin debris, and hair. Periodic plugging of the sinus opening leads to cellulitis, infection, and abscessation. Sinuses are classified as one of six types and one of three subtypes (see Table 15.8). Myelitis, meningomyelitis, or encephalitis can occur if the infected sinus extends to the spine. Cervical radiographs and advanced imaging (CT or MRI) should be performed to determine the extent of involvement (Fig. 15.63).

Congenital dermoid sinuses have also been reported on the dorsal nasal midline of golden retrievers, spaniels, and English bull terriers. Presenting signs are usually a fluctuant, nasal swelling and intermittent discharge. An opening, or "nasal pit," is usually found immediately behind the nasal planum (Fig. 15.64). It courses caudally and extends through an incomplete suture into the nasal septum. Embryogenesis of these sinuses is not completely understood, but it is believed to be a failure of the foramen cecum to close during maturation of the skull. The foramen cecum is a canal in the frontal bone at the base of the skull, which allows an outpouching of the meninges into the prenasal space and into contact with the somatic ectoderm. Dogs are usually presented in early adulthood for treatment of sebaceous discharge or secondary infection from the sinus. The sinus may be difficult to identify, and diagnostic imaging may be helpful in determining its caudal extent.

TABLE 15.8 Classification of Dermoid Sinuses

Type I	Tract extends as far as the supraspinatus ligament or nuchal ligament and attaches there.
Type II	Tract does not extend to the ligament but is attached by a fibrous band.
Type III	Tract does not extend to or attach by fibrous band to the ligament.
Type IV	Tract attaches to the dura mater.
Type V	Sinus has no connection to the skin surface; closed epithelial-line sac
Type VI	Tract extends to supraspinatus ligament and connects to dura mater by a fibrous cord.
Subtype a	Dorsal midline
Subtype b	Head, excluding nose
Subtype c	Nose



FIG. 15.62 Severely infected dermoid sinus in a 2-year-old dog.

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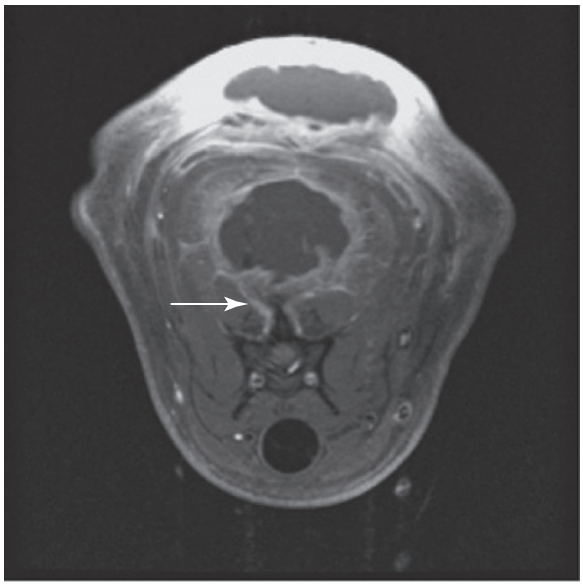


FIG. 15.63 Transverse T1-weighted magnetic resonance image of the cervical spine of the dog in Fig. 15.62. Note communication of the infected sinus with the dorsal spinous process of C4 *(arrow)*.



FIG. 15.64 Nasal dermoid sinus in a dog.

Differential diagnoses include foreign bodies, sebaceous cysts, abscesses, epidermal inclusion cysts, follicular retention cysts, and intracutaneous cornifying epitheliomas. Samples should be collected for cytologic studies and microbial culture and sensitivity testing. Metrizamide fistulography and myelography may reveal spinal communications, especially in conjunction with CT or MRI. Excised tissue should be submitted for histologic examination to rule out other causes of draining tracts. The histology of the dermoid sinuses is consistent with normal skin plus adnexa.

Treatment

Dermoid sinuses may be resected if they are associated with drainage or neurologic signs. Treat with antibiotics before surgery if the sinus is infected. Strict asepsis is essential to prevent postoperative meningitis if the sinus extends to the dura. Incomplete excision may occur if the sinus attaches to the dura and causes a chronic draining lesion. Successful treatment of nasal dermoid sinuses requires complete surgical excision with careful dissection between the nasal bones into the nasal septum. They usually terminate in the septum but may communicate with the dura.

Complications include recurrence if incompletely excised, abscesses, and meningitis. The prognosis is good with complete surgical excision. The prognosis is guarded if neurologic signs occur before surgery.

Clip and aseptically prepare a large area around the sinus. Position the dog in ventral recumbency with the spine or nose deviated dorsally; then make an elliptic incision around the sinus opening. Carefully dissect the sinus to its origin and free its attachment. Divide or split the nuchal ligament if necessary. Perform a laminectomy or hemilaminectomy (see pp. 1372 and 1379) if the sinus tract extends to the dura. Lavage thoroughly before closure. If the nuchal ligament has been transected, reappose it with a locking loop or modified Bunnell suture pattern (see p. 1283). Appose muscles and deep tissue with interrupted absorbable sutures to eliminate dead space (e.g., 3-0 or 4-0 polydioxanone or polyglyconate). If a large amount of dead space is still present, place a closed suction drain at the site. Appose subcutaneous tissue and skin routinely. Submit tissue samples for culture and sensitivity testing and histologic evaluation. Give analgesics and antibiotics, and bandage the site after surgery. Neuter affected animals.

INTERDIGITAL PYODERMA

Interdigital pyoderma (granuloma, acne, furunculosis, folliculitis) is a bacterial pododermatitis that may coexist with other conditions. Sometimes erroneously called an *interdigital cyst*, pododermatitis may be caused by parasites, allergies, mycoses, irritants, neoplasms, and metabolic, neurologic, or autoimmune disease. Bacterial infections usually occur secondary to demodicosis, allergy, hypothyroidism, or hyperglucocorticoidism. Immunosuppression is suspected in some animals. The primary bacterial pathogen is *Staphylococcus pseudintermedius;* secondary opportunistic bacteria include *Proteus* spp., *P. aeruginosa,* and *E. coli.* The condition is seen frequently in West Highland white and Scottish terriers, Pekingese, and English bulldogs.

Varying degrees of pruritus, pain, paronychia, swelling, erythema, and hyperpigmentation are common. Papules, pustules or nodules, draining tracts, and ulcers may be present. Chronic infections may produce interdigital fibrosis and pyogranulomas. Antibiotic and steroid therapy may cause remission, but recurrence is common. The underlying cause should be identified and treated. Diagnostics include hematologic and serum biochemistry profiles, urinalysis, skin scraping, culture and sensitivity testing, cytologic studies, and biopsy. Consider referring animals that do not respond to conservative treatment to a dermatologist.

Treatment

Conservative surgical treatment involves incision, exploration, and debridement of all fistulous tracts. Lesions should be medicated with antibacterial agents (e.g., chlorhexidine, povidone-iodine, and nitrofurazone) and bandaged for 24 to 48 hours. Subsequently, they should be soaked with an antibacterial solution for 15 to 20 minutes twice daily. Oral antibiotics based on sensitivity testing are administered for 6 to 8 weeks. Lesions that fail to respond to this treatment may require fusion podoplasty (see p. 265). Excision of all the interdigital tissue is usually curative in cases that fail to respond to medical therapy.

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REDUNDANT SKIN FOLDS

Redundant skin is characteristic of some breeds and is exacerbated by obesity. Chronic skin overlap or apposition creates skin folds of varying depths. Commonly involved areas include the labia, face, vulva, tail head, and leg folds, with severity and frequency varying with breed and conformation. Pyoderma occurs in the skin fold recesses (intertriginous dermatitis) because they provide a poorly ventilated, dark, warm, moist environment that permits the accumulation of surface debris, such as sebum, saliva, tears, urine, or feces. Sebum contains fatty acids that are both bacteriostatic and fungistatic; however, excessive accumulation of sebaceous material allows surface bacteria within the skin fold to proliferate. Coagulase-positive *Staphylococcus* spp. are most commonly involved, but other organisms include streptococci, coliforms (*E. coli,* pseudomonads, *Proteus* spp.), and yeasts, such as *Malassezia* and *Candida* spp. Friction at contact points, retention of secretions, and bacterial proliferation cause skin maceration and superficial ulceration. In addition to impairing the cutaneous barrier, immune function may be impaired. Affected areas become painful and foul smelling causing the animal to further traumatize the area by scooting, rubbing, licking, or scratching. The severity of clinical signs is affected by coexisting skin disease.

Skin fold resection is the most effective treatment for skin fold pyoderma (discussed later). First, medical therapy should reduce infection, inflammation, and secretions or exudates. Medical therapy consists of clipping hair from the folds and surrounding area, applying topical antibacterial solutions and medicated soaps, using antiseborrheic shampoos and astringents, and giving appropriate systemic antimicrobials. Culture and sensitivity testing is necessary to select appropriate antimicrobials. Glucocorticoids are sometimes necessary to reduce pruritus-induced self-trauma. Weight reduction is beneficial for obese animals. Continuous medical therapy is palliative, not curative. The surgical site must be kept clean, dry, and protected from trauma. Continued antimicrobial therapy may be necessary. General complications of skin fold resection and reconstruction are self-trauma, infection, dehiscence, and pyoderma recurrence.

Lip Folds

Breeds with excessive mandibular labial tissue (large pendulous lips) (e.g., spaniels, Saint Bernards, Newfoundlands, Labrador retrievers, golden retrievers, and Irish setters) most commonly have lip fold dermatitis. It may also occur after partial maxillectomy or mandibulectomy. The fold usually occurs behind the mandibular canine tooth, where food and saliva accumulate. Affected dogs rub and paw the face, and the skin becomes inflamed and thickened. Halitosis and pruritus are the most common presenting complaints.

Lip Fold Resection (Cheiloplasty)

Position the anesthetized animal in dorsal recumbency to allow access to both lips. Clip and aseptically prepare the mandibular area (Fig. 15.65A). Make an elliptical incision around the affected area, paralleling the horizontal ramus of the mandible (see Fig. 15.65B). The incision may involve the mucocutaneous junction. *Elevate and remove the outlined skin segment, preserving underlying muscles. Control hemorrhage with ligation, electrocoagulation, and pressure. Assess the adequacy of resection and excise additional skin if necessary. Lavage the site and appose subcutaneous and subcuticular tissue with continuous or interrupted approximating monofilament absorbable sutures (e.g., 3-0 or 4-0 polydioxanone, poliglecaprone 25, glycomer 631, or polyglyconate). Place interrupted appositional sutures in the skin (e.g., 3-0 or 4-0 polypropylene or nylon) (see Fig. 15.65C). Use an Elizabethan collar or bucket to prevent self-inflicted trauma to the surgical site. Keep the area clean and dry, removing food and saliva as necessary.*

Antidrool Cheiloplasty

Dogs whose face and neck are constantly wetted by saliva are candidates for this procedure. Antidrool cheiloplasty (cheilopexy) reduces the loss of food and saliva from the lateral vestibules of the oral cavity when there is excessive eversion or denervation of the lower lip. This is accomplished by suspending the lower lip from the inside of the upper cheek. Oral function is usually normal after surgery, but inflammation and infection occasionally occur at the surgical site. Permanent flap adhesion and cheek scars are expected.

Position the anesthetized patient in lateral recumbency. Clip the lateral face, lavage the oral cavity, and aseptically prepare the skin for surgery. Grasp the lower lip 2 to 3 cm rostral to the commissure, and elevate it dorsally until the lip is taut when the dog's mouth is completely opened (Fig. 15.66A). The site of maximum tautness is usually near the level of the caudal root of the upper fourth premolar. Beginning near an imaginary line between the medial canthus of the eye and the commissure, make a 2.5- to 3-cm, horizontal, full-thickness incision through the maxillary skin at the site of tautness (see Fig. 15.66B). Adjust the length of the incision to match the breed size. Control hemorrhage with ligation or electrocoagulation. The dorsal labial vein lies just dorsal to the proposed incision site. Use scissors to remove a 2-mm strip of mucosa adjacent to the mucocutaneous junction of the lower lip, beginning 2 cm rostral to the commissure and extending 2.5 cm (see Fig. 15.66C). Create 0.5- to 0.75-cm mucosal and skin flaps by undermining on each side of the incision (see Fig. 15.66D). Place stay sutures at the rostral and caudal aspects of the flaps. Evert the flaps through the cheek incision with the stay sutures (see Fig. 15.66E). Secure and bury the flap edges in the cheek skin incision with three to four preplaced vertical mattress sutures (e.g., 2-0 or 3-0 polypropylene or nylon) (see Fig. 15.66F and G). Place additional approximating skin sutures if necessary to achieve good skin apposition. Reposition the patient and repeat the procedure on the other side. Lavage the oral cavity with water after meals. Fit the dog with an Elizabethan collar to prevent self-inflicted trauma if necessary. Remove sutures at 21 days. Delay suture removal because constant lip movement may interfere with healing. Do not permit retrieving or chew toys for 2 months after surgery.

Nasal Folds

Brachycephalic breeds (i.e., English bulldogs, French bulldogs, Pekingese, Boston terriers, pugs, and Persian cats) characteristically have facial or nasal skin folds across the bridge of the nose. Prominent folds cause pyoderma and a foul odor (Fig. 15.67). Hair rubbing on the cornea is associated with keratitis, ulceration, epiphora, pain, and blepharospasm. Facial folds remain moist and become stained secondary to epiphora. Fold resection and ophthalmic medications are necessary. Excision of too much skin may cause ectropion or promote dehiscence.

Nasal Skin Fold Resection

Position the patient in ventral recumbency. Protect the eyes with a petrolatum-based ophthalmic ointment. Clip and aseptically prepare the dorsum of the nose and lips. Estimate the amount of

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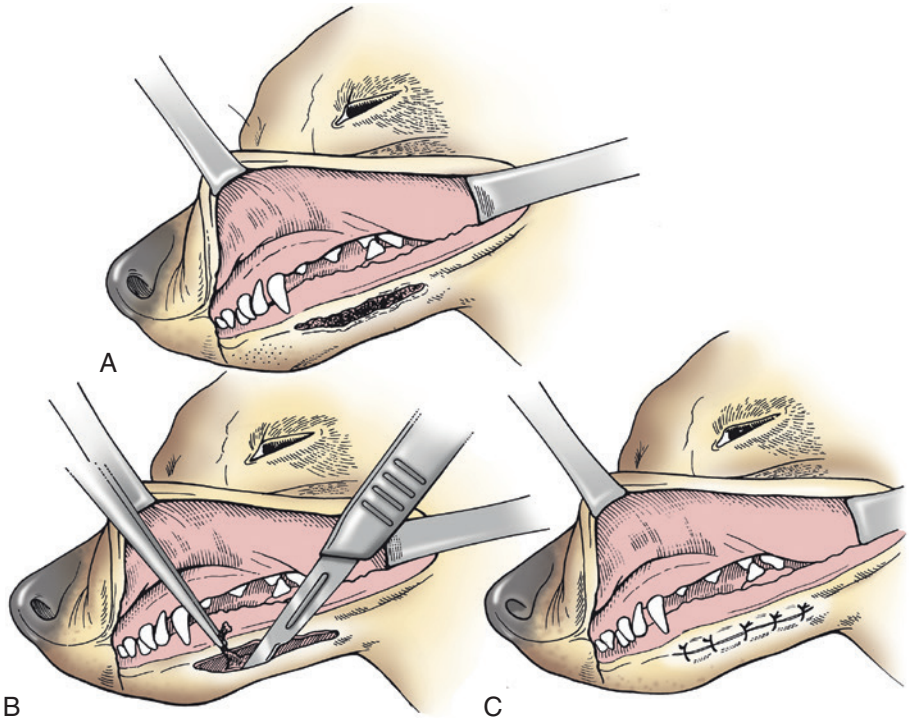


FIG. 15.65 (A) Cheiloplasty for lip fold dermatitis. (B) Make an elliptic incision and excise the infected skin. (C) Appose healthy skin edges with approximating sutures.

skin that must be resected to eliminate the skin folds without causing excess tension or ectropion. Make an elliptical incision around or through the skin folds in unmacerated tissue. Keep the caudal incision approximately 1 cm away from the medial canthus. Undermine and remove the outlined skin segment. Avoid traumatizing the nasolabialis muscle and facial vessels during dissection. Control hemorrhage with ligatures, electrocoagulation, and pressure. Lavage the area with sterile saline. Bury three or four interrupted sutures in the subcutaneous and subcuticular tissues to align and appose the skin edges and assess the adequacy of resection. Resect more skin if skin recesses remain. If necessary, undermine the skin edges to allow apposition without tension. Place additional interrupted, subcuticular sutures (e.g., 4-0 polydioxanone, poliglecaprone 25, glycomer 631, or polyglyconate) with buried knots. Use approximating skin sutures (e.g., 4-0 nylon or polypropylene), and cut the ends short to prevent further corneal irritation. Place an Elizabethan collar to prevent self-trauma. Keep the site free of exudates and ocular discharge. Continue to medicate the eyes.

Vulvar Folds

Vulvar skin folds occur in obese females and those with infantile, recessed vulvas (Fig. 15.68). Urine and vaginal secretions are trapped by the skin fold, resulting in superficial perivulvar dermatitis. Clinical signs include perineal pain, odor, vaginitis, repeated urinary tract infections, urinary incontinence, and pollakiuria. Skin fold pyoderma should be resolved medically before surgery. Episioplasty is a vulvar reconstructive procedure that removes the skin fold (see p. 740 for a description of the technique). Excessive suture line tension may cause dehiscence. Other complications include inflammation, swelling, infection, and recurrent perivulvar dermatitis. Perivulvar dermatitis recurs if the amount of skin excised is inadequate. Urinary incontinence may persist if associated with other abnormalities. Prognosis is good following surgery because most clinical signs are expected to improve.

Tail Folds

Redundant skin often overlaps deformed terminal caudal vertebrae ("screwtails," "corkscrew" tails, ingrown tails) (Fig. 15.69). Tail fold pyoderma occurs most commonly in brachycephalic breeds, but it has also been reported in Schipperke dogs and Manx cats. The skin fold may be quite deep (5–10 cm) with severe pyoderma. The depth of the folds varies with the animal's size, amount of fat, abundance of skin, and degree of vertebral deviation. Fecal contamination, licking, and scooting exacerbate the condition. Signs include perineal pruritus, pain, odor, ulcers, and fistulous tracts. Differential diagnoses should include perianal fistula, anal sacculitis, perianal tumors, trauma, and foreign bodies. Radiographs may be helpful to delineate the degree of caudal vertebral malformation. Draining tracts should be probed or a fistulogram performed to determine their site of origin. To remove all skin recesses at the tail head, complete caudectomy is necessary (see also p. 255).

Tail Fold Resection

Administer perioperative antibiotics based on skin fold culture and sensitivity results, otherwise broad-spectrum empirical antimicrobials should be used. Scrub the skin folds separately from the remainder of the surgical field. Resect the tail and skin folds en bloc, taking care during dissection to avoid penetrating the skin folds or traumatizing the rectum. Manipulate the tail with bone-holding forceps or towel clamps. Ankylosis or severe ventral deviation may make the vertebrae immobile. *Transect the tail cranial to the deviated vertebrae with Gigli wire or a bone cutter if the intervertebral space cannot be located. Smooth sharp bone edges with rongeurs or a bone rasp. Lavage thoroughly and insert closed suction drains (if*

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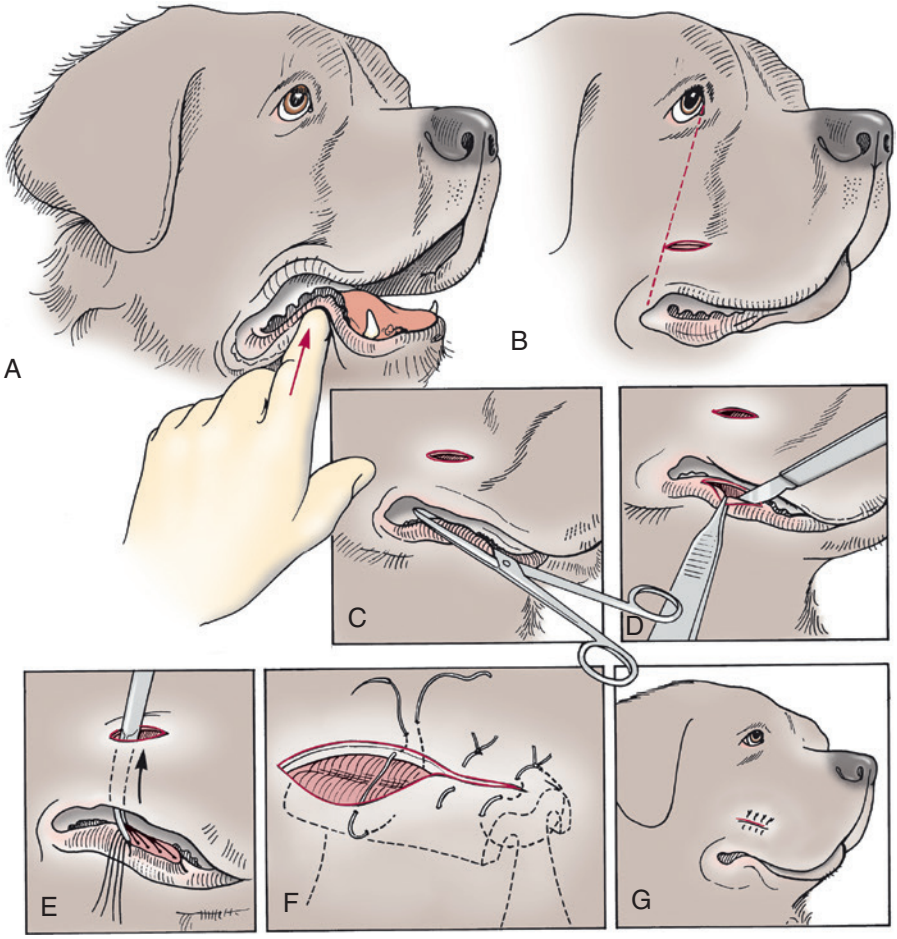


FIG. 15.66 (A) For cheiloplasty in dogs that drool excessively, elevate the everted lip dorsally until it is taut when the dog's mouth is opened maximally. (B) Make a 2.5- to 3-cm horizontal, full-thickness incision through the maxillary skin at the site of tautness near the upper fourth premolar. (C) Remove a 2-mm-wide strip of mucosa 2.5 cm long from the mucocutaneous junction of the lower lip beginning 2 cm rostral to the commissure. (D) Create 0.5- to 0.75-cm flaps. (E) Evert the flaps through the skin incision. (F–G) Secure with vertical mattress sutures.



FIG. 15.67 This English bulldog has a severe facial fold with pyoderma; the nares have been resected.

indicated) before apposing the subcutaneous tissues with absorbable sutures (e.g., 3-0 or 4-0 polydioxanone, poliglecaprone 25, glycomer 631, or polyglyconate). Close the skin with nonabsorbable sutures (e.g., 3-0 or 4-0 nylon, polybutester, or polypropylene). Keep the area clean and free of exudate and fecal contamination by applying warm, moist compresses two or three times daily for 15 to 20 minutes. Remove drains in 3 to 5 days.

SURGERY OF THE TAIL

CAUDECTOMY

Caudectomy, or amputation of a portion of the tail, which is performed to comply with breed standards or tradition, is ethically and morally controversial. *Therapeutic caudectomy* is indicated for traumatic lesions, infection, neoplasia, and possibly perianal fistula. The tail should be amputated with 2 to 3 cm of normal tissue margins when resecting tumors or traumatic lesions. Amputation should be performed near the anus if the end of the tail chronically bleeds because of repeated abrasion or chewing. Amputation near the base is recommended for avulsed tails and

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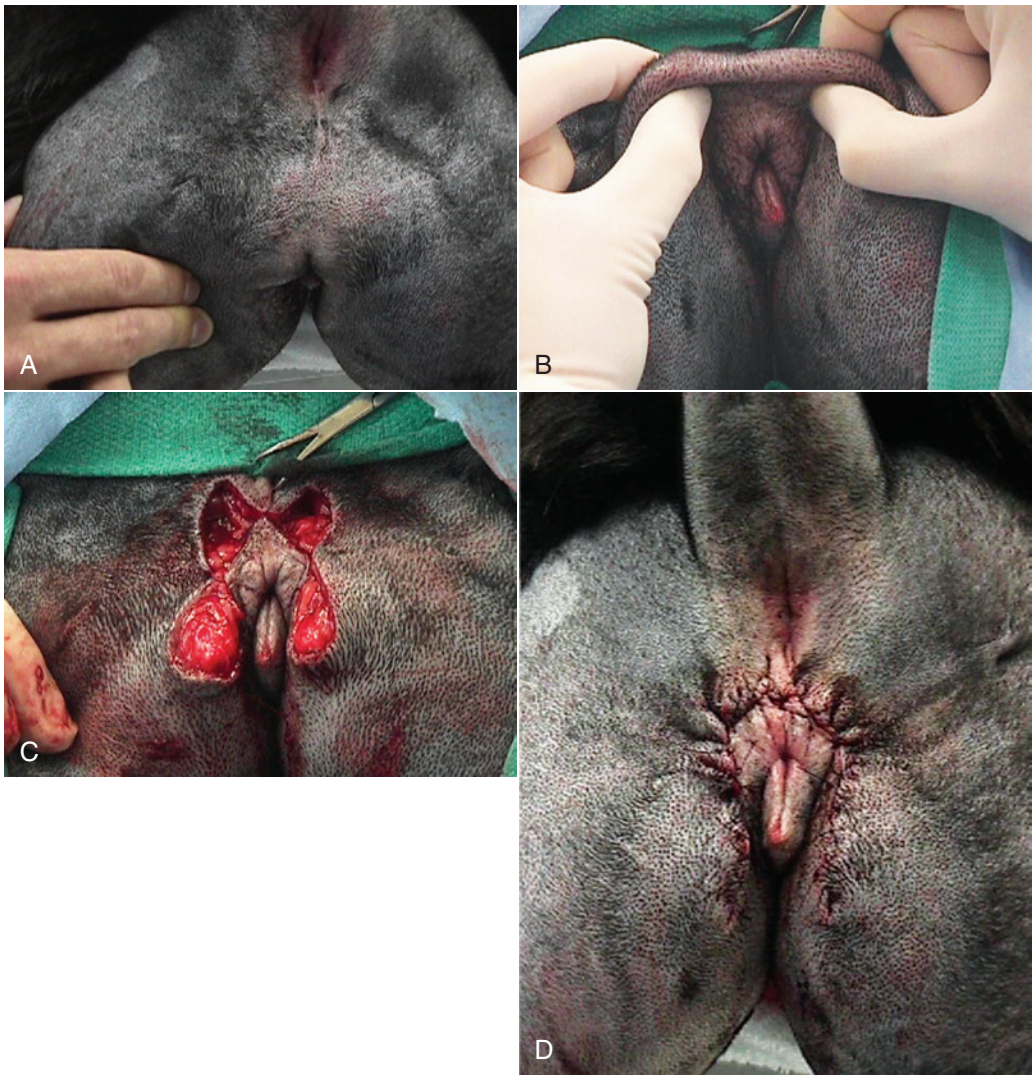


FIG. 15.68 (A) The vulva is completely obscured by the adjacent skin fold. (B) The skin is evaluated to determine the amount to be resected. (C) Skin has been resected and tacked into place to determine if resection was adequate. (D) Appearance of vulva following reconstruction.



FIG. 15.69 Screwtail in an English bulldog.

if necessary for tail fold pyoderma and perianal fistula. *Cosmetic caudectomy* (tail docking) is performed in puppies in order to adhere to breed standards. Many countries and veterinary professional organizations oppose the procedure when performed solely for cosmetic purposes.

Caudectomy in Adults

A caudectomy in dogs and cats older than 1 week requires general or epidural anesthesia. The surgical site should be observed for swelling, draining, inflammation, and pain. Healing after caudectomy is uncomplicated if excess skin tension and self-trauma are prevented. The site should be protected with a bandage or restraining device if necessary. Complications include infection, dehiscence, scarring, fistula recurrence, and anal sphincter or rectal trauma. Incisions that dehisce after partial amputation may heal by secondary intention, which usually leaves a hairless scar. Reamputation may be necessary to relieve irritation and improve cosmesis.

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Partial Caudectomy

Wrap the distal tail with gauze or insert it into an examination glove and secure the covering with tape. Clip a generous area near the amputation site and aseptically prepare it for surgery. Place the patient in a perineal position or lateral recumbency. Position a tourniquet proximal to the transection site. Retract the skin toward the tail head. Make a double V incision in the skin distal to the desired intervertebral transection site. Orient the V to create dorsal and ventral skin flaps that are longer than the desired tail length (Fig. 15.70A). Identify and ligate the medial and lateral caudal arteries and veins slightly cranial to the transection site (see Fig. 15.70B). Incise soft tissue slightly distal to the desired intervertebral space and disarticulate the distal tail with a scalpel blade. If bleeding occurs, place a circumferential ligature around the distal end of the remaining tail or religate the caudal vessels (see Fig. 15.70C). Appose subcutaneous tissue and muscle over the exposed vertebrae with interrupted approximating sutures (e.g., 3-0 polydioxanone, poliglecaprone 25, glycomer 631, or polyglyconate). Position the dorsal skin flap over the caudal vertebrae (see Fig. 15.70D). Trim the ventral skin flap as needed to allow skin apposition without tension. Appose skin edges with approximating sutures (e.g., 3-0 or 4-0 nylon or polypropylene with a reverse cutting needle) (see Fig. 15.70D). Protect the surgical site with a bandage or by placing an Elizabethan collar over the animal's head.

Complete Caudectomy

Anesthetize the patient; clip and aseptically prepare the entire perineum and tail head area. Position the animal in ventral recumbency. Make an elliptic incision around the tail base (Fig. 15.71A). Incise subcutaneous tissues to expose the muscles. Separate the attachments of the levator ani, rectococcygeus, and coccygeus muscles to the caudal vertebrae (see Fig. 15.71B). Ligate the medial and lateral caudal arteries and veins before or after transection. Transect the tail by disarticulation with a scalpel blade at the second or third caudal vertebra. Lavage the site after hemostasis is achieved. Appose the levator ani muscles and subcutaneous tissue with simple interrupted or continuous suture patterns (e.g., 3-0 or 4-0 polydioxanone, poliglecaprone 25, glycomer 631, or polyglyconate). Excise redundant skin if necessary, and appose skin edges with approximating, nonabsorbable sutures (e.g., 3-0 or 4-0 nylon, polybutester, or polypropylene). As an alternative, the tail fold may be preserved; however, skin fold pyoderma may persist.

SURGERY OF THE DIGITS AND FOOTPADS

BIOPSY

Diagnosis of dermatologic conditions of the claw may require biopsy if history, physical examination, cytology, and cultures have not yielded an answer. Submission of an avulsed or sloughed claw is usually not helpful because claw matrix is absent. Punch biopsies are often not diagnostic, and owners are often reluctant to allow distal phalanx (P3) amputation to achieve a definitive diagnosis. The described technique yields appropriate samples of the claw epithelium to allow diagnosis without onychectomy; however, claws may not grow back properly following biopsy. Dewclaws should be biopsied when affected and present to prevent postoperative lameness.

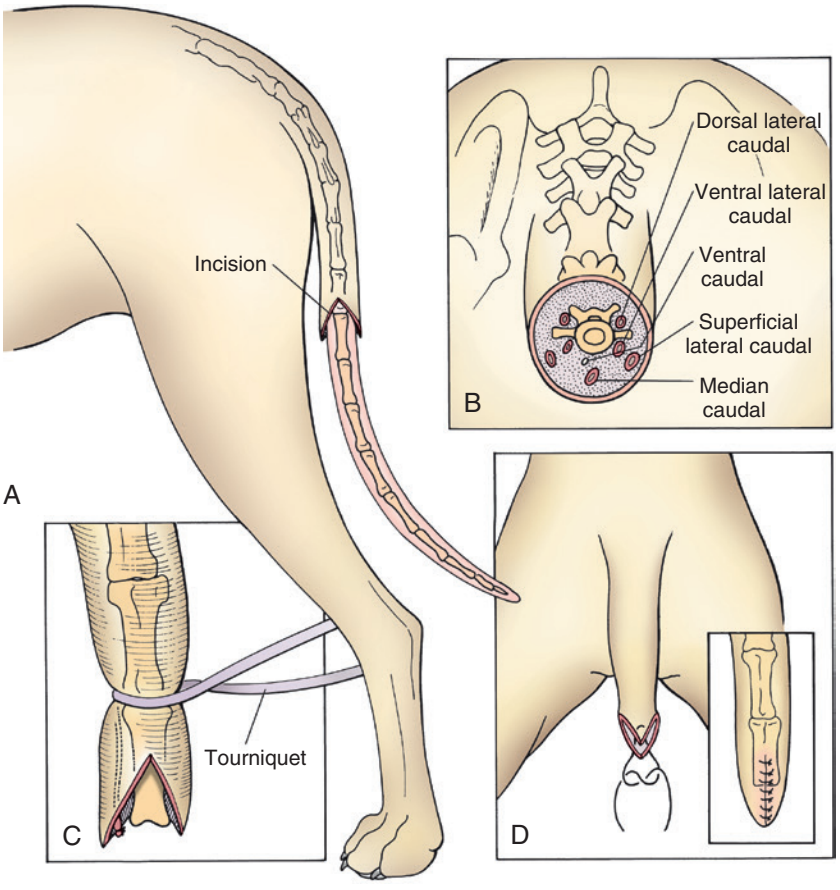


FIG. 15.70 Partial caudectomy in an adult. (A) Retract the tail's skin toward the tail head and make a double V incision in the skin distal to the desired transection site. (B) Ligate the medial and lateral caudal arteries and veins. (C) Transect the soft tissue distal to the desired intervertebral space. Transect the tail through the desired intervertebral space. A proximally placed tourniquet assists hemostasis. (D) Appose soft tissue and skin with approximating sutures.

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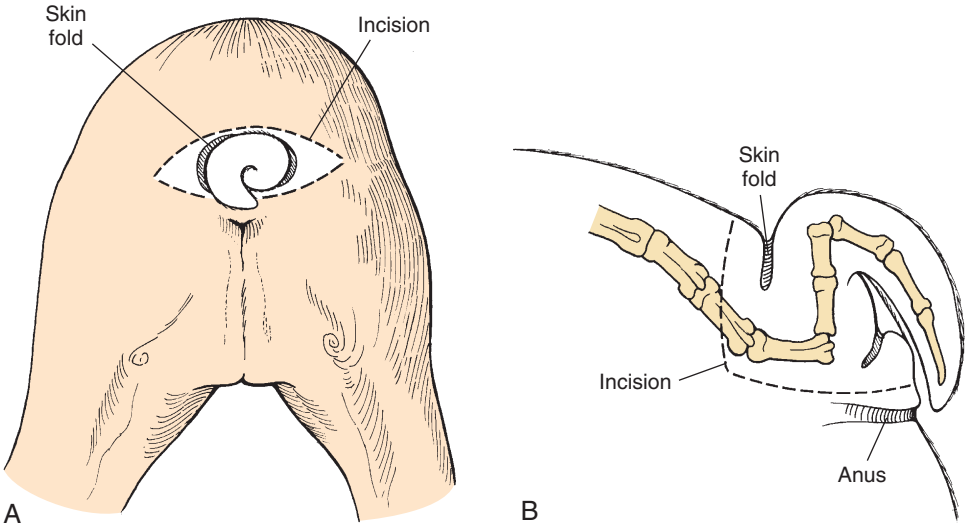


FIG. 15.71 Complete caudectomy with tail fold pyoderma requires (A) an elliptic incision around diseased skin and (B) deep dissection to locate the caudal vertebrae rostral to the vertebral deviation.

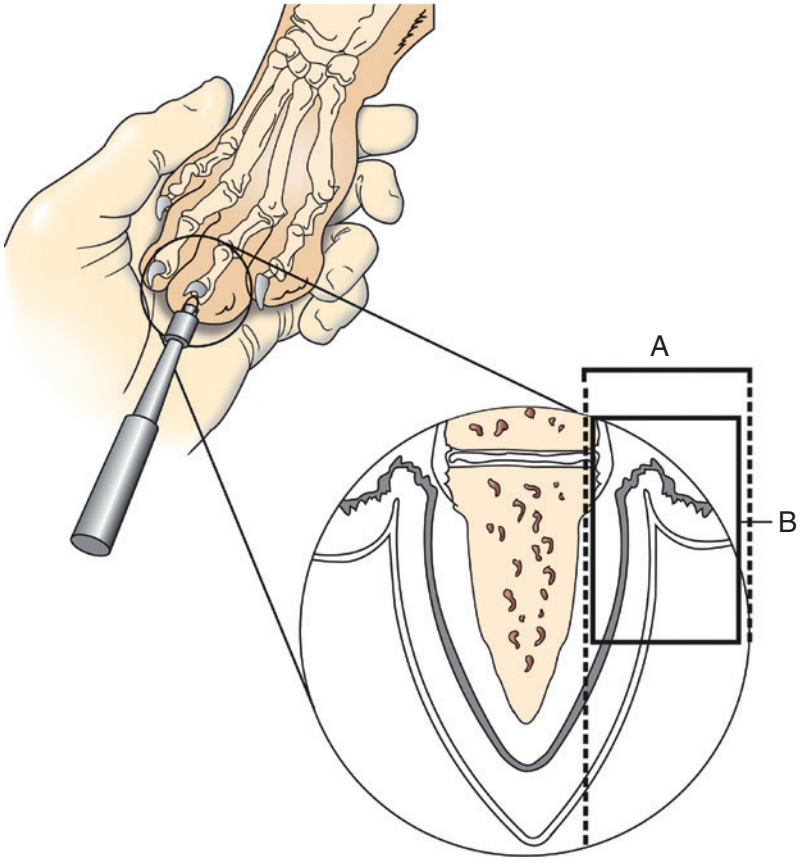


FIG. 15.72 (A) Position an 8-mm-diameter biopsy punch parallel to the long axis of the claw 1 to 2 mm distal to the claw fold. Rotate the punch slowly in one direction deep into the tissue initially through the horn of the claw, then through the bone of the distal phalanx and laterally through normal skin on the lateral aspect of the claw fold. (B) *Inset,* claw segment biopsied.

After the induction of general anesthetic, the paws designated for biopsies are clipped, but not scrubbed to prevent removing important pathologic surface evidence. *Apply a tourniquet to decrease hemorrhage. Position a 6- to 8-mm diameter biopsy punch parallel to the long axis of the claw 1 to 2 mm distal to the claw fold. Rotate the punch slowly in one direction deep into the tissue (Fig. 15.72). Initially, cut through the horn of the claw, then through the bone of the distal phalanx and laterally through normal skin on the lateral aspect of the claw fold. Transect the base of the biopsy with a scalpel blade or iris scissors. Control hemorrhage with pressure.*

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Mark the haired surface for identification and place the specimen in formalin. Place interrupted sutures (3-0 to 4-0 monofilament nonabsorbable) to appose the skin and cover the exposed bone. Give analgesics postoperatively, as the animal will experience pain.

ONYCHECTOMY

Onychectomy (i.e., declawing) is removal of the third digital phalanx (P3) (Fig. 15.73A). Elective onychectomy is a controversial procedure; it is usually performed between 3 and 12 months of age to prevent cats from scratching furniture or people. Usually only the forelimb claws are removed. Although it is recommended that declawed cats be kept indoors because the procedure interferes with a cat's ability to protect itself, those with hind-limb claws can climb to escape some dangers. Alternatives to elective onychectomy are to glue a vinyl cap to each claw every 6 to 8 weeks (SoftPaws) or perform deep digital flexor tenectomy (see p. 260). Vinyl caps blunt the claws to render them less damaging.

Onychectomy or more extensive digit amputation may be required to remove infected nail beds and neoplasms. The most common nail bed tumors are squamous cell carcinomas, melanomas, soft tissue sarcomas, osteosarcomas, and mast cell tumors. Complete excision may require that adjacent phalanges be removed with the affected claw. Onychomycosis, usually caused by *Trichophyton mentagrophytes,* produces dry, cracked, brittle, and deformed nails with inflamed alopecic nail beds. Follicular infections with *Demodex* spp. and staphylococci produce similar lesions.

Onychectomy is performed with the patient under general anesthetic with multimodal analgesia (Table 15.9). Perioperative analgesia (buprenorphine, butorphanol, oxymorphone, meloxicam, or transdermal fentanyl) is recommended for a minimum of 24 to 48 hours after surgery. Additional perioperative analgesia may be provided by blocking regional nerves with 0.5% bupivacaine. The dorsal and palmar branches of the radial, median, and ulnar nerves provide sensory innervation to the feline forepaw. These branches are blocked at four sites using 0.1 to 0.2 mL of 0.5% bupivacaine at each site (not to exceed a total dose of 2 to 3 mg/kg), which provides 4 to 6 hours of analgesia.

Perform the nerve blocks after the induction of anesthesia and preparation of the paw for onychectomy. Fully extend the carpus and palpate the superficial digital flexor tendon along the palmar aspect of the paw. Block the median nerve with 0.15 mL of bupivacaine injected just medial to the superficial digital flexor tendon

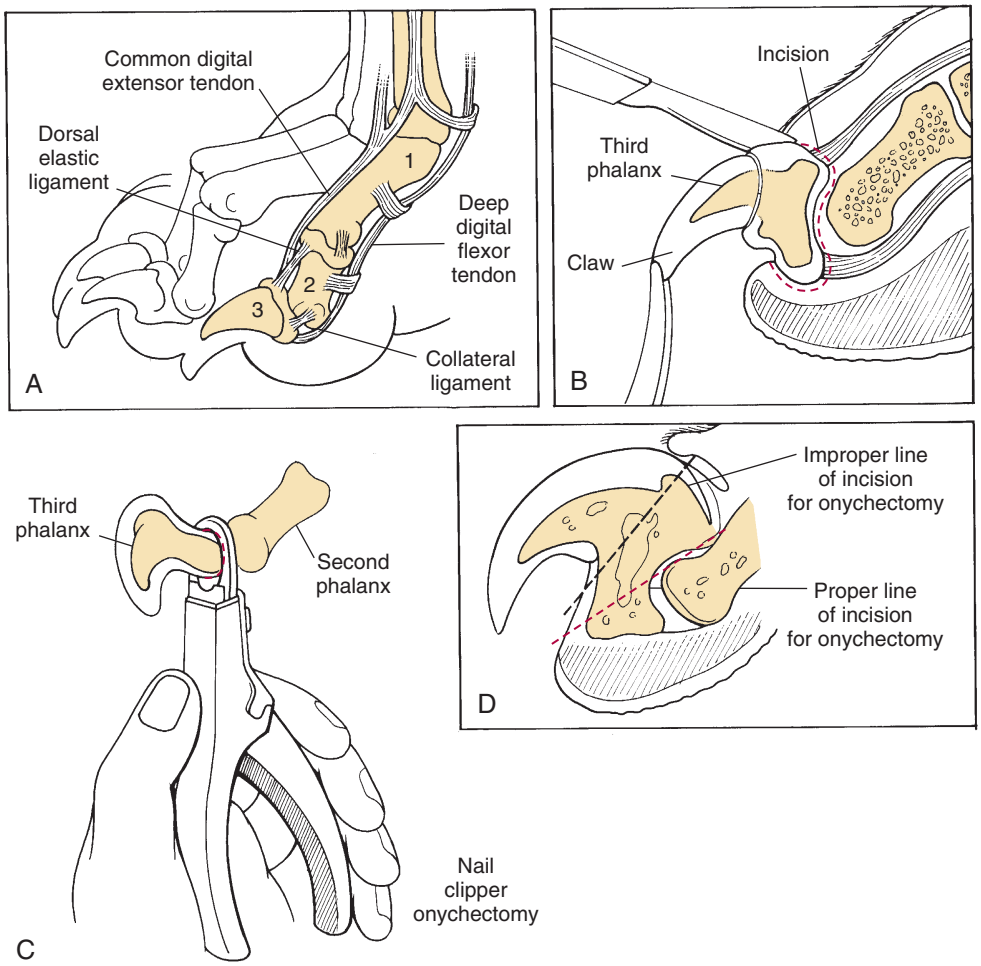


FIG. 15.73 (A) Distal phalangeal anatomy for onychectomy. (B) Dissection onychectomy disarticulates the third phalanx by transecting tendons, ligaments, and other soft tissue attachments. (C) Nail clipper onychectomy should remove the entire ungual crest but often leaves a portion of the ventral flexor process of P3. (D) Proper and improper lines of transection.

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TABLE 15.9 Anesthetic Considerations for Onychectomy in Cats

Preoperative Considerations	
Associated conditions	• Minimal; usually healthy
Blood work	• HCT, TP • In patients >5–7 y, consider electrolytes, liver enzymes, BUN, Cr
Physical exam	• Normal
Premedications	• Hydromorphone[a] (0.05–0.1 mg/kg IV, IM), *or* • Morphine[b] (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM), *or* • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM), *or* • Fentanyl patch placed 6–12 h prior to surgery (see Table 12.1)
Intraoperative Considerations	

Induction	• If premedicated, give:
	  • Ketamine (5.5 mg/kg IV) with diazepam (0.28 mg/kg IV), *or*
	  • Propofol (4–8 mg/kg IV), *or*
	  • Thiopental (8–10 mg/kg IV)
	• If no premeds given, then:
	  • Dexmedetomidine[d] (33 µg/kg IM), *plus*
	  • Butorphanol (0.66 mg/kg IM), *plus*
	  • Ketamine (6.6 mg/kg IM); use ½ of all doses if given IV

Maintenance	• Isoflurane or sevoflurane • Atipamezole (333 µg/kg IM) at end of procedure if reversal of dexmedetomidine is needed
Monitoring	• HR, respiratory rate, SpO_2, temperature, blood pressure
Blocks	• Digital block
Postoperative Considerations	
Analgesia	• Morphine[b] (0.1–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h), *or* • Hydromorphone[a] (0.05–0.1 mg/kg IV, IM q3–4h), *or* • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM q4–8h or 0.01–0.02 OTM q6–12h) • ± Ketamine CRI (2 µg/kg/min IV; if no previous loading dose, then give 0.5 mg/kg IV prior to CRI), *plus* • Meloxicam[e] (0.05–0.1 mg/kg PO, SC once)
Monitoring	• SpO_2, blood pressure, HR, respiratory rate, temperature
Estimated pain score	• Severe

[a]Monitor for hyperthermia in cats.
[b]Give slowly to prevent histamine release.
[c]Buprenorphine is a better analgesic than morphine in cats.
[d]Use only in young animals.
[e]Black box warning added by the Food and Drug Administration in October 2010 identified cases of renal failure and death in cats with repeated uses of meloxicam. Meloxicam is approved for single use only in cats in the United States.
BUN, Blood urea nitrogen; *Cr,* creatinine; *CRI,* constant rate infusion; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *OTM,* oral transmucosal; *PO,* orally; *q,* every; *SC,* subcutaneous; *SpO_2,* oxygen saturation via a pulse oximeter; *TP,* total protein.

at a point approximately one-third the distance between the carpal pad and the first digital pad (Fig. 15.74A). Block the palmar branches of the ulnar nerve at approximately the same level along the lateral aspect of the superficial digital flexor tendon using 0.15 mL bupivacaine (see Fig. 15.74A). Block dorsal digital nerves II through V by inserting the needle from lateral to medial just distal to the carpus. Inject 0.2 mL of bupivacaine as the needle is withdrawn, being careful to block dorsal digital nerve V, which lies lateral to metacarpal V (see Fig. 15.74B). Inject 0.1 mL of bupivacaine to block dorsal digital nerve I. Locate this nerve by inserting the needle from distal to proximal to the point of articulation between metacarpals I and II.

Dissection Onychectomy

In cats, either a scalpel, radiofrequency, or CO_2 laser may be used to remove the third phalanx. Onychectomy performed with CO_2 laser is associated with improved limb function and decreased postoperative pain in the perioperative period. Radiofrequency has been recently shown to be a reasonable alternative to laser for performing onychectomy, as the degree of collateral tissue damage was similar between modalities.

The dissection technique is also the preferred procedure for canine onychectomy. Clipping hair from the paw and around the toes is advised in dogs and long-haired cats. Although most germinal cells are located in the ungual crest, the entire process must be removed to guarantee there will be no claw regrowth.

Aseptically scrub the paw, digits, and nails for surgery. Block the regional nerves to each front paw. Position the animal in lateral recumbency and apply a tourniquet below the elbow to minimize blood loss and improve visualization. In cats, tourniquet application above the elbow may damage the radial, median, or ulnar nerves because feline arm muscles lack protective bulk. *As an alternative, have an assistant compress the brachial artery with a thumb or index finger against the humeral shaft by applying pressure just cranial to the triceps muscle on the medial aspect of the brachium. Drape the feet. Extend the claw by pushing up on the digital pad and by grasping the nail tip with a towel clamp or grasping forceps (e.g., Allis tissue forceps). Circumferentially incise the hairless, cuticle-like skin away from the claw near the articulation between the second and third phalanges (see Fig. 15.73B). Transect the common digital extensor tendon and dorsal ligaments with a No. 11 or No. 12 scalpel blade. Follow the contour of the proximal end*

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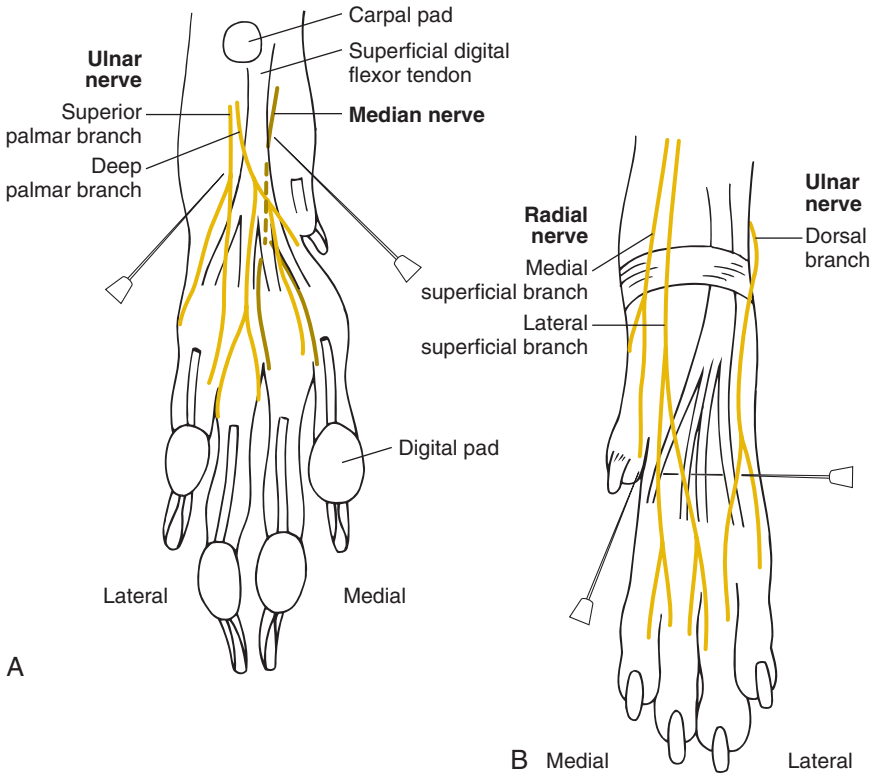


FIG. 15.74 Sites for nerve blocks of sensory innervation to the feline forepaw. (A) Extend the carpus and palpate the superficial digital flexor tendon along the palmar aspect of the paw. Block the median nerve with 0.16 mL of bupivacaine just medial to the superficial digital flexor tendon. Similarly block the palmar branches of the ulnar nerve along the lateral superficial digital flexor tendon. (B) Block dorsal digital nerves II to V by inserting the needle from lateral to medial just distal to the carpus. Inject 0.2 mL of bupivacaine as the needle is withdrawn. Block dorsal digital nerve I at the articulation between metacarpal I and II with 0.1 mL of bupivacaine.

of P3 to transect the deep digital flexor tendon and dissect the phalanx from the digital pad and other soft tissue attachments (i.e., joint capsule, collateral ligaments, and other tendons). Avoid cutting the digital pad. Remove all 10 claws from the front paws for elective onychectomy. Appose skin edges with a bandage, single interrupted cruciate sutures, or cyanoacrylate absorbable tissue adhesive (e.g., Vetbond). Do not place sutures through the digital pads. Apply tissue adhesives over the apposed surface of the skin and not between the cut edges. Apply a bandage from the paw to the distal antebrachium (see p. 203) and remove the tourniquet.

Nail Clipper Onychectomy

Prepare the paws for surgery as described in the section on Dissection Onychectomy. Trim the claws to facilitate positioning the nail trimmer in the interphalangeal space. Position a sharp, guillotine type of nail clipper (Resco nail shears) around the claw (see Fig. 15.73C). Extend the claw by grasping the tip with a towel clamp or grasping forceps (e.g., Allis tissue forceps). Position the blade dorsally at the joint space and transect the extensor tendon or tendons. Rotate the blade ventrally with continuous contact with the skin. Lift the claw dorsally to close the joint space and deviate the flexor process ventrally. After ensuring that the digital pad is proximal to the line of transection and the instrument is in the joint space, close the instrument to amputate the phalanx. Inspect the articular surfaces for complete excision of P3 (see Fig. 15.73D). If a small portion of the palmar aspect of P3 remains, grasp it with a thumb forceps and carefully dissect it from the digital pad with a scalpel blade or sharp-sharp scissors. Some prefer to retain this portion of P3, especially in large cats, to maintain deep digital flexor function. *Remove all 10 claws from the front paws for elective onychectomy. Appose skin edges with a single interrupted suture or absorbable tissue adhesive, if necessary. Apply a bandage from the paw to the distal antebrachium (see p. 203) and remove the tourniquet.*

Postoperative Care and Complications

Mild bleeding should be expected when the bandage is removed after 12 to 24 hours; if bleeding persists, the bandage should be reapplied for 2 to 3 days. Shredded paper, instead of litter, should be used for 2 weeks while the nail beds heal. The cat will usually remove sutures within a week. Convalescence is more rapid in young, growing cats than in older or obese cats. Owner satisfaction with this procedure is high. Complications (i.e., pain, hemorrhage, pad damage, lameness, swelling, infection, claw regrowth, second phalanx protrusion, and palmigrade stance) occur in 50% of patients. Cutting the digital pads prolongs postoperative pain and lameness. Early postoperative pain is more common after blade onychectomy than after the nail clipper procedure. Hemorrhage is most common in older animals and when wounds are not apposed. Late postoperative complications are more common when nail clippers are used. Nonabsorbable tissue adhesives may cause postoperative lameness (frequently non–weight bearing)

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FIG. 15.75 This cat was not confined after onychectomy, and its bandages got wet and were left on longer than desired. All four paws were injured, with the right rear leg requiring amputation.

BOX 15.16 Breeds Recommended for Possible Dewclaw Removal

- Alaskan malamute
- Basset hound
- Belgian malinois
- Belgian sheepdog
- Belgian tervuren
- Bernese mountain dog
- Boxer
- Cardigan Welsh corgi
- Chesapeake Bay retriever
- Dalmatian
- Dandie Dinmont terrier
- Kerry blue terrier
- Komondor
- Lakeland terrier
- Norwegian elkhound
- Papillon
- Puli
- Rottweiler
- Shetland sheepdog
- Siberian husky
- Silky terrier
- Saint Bernard
- Vizsla
- Weimaraner

and infections and may be extruded from the wounds as a foreign body. Improper tourniquet use may cause neurapraxia, tissue necrosis, and lameness. The radial nerve is most often affected, but signs usually resolve in 6 to 8 weeks. Tight bandages can result in ischemic necrosis of the paw (Fig. 15.75). Incomplete removal of the germinal cells in the dorsal aspect of the ungual crest allows claw regrowth. If only a small remnant of the flexor process remains, claw regrowth is not anticipated. Claw regrowth should be suspected if draining tracts develop. The regrown claw is usually deformed. Chronic pain evidenced by behavioral changes (e.g., decreased activity, decreased appetite, or increased aggression) is sometimes observed and may be caused by the windup pain phenomenon associated with unsatisfactory perioperative analgesia (see Chapter 13).

DEEP DIGITAL FLEXOR TENECTOMY

The deep digital flexor tendon inserts on the flexor process of the third phalanx and is needed to flex the phalanx. Claws remain retracted after the deep digital flexor tendons have been severed, which limits the cat's ability to scratch; however, the nails become thick and blunt and must be trimmed regularly. Hemorrhage, infection, and lameness may occur postoperatively. Tenectomy of the superficial digital flexor (which inserts on the proximal aspect of P2) instead of the deep digital flexor results in an abnormal flat-footed stance. Problems may include persistent lameness and ability to scratch, interphalangeal joint immobility, fibrosis, pain, and claw ingrowth into the digital pads. Owners are frequently dissatisfied because of the cat's continued ability to scratch, the unaesthetic appearance of the thickened claws, the long-term lameness, and the necessity for nail clipping, which is difficult to accomplish. Cats may require an onychectomy to relieve clinical signs. For these reasons, this technique is not routinely recommended.

Anesthetize the cat and position it in dorsal or lateral recumbency. Clip and aseptically prepare the paws. Apply a tourniquet below the elbow to minimize hemorrhage. Make a 3- to 5-mm skin incision over the palmar surface of the second phalanx (P2), near the P2-P3 interphalangeal joint and digital pad (Fig. 15.76A). The glistening white tendon of the deep digital flexor lies directly beneath the skin. *Dissect under the tendon with mosquito hemostats or small scissors (see Fig. 15.76B). Excise a 5-mm segment of tendon with a scalpel blade, electrosurgery blade, or laser (see Fig. 15.76C and D). Appose the skin edges with an interrupted suture or absorbable tissue adhesive as for onychectomy. Repeat the procedure on each digit, and trim each claw.*

Use torn paper rather than litter for 2 weeks. Limit activity and discourage jumping for 1 week.

DEWCLAW REMOVAL

The dewclaw is the first digit of the canine rear paws. The first and second phalanges of the digit are inconsistent. Dewclaws are absent in some dogs and double in others. Great Pyrenees and Briards must have double rear dewclaws to meet breed standards. In other breeds, loosely attached dewclaws are removed to prevent inadvertent or self-trauma during hunting or grooming (Box 15.16). Often only the rear dewclaws are removed. Dewclaws can be removed at 3 to 5 days of age with local analgesia. Hemorrhage is more excessive after 5 days of age, and anesthetics are necessary. Complications include hemorrhage, pain, infection, and dehiscence. Premature suture removal may cause scarring. Bandages applied too tightly may cause swelling or ischemic necrosis.

Dewclaw Removal in Puppies

Aseptically prepare the medial aspect of the paw. Have an assistant cup the puppy in his or her hands and immobilize the paw. Facilitate restraint and analgesia by giving a sedative, a local anesthetic, or both (see p. 144). Abduct the digit and transect the web of skin, attaching the dewclaw to the paw with Mayo scissors, electrosurgery, or laser (Fig. 15.77A). Disarticulate the metatarsal/metacarpal-phalangeal joint or transect the bone near the metatarsal or metacarpal bone with a scalpel blade or Mayo scissors, electrosurgery, or laser. Control hemorrhage with pressure or electrocautery. Appose skin margins with a single approximating suture or absorbable tissue adhesive or allow healing by secondary intention.

Dewclaw Removal in Adults

If dewclaw removal is not performed within the first week of life, it should be delayed until after 3 months of age and performed using a general anesthetic. It is convenient to remove dewclaws at the time of neutering.

Position the patient in lateral recumbency. Clip and aseptically prepare the paws for surgery. Make an elliptic incision around the base of the digit where it articulates with the metatarsal or metacarpal bone (see Fig. 15.77B). Abduct the digit and dissect subcutaneous tissue to the metacarpophalangeal or metatarsophalangeal joint.

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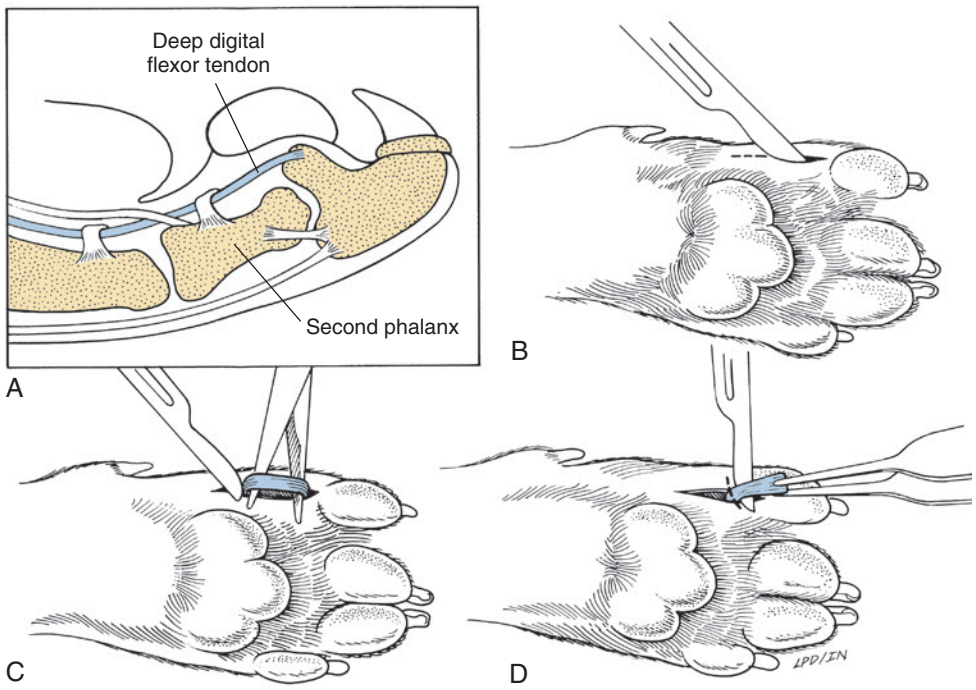


FIG. 15.76 Deep digital tenectomy. (A) Make a 3- to 5-mm incision over the palmar surface of the second phalanx. (B) and (C) Elevate and excise a 5-mm segment of deep digital flexor tendon. (D) shows the relationship of the deep digital flexor tendon to P1, P2, and P3.

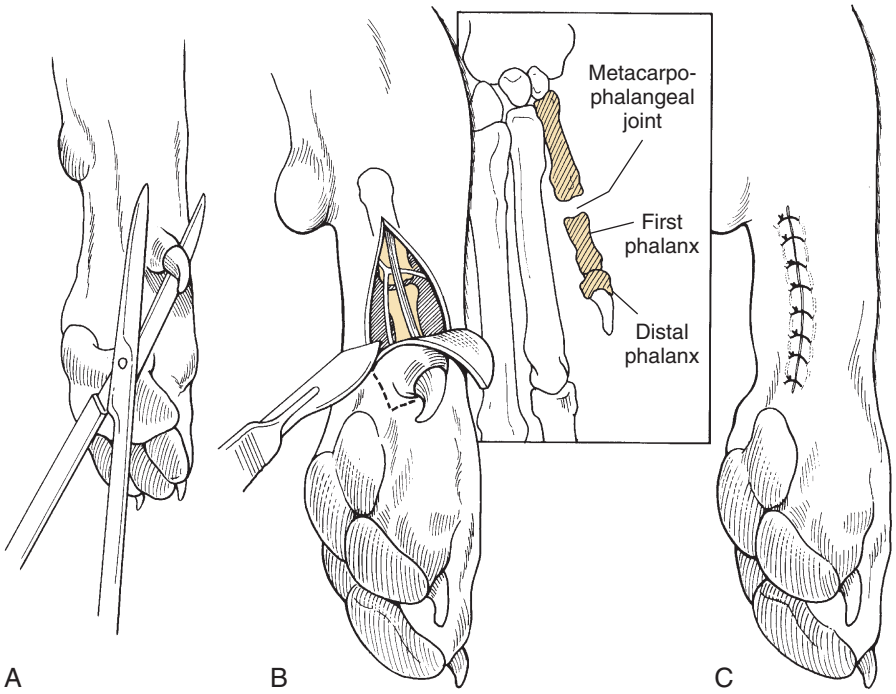


FIG. 15.77 Dewclaw removal. (A) In puppies, abduct and transect the dewclaw with scissors, disarticulating at the metatarsal (carpal)-phalangeal joint. (B) In adults, make an elliptic incision around the base of the dewclaw, dissect subcutaneous tissue, and ligate the dorsal common and axial palmar arteries. Then disarticulate the joint or transect P1 with bone cutters *(inset)*. (C) Appose subcutaneous tissue and skin with approximating sutures.

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If the first and second phalanges are firmly attached, free them with a No. 11 blade. Ligate the dorsal common and axial palmar digital arteries. Disarticulate the metacarpophalangeal or metatarsophalangeal joint with a scalpel blade. The results are less cosmetic when bone cutters are used to transect the first phalanx near the metacarpophalangeal or metatarsophalangeal joint. *Appose subcutaneous tissue with 3-0 or 4-0 simple continuous or interrupted approximating sutures using absorbable suture material (i.e., polydioxanone, polyglyconate, poliglecaprone 25, glycomer 631 or polyglactin 910). Appose the skin with interrupted approximating sutures (i.e., 3-0 or 4-0 nylon or polypropylene) (see Fig. 15.77C). Apply a soft, padded bandage to protect the surgical site for 3 to 5 days. Remove sutures at 7 to 10 days.*

DIGIT AMPUTATION

Digit amputation is performed because of neoplasia, chronic bacterial or fungal infections, osteomyelitis, or severe trauma (Fig. 15.78). Affected digits are swollen and painful with thickened, dystrophic, or absent claws. Squamous cell carcinoma is the most common tumor identified in canine and feline digits; however, numerous neoplastic processes can occur in this location. Occasionally a digit is amputated to facilitate salvage of a weight-bearing pad or portion of the paw.

The level of amputation is determined by the site of the lesion and the disease process. General anesthesia is required. Complications include hemorrhage, infection, dehiscence, and recurrence. Tight bandages may cause swelling or ischemic necrosis. The primary weight-bearing digits are the third and fourth digits. In dogs undergoing removal of one or both central weight-bearing digits, postoperative transient lameness is expected.

Digital tumors occur in older dogs (mean age, 10 years) and more rarely in cats (mean age 12.7 years) and are often initially misdiagnosed as infections. They occur most often in male large- to medium-breed dogs (10 to >30 kg). Bone invasion is common. Clinical signs include lameness, digit swelling and ulceration, and a fixed protruding, deviated, or lost nail. Tumors must be differentiated from paronychia. Squamous cell carcinomas, malignant melanomas, soft tissue sarcomas, osteosarcomas, and mast cell tumors are common digital tumors. Squamous cell carcinomas, mast cell tumors, and melanomas arising in the subungual epithelium are aggressive and sometimes metastatic. Black dogs are predisposed to subungual squamous cell carcinomas. The 1-year survival rate after digital amputation varies from 45% to 100%, depending on the tumor type; digital melanomas have a median survival of 1 year.

Aside from squamous cell carcinoma and fibrosarcoma, digital tumors in cats can also be metastatic, most commonly from pulmonary adenocarcinoma. These tumors typically involve weight-bearing digits and can involve more than one toe. Cats with digital tumors should be carefully evaluated for primary disease. Prognosis for digital tumors in cats is guarded. Median survival in cats with digital squamous cell carcinoma is 73 days, whereas cats with metastatic lesions have similarly poor survival.

Clip and aseptically prepare the paw for surgery. Position the dog or cat in ventral or lateral recumbency with the leg suspended. Place a tourniquet and drape the area. Release the aseptically prepared paw into the sterile field. Begin a dorsal skin incision at the distal end of the appropriate metacarpal (metatarsal) or the proximal end of the first phalanx. Make a transverse encircling incision at the appropriate interphalangeal joint (inverse Y incision) (Fig. 15.79A and B). Preserve the digital pad if only the third phalanx is removed. Transect the flexor and extensor tendons, ligaments, and joint capsule. Ligate the digital arteries and veins with 3-0 or 4-0 absorbable suture. Disarticulate with a scalpel blade or transect the phalanx with bone cutters. Include the sesamoid bones with the excision. Suture the extensor tendon to the dorsal surface of the pad when it is preserved. Appose subcutaneous tissues over the end of the bone with interrupted absorbable sutures (e.g., 3-0 or 4-0 polydioxanone, poliglecaprone 25, glycomer 631, or polyglyconate). Appose skin with approximating sutures (e.g., 3-0 or 4-0 nylon or polypropylene) (see Fig. 15.79C). Apply a padded bandage and, if necessary, an Elizabethan collar. Keep the bandage clean and dry. Change the bandage in 2 to 3 days or as needed to evaluate the wound. Remove the bandage and sutures after 7 to 10 days.



FIG. 15.78 (A) A melanoma caused this digit to enlarge, and the nail was lost. (B) The digit was amputated, and the adjacent toes were fused.

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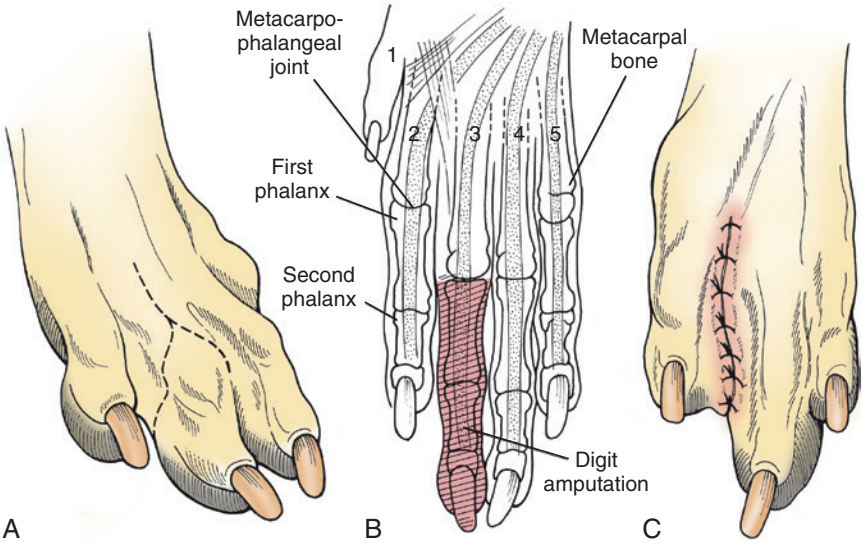


FIG. 15.79 Digit amputation. (A) Begin the skin incision dorsally and extend it laterally on each side of the involved digit. (B) Ligate the digital vessels and transect the tendons, ligaments, and joint capsule to disarticulate the digit between P3–P2, P2–P1, or P1–metacarpal (tarsal). (C) Appose subcutaneous tissue over the end of the bone and approximate skin edges.

FOOTPAD INJURIES

The footpad is the toughest area of the canine skin and is designed to absorb shock, standing, and abrasive forces. The stratum corneum is usually pigmented, thick, and keratinized, with a rough surface of conical papillae. Footpad injuries include laceration, degloving, abrasion, avulsion, burns, and tumors. Injuries may not heal well as a result of the weight-bearing forces and pad loss. Weight-bearing areas without pads may ulcerate.

Superficial Pad Loss

Wounds with superficial pad loss are allowed to heal by secondary intention and treated with nonadherent, absorbent, or semiabsorbent bandages and a spoon splint. A functional pad is maintained if epithelial tissue remains at the periphery and the wound is able to heal by contraction and epithelialization. Splinting the paw promotes healing because wound contractile forces are antagonized by weight bearing, which pushes the wound edges apart. Aloe vera extract gel has a positive effect on early stages of paw pad healing. Acemannan-containing hydrogel wound dressing also stimulates wound healing. Bandaging and splinting the paw should be continued until reepithelialization and some keratinization occur. Exercise should be restricted while the limb is bandaged. After the bandage has been removed, exercise may be gradually allowed on nonabrasive surfaces.

Lacerations

Improperly managed simple lacerations can become chronic nonhealing wounds because forces applied on the pads during standing or walking flatten and spread the pad, separating the lacerated edges. Proper management of lacerated pads includes lavage, suturing, and bandaging. Acute lacerations should be thoroughly lavaged and minimally debrided. Old, severely contaminated or infected lacerations need hydrophilic bandages for several days.

Debride the edges of chronic lacerations to remove necrotic tissue and provide a bleeding edge for apposition. Appose deep layers of the pad with buried simple interrupted absorbable sutures (e.g., 3-0 and 4-0 polydioxanone, poliglecaprone 25, glycomer 631, polyglyconate). Appose epithelial edges with interrupted approximating sutures (simple, vertical mattress, far-near-near-far) (e.g., 2-0 or 3-0 polypropylene or nylon), taking bites several millimeters from the cut edge. After closure, protect the pad with a nonadherent, thick, absorbent bandage with a foam sponge, with or without a splint (see p. 203). Change the bandage every 1 to 3 days, depending on the amount of drainage. Remove the sutures after 10 to 14 days and reapply the splint and bandage for 3 to 4 days. Remove the splint but rebandage the paw for an additional 3 to 6 days to allow the wound to strengthen. As an alternative, protect the pad with a commercial dog boot after suture removal. Restrict exercise while the limb is bandaged. Gradually institute exercise on a nonabrasive surface after bandage removal.

Paw Salvage

Degloving or crushing injuries to the paw may result in a nonfunctional paw. Amputation or multiple surgical procedures may be required to maintain paw function when injury is severe. Free grafts, axial pattern flaps, or thoracic wall pouch flaps, pedicled muscle flaps, and microvascular free tissue transfer have been used to reconstruct distal limb injury. Partial amputation may be performed with carpal pad transfer for weight-bearing or in preparation for a prosthetic device (Fig. 15.80). Indoor cats and small dogs that walk primarily on carpets may function well on grafted skin that does not have pads; however, most dogs require salvage or replacement of the metacarpal-metatarsal pad. Replacement of metatarsal, metacarpal, or weight-bearing digital pads may be achieved by transposing adjacent pads, segmental digital pad grafts, or microneurovascular free digital or carpal pad transfer (Fig. 15.81). Tissue should not be transposed over a pad injury caused by tendon malfunction, bone malalignment, or nerve damage until the cause is corrected. A digital pad is transposed to replace a portion of the metatarsal or metacarpal pad. The phalanges of digit two or five are removed through a palmar incision. The digital pad is maintained on a pedicle of

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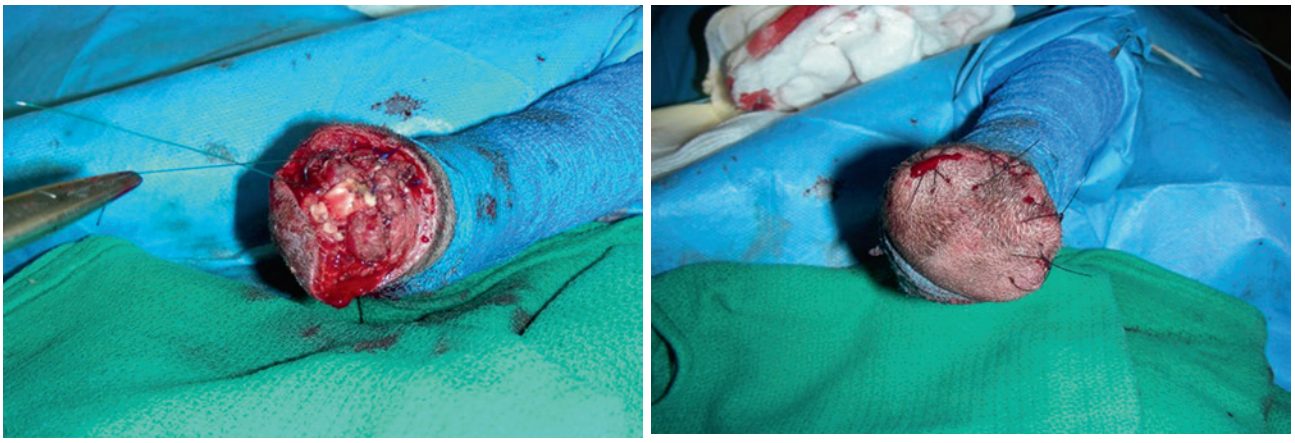


FIG. 15.80 Partial amputation of the distal rear limb in a dog due to severe frostbite of the digits.

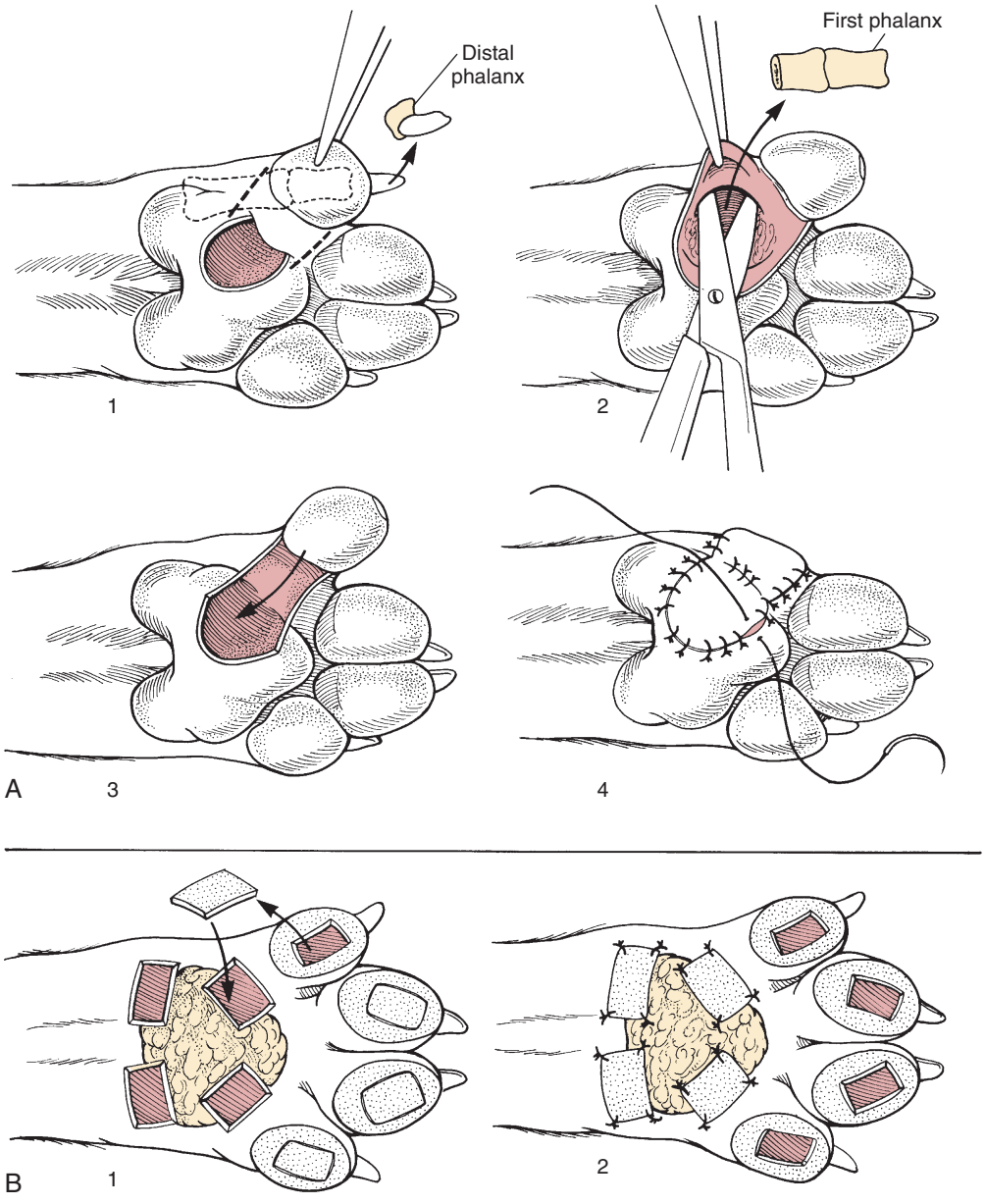


FIG. 15.81 Replacement or salvage of weight-bearing pads. (A) Partly or completely replace a pad by removing P3 and P2 from digit 2 or 5 and transposing the pad and skin to the injured area (phalangeal fillet technique). (B) The metacarpal or metatarsal pad may be salvaged by grafting from digital pads. Place the free segmental graft on a healthy granulation bed and across the skin pad junction. Donor sites are apposed with approximating sutures if tissue is pliable or allowed to heal by secondary intention under a protective bandage.

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skin and is transposed to the metatarsal-metacarpal pad. As an alternative, severe trauma to all digits may necessitate digital amputation and transposition of the metatarsal or metacarpal pad over the bone ends to provide a weight-bearing surface. Free segmental digital pad grafts (6 × 8 mm) sutured into recessed recipient beds in granulation tissue allow pad reepithelialization with a tough, keratinized, and effective weight-bearing surface. Microneurovascular pad transfer requires specialized instrumentation and training. The paw should be bandaged after surgery as described previously for lacerations.

Fusion Podoplasty

Fusion podoplasty is a salvage procedure used to treat chronic interdigital pyoderma that is refractory to medical therapy. The procedure can also be used to palliate animals with chronic flexor tendon injury or to reconstruct the paw after digit amputations. Fusion podoplasty is not recommended on all four paws during a single surgery because of the prolonged operative time and postoperative discomfort.

Clip and aseptically prepare the paw. Position the anesthetized patient in dorsal or lateral recumbency. Consider placing a tourniquet around the distal limb to minimize hemorrhage, but be mindful of the length of the surgical procedure. Use a sterile marking pen to outline interdigital skin to be removed. Excise the interdigital web and skin between the digital and metacarpal-metatarsal pads. Preserve 2 to 3 mm of skin adjacent to the nail. Prevent damage to the digital vessels and nerves during dissection. Control hemorrhage with electrocoagulation or digital pressure; alternatively the dissection can be performed with radiofrequency or laser. Thoroughly lavage the wound. Suture the digital pads in apposition with simple interrupted sutures (e.g., 3-0 polypropylene or nylon). Approximate the digital and metacarpal-metatarsal pads with simple interrupted sutures; direct apposition of epithelial edges may not be possible. Approximate adjacent skin edges dorsally with simple interrupted sutures (e.g., 3-0 polypropylene or nylon). Repeat the procedure on the opposite paw if indicated. Remove the tourniquet.

Following amputation of the third or fourth digit, or both (see p. 262), the weight-bearing portion of the foot can be reconstructed with the second and fourth or fifth digits. The digits and tumor are removed en bloc with the digital pads while the metacarpal or metatarsal pads are preserved.

Close the defect in three layers. First appose the interosseous muscles of the adjacent digits with 3-0 or 4-0 monofilament absorbable interrupted sutures; next appose the subcutaneous tissue on both the palmar (plantar) and dorsal surfaces with absorbable sutures. Finally, appose the dorsal skin remaining on the second digit with the dorsal skin of the fourth or fifth digit and then the plantar or palmar skin between the digits with 3-0 or 4-0 monofilament nonabsorbable appositional sutures.

Apply a nonadherent, absorbent bandage and spoon splint or clamshell type of splint extending from the paw to the elbow or hock. For patients with interdigital pyoderma, change the bandage and splint daily until drainage diminishes, usually within 10 to 14 days. Remove sutures between 10 and 21 days after surgery, but continue bandaging until granulation is complete.

When healing nears completion, a lighter bandage or a paw boot allows transition between a heavy-splinted bandage and full weight bearing. Lameness is expected after digit amputation with fusion podoplasty for 4 or more weeks. Infection may persist in animals with interdigital pyoderma.

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16



Surgery of the Eye

Surgery of the Eyelid

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Meibomian glands line the eyelid margins and produce the oil layer of the tear film that reduces tear evaporation. **Trichiasis** is the condition in which hairs arising from normal haired skin contact the cornea. **Distichia** are hairs growing out of the meibomian gland openings. **Ectopic cilia** are hairs that grow from palpebral conjunctiva to contact the cornea. **Entropion** is eyelid margin inversion, and **ectropion** is eyelid margin eversion. **Lagophthalmos** is an incomplete blink. **Exotropia** refers to eyes that are turned outward (laterally).

PREOPERATIVE CONCERNS

Eyelid function is essential to corneal health, comfort, and vision because the eyelids distribute tears over the cornea surface and protect the eye. Eyelid surgeries should be planned to preserve eyelid function for the health of the cornea. Eyelid margins that are improperly aligned in surgery or become misaligned postoperatively can result in corneal disease. If eyelid function is lost from trauma or neurologic disease, eyelid surgery may be urgently needed to prevent corneal ulcers and disease. Ideally, skin infections should be treated before surgery, and drugs that may interfere with healing should be given at a reduced dose or stopped entirely at the time of surgery.

Correct instrumentation and suture should be acquired before any planned surgery, and the prospective surgeon should have adequate knowledge of the condition, anatomy, procedure, and other requirements before attempting surgery. Complex or unfamiliar procedures should be referred to a veterinary ophthalmologist for the best outcome, particularly those requiring microsurgical equipment and skills to line skin flaps with conjunctiva.

PREPARATION OF THE SURGICAL SITE

Liberally apply sterile eye ointment or antibiotic ophthalmic ointment to both eyes for lubrication. Clip an approximately 2-inch wide (5 cm) area around the eyelids with a No. 40 clipper blade. Gently brush away the clipped hair; avoid using a vacuum cleaner around the eye. Alternatively, tape (as on a lint roller) may be used to remove the clipped hairs from skin. *Prep the skin with 5% povidone-iodine solution (not scrub) on quarter-folded gauze squares or swab sticks. Irrigate the conjunctival sac with eye wash or artificial tear drops to remove excess hair and relubricate. Avoid using chlorhexidine, any surgical scrubs, soaps, or alcohol because these will damage the cornea. After moving into the operating room, reprep the eye with 5% povidone-iodine (e.g., Betadine Swabstick) and drape the eye with sterile quarter drapes and a top surgical drape.*

ANESTHETIC CONSIDERATIONS

General anesthesia is usually indicated when an eyelid surgery requires eyelid margin incisions, when placing sutures, or when using sharp objects such as scalpel blades or suture needles near the cornea. More precise suture placement can be performed when the small animal patient is still, rather than mobile. Although topical anesthetics and minimal sedation are commonly used for some ophthalmic procedures in human patients, the likelihood of unnecessary trauma to the eye is greater in veterinary patients when only these are used.

Preoperative sedatives such as a benzodiazepine with an opioid can be helpful in achieving quiet, struggle-free induction (Table 16.1). If possible, induce the animal in an area where monitors are immediately available after intubation. With ophthalmic patients, in addition to monitoring pulse oximetry, heart rate, respiratory rate, and electrocardiogram (ECG), it is also important to monitor blood pressure and end-tidal carbon dioxide ($EtCO_2$). Common monitoring techniques, such as evaluating palpebral reflex, ocular movements, or jaw tone, are more difficult because the head is draped and is often turned away from the person administering anesthesia. A pulse oximeter with an ear tip sensor that can be secured to the vulva, prepuce, or toes is helpful because repositioning a sensor on the tongue may be difficult once surgery has begun.

Eye and head movement during surgery can lead to iatrogenic eye injuries; therefore depth of anesthesia must be carefully monitored to reduce movement while preventing hypotension and hypoventilation. In addition, positive pressure ventilation may be needed throughout the procedure.

Monitoring heart rate and ECG is important during ophthalmic surgery because of the potential for initiation of the oculocardiac reflex.[1] This reflex consists of a trigeminal (V) afferent and a vagal (X) efferent pathway. Traction on extraocular muscles or pressure applied to the globe can cause asystole or a variety of cardiac arrhythmias, including severe bradycardia, ventricular fibrillation, premature ventricular contractions, and ventricular tachycardia. If this reflex occurs, manipulation of the eye must stop and pharmacologic treatment initiated. For asystole or bradycardia, atropine (0.02–0.04 mg/kg IV) or glycopyrrolate (0.01–0.02 mg/kg IV) should be given. Because atropine has a faster onset and an increased cardiac effect, it is the drug of choice in cases of severe bradycardia or sinus arrest

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ABSTRACT

Ophthalmic surgery requires precision and a detailed knowledge of the anatomy of the eye and adjacent structures. Care should be taken to ensure that appropriate surgical instrumentation and suture material are available before any eye surgery is undertaken.

KEYWORDS

eyelid
third eyelid
cherry eye
conjunctiva
facial folds
nasal folds
corneal ulcers
corneal ulcerations
conjunctivitis
entropion
ectropion
tarsorrhaphy
third eyelid flap
third eyelid prolapse
conjunctival grafts
conjunctival flaps
facial folds
enucleation

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TABLE 16.1 Anesthetic Considerations in the Ophthalmic Patient

Preoperative Considerations

Associated conditions	• May be geriatric with comorbidities
Bloodwork	• Minimum PCV, TP, Cr, ALT but ideally CBC, chemistry panel • If patient >5 years old, CBC, chemistry panel ± UA • Consider cardiac workup if heart murmur is present
Physical examination	• May be painful at presentation depending on the cause • If trauma, stabilize and systemically evaluate

Premedications	• Anticholinergics (caution with cardiac disease, geriatric cats):
	  • Atropine (0.02–0.04 mg/kg IM, SC) *or*
	  • Glycopyrrolate (0.01 mg/kg IM, SC)
	• Anxiolytics:
	  • Diazepam (0.1–0.2 mg/kg IV), *or*
	  • Midazolam (0.1–0.2 mg/kg IM, IV)
	• Analgesics:
	  • Hydromorphone[a] (0.1–0.2 mg/kg IM in dogs; 0.05–0.1 mg/kg IM in cats), *or*
	  • Morphine (0.1–0.5 mg/kg IM, SC in dogs; 0.1 mg/kg IM, SC in cats), *or*
	  • Butorphanol (0.1–0.4 mg/kg IM, SC), *or*
	  • Oxymorphone (0.02–0.1 mg/kg IM) *or*
	  • Buprenorphine[b] (0.01–0.02 mg/kg IM in dogs, 0.005 mg/kg SC in cats or Simbadol 0.24 mg/kg SC qd for up to 3 d in cats)

Intraoperative Considerations

Induction	• Propofol (2–6 mg/kg IV), *or* • Alfaxalone (2–3 mg/kg IV in dogs; 2–5 mg/kg IV in cats) • Use caution with a face mask if the animal has a fragile eye
Maintenance	• Isoflurane or sevoflurane, *plus* • If needed for analgesia: fentanyl (loading dose 10–20 µg/kg plus infusion 0.6–10 µg/kg/min) IV CRI • If severe bradycardia or other dysrhythmias, give atropine (0.02–0.04 mg/kg IV) *or* glycopyrrolate (0.005–0.01 mg/kg IV)
Fluid needs	Crystalloids, 5–10 mL/kg per h
Monitoring	• Blood pressure • HR • Respiratory rate • ECG • $EtCO_2$ • SpO_2 • Temperature • Neuromuscular transmission via a peripheral nerve stimulator (also known as a *train-of-four monitor*) if an NMB agent was used Note: most likely there will be limited or no access to enable assessment of palpebral reflex or jaw tone
Blocks	• Orbital block after closing orbital septum: 0.5% bupivacaine (0.5–2 mg/kg in dogs; 0.2–0.5 mg/kg in cats) (max 2 mg/kg) (see p 152) • Topical analgesia such as proparacaine, tetracaine

Postoperative Considerations

Analgesia	• Oxymorphone (0.02–0.04 mg/kg IV, IM), *or* • Methadone (0.1–0.5 mg/kg IM, SQ q3–4h) *or* • Morphine (0.1–0.5 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs[d]; 0.05–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h in cats), *or* • Hydromorphone[a] (0.1–0.5 mg/kg IV or 0.1–1 mg/kg IM, SC q2-4h in dogs; 0.05–0.1 mg/kg IV or 0.1–0.2 mg/kg IM, SC q2-4h in cats), *or* • Buprenorphine[b] (0.01–0.02 mg/kg IV, IM, SC q4–6h), *plus* • Carprofen (2.2 mg/kg PO, IM q12h in dogs), *or* • Robenacoxib (2 mg/kg PO, SC q24h for 3 doses [extralabel] in dogs; 1–2 mg/kg SC q24h for 3 doses in cats), *or* • Meloxicam[c] (0.1–0.2 mg/kg once SC or PO, then 0.1 mg/kg PO q24h in dogs; 0.3 mg/kg SC once in cats)
Monitoring	• SpO_2 • Blood pressure • HR • Respiratory rate and effort, particularly if NMBs were used • Temperature
Bloodwork	• Usually not needed unless the patient has diabetes mellitus
Estimated pain score	• Can be mild to severe depending on cause and/or procedure

ALT, Alanine aminotransferase; *BUN,* blood urea nitrogen; CBC, complete blood count; *Cr,* creatinine; *ECG,* electrocardiogram; *EBL,* estimated blood loss; *$EtCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IOP,* intraocular pressure; *IV,* intravenous; *NMB,* neuromuscular blocker; *OTM,* oral transmucosal; *PCV,* packed cell volume; *PO,* orally; *PRN,* as needed; *SC,* subcutaneous; *SpO_2,* oxygen saturation via a pulse oximeter; *TP,* total protein; *UA,* urinalysis.

[a]Monitor for hyperthermia in cats.

[b]Buprenorphine is a better analgesic than morphine in cats.

[c]Black box warning added by the Food and Drug Administration in October 2010 identified cases of renal failure and death in cats with repeated uses of meloxicam. Meloxicam is approved for single use only in cats in the United States.

[d]Give slowly to prevent histamine release.

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(see Chapter 12). Premedication with glycopyrrolate or atropine (see Table 16.1) can reduce or prevent this reflex but they should be used with caution in animals with cardiac disease or geriatric feline patients who may not tolerate tachycardia. In these cases, having glycopyrrolate or atropine available in the operating room with the appropriate dose already calculated, so that it may be given rapidly IV if necessary, is recommended.

For eyelid surgeries, induction, intubation, and anesthetic maintenance can be done as for other soft tissue surgeries. Because tear production decreases substantially during general anesthesia, the eyes should be lubricated at least every 2 hours. Reduced tear production may persist for 24 hours postoperatively; therefore continued application of a topical lubricant such as ophthalmic ointment for 1 to 2 days postoperatively should be considered.[2] In addition, ensure that there is no pressure on the opposite eye if the patient is in lateral recumbency.

Whenever possible, extubation and anesthetic recovery need to be done in a quiet environment to avoid head banging that can cause eyelid bleeding and swelling. Because of their sedating and analgesic effects, opioids are helpful in the postoperative period. Low-dose acepromazine (0.005–0.01 mg/kg IV) will enhance the sedative effects of the opioid and may be useful if the patient is dysphoric during recovery (see Chapter 12 and Table 12.1).

ANTIBIOTICS

Perioperative antibiotics are indicated for eyelid surgery because the normal flora of ocular surfaces and eyelids include potential pathogens. Systemic antibiotics should be administered before surgery so that therapeutic blood levels will be achieved by the time of surgery (see Chapter 9). Broad-spectrum topical antibiotics are most commonly chosen for perioperative prophylaxis (Box 16.1), and systemic antibiotics (e.g., IV cefazolin) are typically given preoperatively. If eyelid surgery is extensive, skin infection is present, or tissue is already damaged, systemic antibiotics may be indicated postoperatively for 10 to 14 days.

The most commonly used prophylactic topical antibiotic for dogs is neomycin/polymyxin/bacitracin and for cats is erythromycin or oxytetracycline (see Box 16.1). Cats very rarely have an anaphylactic reaction on their first exposure to neomycin or polymyxin; ideally those medications should therefore not be given for the first time to an anesthetized cat.

BOX 16.1 Examples of Prophylactic and Therapeutic Antibiotics for Eye Surgery

Oral

Amoxicillin, 22 mg/kg PO BID
Cephalexin, 30 mg/kg PO BID to TID
Cefpodoxime, 5–10 mg/kg PO SID

Topical Broad-Spectrum Antibiotics

Neomycin-polymyxin-bacitracin, 0.25 inch (0.6 cm) QID
Neomycin-polymyxin-gramicidin, 1 drop QID
Erythromycin, 0.25 inch (0.6 cm) QID
Oxytetracycline, 0.25 inch (0.6 cm) QID

Topical Gram-Positive Spectrum Antibiotics for Infected Corneal Ulcers

- Cefazolin 5% eye drops made from a 1 g vial of IV injectable cefazolin to be used 1 drop q1–2h initially. Note: the solution must be **refrigerated**; with the injectable it is made from, it expires in 10 days. Use sterile syringes, needles, and containers. To formulate in artificial tears and LRS: remove 3 mL from a 15 mL bottle of artificial tears solution (e.g., Tears Again drops) and add to 1 g injectable cefazolin bottle. Then add 2 mL of LRS to the cefazolin bottle. Reconstitute the cefazolin and mix well. Remove 4 mL of the cefazolin solution and add it back to the 15 mL bottle of artificial tears and mix well. Note: do not use artificial tears with glycerin or viscous tears; use only liquid artificial tears.
- *LRS:* Reconstitute 1 g vial of cefazolin in 10 mL of LRS to make a 100 mg/mL solution. Dilute the solution 1:1 in a sterile container (e.g., red top tube with no additive) with LRS; e.g., for a 3 mL red top tube, draw up 1.5 mL of 100 mg/mL cefazolin solution and add to the red top then add 1.5 mL of LRS to the red top and mix well. Use a needle and syringe to draw up the medication; remove the needle to place a drop of the solution on the eye.
- Moxifloxacin (Vigamox), 1 drop q1–2h initially
- Gatifloxacin, 1 drop q1–2h initially

Topical Gram-Negative Spectrum Antibiotics for Infected Corneal Ulcers

Ciprofloxacin, 1 drop q1–2h initially
Ofloxacin, 1 drop q1–2h initially
Gentamicin 0.9%, 1 drop q1–2h initially
- Can fortify by adding 0.33 mL of 100 mg/mL gentamicin to every 5 mL of 0.3% gentamicin ophthalmic solution

Tobramycin, 1 drop q1–2h initially

BID, Twice daily; *IV,* intravenous; *LRS,* lactated Ringer's solution; *PO,* orally; *QID,* four times daily; *SID,* once daily; *TID,* three times daily.

SURGICAL ANATOMY

The eyelids and orbit house and protect the eye (Fig. 16.1). Eyelids are mobile folds of skin that block light and protect the cornea. The upper lid is slightly larger and more mobile than the lower lid. Upper and lower lids join at the *medial and lateral canthi* (see Fig. 16.1) and are stabilized by the medial and lateral palpebral ligaments. The width of the opening between the lids is controlled by opposing groups of muscles; the orbicularis oculi muscle closes the palpebral fissure, whereas the fissure is widened by the levator palpebrae superioris, levator anguli oculi medialis, and smooth muscles of the periorbita. The upper and lower *nasolacrimal puncta* (see Fig. 16.1), which drain excess tears from the ocular surface to the nose and pharynx via the nasolacrimal duct, are located just posterior to the eyelid margin, approximately 2 to 5 mm from the medial canthus. The eyelids close like a zipper from lateral to medial to move tears toward the nasolacrimal puncta. The medial canthus (inner corner of the eyelids) has the *lacrimal caruncle* that projects small fine hairs, has sebaceous glands, and may be pigmented.

The eyelid margins of dogs and cats are typically devoid of hairs. The upper eyelid skin has long *cilia* projecting from the upper lateral eyelid skin just above the eyelid margin; the lower lid is devoid of cilia (see Fig. 16.1). Long tactile hairs arise from a caruncle dorsomedial to each eye and correspond to human eyebrows. The eyelid margin is a mucocutaneous junction where the conjunctiva meets skin.

The eyelid margins are lined with a row of *meibomian glands* that secrete the oil component of the tear film that helps prevent tear film evaporation (see Fig. 16.1). The meibomian glands are visible through the palpebral conjunctiva at the eyelid margin and contain white infiltrate when diseased. The meibomian gland openings look like a row of small gray dots along the eyelid margin.

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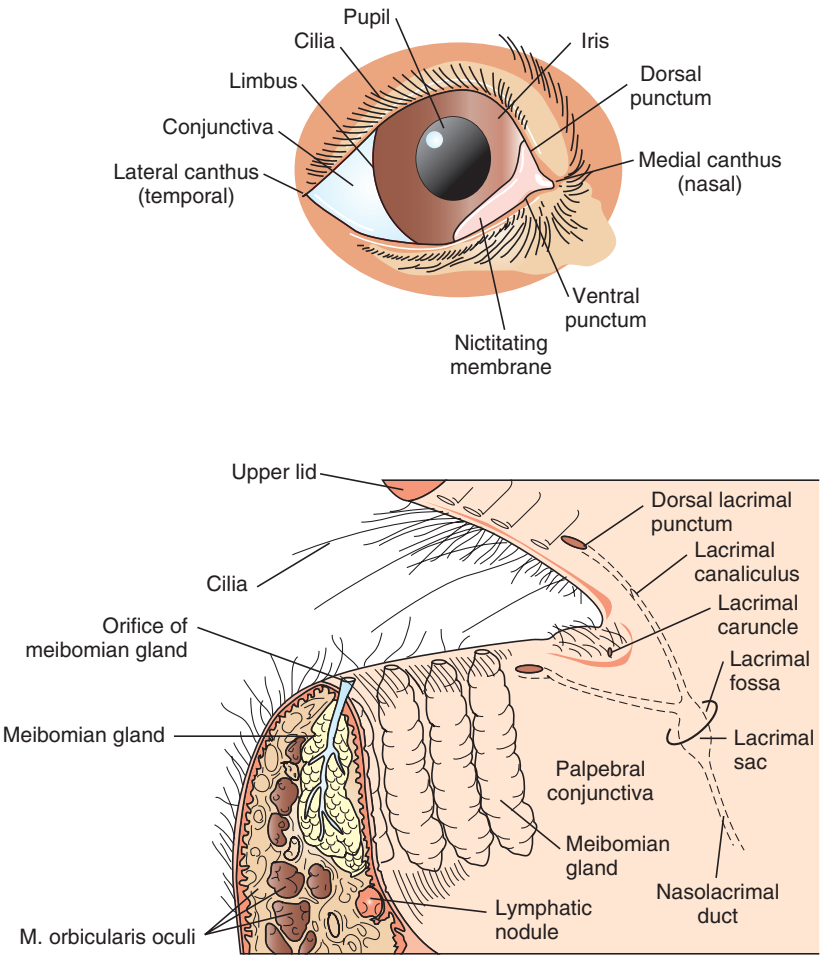


FIG. 16.1 Periocular and eyelid (as viewed from the inside) anatomy.

SURGICAL TECHNIQUES

Eyelid surgery involves handling small amounts of tissue; precision and delicacy are required. Sutures should always be placed partial thickness in the skin, and sutures and knots should always be placed such that they do not contact the cornea.

Temporary Tarsorrhaphy

A temporary tarsorrhaphy is indicated for patients who cannot blink or have traumatic proptosis. This procedure is also used after some eyelid surgeries to reduce tension on the surgery site and to help reduce wound contracture during healing or for temporary treatment of exophthalmos with excessive corneal exposure. A temporary tarsorrhaphy should be avoided for deep corneal ulcers, corneal lacerations, corneal perforations, or any ocular disease not involving excessive corneal exposure because this procedure will not assist healing and it prevents visualization of the diseased area.

Place horizontal mattress sutures at the eyelid margins with 4-0 silk to appose the eyelids, using partial-thickness bites oriented perpendicular to the lid margin (Fig. 16.2). Tie the suture knots snugly because loose sutures could contact the cornea. Stents may be used if needed to reduce pressure of the suture on the lids.

The owner should monitor the animal for a dramatic increase in eye discharge or pain after a tarsorrhaphy has been placed. If this is seen, the sutures should be removed immediately and the eye reevaluated. Uncomplicated tarsorrhaphy sutures should be removed in 14 days, or sooner if they loosen. If the tarsorrhaphy sutures become loose and are not immediately removed they can traumatize the cornea, leading to corneal ulcers.

HEALING OF THE EYELID

The eyelid heals similar to any other skin. Because the eyelids are exposed to more moisture than other skin, moist dermatitis may occur more commonly than at other sites.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

For eyelid surgery, a "soft" suture such as polyglactin 910 (absorbable) or silk (nonabsorbable) should be used. Polyglactin 910 can be left to dissolve, or the sutures can be removed. Being nonabsorbable, silk should be removed after the incision is healed because reaction to silk suture material will eventually develop. For the eyelid margin, suture size should be 5-0; for eyelid skin away from the margin, it should be 3-0 to 5-0. Suture should have a curved, swaged on fine, reverse cutting needle (Box 16.2). Stiffer sutures (polydioxanone, nylon) should be avoided because they can cause tissue distortion and become a hazard to the cornea if they loosen or shift in position.

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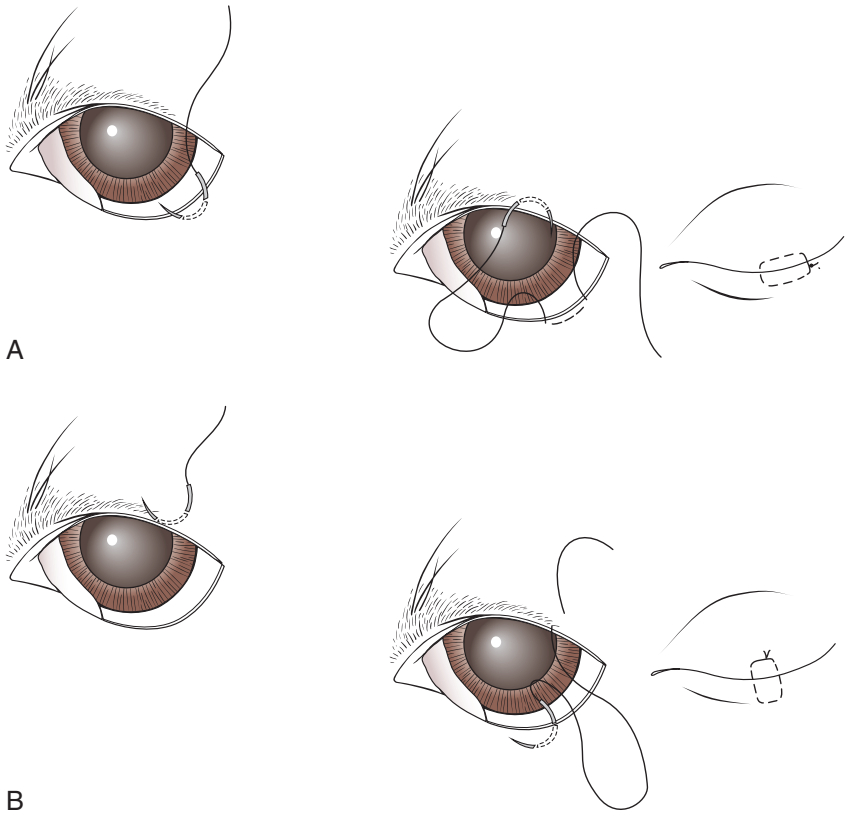


FIG. 16.2 Temporary tarsorrhaphy achieved by orienting horizontal mattress suture (A) parallel or (B) perpendicular to the eyelid margin. Sutures do not penetrate the full thickness of the eyelid.

BOX 16.2 Examples of Specialized Instruments and Materials Needed for Eye Surgeries

Eyelid Surgery

4-0 to 5-0 polyglactin 910 suture, 4-0 silk, or staples, depending on procedure; tissue glue for eyelid tacking
Baby Jones or other towel clamps (4)
Jaeger lid plate
Derf or Pars needle holders
0.8 mm Bishop-Harmon or Brown-Adson forceps
Iris or ocular scissors
Optional: chalazion clamp, Bard-Parker blade handle, and No. 15 blade for eyelid mass resection, mosquito hemostats
Optional: Allis tissue forceps for single lid Hotz-Celsus procedure
Optional: stent material such as sterile IV tubing or holed button

Cherry Eye

5-0 or 6-0 polyglactin 910 suture
Baby Jones or other towel clamps (4 or 6)
Castroviejo or Barraquer curved tip needle holders
Microsurgical forceps such as Colibri 0.3 mm with a tying platform
Bishop-Harmon forceps 0.8 mm
Iris or ocular scissors

Conjunctival Graft

6-0 polyglactin 910 suture
Baby Jones or other towel clamps (4)
Barraquer or Castroviejo eyelid speculum
Stevens tenotomy scissors
Colibri forceps 0.3 mm with a tying platform (2)
Barraquer or Castroviejo needle holders
Iris scissors
Bishop-Harmon forceps 0.8 mm
Magnification

Proptosis

4-0 nylon or silk suture
4-0 or 5-0 polyglactin 910 suture
Mayo scissors
Derf needle holder
Brown-Adson or Bishop-Harmon 0.8 mm forceps
Mosquito hemostats
Jaeger lid plate, smooth blade handle, or other material to help push globe back atraumatically
Iris or other scissors for suture cutting
Optional: stent material

Enucleation

2-0, 3-0, or 4-0 PDS for the orbital septum
2-0, 3-0, or 4-0 polyglactin 910 for the subcutaneous tissue and skin
Towel clamps (4)
Optional Allis tissue forceps
Brown-Adson forceps
Bard-Parker blade and No. 15 scalpel blade
Short curved Metzenbaum scissors
Mayo scissors
Sharp curved enucleation scissors
Curved mosquito or Crile forceps
Large hemoclips
Derf or other small needle holder
Optional: cautery and suction
Optional: silicone orbital implant

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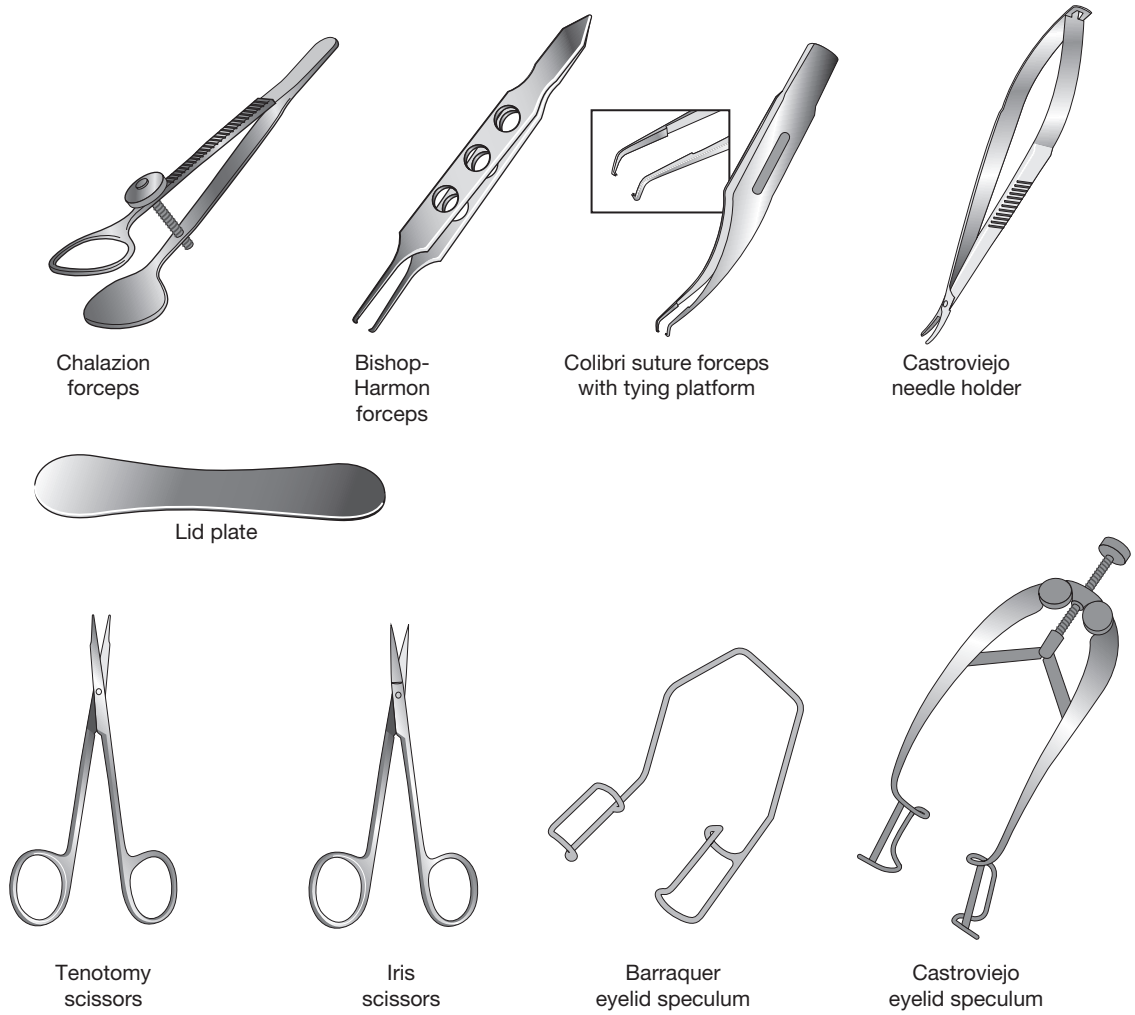


FIG. 16.3 Ophthalmic surgical instruments (not to scale).

Ophthalmic surgery instruments (see Box 16.2) should include 0.8 mm Bishop-Harmon forceps for handling eyelid tissue. Use Derf or Pars needle holders and small ocular or iris scissors to handle and cut 5-0 suture. An autoclavable Jaeger lid plate or chalazion clamp will also provide tissue support and protection to the cornea during eyelid surgery. Chalazion clamps have a solid rounded plate and an open ring that clamp together when compressed (Fig. 16.3). When the chalazion forceps is closed, pressure is applied to tissues under the ring edges, causing a temporary blockage of bleeding to the surgery site. Bleeding should be treated with direct pressure; sterile cotton swabs allow for precise but gentle wiping to allow visualization of the surgery site. A handheld battery-operated Medline or Bovie Medical cautery pen can also be used for surface hemorrhage.

POSTOPERATIVE CARE AND ASSESSMENT

After surgery, the eyelid and facial skin incisions should be protected with a rigid plastic Elizabethan collar (E-collar) that extends just beyond the animal's nose when the head is fully extended. Patients can compromise the surgery site if they traumatize their surgical incisions, which sometimes necessitates additional surgery. Patients usually rub their eyes on objects or the floor, so a properly sized rigid E-collar is more effective than eye patches, inflatable neck collars, or soft E-collars.

NOTE Soft E-collars, rigid neck restraints (e.g., Bite Not collar), and inflatable neck restraints allow the patient to rub the eyes against objects, so they are not effective protection.

Medical therapy after surgery consists of topical prophylactic antibiotics (see Box 16.1) four times daily, oral antibiotics for extensive surgeries, antiinflammatories, and analgesics. Administering medications four times daily can be difficult, but a typical regimen is for the owner to medicate the animal when they wake up, leave the house, get home, and before they go to bed. Mucoid discharge from the eyes can be normal in the initial period after surgery and is typically caused by postoperative conjunctivitis.

Owners should monitor for increased swelling, redness, or discharge from the incision in addition to pruritus, blepharospasm, epiphora, or pain. At the postoperative exam, the cornea should be checked for ulceration, vascularization, or signs of topical irritation. If an incision infection occurs, consider culture, sensitivity, and systemic antibiotics.

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COMPLICATIONS

Eyelid margin imperfections and notches can adversely affect the even distribution of the tear film over the cornea. The incision can dehisce from infection or be disrupted by the patient. Poor eyelid alignment and inappropriate tissue tension may cause entropion, ectropion, or a distorted eyelid, and hair contact from these areas can lead to corneal ulceration, vascularization, granulation, or pigmentation. Sutures rubbing against the cornea can cause ulceration, necessitating suture replacement or removal.

Epiphora may occur if the nasolacrimal puncta or duct is injured. Hypersensitivity to topical neomycin or polymyxin can cause severe eyelid swelling, pruritus, and chemosis (conjunctival edema) or, very rarely, anaphylaxis in cats.

SPECIAL AGE CONSIDERATIONS

Younger animals are more prone to trauma or problems from conformation such as entropion, but they heal faster than older animals.

SPECIFIC DISEASES

EYELID LACERATION

DEFINITIONS

Eyelid laceration is a partial or full-thickness cut on the eyelid.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Eyelid lacerations are caused by trauma such as bite wounds or contact with a sharp object. They should be repaired as soon as possible to protect the cornea and maintain an effective blink response. Healing of lid lacerations by secondary intention can cause eyelid fibrosis and distortion that can secondarily cause corneal scarring, pain, and corneal exposure. Direct reapposition of wound ends is possible if one-third or less of the lid margin is missing. More extensive injuries require advancement flaps or grafts for repair. Preservation of the maximum amount of eyelid margin is essential for normal eyelid function. Even very narrow flaps of eyelid should be replaced rather than excised if they appear to be viable (Fig. 16.4). If the distal tissue does not survive, the eyelid margin may need to be reconstructed if greater than one-third of the eyelid margin is lost.

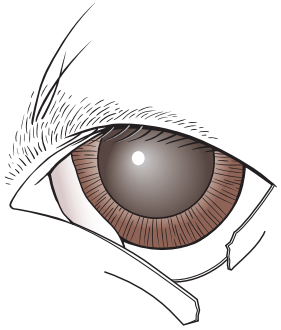


FIG. 16.4 Eyelid laceration involving the lid margin. Preserve even thin strips of tissue to avoid lid margin reconstruction.

DIAGNOSIS

Clinical Presentation

Signalment

Eyelid lacerations can occur at any age and in any breed but are more common in young, active animals.

History

Bleeding, swelling, or a distorted eyelid may be observed. Copious bleeding may be seen at the time of initial injury, but often stops quickly.

Physical Examination Findings

The eyelid margin will look irregular with a full-thickness laceration. Eyelid swelling can be considerable and thus distorting. A strip of tissue at the margin may have been torn away from the rest of the eyelid. Check the cornea, the third eyelid, inside the globe, on the head, and body for other signs of injury.

DIFFERENTIAL DIAGNOSIS

Eyelid distortion from skin disease, scarring, or ulcerative neoplasms could look similar.

MEDICAL MANAGEMENT

Topical and oral antibiotics should be initiated as for other skin injuries after an eyelid laceration. If the cornea is not being covered completely by the eyelids during blinking, lubrication of the cornea is indicated.

SURGICAL TREATMENT

Surgery is indicated when the eyelid margin is affected, incomplete corneal coverage by the eyelids is seen during blinking, or the eyelid defect is large. For eyelid lacerations involving the nasolacrimal duct, or potentially complicated eyelid repairs, referral to a veterinary ophthalmologist is recommended.

Preoperative Management

Initiate treatment for skin contamination or infection before surgery. If the laceration is less than one-third of the eyelid margin length and is still bleeding, fresh, and recent (few hours), then primary closure may be possible. If the laceration is older, the edges of the lacerated tissue will need to be debrided. Keep the cornea lubricated if compromised lid function causes corneal exposure.

Anesthesia

General anesthesia is required due to the precise eyelid margin alignment needed (see pp. 266–268).

Surgical Anatomy

See Fig. 16.1.

Positioning

Position the animal in ventral recumbency with a towel roll under the chin to hold the head up.

SURGICAL TECHNIQUES

After clipping the hair, thoroughly irrigate and gently cleanse the wound with a 5% povidone-iodine solution. Identify the eyelid

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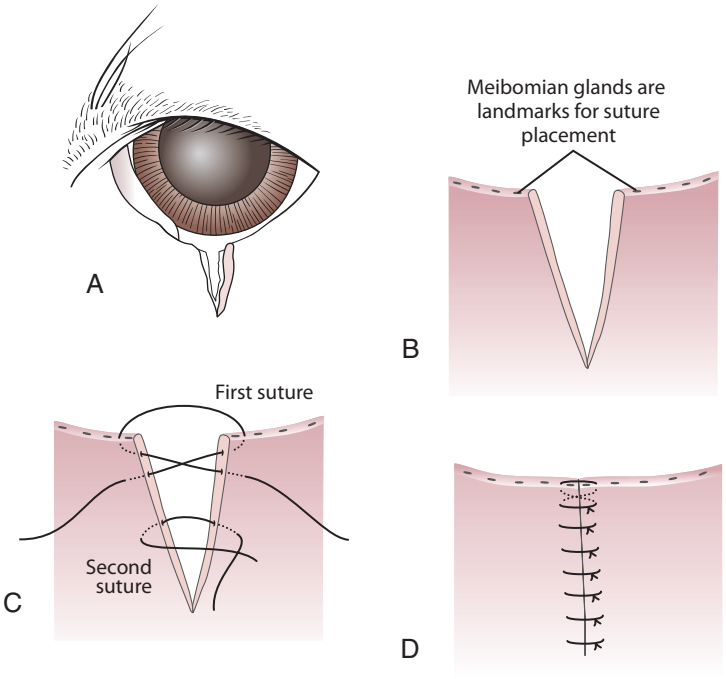


FIG. 16.5 Eyelid laceration. (A) Take care to preserve as much eyelid margin as possible. (B) Align and close the eyelid margin first. Use the meibomian gland openings to guide eyelid margin alignment. (C) Use a partial-thickness modified figure-eight cruciate to align the eyelid margin. Place the second suture in the middle of the laceration site to align the skin. (D) Taking care to place skin sutures at partial thickness, close the skin and align it using a simple interrupted pattern. Place sutures approximately 3 to 5 mm apart.

margin, the meibomian glands openings along the edge of the eyelid margin (see Figs. 16.1 and 16.5B), and the palpebral conjunctiva. For older lacerations, debride the edges in a narrow strip no more than 1-mm wide with iris scissors to freshen the skin edges before closure. Take care to preserve as much eyelid margin as possible (Fig. 16.5A). Align and close the eyelid margin first, ensuring that the lid margin has perfect alignment. Correctly placed sutures on the eyelid margin will be placed along the line of meibomian gland openings (Fig. 16.5B). *Use a partial-thickness modified figure-eight cruciate or simple interrupted suture pattern with 5-0 or 4-0 polyglactin 910 to align the eyelid margin (Fig. 16.5C). Taking care to place skin sutures partial thickness, close the skin and aligne it with a simple interrupted pattern. Place sutures approximately 3 to 5 mm apart, and direct the suture and knot ends away from the cornea (Fig. 16.5D).* When the surgery is complete, the inside of the eyelid should have no visible suture, leaving the conjunctival tissue to heal on its own, and no suture should be touching the cornea.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Surgical instruments and suture should be used as described in the General Principles on pp. 269–271.

POSTOPERATIVE CARE AND ASSESSMENT

A complete temporary tarsorrhaphy may be indicated to protect the cornea after repair of extensive eyelid injuries because of impaired eyelid function and blink reflex. Suture placement should be reevaluated after the patient is awake and periodically after surgery to ensure no corneal contact with sutures.

COMPLICATIONS

Complications of surgery, in addition to the general principles listed previously, include necrosis of eyelid tissue. Corneal disease will occur if there is poor lid conformation and function. Sutures can be removed after 14 days.

PROGNOSIS

If eyelid laceration repairs are done shortly after the trauma, lid margin restoration is good, and the tissue destruction was minimal, the prognosis can be excellent for long-term eyelid function. If the laceration was chronic or extensive tissue damage was present, the prognosis can be guarded to good.

NEOPLASTIC EYELID MASS

DEFINITIONS

Meibomian glands are the oil glands that line the eyelid margins.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Eyelid masses, even if benign, can create ongoing trauma to the cornea, necessitating surgical mass removal. Eyelid masses can cause corneal trauma and discomfort, interfere with eyelid function, and cause keratitis. Chronic eyelid masses are usually less urgent if they are slowly progressive or are small. Eyelid neoplasms in dogs are most often associated with the meibomian glands (see Figs. 16.1 and 16.5B), and 70% to 80% are benign. Sebaceous adenomas and epitheliomas are the most common eyelid tumor in dogs; they sometimes grow rapidly. Malignant eyelid tumors are less common and include squamous cell carcinoma, sebaceous adenocarcinoma, basal cell carcinoma, and fibrosarcoma. Of the eyelid melanomas, approximately 60% are benign and 40% are malignant.

In cats, eyelid neoplasms tend to be malignant, and squamous cell carcinomas (~60% of cases) are the most common. Others include fibrosarcoma, adenocarcinoma, basal cell carcinoma, melanoma, and hemangiosarcoma.

DIAGNOSIS

Clinical Presentation

Signalment

The average age of dogs presenting with eyelid neoplasia is 8 years, whereas cats are on average 10 years old. Beagles, Siberian huskies, English setters, toy and miniature poodles, and Labrador and golden retrievers have an increased prevalence for eyelid tumors.

History

Eyelid masses are grossly visible and can develop slowly or rapidly. Clinical symptoms include ocular discharge, blepharospasm, hemorrhage, and topical irritation. Some animals rub at the eye but many are not bothered by the mass.

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Physical Examination Findings

In affected animals a mass can be visualized or palpated on the eyelid, eyelid margin, or conjunctiva. Evert the eyelid and inspect the underside of the lid to see if the mass extends into the meibomian glands along the eyelid margin (Fig. 16.6). Multiple eyelid masses may be of the same or different types. Approximately 10% of malignant canine lid tumors are locally invasive. Ophthalmic examination may show secondary corneal ulcers, keratitis (corneal blood vessels), or corneal melanosis (pigmentation). In severe cases, periocular excoriations, skin ulceration, or bleeding from the skin may be present.

Laboratory Findings

If large enough, an eyelid mass may be aspirated for cytology; an eyelid plate, scalpel handle, or sterile tongue depressor should be placed between the needle and the eye. Aspiration of enlarged local lymph nodes may also be considered. Eyelid tumors can be associated with secondary infections; therefore culture and sensitivity and treatment with appropriate antibiotics may be considered before surgery to shrink the diseased area if infection is suspected. Histopathologic examination of all excised masses should be performed for a definitive diagnosis and to check surgical margins.

DIFFERENTIAL DIAGNOSIS

Differentials include inflammatory lesions such as chalazion or meibomian gland disease, fungal granulomas from histoplasmosis, eyelid cysts, and cicatricial distortion.

MEDICAL MANAGEMENT

Chemotherapy, radiation therapy, cryotherapy, or photodynamic therapy may be used in combination with or as an adjunct to surgical treatment. In particular, squamous cell carcinoma may be treated with beta-irradiation using a strontium-90 probe.

SURGICAL TREATMENT

Eyelid tumors that are progressively enlarging, contacting the cornea, or causing clinical symptoms should be excised. If less than one-third of the eyelid margin is involved, primary closure may be used after mass excision. Mast cell tumors requiring wide 0.5 cm margins or masses involving more than one-third of the eyelid margin length should be referred to a veterinary ophthalmologist for reconstructive surgery. More eyelid length may be able to be resected in some breeds, such as cocker spaniels, that have excessive eyelid length. Breeds with short, tight-fitting eyelid fissures such as miniature poodles or cats may have less than the expected amounts of resectable lid, making dehiscence, visible lid shortening, reduced palpebral fissure size, facial asymmetry/disfigurement, and epiphora more likely.

Preoperative Management

Treat eyelid infections or conjunctivitis if needed before surgery. Estimate the amount of tissue to be removed before anesthetizing the animal.

Anesthesia

If the patient has metastatic neoplasia involving the lungs or liver, special precautions are necessary when anesthetizing them; however, general anesthesia is usually necessary due to the precision required to perform this surgery.

Surgical Anatomy

Many meibomian adenomas extend proximally and may be more visible from the underside of the eyelid, so this area should always be inspected before starting surgery (Figs. 16.1 and 16.6; also see p. 268).

Positioning

Ventral or lateral recumbency provides the best access for eyelid surgery. In ventral recumbency, elevate the chin on a towel roll to improve the airway and head positioning.

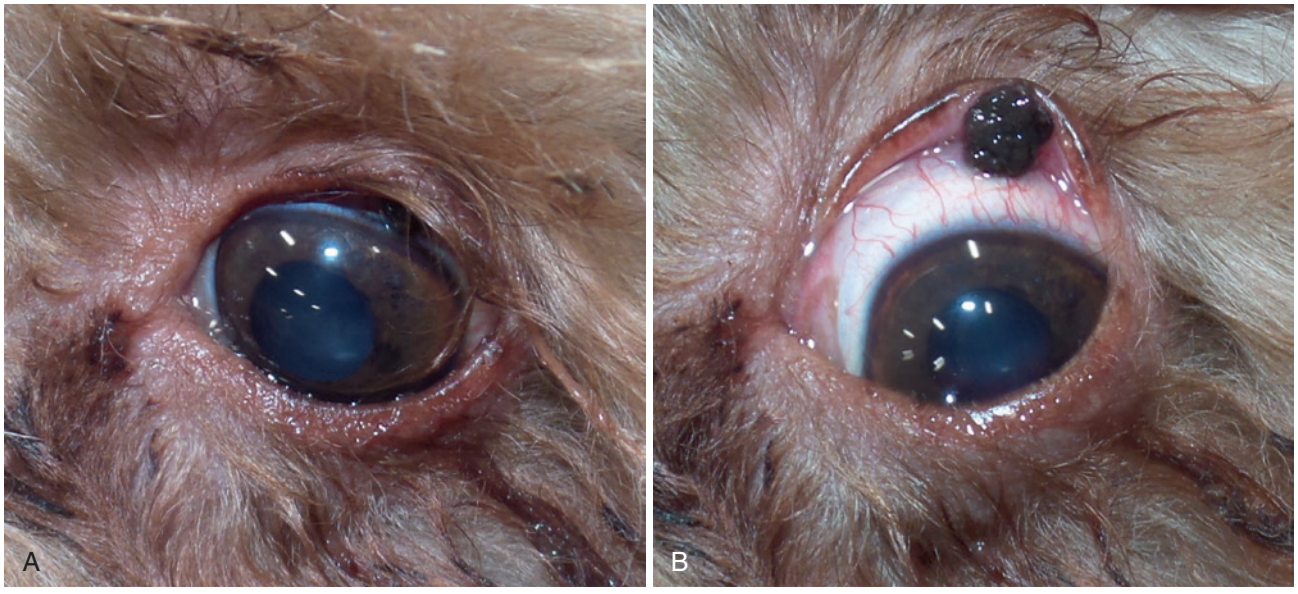


FIG. 16.6 (A) Eyelid mass not visible from the outside of the eyelid. (B) Eyelid mass visible from the underside of the eyelid.

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SURGICAL TECHNIQUES

Primary Mass Removal and Closure

Full-thickness eyelid excisions are usually needed to remove the eyelid mass completely.

Stabilize the lid and provide support with a lid plate or chalazion clamp (Fig. 16.7A). If a lid plate or chalazion clamp is not available, use a sterile tongue depressor to support and stabilize the lid. Ensure that the planned excision area involves less than one-third of the eyelid margin length. Using a No. 15 Bard-Parker blade, make a V or house-shaped incision around the mass with a 1 to 2 mm grossly visible margin. Cut any conjunctiva with iris or tenotomy scissors to fully loosen and remove the mass (Fig. 16.7B). Control hemorrhage with direct pressure or a battery operated handheld cautery pen. Align and close the eyelid margin and skin first, ensuring that the lid margin has perfect alignment (Fig. 16.7C, inset). The line of punctate meibomian gland openings along the eyelid margin can be used as a landmark to place the eyelid margin sutures in the correct position. *Use a partial-thickness modified figure-eight cruciate (Fig. 16.7C) or simple interrupted suture pattern with 5-0 polyglactin 910 to align the eyelid margin. While taking care to place skin sutures at partial thickness, close and align the skin using a single-layer simple interrupted pattern.[3] Put the second suture in the middle of the incision to bring the cut skin edges margins together and space remaining skin sutures approximately 2 to 4 mm apart. Direct suture knot ends away from the cornea.* When the surgery is complete, the inside of the eyelid should have no visible suture (Fig. 16.7D) and no suture should be touching the cornea anywhere along the incision.

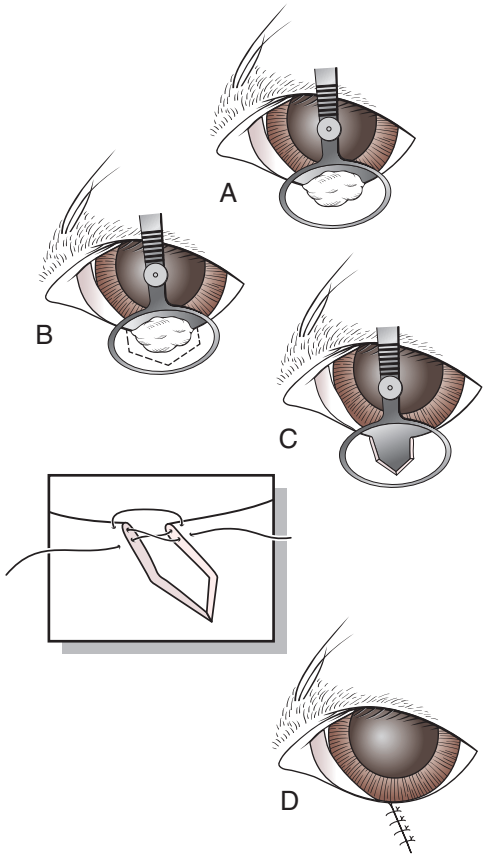


FIG. 16.7 Eyelid mass resection. (A) Stabilize the lid and provide hemostasis with a lid plate or chalazion clamp. (B) Make a V or a house-shaped incision around tumors involving less than one-third of the eyelid margin. (C) Align the eyelid margin first with a figure-eight modified cruciate suture. (D) Close the remaining skin partial thickness in a simple interrupted pattern with skin sutures 2 to 4 mm apart.

NOTE The suture at the eyelid margin is the most critical for eyelid alignment and to reduce the likelihood that a notch will develop.

Small (<3 mm) pedunculated conjunctival masses that do not involve deeper tissues may be removable with only a topical anesthetic in a still patient. Deeply attached or extensive conjunctival mass removals require general anesthesia.

Expose masses arising from the conjunctiva by everting the lid with a chalazion clamp. Excise the mass with the conjunctiva surrounding the mass using tenotomy scissors, then allow the palpebral conjunctival defect to heal by secondary intention.

Masses involving more than one-third of the eyelid margin length require advancement flaps for reconstruction, and referral to a veterinary ophthalmologist is recommended. If referral is not possible, an H-plasty skin advancement or semicircular graft may be considered to reconstruct the eyelid when more than one-third of the eyelid margin was removed.

H-plasty Pedicle Advancement Flap

AP An H-plasty advancement flap should only be performed on the lower eyelid, should not be performed on the upper eyelid, and is more appropriate for masses away from the canthi.

After resecting the mass with parallel incisions perpendicular to the lid margin, create a single-pedicle advancement flap by extending these incisions away from the eyelid margin (Fig. 16.8A). Make the length of the advancement flap 1 to 2 mm longer than the defect left from the mass removal. Excise small triangular sections of skin on either end of the distal incision (Fig. 16.8B). Advance the flap to the lid margin, align it carefully with the remaining lid, and suture it with 5-0 polyglactin 910 into place with a figure-eight suture pattern (Fig. 16.8C) at the eyelid margin. Suture the rest of the graft skin to the adjacent skin using 4-0 to 5-0 absorbable polyglactin 910 (Fig. 16.8D). Remove the external haired dermis from distal edge of the graft to create a hairless area in the area closest to the cornea.

Semicircular Flap

AP A semicircular incision at the lateral canthus may be used to advance the lateral skin into a defect on the upper or lower lateral eyelid; it is therefore most appropriate for large, laterally located eyelid lesions.

Excise and remove the eyelid mass (Fig. 16.9A). Following the curvature of the lateral eyelid, make a lateral semicircular skin incision where the length of the curve is approximately twice the length of the excised portion of the eyelid, with the peak/trough of the curve in the center of the incision (Fig. 16.9A). Excise a triangular area of skin at the lateral incision to remove the dog ear formed when the graft is slid into the defect (Fig. 16.9B). Slide the skin graft medially into the eyelid defect and align the eyelid margin with the skin graft with a figure-eight modified cruciate suture (Figs. 16.9B–D). Next, align the peak/trough of the semicircular skin graft to the lateral canthus with another figure-eight modified cruciate suture (see Fig. 16.7D). Close the skin with simple interrupted

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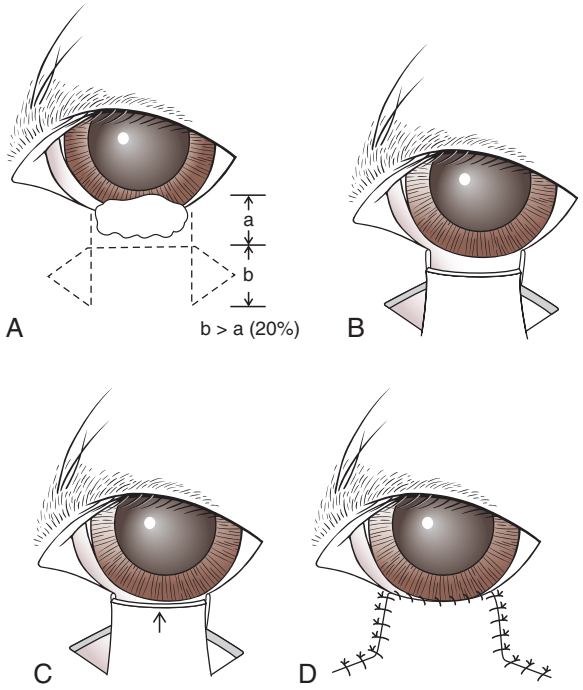


FIG. 16.8 H-plasty to reconstruct the lower eyelid margin after more than one-third of the eyelid margin length was removed. (A) Resect the mass with a rectangular incision, including 2-mm margins of normal skin. Create an advancement flap by extending proximally the medial and lateral incisions. (B) Remove triangles of tissue on each side of the flap to prevent dog ears. Undermine and mobilize the conjunctiva. (C) Advance the flap to the lid margin, aligning it carefully. (D) Suture conjunctiva and skin at the new lid margin with a simple continuous pattern, and try to align so no growing hairs will touch the eye. Place interrupted sutures to appose the remaining incision.

sutures (4-0 to 5-0 absorbable polyglactin 910; Fig. 16.9E). Along the lateral free skin graft margin, remove a narrow 2- to 3-mm margin of superficial haired dermis, taking care to preserve the deeper skin graft tissue. The goal is to create a hairless skin margin to try to reduce trichiasis.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Surgical instruments and suture should be used as described previously in the General Principles, pp. 269–272. Sliding eyelid grafts should be ideally lined by conjunctiva; microsurgical techniques are needed for conjunctival tissue handling, so these surgeries are best referred to a veterinary ophthalmologist.

POSTOPERATIVE CARE AND ASSESSMENT

In addition to the eyelid General Principles on p. 271, expect postoperative eyelid swelling that should resolve in 2 to 4 weeks. Apply first cold and then later warm compresses to reduce eyelid swelling and promote circulation if major reconstruction or grafts were used. Reserve assessment of the repair for 2 to 4 weeks, when swelling subsides. Remove the sutures after 14 days when the skin is healed. Maintain the E-collar for 2 to 3 days after suture removal if the animal wants to scratch at the site.

COMPLICATIONS

In addition to the eyelid General Principles on p. 272, dehiscence following repair can occur if there has been excessive tension on the site or incision infection. Tumor recurrence can occur if the surgical margins still contain neoplastic cells.

Trichiasis causing chronic irritation is common after surgeries in which the eyelid margin is not maintained or recreated (H-plasty, semicircular, and other flaps) and can be a significant complication that can lead to discomfort, corneal ulceration and scarring, epiphora, self-trauma, and so forth and may require additional procedures.

PROGNOSIS

Prognosis depends on the type of tumor identified and its biologic behavior as well as the margins achieved.

ENTROPION AND ECTROPION

DEFINITIONS

Entropion is the inward rolling of the eyelid margin; **ectropion** is an outward rolling of the eyelid margin.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Entropion and ectropion are often an inherited breed characteristic or can occur if an eyelid laceration did not heal properly. Primary entropion is most common in young adult dogs of predisposed breeds. Entropion may worsen over time, so clinical disease may show up later in life. Enophthalmos (globe sunken inwards) or a smaller than normal globe (microphthalmos, phthisis bulbi) can also cause entropion. In certain breeds (e.g., English bulldogs, cocker spaniels, and giant breed dogs) excessive eyelid length can lead to both entropion and ectropion in the same eyelid. In cats, entropion can develop with chronic conjunctivitis,[4] weight loss, corneal ulceration, and age; feline conjunctivitis is usually caused by infection from feline herpesvirus-1, chlamydia, and mycoplasma. Chronic entropion in cats can commonly lead to the formation of a corneal sequestrum. Severe ectropion can lead to excessive corneal exposure and disease.

DIAGNOSIS

Clinical Presentation

Signalment

Entropion and ectropion in dogs most often occur due to breed conformation. The most common dog breeds that are severely affected include shar-peis, chow chows, English bulldogs, Rottweilers, English and springer spaniels, bloodhounds, basset hounds, Bouvier des Flandres, and giant breed dogs. Breeds that may have lower eyelid entropion include Labrador retrievers, golden retrievers, Chesapeake Bay retrievers, and Irish setters. Medial lower eyelid entropion is commonly seen in brachycephalic breeds, including Boston terriers, pugs, and shih tzus. Brachycephalic cats (e.g., Persians, Himalayans) may have conformational lower medial entropion or enophthalmos.[5] Feline entropion may occur from trauma or from ongoing blepharospasm related to irritation of the globe associated with infectious conjunctivitis or corneal ulceration.

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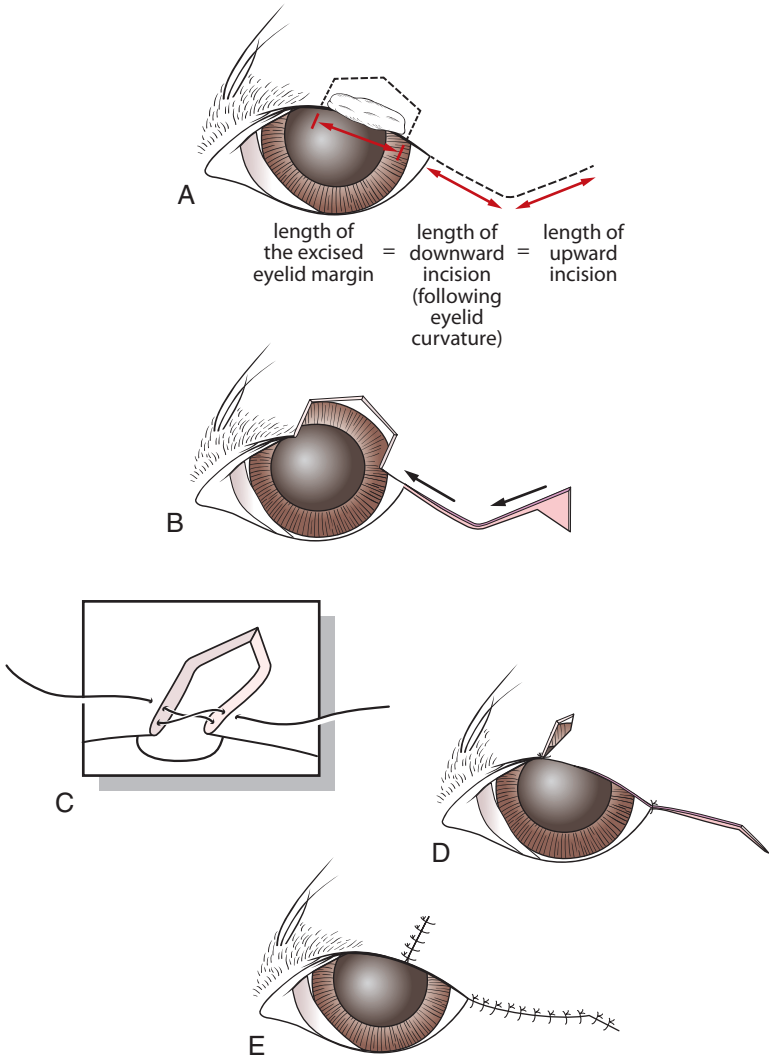


FIG. 16.9 Semicircular graft to reconstruct the eyelid after more than one-third of the eyelid margin length has been removed. (A) Remove the eyelid mass. (B) Following the curvature of the lateral eyelid, make a lateral semicircular skin incision where the length of the curve is approximately twice the length of the excised portion of the eyelid with the peak/trough of the curve in the center of the incision; at the lateral end of the incision, excise a triangular area of skin. (C) Advance the skin flap into the defect and close the eyelid margin with a figure-eight suture. (D) Appose the skin of the new lateral canthus with the peak/trough of the skin graft using a figure-eight suture. (E) Close the skin in a simple interrupted pattern.

History

Animals with entropion typically have a history of blepharospasm, ocular discharge, and rubbing at their eyes. Symptoms can be intermittent early in the disease but worsen over time. Vision may be impaired in chronic cases in which secondary corneal scarring (vascularization, pigmentation) develops.

Physical Examination Findings

Entropion causes epiphora, blepharospasm, and conjunctivitis. Excessive tearing and moisture can lead to eyelid discoloration, moist dermatitis, alopecia, and skin excoriations. Apply a topical anesthetic (e.g., proparacaine) to the eye before evaluating for entropion because spastic entropion should resolve with the topical anesthetic. Be aware that restraining the animal to examine the face may itself stimulate squinting that can cause the appearance of entropion. Restraint of the head may also push skin toward the eyes, causing the appearance of entropion. Examine the cornea for hair contact that sometimes may look like a disrupted or raised area in the tear film meniscus. Roll the eyelid margins out and see if the eyelid retains that position or rolls back in. Eyelids with entropion may have alopecia or a color change from the constant moisture. Corneal ulceration, vascularization, granulation, and pigmentation (pigmentary keratitis) will develop over time in the area of hair contact as the cornea strengthens itself to withstand the ongoing trauma from entropion. Pull the lateral canthus laterally and examine the palpebral fissure size compared to the corneal diameter to evaluate for macropalpebral fissure and ectropion.

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DIFFERENTIAL DIAGNOSIS

Spastic entropion should resolve after a topical anesthetic has been applied and does not require surgery. Distichiasis, trichiasis, ectopic cilia, imperforate lacrimal puncta, dacryocystitis, foreign bodies, KCS, and corneal ulcers are other causes of excessive eye discharge, blepharospasm, secondary dermatitis, and pigmentary keratitis.

MEDICAL MANAGEMENT

A topical eye ointment will lubricate the surface and may reduce irritation from trichiasis until surgery can be performed. Topical anesthetics such as proparacaine should not be used because they mask symptoms of corneal ulceration, and chronic use is toxic to the cornea.[6] In cats, treat conjunctivitis with topical antivirals (e.g., 0.1% idoxuridine or trifluridine, 1 drop five times daily) and antibiotics (e.g., erythromycin or Terramycin [oxytetracycline/polymyxin 0.25 inch (0.6 cm), four times daily]). Avoid topical corticosteroids because feline conjunctivitis occurs from infection (e.g., feline herpesvirus-1, chlamydia, mycoplasma).[4]

SURGICAL TREATMENT

Surgery is indicated when the entropion or ectropion is causing secondary corneal disease or discomfort. After application of a topical anesthetic, estimate the amount of correction needed on an awake animal. Surgery is not always required in cases of ectropion.

Preoperative Management

Initiate treatment for corneal ulcers before performing surgery if necessary. Treat feline conjunctivitis with topical antivirals and antibiotics if needed.

Anesthesia

See pp. 266–268. General anesthesia is required to achieve exact alignment.

Surgical Anatomy

See p. 268. Examine the lateral canthus for laxity and entropion before surgery.

Positioning

Position the animal in ventral recumbency with a towel roll under the chin to hold the head up. Make sure the skin and ears are placed in a natural position, and that draping and towel clamps are not distorting the eyelid position.

SURGICAL TECHNIQUES

Keep tissue handling minimal, tissue resection precise, and hemostasis well controlled.

Eyelid Tacking

Entropion in the neonate or young animal (≤20 weeks of age), especially shar-pei puppies, may be treated by temporarily rolling out the eyelid margin and may eventually resolve with growth. Temporary eyelid tacking may also be used for spastic entropion to provide relief from corneal irritation until the primary cause is corrected.

Use a sterile eyelid plate or tongue depressor between the eyelid and globe to protect the eye. Use the wooden end of a long sterile cotton swab parallel to the eyelid margin and compress the skin approximately 5 to 7 mm away from the eyelid margin to evert it. Place skin staples perpendicular to the eyelid margin over the swab stick and approximately 0.5 to 1 cm apart to hold the eversion (Fig. 16.10). The staples should not go over the eyelid margin, touch the cornea, or be visible inside the eyelid.

Alternatively, use skin sutures to evert the eyelid skin. Using 3-0 or 4-0 polyglactin 910, place a partial-thickness vertical mattress pattern perpendicular to the eyelid margin, with one bite 3 to 5 mm from the eyelid margin that is approximately 5 mm wide, and place another bite into the skin over the rim of the orbit (see Fig. 16.10). Do not penetrate the eyelid margin or conjunctiva. Tie the suture to create a furrow in the skin. Place additional sutures as necessary to correct both upper and lower lid entropion. Place a few drops of surgical glue within the created furrow to reduce tension on the suture line. Glue keeps the sutures in place longer and helps keep them from being rubbed out.

If necessary, the tacking procedure may be repeated in young dogs until they have outgrown their entropion. Depending on the breed, permanent correction by excision of tissue may be needed in dogs approximately 5 months or older. Severely affected dogs such as shar-peis, chow chows, and English bulldogs should be referred to a veterinary ophthalmologist.

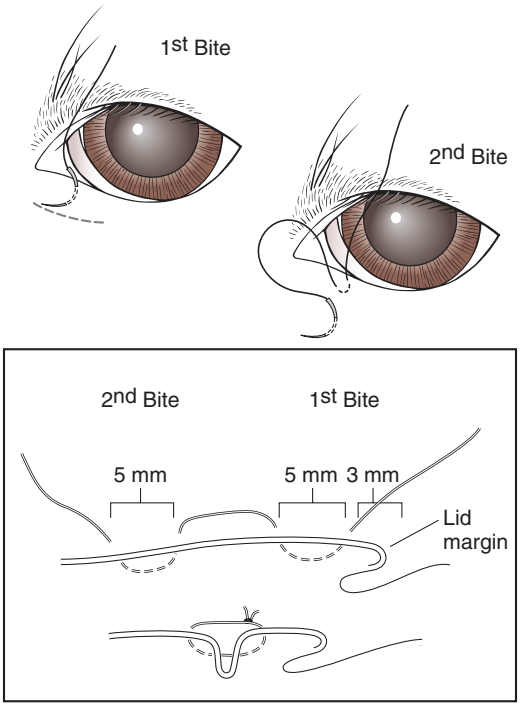


FIG. 16.10 Entropion eyelid tacking. Use vertical mattress sutures in the neonate to temporarily evert the eyelid margin. The first bite is 5-mm wide and begins 3 mm from the eyelid margin *(inset)*. Position the second 5-mm-wide bite over the rim of the orbit, biting into the orbital fascia. Tie the suture, inverting a furrow of skin. A few drops of surgical glue placed within the created furrow minimizes tension on the sutures and helps maintain their position if the patient rubs at the eyes.

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Modified Hotz-Celsus

Various adaptations of the Hotz-Celsus procedure provide a definitive surgical correction for entropion. Skin excision surgery may need to be combined with palpebral fissure reduction in some breeds. Animals with severe entropion, macropalpebral fissure, or distichia should be referred to a veterinary ophthalmologist for correction because specialized equipment is needed to treat distichia with cryosurgery, and severe cases may also need concurrent eyelid or head skin lifts.

Starting 3 to 5 mm from the eyelid margin, estimate the size of the crescent-shaped skin to be removed by crimping the skin to be excised with a curved mosquito, Kelly, or Crile forceps. (Fig. 16.11A). In severe cases for which a large correction will be needed, Allis tissue forceps can also be used to crimp the skin. The lower and upper eyelid margins should be aligned with no entropion after the forceps have been removed and the crimped skin approximates the amount of skin to be removed.

Stabilize the eyelid by placing a Jaeger lid plate or tongue depressor under the affected eyelid (Fig. 16.11B). Using a No. 15 blade, incise the skin partial thickness and parallel to the eyelid margin along the length of the crimped skin. Place the incision 3 to 5 mm from the lid margin (Fig. 16.11B). Make a second, crescent-shaped skin incision on the other side of the crimped skin and remove the strip of skin. The orbicularis muscle may or may not be removed, but removal of muscle will make the entropion correction more secure, although more bleeding will occur. *Control hemorrhage with direct pressure. Do not incise the palpebral conjunctiva. Align and suture one bite at the lateral canthus and then close at the center of the defect with simple interrupted 4-0 to 5-0 polyglactin 910 skin sutures (Fig. 16.11C). Place each suture precisely to appose the skin (Fig. 16.11C, inset). Split the distances of the remaining defects when placing additional sutures, so that ultimately the sutures are spaced 2 to 4 mm apart (Fig. 16.11D–F). To prevent corneal irritation, cut the suture ends short enough and orient them so they will not contact the cornea.*

Whenever lateral canthus entropion is present or both the upper and lower lateral eyelids are operated, the skin excisions should be extended around the lateral canthus in combination with the lower and upper eyelid skin excisions.

Make a horizontally oriented U-shaped resection at the lateral canthus, carrying the elliptical incision around the lateral canthus, staying 3 to 5 mm from the eyelid margin (Fig. 16.12A). The lateral canthus incision should be an extension of the upper and lower eyelid incisions. *Place the first suture at the most lateral part of the lateral canthus (Fig. 16.12B). Split the distances of the remaining defects when placing additional sutures (Fig. 16.12C), so that ultimately the sutures are spaced 2 to 4 mm apart (Fig. 16.12D).*

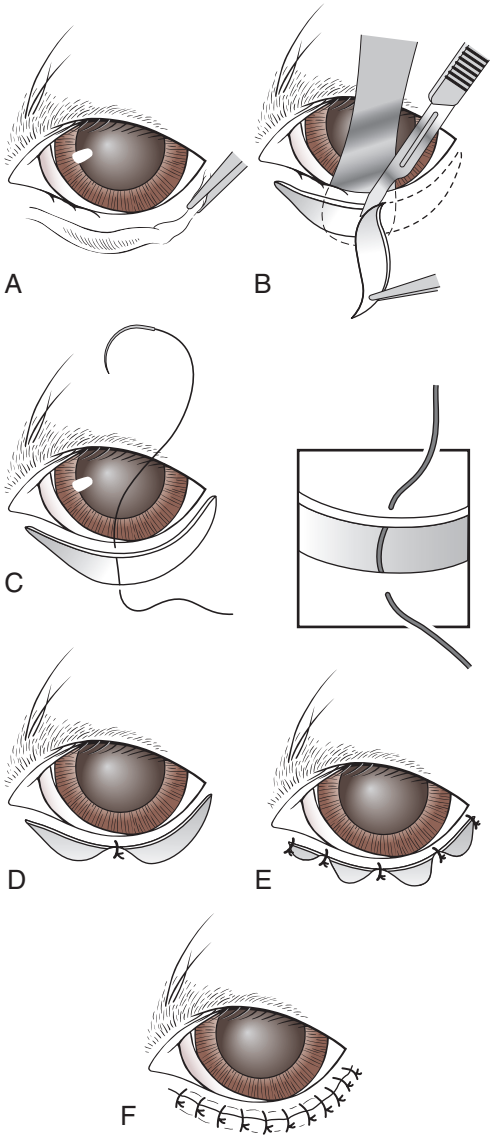


FIG. 16.11 Hotz-Celsus procedure for entropion repair. (A) Starting 3 to 5 mm from the eyelid margin, crimp the skin to be excised with a curved mosquito, Kelly, Crile, or Allis tissue forceps to estimate the size of the skin to be removed. (B) Stabilize the lid by placing a Jaeger eyelid plate under the affected eyelid. Using a No. 15 blade, incise the skin partial thickness and parallel to the eyelid margin along either side of the crimped skin and remove the strip of skin. (C–D) Begin closing the defect at the center of the wound with a simple interrupted split-thickness skin suture *(inset)*. (E) Split the distance of the remaining defects when passing additional sutures. (F) The final sutures are spaced 2 to 4 mm apart.

Permanent Lateral Tarsorrhaphy

Permanent lateral tarsorrhaphy reduces the palpebral fissure size in animals with macropalpebral fissure (large eyelid openings) that have ectropion, entropion, excessive corneal exposure, or risk for traumatic proptosis.[4] A medial canthoplasty is generally preferred in brachycephalic breeds with exotropia to reduce lagophthalmos by correcting the ventromedial entropion, as well as reducing the palpebral fissure size. Due to the nasolacrimal ducts and vasculature at the medial canthus, referral to an ophthalmologist is recommended for medial canthoplasty.

For permanent lateral tarsorrhaphy surgery, determine the amount of eyelid margin to be removed laterally by examining the eyelid opening size relative to the horizontal corneal diameter. After the tarsorrhaphy, the palpebral fissure opening should cover most of the exposed bulbar conjunctiva but should not cover the cornea.

Rather than making the initial part of the incision through the eyelid margin perpendicular to the eyelid margin (as would be done to remove an eyelid mass), angle the incision so that it's more parallel to a line that bisects the canthus; the angled incisions will make the new lateral canthus more natural looking (Fig. 16.13A).

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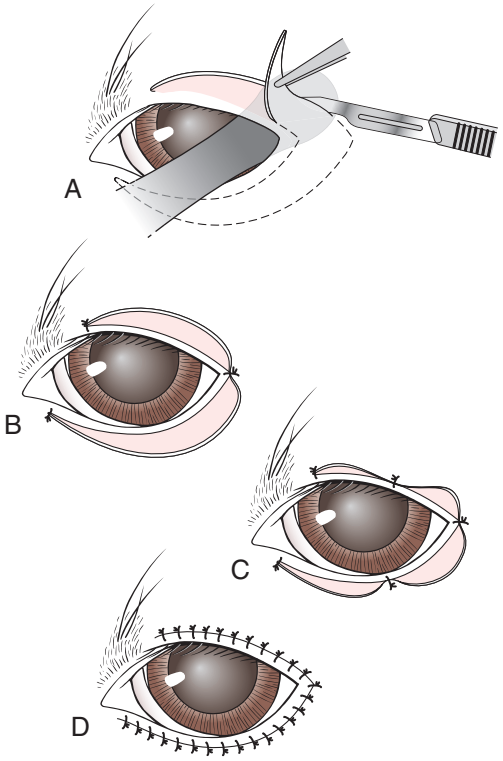


FIG. 16.12 Modification of the Hotz-Celsus procedure is performed when there is lateral canthal entropion (i.e., affecting both the upper and lower lateral lids). (A) Make a U-shaped resection around the lateral canthus staying 3 to 5 mm from the eyelid margin. (B) Place the first suture at the most lateral aspect of the excised area. (C) Split the distances of the remaining defects when placing additional sutures. (D) Skin sutures are placed 2 to 4 mm apart.

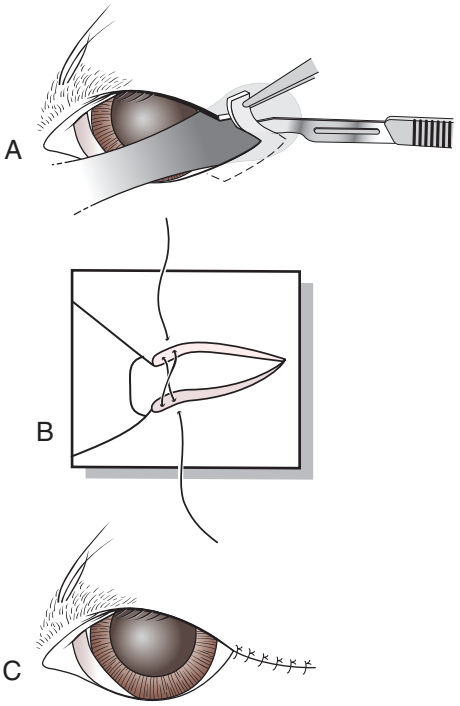


FIG. 16.13 Permanent lateral tarsorrhaphy. (A) Excise upper and lower lid margins at the lateral canthus, removing a V-shaped segment of skin. (B) Align the lateral eyelid margins with a figure-eight suture at the new lateral canthus. (C) Place skin sutures to precisely align the eyelid margins and appose the skin.

Using either a Jaeger lid plate and No. 15 scalpel blade or iris scissors, make a full-thickness eyelid incision into the upper and lower eyelid margins so the incisions on the upper and lower eyelids are equidistant from the lateral canthus (Fig. 16.13B). Remove a 3 to 4 mm wide section of lateral eyelid margin, including the meibomian glands from the upper and lower eyelids. The amount of eyelid margin removed will directly correlate to the size of the palpebral fissure reduction. *Align the eyelid margins in a figure-eight suture (Fig. 16.7C) and close the skin in a simple interrupted pattern with 5-0 or 4-0 polyglactin 910 taking care to place sutures partial thickness (Fig. 16.13C) so that no suture contacts the cornea.*

Ectropion causing folds and lid drooping can be corrected by wedge resection, as with an eyelid mass removal. The wedge is often placed laterally but ultimately depends on the eyelid shape of the individual.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Use 4-0 to 5-0 absorbable polyglactin 910 or silk suture and instruments as listed on pp. 269–271.

POSTOPERATIVE CARE AND ASSESSMENT

See p. 271. Use a rigid plastic E-collar, topical and oral antibiotics (see Box 16.1), antiinflammatories (such as nonsteroidal antiinflammatory drugs [NSAIDs]), and analgesics if not otherwise contraindicated. Prophylactic antibiotic treatment should be continued until the incisions are healed. The increased moisture at eyelid sites make these areas more prone to infection. With aggressive animals, suture such as polyglactin 910 can be left to dissolve, but silk sutures must be removed because they will not dissolve, and leaving them will predispose to infection. Maintain the E-collar for 2 to 3 days after suture removal if the animal wants to rub or scratch the eyelids.

After eyelid surgeries, tissue swelling will typically peak on the day after surgery and will take 2 to 4 weeks to resolve. Because of inflammation, ectropion may be present postoperatively; therefore delay assessment of the adequacy of correction until swelling subsides (4 weeks to several months). If undercorrection has occurred or if entropion recurs, the procedure may need to be repeated.

NOTE Warn owners of young animals that more that one surgery may be needed to fully correct the entropion or ectropion.

Corneal lesions should rapidly resolve after surgical correction. Urgent additional surgery will be needed if corneal ulceration is worsened by the eyelid changes.

COMPLICATIONS

Complications include undercorrection and overcorrection of the entropion or ectropion. Incisions made too far from the

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eyelid margin may not effectively correct the entropion, and those made too closely may overcorrect it. If the eyelid margins are not aligned at the end of surgery, the upper eyelid entropion may worsen after the lower entropion is corrected in severely affected breeds. If suture ends are left too long or sutures are placed full thickness through the eyelid, the sutures could contact the cornea and cause a corneal ulcer. Correction of severely affected animals, especially shar-peis, should be performed by an experienced ophthalmologist.

Incision infection and dehiscence from self-trauma are complications that can be conservatively managed but may also lead to additional surgery. Facial appearance may be altered by eyelid surgery.

PROGNOSIS

The prognosis is good for comfort and vision if surgical correction has adequately resolved the problem.

Surgery of the Conjunctiva and Third Eyelid

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Bulbar conjunctiva is the conjunctiva on the globe, and **palpebral conjunctiva** is the conjunctiva on the inside of the eyelids. The **bulbar** surface of the third eyelid refers to the conjunctival surface of the third eyelid facing the globe (posterior side of the third eyelid). The **palpebral** surface of the third eyelid refers to the conjunctival surface of the third eyelid facing the external eyelid (rostral or anterior side of the third eyelid). **Cherry eye** is a prolapse of the third eyelid gland.

The limbus is the junction of the cornea and sclera. **Hypopyon** is white blood cells in the anterior chamber, and **hyphema** is red blood cells inside the anterior chamber. **Keratoconjunctivitis sicca** (KCS) is ocular surface inflammation caused by a lack of aqueous tears that is usually immune mediated in the dog and caused by feline herpesvirus in the cat. **Anterior uveitis** is inflammation of the anterior uvea causing a breakdown of the blood-eye barrier. **Glaucoma** is the disease of high pressure inside the eye causing vision loss. **Leukoma** is a white corneal scar.

PREOPERATIVE CONCERNS

Procedures involving the conjunctiva, third eyelid, and third eyelid gland should be performed by an individual who is trained and comfortable with the diagnosis, surgical procedure, follow-up, and possible complications. Surgeries requiring microsurgery, corneal suturing, or surgery inside the eye should be referred to a veterinary ophthalmologist.

Each animal should be thoroughly evaluated for concurrent and contributing abnormalities (e.g., corneal ulcers caused by a foreign body inside the eyelid, concurrent dry eye, neurologic disease). A complete ocular exam should include examination of the cornea, anterior chamber, and fundus (optic nerve head and retina). Systemic diseases such as hyperadrenocorticism or treatments with systemic corticosteroids can also adversely affect healing and should be taken into consideration with the treatment plan.

Successful ophthalmic surgery requires a correct diagnosis, appropriate choice of surgical procedure and materials, attention to detail, proper instrumentation and equipment, and suitable follow-up. With surgical treatment, appropriate postoperative medical therapy is essential to success. Infected corneal ulcers, for example, require continued monitoring and medical management after surgery.

The conjunctiva rapidly swells and becomes inflamed with any tissue handling. Conjunctiva is highly vascular, whereas the cornea is avascular. Antiinflammatory drugs (NSAIDs or steroids) given topically or systemically can minimize swelling but will slow corneal vascularization and healing; they should be used conservatively, if at all, in cases for which ocular surface healing is needed.

NOTE Intraocular, corneal, and microsurgical procedures are best referred to a veterinary ophthalmologist or an individual trained and experienced in these procedures.

PREPARATION OF THE SURGICAL SITE

For conjunctival and third eyelid procedures, treat with a prophylactic sterile broad-spectrum antibiotic ophthalmic ointment (e.g., neomycin, polymyxin, bacitracin) to keep the corneal surfaces lubricated and reduce surface bacteria (see Box 16.1). Prep and drape as previously described (p. 266).

NOTE Identification of tissue layers and accurate placement of sutures with conjunctiva are facilitated by use of magnifying loupes. Surgical loupes and appropriately high magnification are necessary for any procedures near the cornea.

ANESTHETIC CONSIDERATIONS

Conjunctival surgery and surgery near the cornea require precision and delicacy; therefore general anesthesia is necessary (see pp. 266–268). In addition, animals with conjunctival or corneal disease can have fragile eyes, thus minimizing head movements and thrashing is necessary to avoid additional eye trauma.

Many brachycephalic breeds have shallow orbits, and prominent eyes and are particularly susceptible to ocular disease. These animals also tend to have elongated soft palates, small tracheas, and they may have chronic airway disease; thus diligent monitoring and support are necessary before, during, and after anesthesia. Premedications may be administered both to calm and relax them and to avoid the oculocardiac reflex that can cause severe bradycardia or cardiac sinus arrest (e.g., glycopyrrolate or atropine; see p. 266 and Table 16.1).

Propofol is an excellent option for induction because of its rapid, titratable effect and rapid elimination. Alfaxalone may cause less respiratory and cardiac depression than propofol, allowing for a rapid and smooth induction, particularly in brachycephalic dogs, heart patients, and cats (see Table 12.1 and p. 134). Both alfaxalone and propofol may increase intraocular pressure (IOP) in healthy dogs,[7] though this may not be clinically significant. Induction with a face mask may place pressure on

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or near the eyes and should be avoided when possible. Ketamine should also be avoided because it causes undesirable eye movements. Anesthesia should be maintained with inhalants (see Table 16.1); injectable anesthetics alone (unless given as a constant-rate infusion) will not typically last sufficiently long or provide sufficient immobilization for delicate, precise eye surgery (see Chapter 12). Use a cuffed, wire-reinforced, guarded endotracheal tube to prevent airway collapse if the neck is bent to position an eye for surgery. For other anesthetic considerations (fluid therapy, monitoring depth of anesthesia) see Chapter 12.

With eye surgery, neuromuscular blockers (NMBs) and a ventilator may be used to prevent any eye movement. These drugs require specialized equipment and training to use safely. NMBs cause muscle paralysis, including the respiratory muscles, and even low doses are dangerous without ventilation respiratory support. In these cases, measuring $EtCO_2$ is imperative to evaluate anesthesia. Staff need to be educated and experienced with supporting ventilation manually or mechanically in all patients given NMBs, and they should also be skilled in interpreting anesthetic monitors and parameters. Analgesics (see Chapter 13) must be administered to these patients because, although NMBs prevent movement, they do not provide analgesia. The animal must continue to be ventilated until the NMB agents have worn off as determined with a nerve stimulator.

Whenever possible, a smooth recovery that minimizes excessive head motion is ideal, especially if the eye is fragile from a deep corneal ulcer. Extubation should be performed after the animal can maintain its own oxygenation. Brachycephalic animals should be extubated as late as possible and monitored carefully for a few hours after anesthesia. Postoperative analgesics (e.g., opioids; see Chapter 13) will also help smooth the recovery.

ANTIBIOTICS

Perioperative and postoperative antibiotics are indicated prophylactically with conjunctival and cornea surgery. Topical medications that can penetrate through the cornea will reach the anterior chamber of the eye, whereas systemic antibiotics are needed to reach the posterior segment.

NOTE Avoid using topical ointments on deep corneal ulcers or perforations because ointments inside the eye cause uveitis.

SURGICAL ANATOMY

The special mucous membrane on the inner aspect of the lids is called the *palpebral conjunctiva* (see Figs. 16.1 and 16.15). At the level of the orbital rim, the palpebral conjunctiva reflects at the conjunctival *fornix* onto the eye to become the *bulbar conjunctiva.* The palpebral conjunctiva has goblet cells, whereas the bulbar conjunctiva is thinner and is without goblet cells. Conjunctival goblet cells secrete mucin that makes up the innermost layer of the tear film and adheres the tear film to the ocular surfaces. Lymphatic nodules are found throughout the conjunctiva but are especially prominent on the bulbar surface of the third eyelid. Lymphatic drainage from the conjunctiva empties into the parotid lymph nodes. The *third eyelid* is located ventromedially, is covered with conjunctiva, and is mobile enough to cover the entire anterior face of the cornea. A T-shaped piece of hyaline cartilage stiffens the third eyelid (Fig. 16.14). The *third eyelid gland*, a mixed seromucous gland that contributes the middle aqueous layer of tear film, surrounds the base of the cartilage and is normally tucked in the orbit out of view. The third eyelid is important in protecting the cornea and spreading the tear film. The wall of the globe is comprised of the transparent, multilayered cornea anteriorly and the yellow-white, opaque *sclera* posteriorly (Fig. 16.15). The two meet at the corneoscleral junction or limbus. The *cornea* is usually less than 1 mm thick and is composed of four layers: (1) epithelium with its basement membrane, (2) stroma, (3) Descemet's membrane (basement membrane of endothelium), and (4) endothelium.

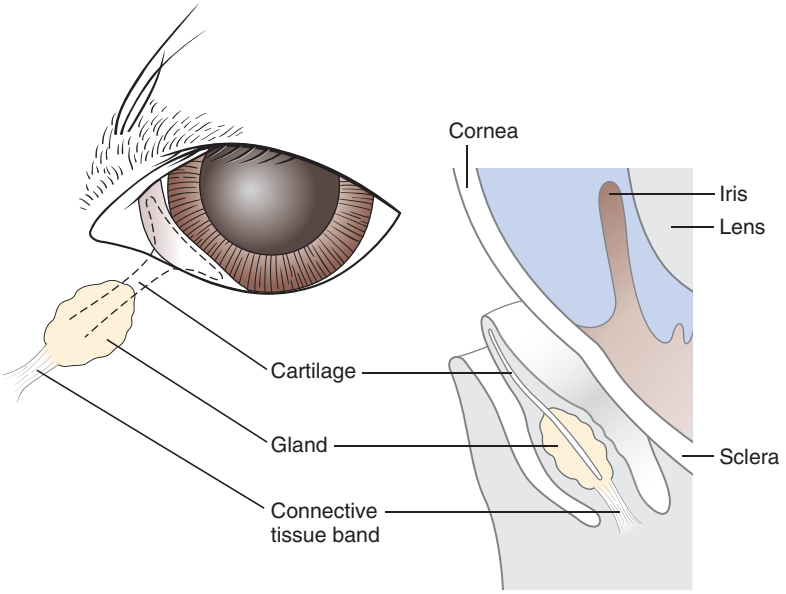


FIG. 16.14 Anatomy of the conjunctiva, third eyelid, and third eyelid gland. (Modified from Severin GA. *Severin's Veterinary Ophthalmology Notes.* 3rd ed. Lakewood, CO: American Animal Hospital Association; 1995.)

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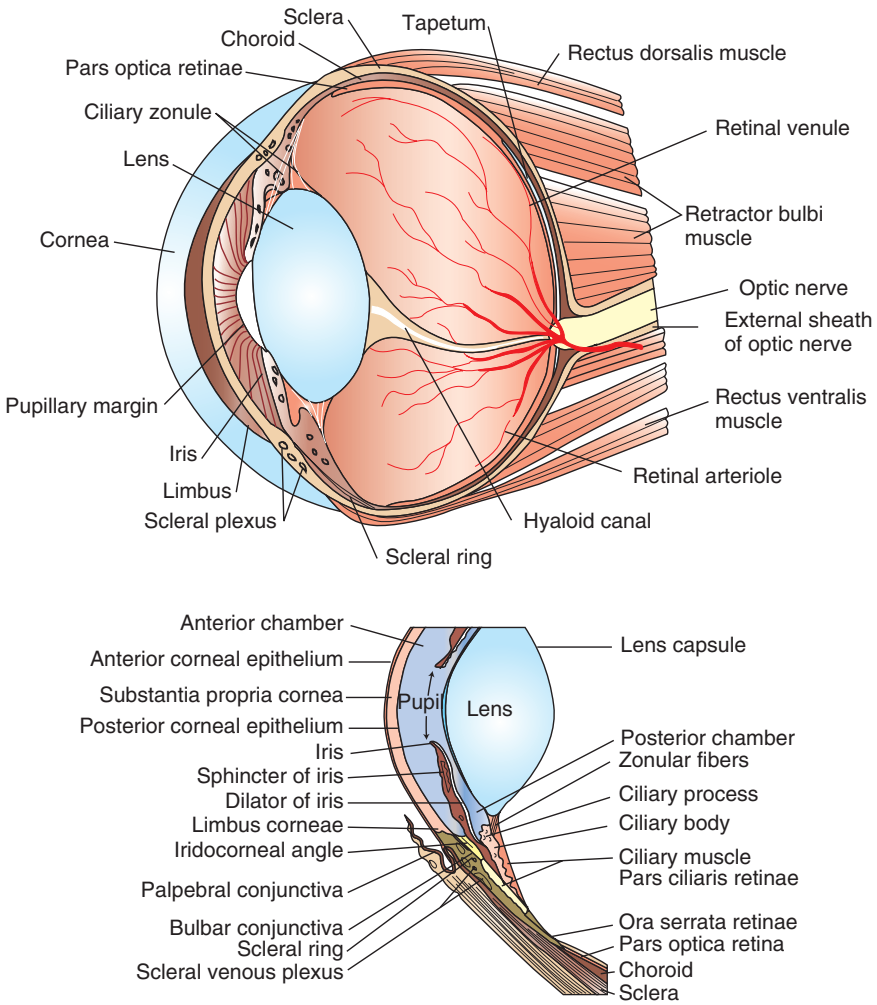


FIG. 16.15 Intraocular anatomy.

SURGICAL TECHNIQUES

Third Eyelid Flap

Third eyelid flaps can serve as bandages (similar to an opaque contact lens) to mechanically protect the cornea after trauma. It is important to note that they do not function like conjunctival grafts because conjunctival grafts provide vascularization, as well as metabolic and tectonic support, to the cornea by integrating into the cornea. Third eyelid flaps should *not* be used on deep corneal ulcers, descemetoceles, or corneal perforations. The raised third eyelid can place pressure on the delicate cornea, increasing the risk of rupture. Third eyelid elevation also prevents any direct monitoring of the cornea during treatment. Indications for a third eyelid flap include bullous keratopathy and indolent corneal ulceration where the corneal epithelium is not adhering well to the underlying corneal stroma.

Exteriorize and extend the third eyelid by grasping it with forceps and elevating it. Pass a cutting needle with swaged-on 3-0 or 4-0 suture monofilament absorbable, such as PDS, through the upper eyelid into the dorsolateral conjunctival fornix (Fig. 16.16A). Direct the needle through the external surface of the third eyelid under the crossbar of the T-shaped cartilage, and exit through the external surface of the third eyelid on the opposite side of the cartilage. Alternatively, direct the suture around the cartilage but do not penetrate the conjunctiva on the bulbar surface of the third eyelid. Direct the suture back through the dorsolateral conjunctival fornix and out the upper eyelid (see Fig. 16.16B). Apply tension on the suture, pulling the third eyelid over the cornea. Tie the suture and use a stent (sterile button or sterile piece of tubing), if needed, to distribute suture knot pressure on the skin (see Fig. 16.16C).

Remove the third eyelid flap sutures 14 days after placement, or sooner if the suture loosens. Complications of third eyelid flaps include eyelid or skin necrosis, corneal irritation from suture contact, cartilage deformation, and persistent third eyelid protrusion following release. Eyelid swelling is expected. Because the cornea is covered by the third eyelid flap, the corneal disease could worsen without being evident, so the patient should be monitored closely for increased ocular pain.

HEALING OF THE CONJUNCTIVA, THIRD EYELID, AND GLAND

Conjunctiva heals rapidly due to its extensive vascularization.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Magnification of ×2 to ×3 using a lighted MagniVisor, Optivisor, or surgical loupe is recommended when operating on the

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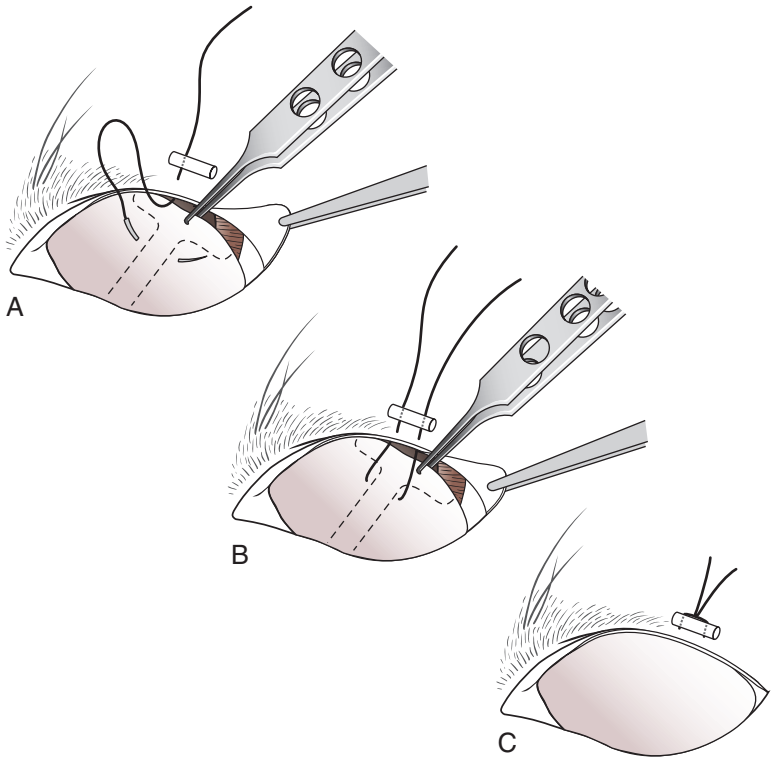


FIG. 16.16 Third eyelid flap. (A) Pass a cutting needle through the upper eyelid in the dorsolateral conjunctival fornix. Exteriorize and extend the third eyelid with forceps and direct the needle through the external surface of the third eyelid, around the cartilage cross-bar but keeping the suture buried so it does not penetrate the posterior surface of the third eyelid. (B) Direct the suture through the dorsolateral conjunctival fornix and out the upper eyelid. (C) Tie the suture in a simple interrupted tightly. Use a stent if needed to distribute pressure.

conjunctiva or near the cornea. Consider using a focal light source with loupes because lighting assists magnification. For suturing conjunctiva, an absorbable small-gauge soft suture such as 6-0 polyglactin 910 with a reverse cutting ¼ curvature swaged-on needle (see Box 16.2) is recommended. To handle such small sutures, microsurgical forceps, needle holders, and scissors should be used because these instruments are specially designed to be delicate with fine tips and unique curves for specific purposes (see Fig. 16.3). Microsurgical techniques should be applied with 6-0 sutures or smaller to avoid damaging both sutures and fragile instruments (i.e., grasp the needle with only needle holders because grasping the needle with tying forceps will damage the forceps; handle suture with only microsurgical instruments and not hands to avoid damaging the suture). An eyelid speculum to hold the eyelids open for surgery on the conjunctiva or third eyelid is recommended (see Fig. 16.3).

POSTOPERATIVE CARE AND ASSESSMENT

The small, delicate sutures placed in the conjunctiva or third eyelid are easily torn or disrupted by the patient. Use a rigid E-collar as discussed on p. 271 that extends just beyond the nose when the head is extended. Soft E-collars and neck-only restraints (e.g., Bite Not collar) will not prevent the animal from rubbing the eye against objects. Topical antibiotics (see Box 16.1) should be administered until the eye is healed.

COMPLICATIONS

Complications of conjunctival and third eyelid surgeries are primarily hemorrhage and dehiscence. Conjunctiva is extremely vascular so infection is rare, but the delicate tissue can tear easily.

SPECIAL AGE CONSIDERATIONS

Cherry eye usually starts at a young age but may not be noticed until later in life. Corneal ulcers can occur at any age.

SPECIFIC DISEASES

THIRD EYELID GLAND PROLAPSE (CHERRY EYE)

DEFINITIONS

Cherry eye is defined as a prolapsed third eyelid gland. **Chemosis** is conjunctival edema resulting in visible swelling.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Prolapse and protrusion of the third eyelid gland are most likely caused by laxity in the attachments between the third eyelid gland and periorbita. Prolapsed third eyelid glands usually occur

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in the first few years of life as a primary condition with genetic risk factors. Trauma can also cause a third eyelid gland prolapse. Uncommon contributing factors may be primary or secondary adenitis, breed-related fascial attachment abnormalities, or specific pathogens affecting the glands. The condition is not usually caused by primary inflammation, neoplasia, or hyperplasia. KCS occurring after protrusion (sometimes years later) suggests involvement of both the lacrimal and third eyelid glands. The hypertrophied, protruding gland, which extends beyond the leading edge of the third eyelid, can become abraded and dry, resulting in secondary inflammation and swelling. Protrusion may be unilateral but is more frequently bilateral. Adenitis is found on histologic examination.

DIAGNOSIS

Clinical Presentation

Signalment

Breeds most often affected are cocker spaniels, English bulldogs, beagles, Pekingese, Boston terriers, basset hounds, shih tzus, and Lhasa Apsos. Most are first affected at a young age (usually <1 year). The condition is more common in dogs than in cats; the Burmese is the most commonly affected cat breed.

History

Owners may notice a sudden third eyelid mass, ocular discharge (serous or mucoid), and/or conjunctivitis. The condition usually begins unilaterally but eventually may become bilateral. Prolapse may be intermittent.

Physical Examination Findings

If protruding, the prolapsed third eyelid gland will look like an obvious round to oval, pink, fleshy mass behind the third eyelid margin near the medial canthus. Lymphoid follicles on the anterior surface of the protruded gland may be seen. If not visible, elevating the third eyelid by gently retropulsing the globe will show a distinct oval pink mass on the palpebral surface of the third eyelid. Ophthalmic exam may show conjunctivitis and mucoid discharge. If KCS is also present, the cornea may have vascularization and/or pigmentation (pigmentary keratitis). Sometimes the third eyelid gland is abraded or has cysts.

Laboratory Findings

Culture of the mucopurulent discharge is not indicated. Cytology of the protruding gland will reveal normal conjunctival epithelial cells and possibly inflammation and glandular cells and is also generally not indicated.

DIFFERENTIAL DIAGNOSIS

Differentials include third eyelid neoplasia, adenitis, hyperplastic lymphoid follicles, elevated third eyelid (Horner syndrome, Haws syndrome, enophthalmos), cyst, everted third eyelid cartilage, and malformation of the third eyelid.

MEDICAL MANAGEMENT

If the cornea is fluorescein negative and the gland is swollen, topical antibiotics with corticosteroids can be used to reduce the swelling and size of the prolapsed third eyelid gland so that it becomes smaller, although this will generally not resolve the prolapse.

SURGICAL TREATMENT

Preoperative Management

Clipping the periocular hair is not required. Remove exudates from the corneal and conjunctival surfaces with sterile gauze and cotton swabs moistened by eye wash or lactated Ringer's solution (LRS). Keep the cornea lubricated.

Anesthesia

See pp. 266 and 281 for anesthetic considerations in young animals.

Surgical Anatomy

The third eyelid is a roughly triangular fold of mucosa located at the medial canthus and covered by conjunctiva (see Figs. 16.1 and 16.14). The base of the triangle is the free or leading margin. A T-shaped piece of cartilage lies within the third eyelid providing internal structure to it, and the "arms" of the T extend along the leading margin. The third eyelid gland is typically within the bulbar conjunctiva at the base of the third eyelid. Lymphoid follicles found primarily on the bulbar surface of the third eyelid appear as small raised translucent bumps. Blood supply to the third eyelid comes from branches of the internal maxillary artery. Lacrimal excretory ductules leave the gland and emerge in the middle section of the bulbar mucosal surface.

Positioning

Position the animal in ventral recumbency with a rolled towel placed under the chin to hold the head level.

SURGICAL TECHNIQUES

Replacement, not removal, of the prolapsed gland is the standard of care and is recommended to reduce the incidence of KCS later in life. Goals of surgical treatment include replacing the protruding gland back into its normal anatomic position on the bulbar third eyelid, maintaining third eyelid mobility, and preserving glandular tissue and excretory ducts. Described techniques include creating an envelope or pocket in adjacent conjunctiva and covering it, with or without scarification of the gland surface. In addition to pocket procedures, anchoring the third eyelid to the base of the third eyelid cartilage,[8] extraocular muscles,[9] sclera, periorbital fascia, or periorbital bony rim[10] have been described. Anchoring procedures interfere with third eyelid mobility, whereas pocket procedures may damage excretory ducts. Combined pocket and tacking procedures may be more successful for larger and more chronic protrusions than pocket procedures alone. Pocket procedures without tacking may be more effective in young animals and those with mild protrusions.

Pocket procedure: *Place an eyelid speculum to maximize exposure. Grasp and extend the third eyelid with two small towel clamps or mosquito forceps placed on either side of the third eyelid gland. Make a linear conjunctival incision between the protruded gland and cornea (i.e., proximal to the gland), taking care to retain at least 5 to 7 mm of conjunctiva at the limbus (Fig. 16.17A). Deepen the incision until prominent conjunctival blood vessels are visible. Make a second parallel incision into the conjunctiva between the third eyelid margin and gland (i.e., distal to the gland), taking care to retain at least 4 to 5 mm of conjunctiva along the third eyelid margin (see Fig. 16.17A). At one end of the incision, place a suture knot of 6-0 polyglactin 910 on the external palpebral surface of the third eyelid with tying forceps (e.g., Colibri) and nonlocking Castroviejo needle holders or the equivalent (see Box 16.2). Pass the needle through the third eyelid*

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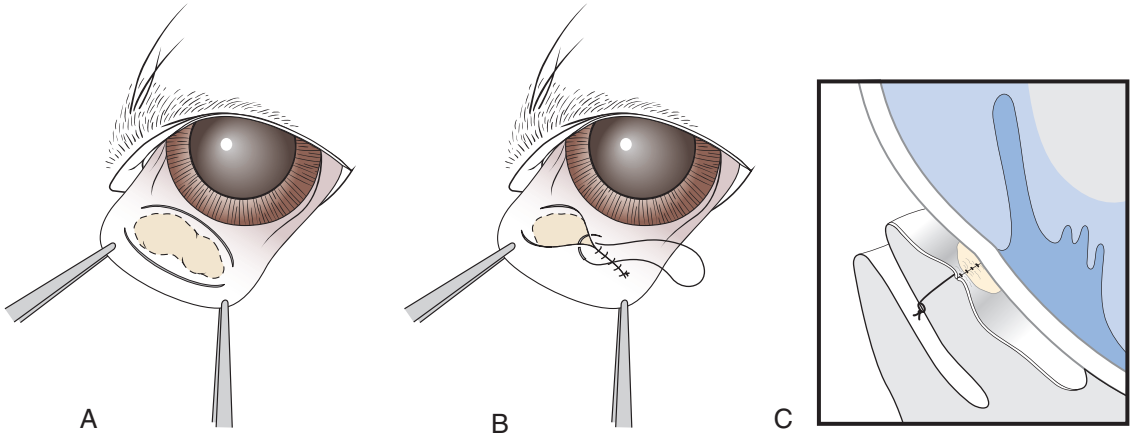


FIG. 16.17 Pocket procedure for cherry eye. (A) Make a linear conjunctival incision on either side of the protruded gland, taking care to retain a least 4 to 5 mm of conjunctival tissue on either side for suturing. (B) Anchor the first knot of 6-0 polyglactin 910 partial thickness on the anterior surface of the third eyelid, and then pass the suture through the third eyelid into the incision. Close the incision in a simple interrupted pattern taking bites on either side of the gland, while pushing the gland into the resulting pocket, keeping the suture snug and unexposed. (C) Pass the suture back through the third eyelid and anchor the knot on the anterior third eyelid surface.

from the palpebral to the bulbar surface of the third eyelid so the needle exits inside one end of one of the conjunctival incisions. Tuck the gland into its normal position by suturing (imbricating) the two incisions together over the gland in a simple continuous pattern with 6-0 polyglactin 910 (see Fig. 16.17B). Apply downward pressure to the gland with a cotton-tipped applicator as the suture is tied, tucking or "pocketing" it into the bulbar conjunctiva. Pass this suture back through the third eyelid and place the final bite and knot on the external palpebral surface of the third eyelid (Fig. 16.17C). Take care to avoid corneal contact or tearing the conjunctiva, and avoid grasping the needle with the microsurgical tying forceps because the needle can damage the forceps.

NOTE Removal of the gland of the third eyelid increases the risk of dry eye (KCS) and is contraindicated.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

For smaller dogs and cats, use 6-0 polyglactin 910 and microsurgical forceps, needle holders, and scissors (see Box 16.2). For very large dogs, either 5-0 or 6-0 polyglactin 910 can be used to suture conjunctiva.

POSTOPERATIVE CARE AND ASSESSMENT

Use a rigid E-collar that extends just beyond the nose when the head and neck are extended as discussed previously (see p. 271). Administer a topical antibiotic (e.g., neomycin, polymyxin, bacitracin) four times daily until the conjunctiva is healed in 10 to 14 days (see Box 16.1). Avoid topical corticosteroids for several weeks after surgery because they adversely affect healing. Swollen glands may take several weeks to return to a normal size.

COMPLICATIONS

Complications include suture exposure to the corneal surface causing ulceration, and dehiscence of the incision or holes torn in the conjunctival pocket allowing for repeat prolapse of the third eyelid gland.

PROGNOSIS

The prognosis for gland replacement can be good with the pocket procedure. Warping of the third eyelid margin and visible thickened tissue at the base of the gland may be present after surgery, especially if the gland was very large. KCS is much more likely if the gland is partially or completely removed. Recurrence may require additional surgery to try to replace the gland.

DEEP OR INFECTED CORNEAL ULCERS

DEFINITIONS

A **superficial corneal ulcer** is an injury where only the corneal epithelium has been removed. A **stromal corneal ulcer** involves loss of the corneal stroma in addition to the epithelium. A **descemetocele** is a very deep corneal ulcer in which most or all of the corneal stroma is absent, causing a defect that extends down to Descemet's membrane (the basement membrane of the corneal endothelium). Melting corneal ulcers involve liquefaction of the corneal stroma by protease and collagenase proteolytic enzymes; the enzymes are produced by inflammatory and corneal cells. **Infectious endophthalmitis** is infection inside the eye. **Phthisis bulbi** is an acquired shrunken, atrophied eye resulting from chronic diseases such as corneal perforation or chronic uveitis. **Epithelial downgrowth** occurs when corneal epithelium grows down through a corneal perforation and into the eye.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Corneal ulcers often occur from an injury that removes some corneal epithelium with or without stroma. The trauma can be endogenous from dry eye, poor blinking and exposure, ectopic cilia, distichia, entropion, eyelid masses, facial folds, or excoriations. Exogenous causes of corneal ulcers are physical contact of the cornea against an object. With cats, active feline herpesvirus infection also causes corneal ulceration.

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Uncomplicated superficial injuries to the cornea undergo avascular healing. An uncomplicated cornea ulcer is quickly covered by sliding epithelium. When the epithelium is absent from the cornea, hydration of the corneal stromal can occur, causing focal corneal edema in the ulcer bed. With healing, epithelial cells lose cilia, flatten, and begin migration over a corneal wound defect within 1 hour. The entire cornea can be reepithelialized in 7 to 10 days. Once the cells have covered the defect, mitosis occurs and the multilayered epithelial surface is reconstituted. Attachment of the epithelium to the underlying tissues may be fragile for 6 to 8 weeks. Indolent ulcers occur when the epithelium does not adhere to the underlying stroma and typically appear as a superficial corneal ulcer with loose epithelium. Superficial indolent ulcers (and no other kinds of ulcer) should be treated with corneal epithelial debridement and a grid, punctate, or diamond burr keratotomy.

Bacterial and fungal organisms can make up the normal flora of the conjunctival and corneal surfaces. Opportunistic organisms may infect an open corneal wound and cause progressive corneal stromal loss. In infected or destructive stromal corneal ulcers, vascular healing is necessary. Because the cornea is naturally avascular, the immune system has limited access to a corneal injury until neovascularization develops. In response to the infected corneal ulcer, corneal neovascularization will start growing from the limbus after 3 to 5 days and typically grows at a rate of approximately 1 mm per day. The length of corneal vascularization can sometimes provide an estimate of how long the infected corneal ulcer has been present. Corneal vascularization usually takes approximately 4 weeks to reach the center of the cornea. Once corneal vascularization reaches an infected ulcer site, the immune system will eliminate the infection and the corneal tissue can rebuild through granulation. Collagen fibers in the regenerating stroma are irregular and thus decrease corneal transparency. Scar density decreases over time, but a focal white corneal scar (leukoma) typically remains. If a deep corneal ulcer does not vascularize, the ulcer bed will remain as a thin area of cornea that looks "indented" or flat when viewed from the anterior surface and may be more susceptible to future injury or infection.

The cornea contains extensive innervation, and a corneal ulcer will cause reflex anterior uveitis. Uveitis is a breakdown of the blood-eye barrier inside the eye that causes aqueous flare (protein leakage into the aqueous humor), as well as miosis, diffuse corneal edema, and reduced IOP (in the early phase). The severity of the uveitis is proportional to the severity of the corneal infection. As the corneal infection responds to therapy, the reflex uveitis will improve. Monitoring the uveitis allows evaluation of how the corneal ulcer is responding to treatment. If the uveitis worsens despite treatment, the corneal infection may be antibiotic resistant, so therapy should be escalated. Release of various enzymes (collagenases, proteases) from corneal cells and leukocytes can cause corneal stromal melting and liquefaction.

If a corneal perforation occurs, the corneal endothelium will be damaged as well. Infected corneal ulcer pathogens have access into the eye, and intraocular anatomy will be distorted. The body will often place a fragile plug of fibrin, iris, and/or blood in the perforation site. *Anterior synechiae* (adhesion of iris to the cornea) usually develop, and eventually epithelial downgrowth and adhesion formation inside the eye can lead to glaucoma, pain, and blindness. Blood at the ulcer bed when corneal vascularization has not yet reached the site is usually from a corneal perforation.

DIAGNOSIS

Clinical Presentation

Signalment

Any animal can acquire an infected or deep corneal ulcer. Animals with poor ocular surface health (e.g., KCS, lagophthalmos) may be predisposed to ulceration. Brachycephalic animals are predisposed to ulceration due to corneal exposure. Animals with a compromised immune system (i.e., cats with feline leukemia virus [FeLV] or feline immunodeficiency virus [FIV]; dogs with hyperadrenocorticism) or those treated with immunosuppressive drugs are more likely to develop an infected corneal ulcer.

History

Symptoms observed are usually eye discharge, squinting, holding the eye closed, and/or rubbing the eye. Animals usually present within days of the eye problem first being noted, although some owners will try various topical eye drops before presentation; it is therefore important to obtain a careful history. Previous medical therapy, such as the use of corticosteroids on a corneal ulcer, can also be essential to the history and medical management.

Physical Examination Findings

Symptoms of an infected corneal ulcer are listed in Box 16.3.

Descemetoceles usually look like a dent or crater on the cornea that is clear at the bottom although surrounded by cloudy corneal edema. In contrast, corneal perforations have a raised, opaque, white to brown focal bulge in an area that was previously ulcerated. The central bulge represents protruding fibrin and/or iris. The pupil may be distorted or not visible and the anterior chamber may be shallow.

Laboratory Findings

No change in white cell count is expected unless the infection from a ruptured eye is beginning to ascend or spread systemically. Any contributing health problems that may lower immunity should be evaluated because these diseases adversely affect corneal healing.

DIFFERENTIAL DIAGNOSIS

Other causes of ocular pain include ectopic cilia, severe distichia, dry eye, entropion, and an eyelid mass. Other ocular diseases, such as anterior uveitis and glaucoma, can also cause diffuse

! BOX 16.3 Symptoms of an Infected Corneal Ulcer

- Inappropriately severe anterior uveitis as noted by:
  - Diffuse corneal edema
  - White blood cells *(hypopyon),* red blood cells *(hyphema),* or fibrin in the anterior chamber
  - An extremely miotic pupil
  - Aqueous flare ("flashlight in fog") caused by protein in the aqueous humor
  - Significantly reduced intraocular pressure especially when compared to the normal eye
- Ulcer edges with an opaque white or yellow color (presence of white blood cells)
- Visible "denting" or progressive deepening of the corneal ulcer bed
- Melting or liquefaction of the corneal ulcer tissue
- Significant, diffuse corneal edema associated with the ulcer

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corneal edema. If a corneal dent/defect is visible with a comfortable eye and negative fluorescein stain, a previous stromal ulcer may have occurred in the past, leaving an epithelialized thin area of cornea (a corneal facet).

MEDICAL MANAGEMENT

Simple uninfected corneal ulcers are typically treated prophylactically with topical antibiotics, and most heal rapidly without complications. Corneal ulcers should not be treated with systemic or topical corticosteroids because they suppress the local immune system and potentiate corneal ulcer infection. Topical NSAIDs are also not generally advisable. Infected corneal ulcers, descemetoceles, and corneal perforations should ideally be referred to a veterinary ophthalmologist. Apply a rigid E-collar as described on p. 271.

Deepening of infected corneal ulcers can occur rapidly (i.e., over a period of several hours). Medical therapy can be pursued until the animal can be seen by an ophthalmologist or if the owner declines referral. Some corneal ulcers can be managed medically, but severe corneal ulcers may require surgery to save the eye.

Before starting antibiotic therapy, a culture and sensitivity of the ulcer bed, while possibly risky, may help direct medical therapy. Antibiotic resistance has increased, making sensitivity results nearly essential to successful treatment of a corneal ulcer. *After adequate topical anesthesia and immobilization, carefully swab the ulcer edges for culture and sensitivity with a sterile cotton-tipped applicator that is then placed in blood culture broth or a culturette. Because the sample size will be small, ask the microbiology laboratory to start the culture in enrichment broth to amplify the organisms before plating.*

Use topical antibiotics for corneal ulcers because the cornea has no inherent blood supply, which prevents systemically administrated antibiotics from reaching adequate concentrations. Bacterial numbers can double as rapidly as every 30 minutes, so frequent treatment is essential to try to control an active infection. Diligent treatment with antibiotic drops every 1 to 2 hours around the clock for at least 24 to 48 hours can interrupt the bacterial growth cycle. While awaiting culture results, broad-spectrum antibiotics should be administered or selected based on a Gram stain (see Box 16.1). For gram-negative bacteria, use topical antibiotics such as fortified 0.9% gentamicin (add 0.33 mL of 100 mg/mL IV gentamicin to every 5 mL of 0.3% gentamicin) or a second-generation fluoroquinolone (e.g., ofloxacin; see Box 16.1). For gram-positive organisms, use topical antibiotics such as fourth-generation fluoroquinolones (e.g., moxifloxacin or 50 mg/mL cefazolin [add LRS to the IV vial to make 100 mg/mL then dilute 1:1 by removing 5 mL of cefazolin solution and adding 5 mL of LRS to make 5% or 50 mg/mL]; dilution can also be made in a sterile dropper bottle using artificial tears solution). Cefazolin needs to be refrigerated in the same way as the IV solution to maintain its efficacy, and it expires similarly to the IV solution. After the first 24 to 48 hours, reduce the topical antibiotic frequency to every 4 to 6 hours depending on the response to treatment. If a corneal ulcer is treated too frequently in the long-term, corneal neovascularization will slow or stop such that blood vessels may not reach the ulcer site. A reduced frequency of treatments encourages the corneal ulcer to vascularize. As the treatment frequency is decreased, corneal infection may worsen; thus topical treatments should be balanced in frequency to encourage neovascularization while also trying to prevent progression of the corneal infection. If the corneal infection progresses, the ulcer will deepen and the anterior uveitis will worsen. Topical NSAIDs should be avoided with corneal ulcers because they can slow corneal ulcer healing and some (i.e., diclofenac) may even cause sterile corneal melting.[11] Continue medical treatment until the corneal ulcer has either filled completely with vascularization or the vascularization stops progressing. Systemic antibiotics are indicated if the corneal ulcer has ruptured because this is associated with a risk of internal eye infection.

Symptomatic treatment of secondary anterior uveitis is also essential to ulcer management because uveitis can lead to internal iris adhesions that can cause glaucoma. Glaucoma is progressive over time and causes pain and blindness. Atropine administered topically reduces painful ciliary muscle spasm and minimizes adhesions of iris to lens *(posterior synechiae)* by causing pupil dilation, although atropine usage may increase the risk of glaucoma in an eye that is anatomically predisposed to this condition.

Positive indicators of control of corneal infection are reduced pain, reduced corneal edema, reduced aqueous flare and anterior uveitis, and more effective or longer lasting pupil dilation from atropine as the uveitis decreases. Negative indicators are increased pain, increased corneal edema, increased anterior uveitis, and deepening of the corneal ulcer. If the ulcer is worsening, additional antibiotics or surgery may be needed.

Melting corneal ulcers are very unstable because the liquefied tissue is physically very soft and can slough from the eye. In addition, melting corneas are not strong tissue for suture placement. Collagenase inhibitors such as 10% acetylcysteine, serum that contains macroglobulins, or ethylenediaminetetraacetic acid will help stabilize melting as the ulcer is treated. These cases are best referred to an ophthalmologist and may need to have a conjunctival graft placed to try to save the eye.[12]

If a corneal ulcer ruptures, the anterior chamber depth (spacing between the cornea and iris) may become narrow, and anterior synechiae, pupil distortion, hyphema, and other anatomic abnormalities may be seen. These patients should be referred to an ophthalmologist to best evaluate the prognosis and determine appropriate therapy to save the eye, or determine if enucleation is indicated. If the owners decline referral, medical therapy may be considered for nonleaking corneal perforations less than 5 mm in size (see Seidel test on p. 289). Medical therapy is the same as for an infected corneal ulcer. Deep but small nonleaking corneal ruptures might vascularize and heal, but the prognosis is generally very guarded to poor for saving the eye or vision. Rerupture and further intraocular anatomic derangement, as well as glaucoma, blindness, and ongoing pain, are risks in such cases. Enucleation may be indicated for large, leaking corneal ruptures with extensive intraocular anatomic derangement, extensive hyphema, and/or active infection. If corneal infection progresses to internal eye infection *(endophthalmitis),* the eye will become very painful and require enucleation. If the infection spreads into the orbit and the infection is not contained to the orbit by enucleation, the infection could ascend into the cranium through the optic canal and become life-threatening.

SURGICAL TREATMENT

Surgery for infected corneal ulcers is indicated when the ulcer is very deep and/or large, the ulcer and uveitis are worsening despite aggressive medical therapy, and/or when corneal blood vessel growth is inadequate. Such ulcers are generally best treated by referral to an ophthalmologist.

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Preoperative Management

Use a wire-reinforced, guarded endotracheal tube to prevent airway collapse when the neck is bent to position the eye for surgery.

*Perform a **Seidel test** to determine if a corneal ulcer has reached full-thickness and ruptured. Fill a 3-mL syringe with sterile eye wash or LRS after removing the plunger. Dip the stained tip of the fluorescein strip into the solution repeatedly until the solution is a dark, dense yellow. Have an assistant drip the fluorescein solution onto the cornea ulcer site while you watch the area with magnification and a cobalt light. Alternatively, moisten the tip of a fresh sterile fluorescein strip with a full drop of sterile eye wash, and allow this drop to fall onto the top of the globe; monitor the site in question under magnification with a cobalt light. Observe to see whether a clear rivulet of aqueous humor is seen running through the fluorescein stain.* A positive Seidel test indicates aqueous humor leakage and that the corneal ulcer has ruptured. If a corneal perforation is leaking, referral to an ophthalmologist is indicated because accurate evaluation and repair requires use of specialized skills and an operating microscope.

NOTE Corneal perforations and conjunctival grafts needing corneal sutures require an operating microscope with specialized microsurgical techniques and are best referred to a veterinary ophthalmologist.

Anesthesia

See pp. 266 and 281.

Surgical Anatomy

The white part of the eye has three layers: the *conjunctiva*, *episclera,* and *sclera* proper (collagen fibers and fibroblasts), which make up the structural wall of the eye (Fig. 16.18). The episclera and sclera are covered anteriorly by the conjunctiva; posteriorly, muscles insert around vessels and nerves that penetrate the globe surface. The conjunctiva is a thin, vascularized tissue that has epithelium on the external surface. The underside of the conjunctiva is nonepithelialized and thus used in graft surgery.

Positioning

Position the animal in dorsal recumbency with the eye directed straight up (iris parallel to the floor). Sand bags with vacuum (e.g., Olympic Vac-Pac) will assist with head positioning. Be sure the endotracheal tube is guarded with wire reinforcement. The surgeon may prefer to be seated for surgery.

SURGICAL TECHNIQUES

Conjunctival Flap

AP Conjunctival flaps are typically harvested from the perilimbal bulbar conjunctiva and are placed over corneal ulcers. A conjunctival graft provides blood supply to the ulcer and bypasses the slower healing that occurs with endogenous corneal vascularization. Although a graft may block vision in the healed eye and surgery generally leaves more scarring than medical therapy, grafts are much more likely to save an eye with a severe ulcer than is medical treatment. The conjunctiva is a thin, elastic tissue with extensive blood supply that also delivers systemic antibiotics to an ulcer. The conjunctival tissue provides strength to the ulcer bed and replaces the tissue lost to corneal infection when it integrates into the corneal tissue.

Smaller (pedicle or rotational) grafts are preferable in almost all cases where a conjunctival graft is indicated. However, these require an operating microscope and are generally best done by an ophthalmologist or a surgeon well versed on microsurgical techniques. Examples of larger grafts that block more of the cornea, but do not require corneal suture placement, are detailed here. Of the two grafts described below, the hood graft is more appropriate for a small and/or peripheral lesion. The 360-degree graft is more appropriate for a large and/or central corneal lesion, although it will cause complete corneal opacity. In addition to potentially causing blindness, the 360-degree graft also prevents any visualization of intraocular structures and cannot be evaluated for healing or complications. These grafts may be able to be trimmed of nonadherent attachments at a later date, to try to expose the cornea, though blindness may still result.

Plan the conjunctival graft or flap so it will be several millimeters larger than the defect it must cover; conjunctiva will contract after it is cut so the graft will become smaller after harvesting. Prepare a thin, sliding conjunctival flap by grasping the conjunctiva with ophthalmic tying forceps such as 0.3 mm Colibri forceps (see Box 16.2 and Fig. 16.3) approximately 2 mm from the limbus. Apply traction to tent the conjunctiva, and incise a few millimeters from the limbus with a pair of Stevens or Wescott tenotomy scissors (Fig. 16.19A). Elevate the cut edge, bluntly dissecting under the

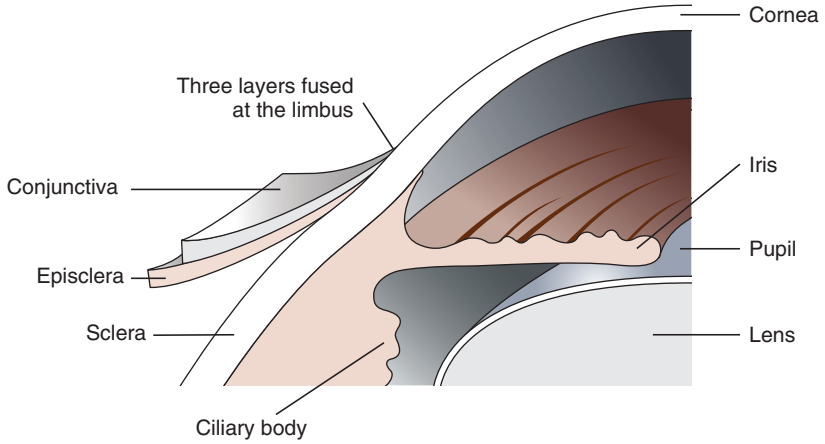


FIG. 16.18 Anatomy of conjunctiva, episclera, and sclera.

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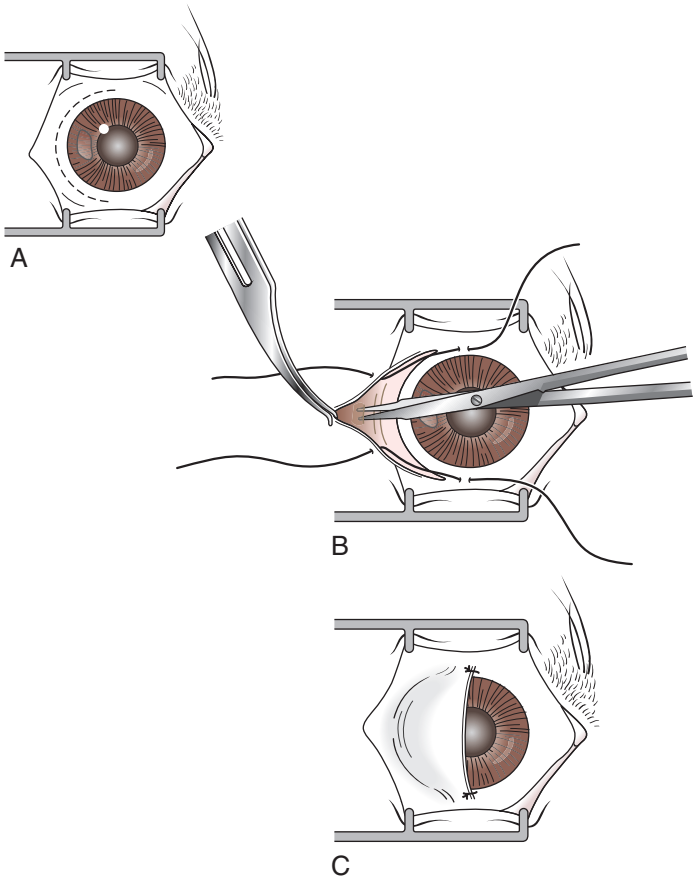


FIG. 16.19 Hood flap. (A) Incise the bulbar conjunctiva adjacent to the lesion parallel to the limbus, and undermine it. (B) Carefully, bluntly dissect the conjunctiva from the fibrous episcleral tissue using tenotomy scissors. The final graft should have no tension. (C) Advance the graft to cover the lesion and suture it in place, usually with two to four simple interrupted sutures into the sclera at the limbus.

conjunctiva and parallel with the limbus, using tenotomy scissors to separate the conjunctiva from the underlying sclera. Starting at the free edge of the tissue flap, use the tenotomy scissor tips directed at a 90-degree angle to the flap and make small cuts with the tips to sharply and bluntly dissect the fibrous episclera from the more superficial thin conjunctiva, taking great care not to make any holes in the conjunctiva (such holes only enlarge with further manipulation and may ruin the elevated graft), yet keeping the graft tissue as thin as possible (Fig. 16.19B). After dissection, the conjunctival flap becomes much more mobile. Position the conjunctival graft over the defect; it should lay over and beyond the ulcer bed on its own with no tension. Carefully debride the corneal epithelium from the corneal ulcer bed and edges with cellulose spears, taking great care not to rupture the ulcer, before placing the conjunctival graft on top so the corneal stroma (and not epithelium) will be in contact with the bleeding underside of the conjunctiva (Fig. 16.19C). Secure the conjunctiva into position so that it can heal and integrate with the corneal stroma. Note that suture should never penetrate the cornea full thickness.

NOTE Stevens or Wescott tenotomy scissors and microsurgical forceps, such as Colibri forceps, are required for this procedure (see Fig. 16.3).

Hood Conjunctival Flap

AP *Dissect a hood flap from the bulbar conjunctiva dorsal to the lesion or from the bulbar conjunctiva as close to the corneal ulcer as possible (see Fig. 16.19A). Dissect a semicircular area of conjunctiva as described previously and place it over the ulcer and at least several mm beyond. Ensure that the conjunctiva lies flat on the cornea with no traction; this will occur if the episclera has been well dissected from the conjunctiva with tenotomy scissors. Place two absorbable sutures (6-0 polyglactin 910; see Box 16.2) in a simple interrupted pattern at the limbus 180 degrees apart to position the flap over the cornea. Provide tension across the free edge of the flap to keep it in place over the corneal ulcer; corneal sutures are not required. Place partial-thickness sutures into the episclera in a simple interrupted pattern with 6-0 polyglactin 910, taking care not to place the sutures full-thickness through the eye wall. If needed, place additional limbal sutures to further anchor the flap (see Fig. 16.19C).*

360-Degree Conjunctival Flap

AP *Starting 2 to 3 mm from the limbus, make an incision in the conjunctiva and use tenotomy scissors under the perilimbal tissue to bluntly dissect the conjunctiva from the globe 360 degrees (Fig. 16.20A). Separate the conjunctiva from the episclera (see Fig. 16.20B) as described earlier. Use caution near the area of the third eyelid when harvesting the donor conjunctiva and make every effort to minimize involvement of the third eyelid into the graft; keep the natural mobility of the third eyelid in mind.*

After the episclera has been adequately dissected from the conjunctiva, the conjunctiva should stretch easily over the corneal ulcer bed with minimal traction.

If the graft has a great deal of tension, continue to dissect the episclera off the conjunctiva, starting at the free edge of the conjunctiva and moving the tenotomy scissors down to the globe, taking care not to cause button-holes in the conjunctiva. After the conjunctival graft has been harvested 360 degrees, suture the free edges of the conjunctival graft together in a line with a horizontal mattress pattern (see Fig. 16.20C–D) using absorbable suture (6-0 polyglactin 910 or smaller). Ensure that the sutures are tight, so that no corneal contact is made by sutures. The conjunctival graft will be sutured to itself and not to the cornea.

It is important to get the undersides of the conjunctival edges apposed with the sutures. The tissues cannot heal together if the conjunctival underside of one side is sutured to the epithelial surface of the other side.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Conjunctiva is a thin, delicate tissue; therefore small sutures such as 6-0 polyglactin 910 should be used (see Box 16.2). Larger sutures and associated suture needles can tear conjunctiva; a large suture can also cause iatrogenic problems if it contacts the cornea. Piercing of the conjunctiva and cornea with the suture needle will create holes that will not seal, so precise needle placement is required (and repeated needle piercings are to be avoided). Forceps not designed for handling conjunctiva can also damage any harvested graft material, and proper instrumentation is critical. Be sure use have Stevens or Wescott tenotomy scissors and microsurgical forceps (see Fig. 16.3).

POSTOPERATIVE CARE AND ASSESSMENT

Eyes with deep corneal ulcers are extremely fragile and susceptible to injury upon recovery from anesthesia; therefore recovery

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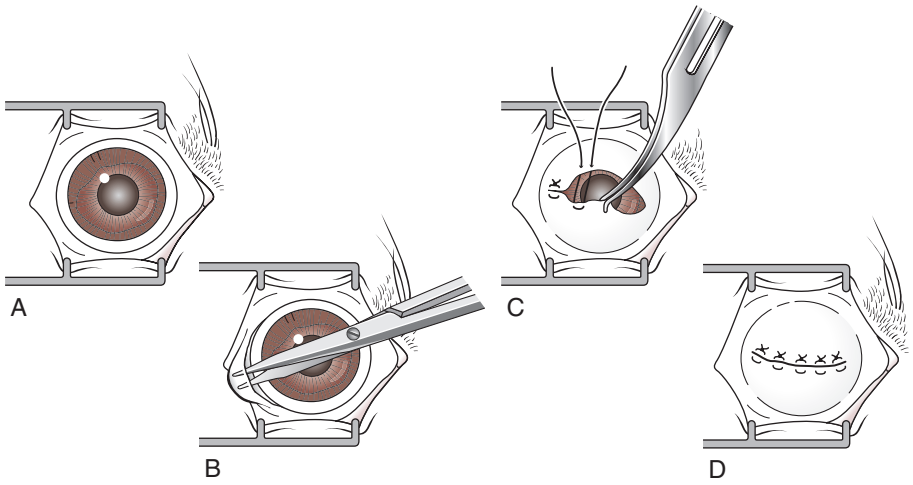


FIG. 16.20 Total or 360-degree conjunctival graft/flap. (A) Start the flap by making a 360-degree conjunctival incision adjacent to the limbus. (B) Undermine the conjunctiva then dissect the episcleral tissues from the conjunctiva using tenotomy scissors and advance it to the center of the cornea. (C–D) Close the conjunctiva by suturing it to itself and not to the cornea in a horizontal mattress pattern.

should be slow, calm, and smooth to prevent head thrashing or self-trauma. Use a rigid E-collar as discussed previously (see p. 271) sized to just beyond the nose when the head is extended (soft E-collars and inflatable neck-only collars will not prevent the animal from rubbing the eye against objects).

Topical antibiotics (see Box 16.1) should be administered until the eye is healed. Analgesics (e.g., opioids) are essential to prevent pain (see Chapter 13). Postoperative sedatives (e.g., acepromazine; see Chapter 12) may be needed to calm the patient. Systemic NSAIDs can be used briefly after surgery because they will help reduce postoperative uveitis, pain, and conjunctival swelling, but they also will slow corneal vascularization and healing. Topical NSAIDs should be avoided with corneal ulceration because some (e.g., diclofenac) can cause corneal melting.[11] The cornea has denser nerve receptors than anywhere else on the body; therefore analgesics like opioids may be prescribed for corneal pain.[13] Use 1% atropine drops once to twice daily to treat secondary uveitis.

Perform an eye exam in a few days and then every 1 to 2 weeks following conjunctival graft surgery. Continue topical antibiotic drops as guided by culture and sensitivity (see Box 16.2) until the conjunctival graft has integrated into the cornea ulcer or the corneal ulcer has completely vascularized. Tissue over the ulcer site should not be removed; it should be left integrated into the cornea. Scar remodeling occurs over several months, and final results vary from an insignificant opacity to a dense leukoma causing significant corneal opacity and vision loss.

COMPLICATIONS

If a corneal ulcer ruptures under the conjunctival graft and intermittent or continuous leaking occurs, the eye should be enucleated. Ruptured corneal ulcers that heal will leave synechiae (adhesions) inside the eye that can later lead to secondary glaucoma. Blindness with phthisis bulbi may also result if there is extensive leaking and/or intraocular damage.

If the conjunctival graft pulls off the corneal or uncovers the ulcer bed, the ulcer still may heal by vascularization. If the conjunctival graft detaches from the cornea and the ulcer ruptures, enucleation will likely be needed. Infectious endophthalmitis can develop even weeks after intraocular contamination and will present with hypopyon and severe pain. These eyes should be enucleated. Conjunctival grafts may fail if the corneal epithelium was not adequately debrided from the ulcer site, if surgical technique was poor, or if local bacterial infection develops. Too much tension on the conjunctival graft may cause premature graft retraction or dehiscence.

PROGNOSIS

With surgical management for infected or deep corneal ulcers, the prognosis for saving the eye is guarded.

Surgeries of the Face and Orbit

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Proptosis is an outward displacement of the eye from its normal position in the orbit. **Exophthalmos** is forward displacement of the globe. **Buphthalmos** is enlargement of the globe from chronic glaucoma. **Enucleation** is removal of the globe, nictitating membrane, orbital glands, and eyelid margins. **Evisceration** is removal of the internal contents of the globe including the iris, uveal tract, lens, and retina. **Intrascleral prosthetic** (ISP) is an eye that has been eviscerated and the internal contents have been replaced with a black round silicone prosthetic (implant).

PREOPERATIVE CONCERNS

If head trauma has occurred, ensure the patient is stable before focusing on the eye problem. Treat any shock or life-threatening problems first.

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PREPARATION OF THE SURGICAL SITE

All members of the surgery staff need to confirm that the correct eye is being prepped and operated on. Antibiotic ophthalmic ointment is applied to both eyes for lubrication and prophylaxis (see Box 16.1). If the eyelid margins are visible, hair around the surgery site should be clipped in a 2-inch (5 cm) wide area with a No. 40 clipper blade. Prep and drape as previously described (see p. 266).

NOTE Procedures requiring surgery inside the eye are best referred to a veterinary ophthalmologist.

ANESTHETIC CONSIDERATIONS

General anesthesia should be performed for globe and orbital surgeries. Globe surgeries are often painful, and perioperative opioids should be considered for analgesia (see Chapter 13). As with other ophthalmic surgeries, consider premedicating patients with glycopyrrolate or atropine to reduce the risk of the oculocardiac reflex that can cause bradycardia (see p. 266); these drugs may need to be repeated intraoperatively.

For enucleation, local adjunctive anesthesia may be considered intraoperatively to improve analgesia (see pp. 152 and 298).[14] Retrobulbar injections of local anesthetics before surgery may increase bleeding during surgery or cause globe perforation if inadvertently injected into the globe. Preoperative retrobulbar injections also risk an intraarterial or intrathecal injection that can cause direct damage to the optic nerve, apnea, or seizures.[15] Systemic toxicosis is unlikely, but maximum doses should be determined and not exceeded in very small animals. Local anesthetics can also be given after the globe is removed and the orbital septum has been closed.

ANTIBIOTICS

Orbital infections can be dangerous due to the proximity of the brain and optic nerve; thus perioperative prophylactic IV antibiotics and postoperative oral antibiotics are indicated (see Chapter 9).

SURGICAL ANATOMY

The orbit contains several secretory tissues that produce tear film fluids. The conjunctival fornix at the intersection of the bulbar and palpebral conjunctiva contains cells that produce mucin, the inner layer of the tear film. The aqueous layer of tears is produced by the lacrimal gland in the dorsolateral orbit and the third eyelid gland on the bulbar side of the base of the third eyelid. The meibomian glands that line the eyelid margins produce an oil that prevents tear film evaporation.

The orbit is the cavity in the skull containing the eye, fat, and the ocular adnexa (Fig. 16.21). The bony orbital margin (frontal, lacrimal, and zygomatic bones) encompasses approximately four-fifths of the orbit circumference; the remainder is completed by the orbital ligament. The orbital ligament is a thick, fibrous band connecting the zygomatic process of the frontal bone with the frontal process of the zygomatic bone. Both the orbicularis oculi muscle and the lateral palpebral ligament attach to the orbital ligament. The medial wall and part of the roof of the orbit are bony (frontal, lacrimal, presphenoid, and palatine bones). Five foramina are present in the medial wall: the optic canal, orbital fissure, lacrimal canal, and two small ethmoidal foramina. The optic canal is rostral to the other foramina in the orbit and is traversed by the optic nerve and the internal ophthalmic artery. The orbital fissure between the basisphenoid and presphenoid bones gives passage to the oculomotor, trochlear, ophthalmic, and abducens nerves; the anastomotic branch of the external ophthalmic artery; and the orbital venous plexus. The retractor bulbi muscle originates in the orbital fissure. The rostromedial orbit houses the lacrimal canal, through which the nasolacrimal duct travels. The *nasolacrimal duct* connects to the two canaliculi, whose two puncta are found at the medial canthus. A fossa for the lacrimal gland is present in the ventral surface of the zygomatic process of the frontal bone, where the orbital ligament originates. The lateral orbital wall and floor are formed by soft tissues: the

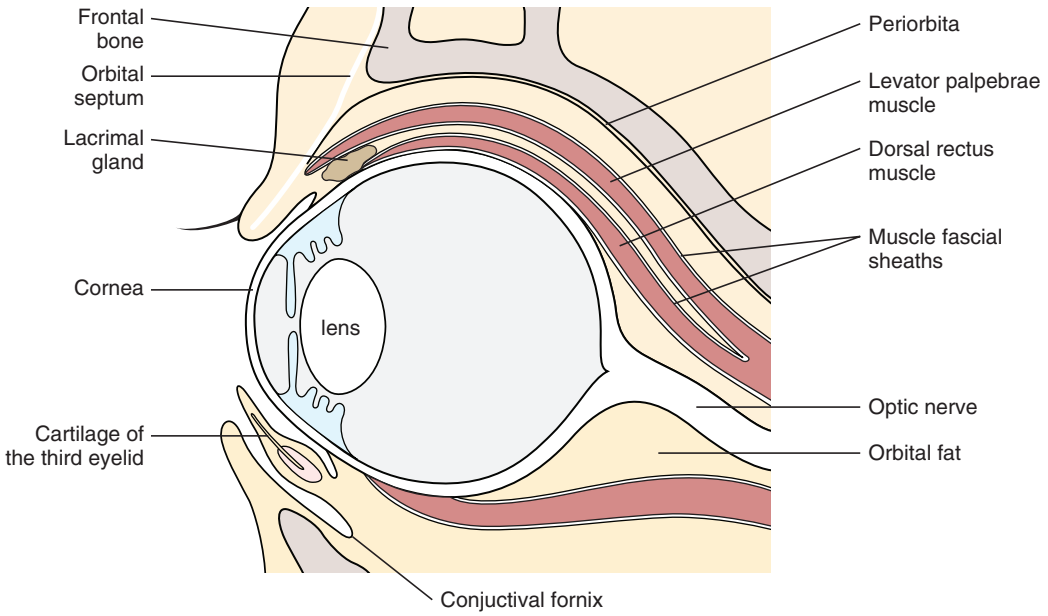


FIG. 16.21 Globe and orbital anatomy.

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medial pterygoid muscle, the temporalis muscle, and the zygomatic gland. The maxillary artery and nerve cross the floor of the orbit near its apex. Orbital fat is present at the caudal pole of the eye, surrounding the optic nerve with spaces between extraocular muscles. This fat serves as a cushion and permits rotation and retraction of the eye. Extraocular muscles insert on the sclera and function to rotate and retract the eye. These muscles include the rectus muscles (dorsal, ventral, medial, and lateral), the oblique muscles (dorsal and ventral), and the retractor bulbi muscle. The orbit, extraocular muscles, and other orbital structures are covered with fascia (periorbital, muscular, and bulbar).

SURGICAL TECHNIQUES

Facial Fold Removal

Facial folds are breed-related skin folds rostral to the eyes on the bridge of the nose that may be large enough to touch the corneal surface. Affected breeds include the pug, Pekingese, and bulldog. Facial fold removal surgery is indicated when they touch the cornea, as they increase the risk for corneal trauma and ulceration. Facial skin infection should ideally be treated and controlled before surgery.

NOTE Warn the owner that removing the facial fold will change the shape and appearance of the face.

Consider the breed in determining how much to reduce or remove the facial fold. Using a No. 15 scalpel blade or Mayo scissors, make parallel incisions along the base of each skin fold or approximately 0.5 to 1 inch (1.3–2.5 cm) above the base of the skin fold to remove the excessive skin (Fig. 16.22A). Align the skin with buried interrupted 3-0 absorbable sutures. Use a simple continuous subcuticular pattern to close the skin incision with 3-0 to 4-0 absorbable intradermal sutures. Close the skin routinely with simple interrupted 3-0 to 2-0 sutures (see Fig. 16.22B). Due to the moisture at the site of the facial folds, continue the oral antibiotics while the skin heals, or for at least 4 weeks if skin infection is present.

HEALING OF THE FACE AND ORBIT

The face and orbital skin heal like other skin. Skin sutures should be tied tightly.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

A typical soft tissue surgery set is used for most of the procedures described.

POSTOPERATIVE CARE AND ASSESSMENT

After surgery, use a rigid E-collar as discussed previously (see p. 271). Antibiotics (see Box 16.1) should be administered until the surgery site is healed.

COMPLICATIONS

Hemorrhage, infection, and incision dehiscence are the major complications of orbital surgeries.

SPECIAL AGE CONSIDERATIONS

Younger animals are more prone to trauma and conformation issues such as facial folds. Glaucoma can be congenital or can affect animals of any age.

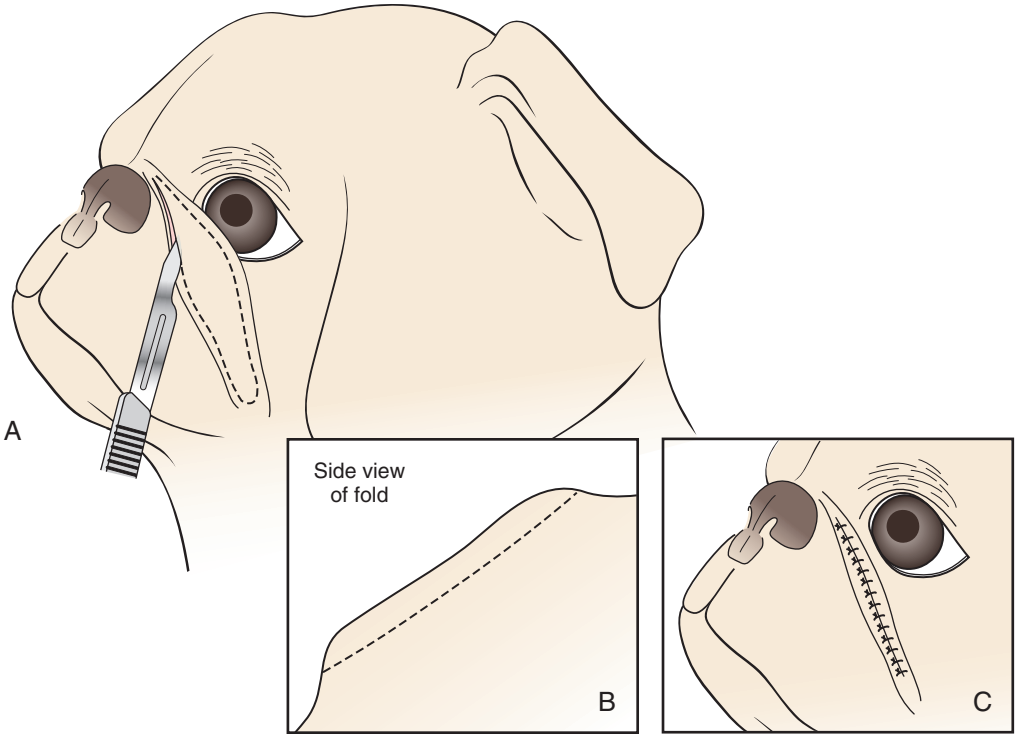


FIG. 16.22 Facial fold removal. (A–B) Start by making parallel skin incisions just above the base of each facial fold to reduce the height of the fold and eliminate corneal contact with the hair and skin. (C) Align and close the subcutaneous layer and then the skin.

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SPECIFIC DISEASES

TRAUMATIC PROPTOSIS

DEFINITIONS

Traumatic proptosis is a forward displacement of the globe from the orbit by a traumatic episode resulting in entrapment of the eyelids behind the eye. **Tarsorrhaphy** is closure of the eyelids.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Blunt head trauma, combined with retrobulbar hemorrhage and/or swelling and orbital fractures, can cause acute forward displacement of the globe beyond the bony orbit and eyelids. Excessive restraint of animals with large palpebral fissures and shallow orbits can also cause globe proptosis. Once displaced, contraction and inward rolling of the eyelids and spasms of the orbicularis oculi muscle prevent return of the prolapsed globe to its normal position. Ocular muscle damage, hemostasis, conjunctival swelling, and corneal exposure leading to corneal ulcers may occur. **Traumatic proptosis is an emergency.**

In brachycephalic dog breeds, proptosis occurs easily because they have shallow orbits and large palpebral fissures that allow their globes to move forward more easily. With these dogs, less severe trauma (e.g., restraint) can cause proptosis and usually results in less tissue damage than nonbrachycephalic dogs.

In dolichocephalic dog breeds and in cats, the naturally deeper orbits mean that severe trauma (e.g., vehicular accident or animal fight) is needed to cause a traumatic proptosis, and the orbital tissue damage is usually more severe. Head trauma can also cause skull, mandibular, and brain injuries. Longer eye exposure times result in more extensive corneal damage and more severe retrobulbar hemorrhage and edema. Pressure and stretching may damage the optic nerve, extraocular muscles, and orbital tissues (e.g., the lacrimal gland).

DIAGNOSIS

The globe will be severely protruded, and the eyelid margins will not be visible because they are entrapped behind the eye. Subconjunctival hemorrhage, chemosis, corneal exposure, periocular bruising, and other signs of trauma are usually present.

Clinical Presentation

Signalment

Any animal may suffer proptosis with trauma; however, this is seen most commonly in brachycephalic breeds (e.g., Pekingese, Lhasa Apso, shih tzu, Boston terrier, pug). Sexually intact males are most often affected.

History

The event causing the proptosis may or may not be witnessed by the owner; events that are not witnessed may lead to a delay in treatment.

Physical Examination Findings

A thorough physical examination should be conducted to evaluate other injuries, and the most life-threatening problems or shock should be treated first. Triage the patient and check for evidence of shock, head trauma, pneumothorax, or hemothorax, and palpate the skull for fractures. Puncture wounds, orbital rim fractures, and other injuries should be identified. If cranial trauma has occurred, ensure the patient is stable before focusing on the eye problem.

The eye should be evaluated and lubricated as soon as possible. Check the pupillary light reflexes and, if possible, examine the intraocular structures for hyphema, retinal detachments, or uveitis. The globe should be rapidly evaluated to determine whether it can be replaced. If the optic nerve is torn, the posterior pole of the globe is clearly visible, or a large perforation of the eye wall is present (may be indicated by a deflated globe or ocular hypotony), enucleation should be performed because the prognosis for globe salvage is grave. If three or more extraocular muscles are torn (typically leading to marked dorsolateral strabismus), the orbit is fractured, or the pupil is not visible owing to diffuse hyphema, the prognosis for saving vision is poor, although the globe itself may be salvageable. Pupil size itself is not always a good predictor of vision prognosis because this is determined by multiple factors. Positive prognostic indicators include a brachycephalic dog signalment, intact direct and consensual pupillary light reflexes, intact vision (indicated by tracking of a thrown cotton ball or head retraction when the eye is menaced), a normal fundus appearance, and short duration of proptosis before treatment.[16]

Diagnostic Imaging

Imaging the thorax may reveal concurrent pneumothorax, pleural fluid, or pulmonary contusions. Imaging of the skull may reveal fractures of the orbit and mandible.

Laboratory Findings

The animal may be anemic if significant hemorrhage has occurred.

DIFFERENTIAL DIAGNOSIS

Other possible causes of exophthalmos (forward displaced globe) include retrobulbar abscess, orbital neoplasia, orbital cellulitis, salivary mucocele, or other space occupying orbital disease. Buphthalmos (enlarged globe) is caused by chronic glaucoma (see later).

MEDICAL MANAGEMENT

While stabilizing the patient, immediately and frequently apply topical ointments or gels to the exposed cornea to keep it moist and minimize damage. In many cases, the animal will not allow topical treatments to be applied, but an attempt should be made. The animal should be kept without food and water, and self-trauma should be prevented with a rigid E-collar sized as previously discussed (p. 271).

SURGICAL TREATMENT

Once the patient is stable, replacement of the globe should be done as rapidly as possible because prolonged or progressive retrobulbar swelling may eventually prevent replacement.

Preoperative Management

Keep the eye moist with topical lubricants such as ointments, gels, or an irrigating solution (eye wash or LRS) until the animal is anesthetized. Lubricate the eye with a sterile ophthalmic viscous lubricant or antibiotic ointment. Administer antiinflammatory

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drugs (corticosteroids or NSAIDs) for optic neuritis and orbital edema if not systemically contraindicated. Administer a perioperative IV antibiotic with a gram-positive spectrum (e.g., cefazolin or ampicillin). Significant systemic conditions (pneumothorax, shock, bleeding, head trauma, etc.) need to be addressed first; the animal must be stable enough to tolerate surgery.

Anesthesia

Anesthetic drug selection should consider any cranial or thoracic injuries, and drugs with a short duration of action should be used (see Chapter 12).

Surgical Anatomy

See p. 292.

Positioning

Position the animal in lateral or ventral recumbency. In ventral recumbency, elevate the chin on a towel roll to improve the airway and head positioning.

SURGICAL TECHNIQUES

If possible, make a lateral canthus incision using a Mayo or iris scissor to enlarge the palpebral fissure enough to move the eyelids in front of the globe (Fig. 16.23A). Lubricate the cornea with sterile ophthalmic antibiotic ointment. Reduce entrapment of the lid margins using atraumatic forceps. Place two to four partial-thickness temporary tarsorrhaphy sutures of 4-0 nylon or silk, but do not tie them, leaving the area of the medial canthus open (see Fig. 16.23B). Take care to allow the sutures to exit the lid margins so that sutures will not touch the cornea when tightened. Grasp the ends of the preplaced sutures with a hemostat and pull them anteriorly while applying gentle posterior pressure to the well-lubricated globe with a Jaeger lid place, smooth Bard-Parker scalpel blade handle, or the cut finger of a surgical glove (see Fig. 16.23C). Tie the sutures with the ends left long; place stents if the sutures are excessively tight (see Fig. 16.23D). Close the canthotomy incision with a figure-eight suture at the lateral canthus (see Fig. 16.7C) and simple interrupted sutures of 4-0 to 5-0 absorbable polyglactin 910 (see Fig. 16.23E). Place the sutures partial thickness in the eyelid to prevent corneal contact.

Some have advocated tying the tarsorrhaphy sutures in a bow for ease of opening and retying, but this may not be necessary. The cornea may not be visible after the sutures are placed.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

A lid plate, Derf needle holders, and Bishop-Harmon forceps facilitate the procedure. Suture with excellent knot security (e.g., 4-0 silk or nylon) is needed for a temporary tarsorrhaphy. The tarsorrhaphy should remain in place without loosening for at least 14 days.

POSTOPERATIVE CARE AND ASSESSMENT

Monitor for pain, increased mucopurulent exudate, malaise, and/or pyrexia. The temporary eyelid sutures may need to be removed for reevaluation of the eye if it becomes increasingly painful. Topical medications should be applied at the medial opening in the palpebral fissure. Administer a broad-spectrum topical ointment (see Box 16.1) four times daily, 1% atropine ointment every 12 to 24 hours, and an oral antibiotic with gram-positive coverage (e.g., amoxicillin or cephalexin). For optic nerve and retrobulbar swelling, consider administering systemic corticosteroids (e.g., prednisolone) or NSAIDs at an antiinflammatory dose for 2 weeks and then tapering weekly. Decreasing optic nerve swelling is a critical component of treatment to try to restore vision. A rigid E-collar (see p. 271) should be placed on the

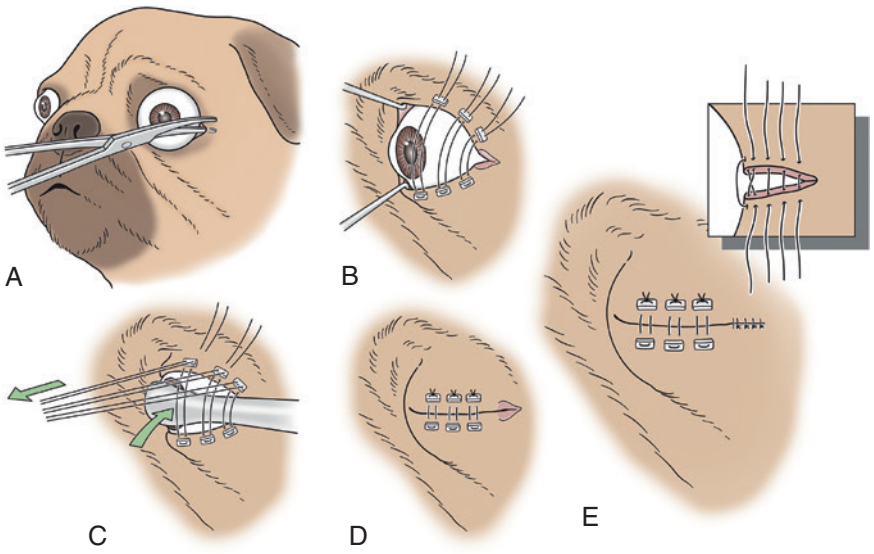


FIG. 16.23 Replacement of a proptosed globe. (A) A lateral canthotomy is often helpful to increase lid laxity, making lid reduction easier. (B) After the lid margins are pulled from their entrapped position, preplace several partial-thickness tarsorrhaphy sutures, but do not tie them. Do not place sutures in the medial aspect of the palpebral fissure. (C) Grasp the sutures simultaneously and pull them anteriorly, while pushing the globe posteriorly with a lid plate or a similar broad object while protecting the corneal surface. (D) Tie the sutures. Do not allow sutures to have contact with the cornea. (E) Close the canthotomy routinely. Use a figure-eight suture at the canthus.

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animal to prevent self-trauma. To reduce swelling, if the animal will allow it, cold compresses should be used postoperatively for the first day, and then warm compresses applied three to four times a day for several more days. The sutures may need to be replaced or tightened if the eyelids open as swelling decreases. Loose sutures should be immediately removed or replaced to prevent corneal contact and ulceration. The tarsorrhaphy sutures are removed from medial to lateral after 14 days; this may be done in a stepwise fashion over a few weeks. If the eye is still exophthalmic, retain the more lateral sutures for a longer time or until they loosen.

COMPLICATIONS

Short-term complications requiring treatment include progressive corneal ulceration secondary to suture irritation, exposure, or infection. Long-term complications after proptosis include blindness and strabismus (very common), corneal scarring, KCS, lagophthalmos, corneal hypoesthesia, and phthisis bulbi. Post-proptosis strabismus improves over 6 to 9 months in many cases and often does not require repair.

PROGNOSIS

The prognosis for vision in proptosed globes is guarded to poor. The prognosis for retaining the globe is good although the degree of eventual ocular cosmesis and comfort is variable. The prognosis for vision and a cosmetically acceptable eye depends on the duration and severity of the proptosis. The prognosis is better with a visual eye, mild proptosis, short duration, absent hyphema, minimal extraocular muscle damage, normal fundic exam, and in brachycephalic breeds. Blindness occurs in approximately 60% to 70% of dogs and in 100% of cats. Postproptosis strabismus occurs in approximately 36% of dogs, most frequently resulting from medial rectus muscle avulsion. Deviation of the globe may improve over 6 to 9 months if the muscle damage was not a complete avulsion. Blindness, lagophthalmos (incomplete blink), deviation of the globe, corneal ulcers, corneal perforations, keratitis, hyphema, phthisis bulbi, chronic uveitis, and glaucoma are possible sequelae to traumatic proptosis.

END-STAGE GLAUCOMA

DEFINITIONS

IOP is intraocular pressure. **Glaucoma** is high IOP. **Buphthalmos** is enlargement of the globe from chronic glaucoma.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Normal IOP in dogs is 10 to 20 mm Hg and in cats is 10 to 25 mm Hg. Glaucoma or high pressure inside the eye occurs when IOP is over 25 mm Hg in dogs and 30 mm Hg in cats. Glaucoma can be primary or inherited where the drainage area of the eye is abnormal, leading to aqueous humor outflow obstruction. Glaucoma can also be secondary to chronic uveitis, intraocular neoplasm, or anterior lens luxation, causing the blockage of aqueous outflow from the eye. Chronic uveitis leads to synechiae (adhesions) inside the eye and the accumulation of inflammatory debris (fibrin, red blood cells, white blood cells) over the irido-corneal drainage angle.

DIAGNOSIS

Clinical Presentation

Signalment

Primary glaucoma in dogs is more frequently seen at middle age in certain breeds (cocker spaniel, basset hound, shar-pei, chow, Bouvier des Flandres, shiba inu). Glaucoma in cats is typically secondary to chronic uveitis or other intraocular disease. Feline primary glaucoma is rare but may be seen in Siamese and Persian breeds. Dogs and cats can also be born with congenital glaucoma.

History

Many owners notice diffuse corneal edema, episcleral redness, or the development of buphthalmos, but they may not notice that the animal is blind in one eye. Blindness is easiest to observe when animals lose vision in both eyes and are in unfamiliar surroundings. Eye problems have usually been long-standing before buphthalmos is noted; onset of blindness due to glaucoma is usually reported to have occurred suddenly.

Physical Examination Findings

Mature dogs with chronic glaucoma will present with a blind, buphthalmic eye that has diffuse corneal edema, an episcleral bloodshot appearance, and possibly an aphakic crescent from a lens subluxation. The cornea may have 360 degrees of corneal vascularization and linear tracks that are splits in the corneal endothelial layer from globe stretching *(striate keratopathy)*. The pupil may be fixed and dilated if primary (inherited) glaucoma is present or may be small or irregular from posterior synechiae if the glaucoma occurred secondary to chronic uveitis. The exception to buphthalmic eyes being blind is in some puppies, shar-peis, and cats, who may sometimes develop buphthalmos yet retain some degree of vision. If the eye is already blind and buphthalmic, vision will not improve even if the IOP can be reduced. Less commonly, a chronically enlarged eye may have an IOP in the normal range because the globe stretching has accommodated for the elevated pressure, and/or ciliary atrophy has occurred.

Diagnostic Imaging

In opaque eyes with glaucoma, ocular ultrasound with 7.5, 10, or 20 MHz probes may reveal as little as a lens subluxation or as much as intraocular masses and/or retinal detachments. If the glaucoma is caused by uveitis due to a systemic disease such as metastatic neoplasia or systemic fungal infection, thoracic radiographs and abdominal ultrasound may reveal the underlying problem.

Laboratory Findings

If glaucoma is secondary to uveitis due to systemic infectious, inflammatory, or neoplastic disease, laboratory testing may reflect the underlying cause. Chronic uveitis in dogs can be caused by systemic infection (fungal disease, rickettsial disease, leptospirosis, *Neospora canis*, *Brucella canis*, Chagas disease, canine distemper, etc.) intraocular or metastatic neoplasia, and immune-mediated disease. In cats, chronic uveitis can be caused by infection (FeLV, FIV, feline infectious peritonitis, toxoplasmosis, *Bartonella felis*, systemic fungal disease), neoplasia, and immune-mediated disease. Immune-mediated disease is diagnosed by excluding other causes of uveitis or by histopathology. Other primary ocular conditions may also cause uveitis severe enough to result in glaucoma, including lens-induced uveitis, immune-mediated ocular disease,

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migrating ocular parasites, or foreign bodies. In these cases, results of hematologic and biochemical analyses may be normal. Surgically removed ocular tissues should always be evaluated histologically to help determine the cause of the glaucoma.

DIFFERENTIAL DIAGNOSIS

Exophthalmos, where the globe is pushed outwards from the orbit, is typically caused by retrobulbar disease. With exophthalmos, the corneal diameter is symmetric with the normal eye, whereas with buphthalmos, the corneal diameter is larger in the diseased eye than the normal eye.

MEDICAL MANAGEMENT

For acute glaucoma with a normal-sized eye, aggressive medical treatment with IV mannitol and topical antiglaucoma medications should be initiated to try to restore and preserve vision. Medical treatment with topical carbonic anhydrase inhibitors (e.g., dorzolamide, brinzolamide), β-blockers (timolol), or prostaglandin analogues (i.e., latanoprost, travoprost) should be considered with any glaucoma while diagnostics are pursued (Box 16.4). Miotics (i.e., demecarium bromide, pilocarpine) and prostaglandin analogues can potentiate uveitis and cause iris muscle spasm pain. The alpha-2 agonists (i.e., brimonidine, apraclonidine) can cause severe bradycardia or death in some small animals with heart disease and should therefore be avoided.

Glaucoma is ultimately a painful, incurable, progressive disease. If the animal lives long enough, glaucoma will generally progress until medical therapy no longer controls IOP and pain.

Referral for chemical cycloablation (to try to reduce the aqueous humor production) with a vitreal aspirate and injection of gentamicin and dexamethasone may be considered in dogs with uncomplicated primary glaucoma. This procedure should be avoided in cats due to the risk of posttraumatic sarcoma. Chemical cycloablation may be an option when owners have financial constraints or when patients have significant risks with general anesthesia. Chemical cycloablation succeeds approximately 50% of the time at controlling glaucoma and chronic medical treatment is frequently needed for uveitis. Additional risks include recurrence or persistence of glaucoma, lens luxation, cataract formation, uveitis with chronic discomfort, recurrent intraocular bleeding, and phthsis bulbi. These patients should be treated surgically.

BOX 16.4 Examples of Chronic Glaucoma Medication Dosages

Carbonic Anhydrase Inhibitors
Dorzolamide, 1 drop BID to TID
Brinzolamide, 1 drop BID to TID

β-Blockers
Timolol, 1 drop BID to TID

Prostaglandin Analogs
Latanoprost, 1 drop SID to BID
Travoprost, 1 drop SID to BID

Miotics
Demecarium bromide, 1 drop BID to TID

BID, Twice daily; *SID,* once daily; *TID,* three times daily.

SURGICAL TREATMENT

Surgical treatment for end-stage glaucoma causing permanent blindness is intended to be palliative and eliminate the need for chronic medical therapy. When medical therapy can no longer control glaucoma, surgery (enucleation or evisceration and implant) should be performed to alleviate the pain.

Preoperative Management

Initiate medical treatment for glaucoma and corneal ulcers until surgery is performed.

Anesthesia

The animal should be evaluated for underlying systemic diseases when the glaucoma occurs secondary to chronic uveitis. See also p. 292.

Surgical Anatomy

Orbital anatomy is discussed on p. 292, and the wall of the eye is composed of the sclera and cornea as discussed on p. 282. The interior of the eye is divided into chambers (Fig. 16.15). The *anterior chamber* is the space between the cornea and the iris. The *posterior chamber* is the narrow space between the iris and the lens. Both chambers are filled with aqueous humor. The *posterior segment,* located behind the lens, is filled with the semisolid gel-like vitreous that is connected to the posterior lens capsule and the retina. The *uvea* (see Fig. 16.15) is the vascularized tissue inside the eye made up by the *iris*, *ciliary body,* and *choroid*. The iris is the colored part of the eye visible through the cornea and controls pupil size to regulate how much light reaches the retina. The ciliary body is a ring of tissue folds that produce aqueous humor and contains the muscles that regulate the lens shape. The lens is a transparent structure suspended just behind the pupil by 360 degrees of *zonules* or modified cilia arising from the ciliary body. The *retina* is the nerve layer lining the back of the inside of the eye and contains the rod and cone photoreceptors that convert light into the nerve impulses that travel through the optic nerve to the brain. The choroid lies between the retina and sclera, and this vascular layer provides nutrient supply to the retina.

Positioning

Position the animal in ventral recumbency with a rolled towel placed under the chin to hold the head level.

SURGICAL TECHNIQUES

Enucleation

Enucleation involves removing the eye along with the tear-producing tissues around the eye (lacrimal glands and third eyelid, conjunctival fornix, and eyelid margins with the meibomian glands). Besides end-stage glaucoma, enucleation may also be performed for irreparable corneal or intraocular injury, neoplasia, severe proptosis, and chronic uveitis causing blindness. Owners may resist having this surgery performed even though it may improve the animal's quality of life.

Transconjunctival, transpalpebral, and lateral enucleation techniques are described here.[17] With all techniques, excessive traction on the globe and optic nerve should be avoided (particularly in cats) because traction on the optic nerve and chiasm can blind the remaining eye. The globe should not be incised or drained because that can spread disease locally and may also

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cause artifacts in tissues submitted for histopathology. Histopathologic evaluation of the globe and any other removed tissue of concern should always be performed.

Transconjunctival Enucleation

The transconjunctival enucleation technique is commonly used. Advantages of this approach include reduced orbital tissue loss (reduced postoperative orbital sinking) and reduced intraoperative bleeding. However, this technique is not preferred for eyes with infected corneal ulcers or perforations. The sterile surgical site will be exposed to a contaminated ocular surface, allowing for possible spread of infectious agents into the orbit. This situation is worsened by rupture of the cornea because intraoperative pressure on the globe may expel infected material from the eye. In these cases, transpalpebral enucleation would be a better choice, especially in dogs.

Using Mayo scissors, make a wide lateral canthotomy to improve exposure (Fig. 16.24A). The area to be cut may be clamped with a straight hemostat before incising to reduce hemorrhage. Placing an eyelid speculum will improve visibility. Make a 360-degree bulbar conjunctival incision a few mm from the limbus using small blunt scissors, such as Stevens tenotomy or small Metzenbaum scissors (see Fig. 16.24B). Palpate the bony dorsal orbital rim so it can be used as a landmark. At the level of the dorsal orbital rim, deepen the incision by dissecting bluntly and sharply with the tip of the scissors until the yellowish sclera and extraocular muscle tendons are exposed (Video 16.1). Incise the tendons with scissors close to the globe (see Fig. 16.24C). Optionally use small hemostats to clamp these areas before cutting to reduce hemorrhage. While grasping the remaining conjunctival tissue at the limbus to prevent pulling on the eye, carry the incision 360 degrees around the globe. The globe should be able to freely rotate once the tissues around the globe are cut; but do not rotate the globe more than 90 degrees; rotate just enough to determine where further dissection is needed.

Do not pull on the eye because this could apply traction to the optic chiasm and blind the remaining eye. While grasping the globe to prevent pulling the eye, clamp the orbital stalk containing the orbital blood vessels, optic nerve, and retractor bulbi muscle with curved hemostats to crush the tissues before cutting (see Fig. 16.24D). While clamping, feel for the back of the globe and the back of the orbit. Incise the orbital stalk with enucleation scissors (strong curved ideally) or curved Metzenbaum scissors approximately halfway between the posterior globe and back of the orbit. Take care to avoid cutting the posterior sclera and rupturing the eye; this could spread disease or adversely affect histopathology. Control hemorrhage from the orbit with clamping the vessels extending from the medial orbit, direct pressure, hemoclips, and/or calcium alginate.

Remove the lid speculum and broadly incise the base of the nictitans, clamping before cutting again to reduce hemorrhage. Remove the entire nictitans, including cartilage and gland at the base. Ensure that all the orbital lacrimal gland, third eyelid with gland, and all the conjunctiva have been removed because these can be fluid producing tissues (see Fig. 16.24E). Remove the eyelid margins with heavy straight scissors, starting at the lateral canthotomy (see Fig. 16.24F). Fully excise the smooth hairless lid margin so that the skin edges will close without excessive dead space or tension. Ensure that the meibomian oil glands along the eyelid margins have been completely removed. If any fluid producing tissue is left behind, the orbit can ultimately develop orbital cysts, infection, or chronic drainage, which may require another surgery. The medial canthal area must be removed. The medial canthus is closely adherent to its deeper attachments, so sharp dissection is sometimes required. Large vessels are occasionally encountered at the medial canthus.

Copiously flush the orbit with sterile saline. Grasp and pull the tissues inside the eyelid to feel for the orbital septum (dense fascia within the eyelid); when the orbital septum is pulled, the whole head will move (Video 16.2). When only subcutaneous tissue without the orbital septum is pulled, much less traction is applied to the head. *Close the orbital septum tightly with 3-0 or 4-0 PDS in a horizontal mattress pattern. Use the tips of your needle holders to feel for openings in the orbital septum. Close any openings so there is no area that will sink in.*

Inject 1 to 3 mL of 0.5% bupivacaine (0.2–0.5 mg/kg cat, 0.5–2 mg/kg dog) into the orbit through the orbital septum using a 23-gauge needle; local anesthesia will improve analgesia for

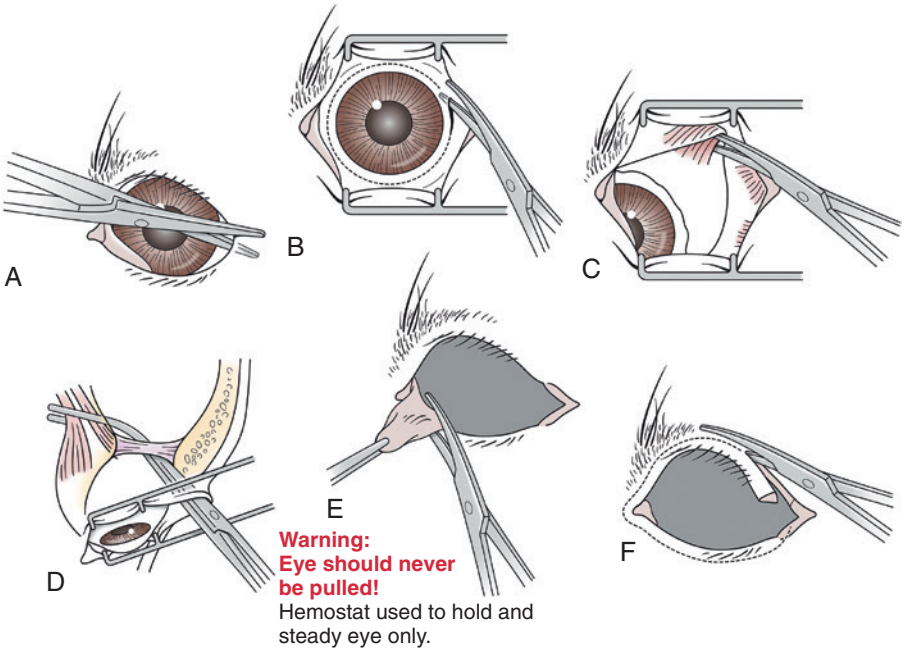


FIG. 16.24 Transconjunctival enucleation. (A) Perform a lateral canthotomy with heavy scissors. (B) Make a 360-degree peritomy (conjunctival incision) with tenotomy scissors. (C) Identify the extraocular muscle attachments and incise them close to the sclera. Optionally, clamp the muscles before incision to reduce bleeding. (D) Clamp the optic nerve and retractor bulbi muscles with a curved hemostat and then incise them. (E) Sharply excise the nictitans and all associated conjunctiva. (F) Sharply excise the lid margins before closing the orbit.

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recovery and the postoperative period (Video 16.3). Close subcutaneous tissues and skin routinely.

To create more space around the globe and improve access to the posterior pole of the globe before it has been removed, remove the eyelid margins and third eyelid after the lateral canthotomy and before subconjunctival enucleation.

Other techniques used to improve cosmesis and reduce skin sinking over the orbit after enucleation include orbital mesh placement, sphere implantation (see later), a combination of the two, or tissue reconstruction (e.g., transposing the temporalis muscle to the orbit). Placing an orbital meshwork is simple. A taut continuous pattern of 3-0 to 5-0 monofilament nonabsorbable suture (nylon or polypropylene) is placed with bites 2 to 4 mm apart in the periosteum of the anterior orbital rim. Meshwork may be placed both horizontally and vertically. All gauze that has been placed in the orbit should be removed before the final bites are placed.

Solid implants of methylmethacrylate or, more commonly, silicone have been placed in the orbit to fill its volume. Spherical implant sizes of 16 to 22 mm diameter are typically used for dogs and cats, with brachycephalic breeds generally tending to require smaller implants and dolichocephalic breeds requiring larger ones. Implants should be gas sterilized and rinsed just before use. Removal of the anterior 25% of a solid silicone sphere (such as those used for ISP surgery, discussed later) has been advocated before the implant is placed into the orbit with the flat side facing forward. The implant may then be retained in position with closure of the orbital septum layer or an overlying mesh on the orbital rim, as described. Alternatively, scarifying the sphere has been used to improve retention.

Lateral Enucleation

Advantages of the lateral approach include better visualization of retrobulbar tissues before the globe has been removed, especially in those with deep orbits; improved confinement of the ocular surface from the sterile surgical site; and retention of more orbital tissue than occurs with the transpalpebral technique. Disadvantages compared with the transconjunctival technique include greater loss of orbital tissue and potentially increased bleeding (because much of the dissection occurs more superficially than the sub-Tenon's space).

Perform a wide lateral canthotomy using straight Mayo scissors (Fig. 16.25A). Bluntly dissect the cut surface of each lid into an anterior skin-orbicularis oculi layer and a posterior tarsoconjunctival layer using a curved Metzenbaum scissor (see Fig. 16.25B). Continue blunt dissection medially until the medial canthus is reached. Remove the scissors, replace one blade of the scissor in the subcutaneous pocket just created, and cut the anterior layer parallel to the lid margin as close to the medial canthus as possible (see Fig. 16.25C). Close the lids with Allis tissue forceps or with a 3-0 or 4-0 simple continuous suture, leaving the ends long (see Fig. 16.25D). Working from the lateral aspect of the globe, use blunt and sharp dissection

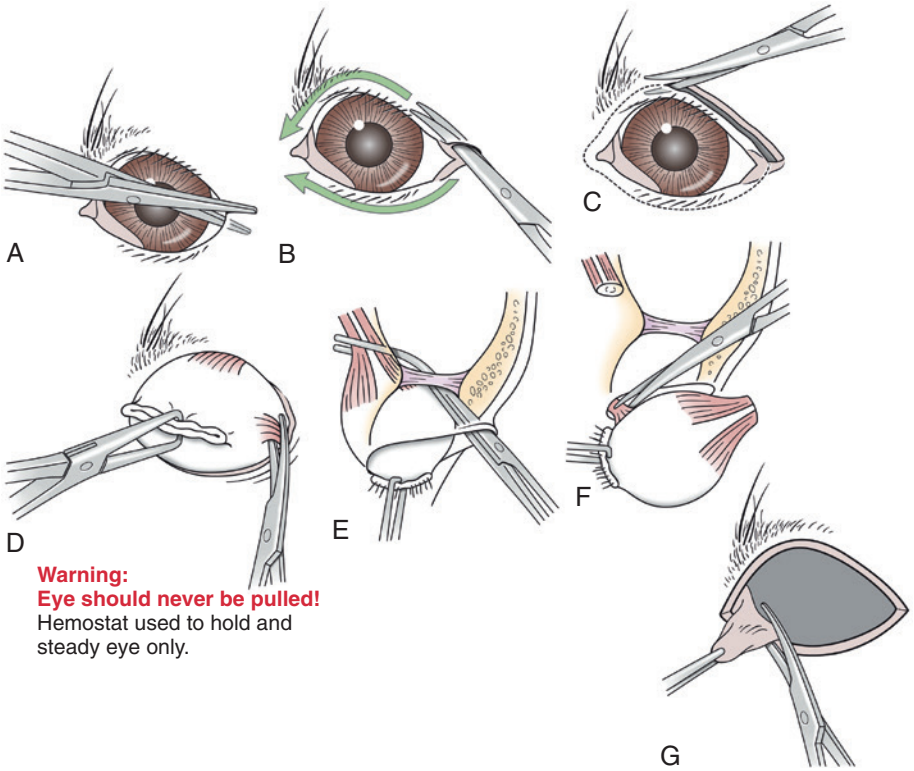


FIG. 16.25 Lateral enucleation (left eye). (A) Perform a lateral canthotomy with heavy scissors. (B) Separate the lid tissues with a curved Metzenbaum scissor to the medial canthus into an anterior skin-orbicularis layer and a posterior tarsoconjunctival layer. (C) Incise the skin-orbicularis layer. (D) Close the palpebral fissure and identify the extraocular muscle attachments; incise them close to the sclera starting with the lateral aspect and continuing posteriorly. The muscles may be clamped before incision. (E) Clamp the optic nerve and retractor bulbi muscles and then incise. (F) Similarly incise the medially located extraocular muscles. (G) Sharply excise the nictitans and all associated conjunctiva.

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of tissues around the lateral globe outside the conjunctival sac while clamping before incising the extraocular muscles close to their attachments on the globe (see Fig. 16.25E). Gently retract and rotate the dissected orbital tissues medially as the dissection is performed. Do not pull on the eye because this could apply traction to the optic chiasm and blind the remaining eye. Clamp the tissues of the posterior pole of the globe with a curved hemostat; then cut the orbital stalk between the back of the globe and the posterior orbit. Roll the globe out of the orbit from lateral to medial, and transect the remaining extraocular muscles, the medial canthal tendon, and the attachments of the medial canthus to the orbit from posterior to anterior (see Fig. 16.25F). Remove the third eyelid with gland, and excise any remaining conjunctiva (see Fig. 16.25G). Close the orbital septum, subcutaneous tissue, and skin, and place local anesthetics as previously described for transconjunctival enucleation (p. 298). Procedures to improve cosmesis (orbital sutures and/or implant) may be considered as for transconjunctival enucleation (p. 299).

Transpalpebral Enucleation

The transpalpebral enucleation technique takes the level of dissection slightly further external to the globe. The main advantage of this technique compared with the other methods of enucleation is the superior confinement of the ocular surface from the surgery site; this is particularly important when infection or neoplasia is present on the surface of the eye. Disadvantages include increased bleeding and increased traction on the optic nerve, a particular concern in all cats and in dogs with deep orbits.

Start with closure of the eyelids. Clamp the eyelids closed with Allis tissue forceps or suture the lids closed with a simple continuous suture, leaving the ends left long; these may be used to position the globe (Fig. 16.26A). Circumferentially incise the anterior skin-orbicularis layer of the eyelids parallel to and 4 to 5 mm from the lid margins with a No. 15 Bard-Parker scalpel (see Fig. 16.26B) or electrocautery. From the initial skin incisions, bluntly dissect the subcutaneous tissues with a small curved Metzenbaum scissor around the globe, while keeping the plane of dissection outside the conjunctival sac (i.e., posterior to the conjunctival fornix) (see Fig. 16.26C–D). Use Allis tissue forceps on the subcutaneous tissue inside the incision to keep the eyelids open dorsally and ventrally. Cut the medial and lateral canthal tendons with Mayo scissors to free up the canthal areas (Video 16.4). Palpate the bony dorsal orbital rim so it can be used as a landmark. At the level of the dorsal orbital rim, deepen the incision by bluntly and sharply dissecting with the tip of the scissors into the tissue until the yellowish sclera and extraocular muscle tendons are exposed (see Video 16.1). Bluntly and sharply dissect 360 degrees around the globe, making the incision parallel to the limbus. Free rotation of the dissected sac indicates that most of the attachments of the globe have been freed.

Do not pull the eye because it could place traction on the optic nerve and cause trauma to the optic chiasm, resulting in blindness of remaining eye. Clamp the tissues at the posterior pole of the globe then remove the hemostat. With enucleation scissors (strong curved, ideally) or curved Metzenbaum scissor, cut the optic stalk halfway between the back of the eye and posterior orbit (see Fig. 16.26E). Remove the block of tissue containing the globe, lid margins, orbital lacrimal gland, conjunctiva, and third eyelid with gland (see Fig. 16.26F). Flush the orbit.

Close the orbital septum (see Video 16.2), subcutaneous tissue, skin, and place local anesthetic (see Video 16.3) as previously described for transconjunctival enucleation (p. 298). Procedures to improve cosmesis (orbital sutures and/or implant) may be considered as for transconjunctival enucleation (p. 299); however,

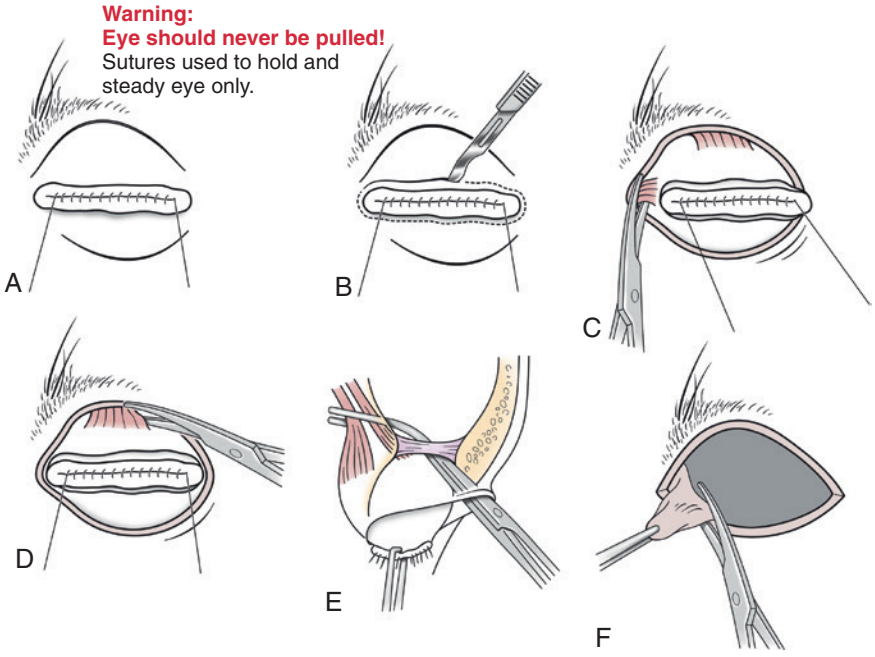


FIG. 16.26 Transpalpebral enucleation (left eye). (A) Suture together the lid margins and leave the ends long to allow for traction. (B) Make a sharp incision around the palpebral fissure through the skin. (C–D) Dissect the subcutaneous tissues to identify the extraocular muscle attachments and isolate and incise them close to the sclera. To reduce bleeding, try to clamp the muscles before incising them. (E) Clamp the optic nerve and retractor bulbi muscles with a curved hemostat and then incise. (F) Sharply excise the nictitans and all associated conjunctiva if any remain.

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if contamination of the surgery site may have occurred, the cosmetic benefits of orbital prosthetic placement should be weighed against the possibility of introducing foreign material into a potentially infected space.

Evisceration and Implant

AP Evisceration and implant surgery (ISP) involves removing the internal contents of the globe while preserving the sclera, cornea, and attachments to the globe, including the extraocular muscles. Due to the retention of the ocular wall, cosmesis can be much greater than with enucleation because after surgery, the globe still remains, and the lids can blink and globe motility remains. Because ISP involves intraocular surgery and requires having a variety of implant sizes on hand, these patients should be referred to a veterinary ophthalmologist.

The primary indication for evisceration and ISP placement is chronic uncontrollable primary glaucoma that is painful. The goal of surgery is to provide long-term comfort with minimal long-term medications. Other indications include certain types of chronic secondary glaucoma, chronic uveitis, progressive phthisis bulbi, and blinding corneal trauma, but only when there is no evidence of infection or neoplasia. Globes suspicious for intraocular infection or neoplasia are generally poor candidates for ISP surgery because of the potential for recurrence. Globes undergoing progressive phthisis may benefit from ISP surgery because current globe size is maintained although the globe size cannot be enlarged. Eviscerated ocular contents should always be submitted for histopathologic evaluation to determine the cause of the intraocular disease.

Contraindications for ISP surgery include corneal ulceration, uncontrolled KCS, severe corneal edema, and intraocular neoplasia. Corneal disease is a concern with ISP surgery because the patient retains the ocular surface, along with any preexisting problems it may have. Ideally before ISP surgery, the corneal disease or dry eye should be well controlled and the cornea should be free of ulceration. Buphthalmic enlarged eyes from glaucoma will shrink over 2 to 3 months after surgery to fit the implant size.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

With enucleation surgeries, strong curved enucleation scissors can greatly assist in incising the optic nerve stalk and removing the eye.

POSTOPERATIVE CARE AND ASSESSMENT

Skin incision care and monitoring are the same as for other soft tissue surgeries. The skin incision should be monitored for increased swelling, redness, or discharge.

COMPLICATIONS

Like any soft tissue surgery, enucleations can have complications with infection and incision dehiscence. A more significant complication is chronic discharge, fluctuance, or swelling of the orbit after surgery. If air or fluid intermittently increases in the orbit, some pathway into the orbit exists and could be a route for the entry of infection. In any fluid-producing tissues (lacrimal gland, third eyelid gland, conjunctival fornix tissue, or meibomian glands) remain, fluid will be produced in the orbit and could lead to orbital infection. If the enucleation site has chronic drainage, orbital cyst formation or the intermittent accumulation of air or fluid, the site should be reoperated to remove any fluid-producing tissues, cysts, the medial/lateral canthal tissues, and/or to ablate the nasolacrimal duct openings.

PROGNOSIS

Long-term prognosis for comfort after enucleation is typically excellent.

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17

Surgery of the Ear



GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Otitis externa is inflammation of the vertical or horizontal ear canal or both; **otitis media** is inflammation of the tympanic cavity and membrane. **Otitis interna** is inflammation of the inner ear that typically causes vestibular disease in dogs. It is nearly always caused by extension of infection into the petrosal bone from otitis media. **Presbycusis** is a term used to describe age-related hearing loss.

PREOPERATIVE CONCERNS

To anticipate surgical complications in animals undergoing ear surgery, it is imperative to determine the extent and severity of disease. Thickening and calcification of the ear canal indicate irreversible inflammatory disease. A sharp pain response on deep palpation of the ear may indicate middle ear infection, whereas a head tilt may indicate severe pain in the ear on the lower side or otitis media or interna (see p. 317). The latter conditions should be suspected if the head tilt is associated with circling, nystagmus, and/or vestibular dysfunction (loss of balance). Periorbital and retrobulbar abscesses may be associated with chronic otitis externa, media, and interna. Facial nerve deficits in patients with chronic otitis externa (e.g., poor palpebral reflex, lip droop, and facial spasms) suggest that the facial nerve is embedded in the horizontal canal, or that serious concurrent middle ear disease is present. Such abnormalities should be noted before surgery to avoid confusion with problems caused by intraoperative trauma during total ear canal ablation (TECA). Soft palate hypoplasia/malformation has been associated with middle ear disease in dogs.

Otoscopic examination should determine whether the tympanic membrane is intact and should define the severity of change in the horizontal and vertical canals. Digital video otoscopy under anesthesia is commonly used for diagnostic examination and treatment of the external ear canal. Always inspect both ear canals, even if the animal has unilateral clinical signs. Skull radiography or computed tomography (CT) should be performed to determine whether concurrent middle ear disease or neoplasia exists (see p. 317). Proliferation of cartilage or bone around the horizontal ear canal should be noted. After imaging, the ear should be cleaned; however, do not use chlorhexidine in a solution stronger than 0.2%, nondilute iodine or iodophors, ethanol, benzalkonium chloride, or some aminoglycosides if the tympanic membrane has ruptured. Drugs and solutions that may be used in animals with a ruptured tympanic membrane are listed in Box 17.1.

Owners' expectations must be considered before surgery is planned in animals with ear disease. Always question owners as to their perception of the ability of their dog to hear before surgery is planned, as TECA may diminish hearing and may be considered unacceptable. Most owners of dogs with severe, chronic otitis externa or media do not report substantial changes in their animal's hearing after this procedure, probably because notable hearing loss occurred before surgery. Similarly, brain stem auditory–evoked response reveals that auditory function declines minimally after TECA in dogs with chronic otitis externa.

NOTE Be sure that the owner is aware of his or her dog's hearing deficits before surgery. This reduces owner dissatisfaction associated with any perceived hearing loss after surgery.

ANESTHETIC CONSIDERATIONS

Ear surgery, particularly TECA, vertical canal resection, and lateral canal resection, is painful. Although butorphanol and buprenorphine are common premedicants in dogs, hydromorphone and morphine are better analgesics for dogs and cats undergoing ear surgery (Table 17.1). Saturating the open surgical site with bupivacaine hydrochloride may provide some analgesia for 4 to 6 hours. A volume of bupivacaine sufficient to cover the area but not exceeding 2 mg/kg should be used. Contact time must be adequate (approximately 15–20 minutes) to allow the local anesthetic to work. The area needs to be relatively free of blood and should not be flushed during the 15 to 20 minutes that bupivacaine is in contact with the tissues. This technique should never be used as the sole method of pain control.

An alternative technique that provides prolonged postoperative analgesia is the constant-rate delivery catheter such as the ON-Q PainBuster (I-Flow Corporation) or the Surefuser (ReCathCo) local anesthetic systems (Box 17.2; Fig. 17.1). The fenestrated catheter is placed in the open wound just before closure by extending the fenestrated end from one end of the wound to the opposite end. This catheter is then attached to an extension set, which consists of an air filter, a flow controller, extra tubing, and a clamp. The extension set is attached to the disposable bulb (the drug reservoir), which has a fill port. These bulbs and their corresponding flow controllers are designed to deliver a specific volume of local anesthetic per hour. Volume and flow rate are determined by identifying which bulb and flow controller are used. Bulbs come in a variety of sizes from 65 to 550 mL, and flow controllers can deliver rates of 0.5 to 10 mL/h (see Box 17.2).

Although high doses of ketamine should not be given to patients with neurologic or renal dysfunction, a small ketamine

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ABSTRACT

Diseases of the ear include those involving the pinna, the canal, and/or the tympanic bulla. Many infectious or inflammatory causes of ear disease are amenable to medical management. When surgery is indicated, knowledge of the regional anatomy, particularly the courses of the facial and vestibular nerves, and the location of the postganglionic sympathetic trunk are essential to minimize complications. Surgical goals include enhancing drainage of the ear canal, improving access for administration of topical medications, and removing diseased or neoplastic tissue.

KEYWORDS

otitis externa
otitis media
otitis interna
tympanic bulla
vestibular dysfunction
Horner syndrome
facial nerve paralysis
aural hematoma
ceruminous gland carcinoma
inflammatory polyps

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BOX 17.1 Partial List of Drugs and Solutions That May Be Used to Flush the Ear in Animals With a Ruptured Tympanic Membrane

Aqueous penicillin
Carbenicillin
Ceftazidime
Ciprofloxacin
Clotrimazole
Enrofloxacin
Fluocinolone (aqueous form)
Miconazole
Nystatin
Ofloxacin
Silver sulfadiazine solution (0.1%)
Squalene (Cerumene)
Ticarcillin
Tolnaftate
Tris-ethylenediaminetetraacetic acid (EDTA)

BOX 17.2 Anesthetic Protocol for Pain Buster/ON-Q or Surefuser Constant-Rate Delivery System in Dogs

- Determine lidocaine dose at 6–18 mg/kg/h.
- If using 2% lidocaine, divide above number by 20 mg/mL to get lidocaine flow rate in mL/h. Use extension set with appropriate flow rate. Range is 0.5–10 mL/h.
- Take above number of mL/h and multiply by number of hours needed to determine total dose of lidocaine needed (in mL). In addition, use this volume to determine the size of the bulb required. Bulbs range in size from 65–550 mL.
- Carefully prime the system before use.
- Overfilling the bulb will cause a decreased rate of delivery of medication. Underfilling the bulb will cause increased rate of delivery of medication.
- When securing the system to the patient, make sure the bulb is close to the wound/incision site. (Attaching to the harness at shoulder height should be sufficient.)
- Discontinue if signs of toxicity occur, such as nystagmus, ataxia, drowsiness, depression, muscle tremors, nausea, or vomiting.
- Leave system in place for 24–48 h.



FIG. 17.1 A constant-rate delivery catheter such as the ON-Q Painbuster (I-Flow Corporation) local anesthetic system allows for prolonged postoperative analgesia after ear surgery.

bolus followed by a ketamine constant-rate infusion (CRI) may facilitate intraoperative management and decrease postoperative pain in dogs or cats that have chronically painful ears (see Table 17.1). Alternatively, a single low-dose bolus of ketamine may be used to augment the pain protocol (see Table 17.1). In dogs, fentanyl, lidocaine, ketamine (FLK) or morphine, lidocaine, ketamine (MLK) CRI may be used intraoperatively or postoperatively for analgesia (see p. 144). Other useful CRIs in dogs include fentanyl and lidocaine; a fentanyl CRI without lidocaine may be used in cats.

Postoperative analgesics should be given after ear surgery. If hydromorphone or morphine was used as a premedicant, it can be readministered 3 to 4 hours after the initial dose (see Table 17.1). However, CRIs are preferred over intermittent administration of opioids postoperatively. If the animal appears dysphoric or anxious, tranquilization or benzodiazepines may be necessary; however, these medications should be used only in animals that have been given sufficient analgesics (see Table 17.1). If a question arises as to whether the animal is in pain, repeating analgesic therapy is nearly always warranted.

ANTIBIOTICS

Preoperative antibiotics are recommended in animals undergoing aural surgery. Severe infection should be treated with systemic and/or topical antibiotics for several weeks before surgery is performed, depending on the site of infection. Otitis externa is best treated with topical therapy because systemic antimicrobials are unlikely to achieve therapeutic concentrations within the fluid and exudates of the external ear canal. Commercially produced topical products typically contain one or more active ingredients (antibacterial, antifungal, or antiinflammatory) in various combinations plus a vehicle and various solubilizers, stabilizers, and surfactants. In contrast, systemic antibiotics are indicated in otitis media because the highly vascularized mucous membrane lining the tympanic cavity of the inflamed middle ear promotes diffusion of drugs from the blood to the bulla. If systemic antibiotics alone do not resolve the infection, the bulla may be instilled with an antimicrobial solution in conjunction with systemic antimicrobials. Selection of systemic antibiotics for treating the middle ear compartment is preferably based on culture and susceptibility testing. Cultures of deep tissues taken during surgery often are more useful than preoperative cultures.

Initial treatment of otitis externa can be empirically based on historical information on the most common isolates and their susceptibility patterns, in conjunction with examination of stained otic swab samples. When treatment failures occur, repeat examination and modification of treatment may be aided by repeat cytology of otic exudate and by submission of cultures. *Malassezia* spp. and *Staphylococcus pseudintermedius* are typically the most common microbial isolates identified in dogs with otitis externa. Most *S. pseudintermedius* organisms are susceptible to cephalothin and oxacillin. Most other bacterial isolates are

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TABLE 17.1 Anesthetic Considerations in the Patient With Ear Disease

Preoperative Considerations

Associated conditions	• Minimal; usually healthy
Blood work	• HCT • TP • In patients older than 5–7 y, consider electrolytes, BUN, and Cr
Physical examination	• Pain may be noted at presentation

Premedications	• If patient is anxious, give the following:
	  • Diazepam (0.2 mg/kg IV), *or*
	  • Midazolam (0.2 mg/kg IV, IM), *plus*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM in dogs; 0.05–0.1 mg/kg IV, IM in cats), *or*
	  • Oxymorphone[e] (0.05–0.2 mg/kg IV, IM), *or*
	  • Morphine[b] (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM in cats, 0.5–1 mg/kg IM in dogs), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM)

Intraoperative Considerations

Induction	• If premedicated, give:
	  • Propofol (2–4 mg/kg IV), *or*
	  • Ketamine (5 mg/kg IV with diazepam or midazolam as above), *or*
	  • Alfaxalone (2–3 mg/kg IV)
	• If no premeds given, give:
	  • Propofol (4–8 mg/kg) IV), *or*
	  • Alfaxalone (2–5 mg/kg IV)

Maintenance	• Isoflurane or sevoflurane, *plus*
	  • Fentanyl (2–10 µg/kg IV prn in dogs; 1–4 µg/kg IV prn in cats) for short-term pain relief, *plus*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV prn in dogs; 0.05–0.1 mg/kg IV prn in cats), *or*
	  • Oxymorphone[e] (0.05–0.2 mg/kg IV, IM), *or*
	  • Morphine[b] (0.1–1 mg/kg IV prn in dogs; 0.05–0.2 mg/kg IV prn in cats), give slowly IV to avoid histamine release *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV prn), *plus* prn
	  • Ketamine (low dose) (0.5–1 mg/kg IV), *or*
	  • Ketamine CRI (0.5 mg/kg IV loading dose, then 10 µg/kg/min IV)

Fluid needs	• Crystalloids, 5–10 mL/kg/h plus 3 × EBL • Colloids, (see p. 32 and Table 4.5) 2–10 mL/kg/h IV; when treating hypotension crystalloids are given first, then a bolus of 2–5 mL/kg of colloids over 10–15 min. If continuing colloids as a CRI, the crystalloid rate should be decreased by 40%–50% to avoid volume overload, particularly in cats
Monitoring	• Blood pressure • HR • ECG • Respiratory rate • SpO_2 • Temperature • End-tidal CO_2
Blocks	• Splash block—bupivacaine (max 2 mg/kg) or lidocaine (max dose 5 mg/kg) placed in the surgical field for 15–20 min, *or* • Constant-rate delivery catheter: lidocaine (see Box 17.2)

Postoperative Considerations

Analgesia	• Morphine[b] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.05–0.2 mg/kg IV or 0.1–0.5 IM q1–4h in cats), *or* • Fentanyl CRI (1–10 µg/kg IV loading dose, then 2–20 µg/kg/h IV), *or* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats), *or* • Oxymorphone[e] (0.05–0.2 mg/kg IV, IM), *or* • Lidocaine CRI (1–2 mg/kg IV loading dose, then 1–3 mg/kg/h IV in dogs) • ± Ketamine CRI (2 µg/kg/min IV, if no previous loading dose, give 0.5 mg/kg IV prior to CRI), *plus* • Carprofen (2.2 mg/kg q12h PO in dogs), *or* • Deracoxib (3–4 mg/kg q24h PO for <7 days in dogs), *or* • Meloxicam[d] (0.1–0.2 mg/kg once SC or PO, then 0.1 mg/kg PO q24h in dogs; 0.05–0.1 mg/kg SC, PO once in cats), *or* • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM q4–8h or 0.01–0.02 OTM q6–12h in cats)
Monitoring	• SpO_2 • Blood pressure • HR • Respiratory rate • Temperature • End-tidal CO_2
Blood work	HCT and TP if large blood loss
Estimated pain score	Can be severe depending on source of pain and/or procedure; many of these patients have been in chronic pain

[a]Monitor for hyperthermia in cats.
[b]Give slowly to prevent histamine release.
[c]Buprenorphine is a better analgesic than morphine in cats.
[d]Black box warning added by the Food and Drug Administration in October 2010 identified cases of renal failure and death in cats with repeated uses of meloxicam. Meloxicam is approved for single use only in cats in the United States.
[e]At the time of publication, oxymorphone was not readily available; however, a dose has been provided throughout in the event it can be compounded or becomes available.
BUN, Blood urea nitrogen; *Cr,* creatinine; *CRI,* constant-rate infusion; *EBL,* estimated blood loss; *ECG,* electrocardiogram; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *OTM,* oral transmucosal; PO, orally; *prn,* as needed; *SC,* subcutaneous; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein.

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susceptible to a number of antimicrobials; however, *Pseudomonas aeruginosa* and *Enterococcus* spp. are often fairly resistant. Ototoxic antimicrobials (e.g., gentamicin, kanamycin, neomycin, streptomycin, tobramycin, amikacin, polymyxin B) should be avoided in animals with otitis. Tris-ethylenediaminetetraacetic acid (EDTA) solution or a combination of tromethamine, EDTA, and benzyl alcohol (see p. 315) may be beneficial in some animals with resistant infection.

SURGICAL ANATOMY

The ear is composed of four parts: (1) the inner ear, which consists of a membranous and bony labyrinth and functions for hearing and balance; (2) the middle ear, which is formed by the tympanic cavity and connects to the pharynx via the auditory tube (eustachian tube); (3) the external ear, which is formed by the auditory meatus and a short canal; and (4) the pinna (Fig. 17.2). The inner ear is located within the osseous labyrinth of the petrous part of the temporal bone. The middle and external ears are separated by the tympanic membrane, and the opening of the horizontal canal into the middle ear is known as the *external acoustic meatus*. Three auditory ossicles (malleus, incus, and stapes) connect the tympanic membrane to the inner ear from the external acoustic meatus to the oval window. Vibrations of the tympanic membrane are transmitted through the chain of these auditory ossicles to perilymph fluid within the vestibule. The tympanic cavity is air filled and, in dogs, is composed of the small dorsal epitympanic recess, the tympanic cavity proper, and the large ventral tympanic bulla. The middle ear connects to the nasopharynx via the auditory tube. Nasopharyngeal polyps (see p. 327) may extend from the nasopharynx into the tympanic cavity and can extrude into the external ear canal.

The tympanic membrane is normally thin and semitransparent but may thicken or rupture when diseased. It is divided into the small, upper pars flaccida and the larger, lower pars tensa. The facial nerve exits the stylomastoid foramen caudal to the ear and courses ventral to the horizontal canal close to the middle ear.

The feline tympanic cavity is divided into two compartments by a thin, bony septum that arises along the cranial aspect of the bulla and curves to attach to the midpoint of the lateral wall (Fig. 17.3). For complete drainage of the middle ear of cats, this bony septum often needs to be perforated. The larger ventromedial compartment is an air-filled tympanic bulla. Most of the lateral wall of the smaller dorsolateral compartment is formed by the tympanic membrane. These compartments communicate through a narrow fissure located dorsally near the cochlear window. Near this fissure, the postganglionic sympathetic nerves form a plexus on a structure known as the *promontory*. Because of their vulnerable location, these nerves can be traumatized during surgical curettage of the feline middle ear, resulting in Horner syndrome (see p. 329).

The canine pinna varies greatly in size and shape among dog breeds. The tip of the pinna is the *apex*, and the *scapha* is the thin, flat pendulous portion. The lateral and medial scaphal margins make up the helix of the ear funneling into the opening of the external ear canal, the *external auditory meatus*. At the base of the ear are several ridges that are important landmarks for surgery (Fig. 17.4): the tragus, the lateral crus of the helix, the pretragic incisure, and the intertragic incisure.

The external ear canal consists of an initial vertical canal and a shorter horizontal canal composed of auricular and annular cartilage. The deepest part of the ear canal (near the tympanic membrane) is osseous. Numerous muscles attach to the cartilage of the ear, allowing it to move to localize sound.

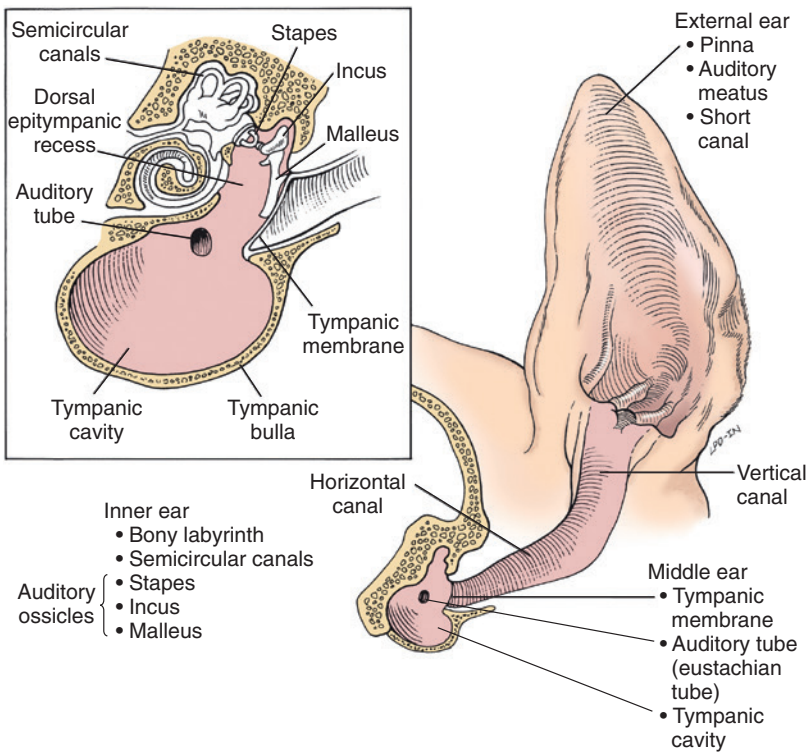


FIG. 17.2 Anatomy of the canine ear.

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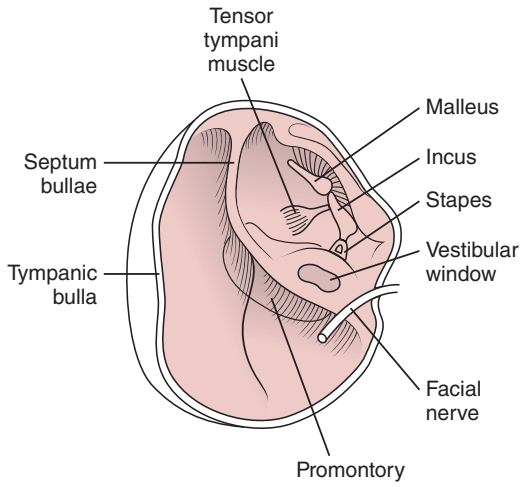


FIG. 17.3 Feline tympanic cavity.

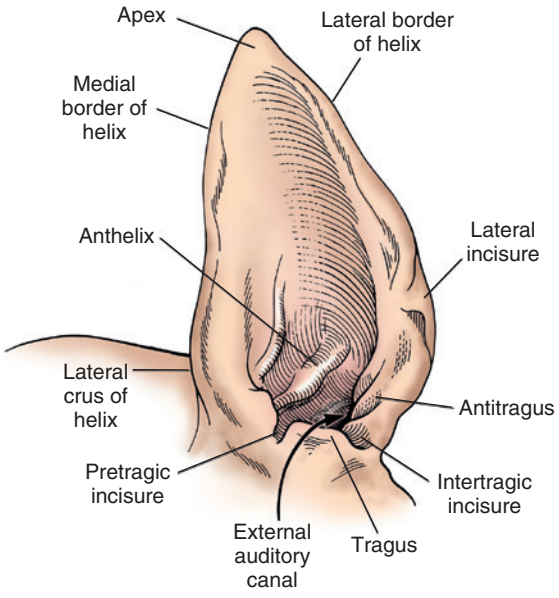


FIG. 17.4 Landmarks of the external ear in dogs.

SURGICAL TECHNIQUES

Numerous surgical techniques have been described for the treatment of ear disease in dogs and cats. When performing a lateral canal resection or a vertical canal ablation, one should be prepared to perform a TECA if the opening of the horizontal canal is stenotic or too narrow to allow adequate drainage.

Lateral Ear Canal Resection

Lateral ear canal resection increases drainage and improves ventilation of the ear canal. It also facilitates placement of topical agents into the horizontal canal. Lateral ear canal resection is indicated in patients with minimal hyperplasia of the ear canal epithelium or with small neoplastic lesions of the lateral aspect of the vertical canal. It should not be performed in animals with obstruction or stenosis of the horizontal ear canal or concurrent otitis media (unless performed in conjunction with ventral bulla osteotomy; see p. 310) or in patients with severe epithelial hyperplasia. Dogs with underlying disease (e.g., hypothyroidism, primary idiopathic seborrhea) often respond poorly to this surgery. Owner counseling is extremely important before a lateral ear canal resection is performed as most studies have shown that owner satisfaction is low when this procedure is performed for chronic otitis externa in dogs. A modification of the original technique for lateral ear resection establishes a "drainboard" and is known as the *Zepp procedure* (Fig. 17.5). The drainboard restricts hair growth at the horizontal canal opening.

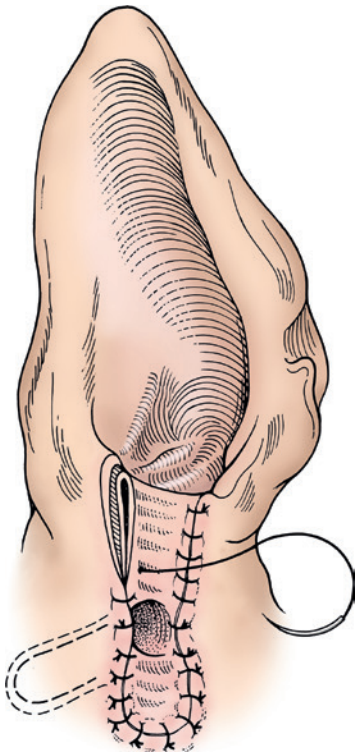


FIG. 17.5 Lateral ear canal resection (Zepp procedure).

NOTE Make sure the owner understands that lateral ear canal resection is not a cure and that medical management of the ear will likely be necessary for the remainder of the animal's life.

Clip the entire side of the face and both sides of the pinna. Gently flush the ear and remove as much debris as possible. Position the animal in lateral recumbency with the head elevated on a towel and prepare the pinna and surrounding skin for aseptic surgery. Place quarter drapes around the ear with the entire pinna draped into the surgical site. Stand at the ventral aspect of the dog's head and position a forceps into the vertical ear canal to determine its ventral extent. Mark a site below the horizontal ear canal that is half the length of the vertical ear canal (Fig. 17.6A). Make two parallel incisions in the skin lateral to the vertical ear canal that extend from the tragus ventrally to the marked site (see Fig. 17.6B). These incisions should be 1.5 times the length of the vertical ear canal. Connect the skin incisions ventrally and, using a combination of sharp and blunt dissection, reflect the skin flap dorsally, exposing the lateral cartilaginous wall of the vertical ear canal. During dissection, stay as close as possible to the cartilage of the ear canal to avoid inadvertently damaging the facial nerve. Note the parotid

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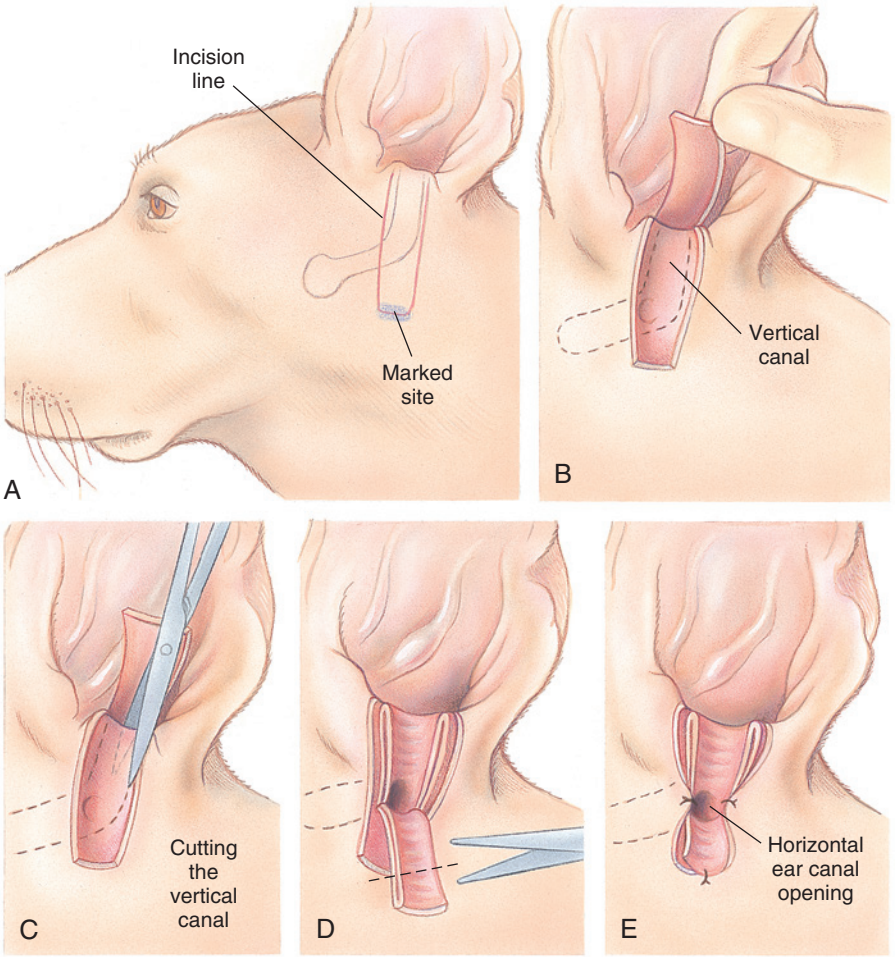


FIG. 17.6 Lateral ear canal resection. (A) Mark a site one-half the length of the vertical ear canal below the horizontal ear canal. (B) Lateral to the vertical ear canal, make two parallel incisions that extend from the tragus ventrally to the marked site. (C) Connect the skin incisions ventrally and reflect the skin flap dorsally, exposing the lateral cartilaginous wall of the vertical ear canal. Use Mayo scissors to cut the vertical canal. (D) Reflect the cartilage flap distally and inspect the opening of the horizontal canal. Resect the distal half of the cartilage flap to make the drainboard, and remove the skin flap. (E) Place sutures from the epithelial tissue to the skin. Begin suturing at the opening of the horizontal canal, then suture the drainboard.

gland at the ventral extent of the incision and avoid damaging it. Standing at the dorsal aspect of the animal's head, use Mayo scissors to cut the vertical canal (see Fig. 17.6C). Place one blade of the scissors in the canal at the pretragic or tragohelicine incisure at the cranial (or medial) aspect of the external auditory meatus and, with the scissors at a 30-degree angle, incise the canal ventrally to the level of the horizontal canal. Repeat the process beginning at the intertragic incisure (caudal or lateral aspect of the external auditory meatus). Do not allow the incisions to converge toward the lateral aspect of the canal, or the drainboard will be too narrow. Be sure to extend the incisions as far distally as the beginning of the horizontal canal, or the drainboard will not lie flat against the skin. Reflect the cartilage flap distally, and inspect the opening of the horizontal canal; if indicated, obtain cultures (see Fig. 17.6D). Occasionally, the opening can be widened by making two small cuts at the cranial and caudal aspects. *Resect the distal half of the cartilage flap to make the drainboard and remove the skin flap.* The ligament between the horizontal and vertical flaps usually acts as a hinge to allow the drainboard to lie flat, but in some cases scoring the cartilage on the ventral aspect of the drainboard facilitates this. *Place absorbable or nonabsorbable monofilament sutures (3-0 or 4-0) from the epithelial tissue to the skin (see Fig. 17.6E). Begin suturing at the opening of the horizontal canal, then suture the drainboard. Last, suture the cranial and caudal aspects of the medial wall of the vertical ear canal to the skin (see Fig. 17.6E).*

Vertical Ear Canal Ablation

Vertical canal ablation can be performed when the entire vertical canal is diseased but the horizontal canal is normal. It may be the technique of choice when neoplasia is confined to the vertical canal or in some animals with chronic otitis externa. Total removal of the vertical canal may result in less postoperative exudation and pain. This technique may provide a better cosmetic appearance of the ear than is provided by lateral ear canal resection when an abundance of hyperplastic tissue is present in and around the vertical canal (Fig. 17.7).

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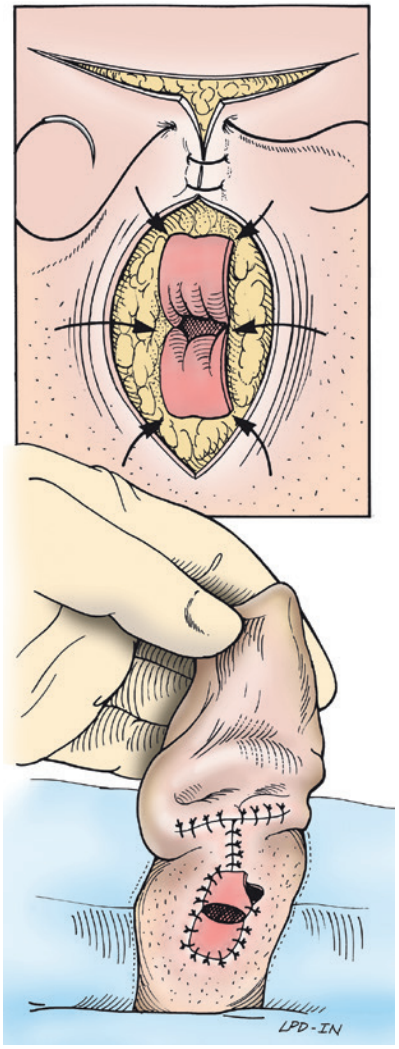


FIG. 17.7 Vertical ear canal resection may be performed when the entire vertical canal is diseased but the horizontal canal is normal.

Position and prepare the animal as for a lateral ear canal resection. Make a T-shaped incision with the horizontal component parallel and just below the upper edge of the tragus (Fig. 17.8A). From the midpoint of the horizontal incision, make a vertical incision that extends to the level of the horizontal canal. Retract the skin flaps, reflect loose connective tissue, and expose the lateral aspect of the vertical canal (see Fig. 17.8B). Continue the horizontal incision through the cartilage around the external auditory meatus with a scalpel blade. Remove as much of the diseased tissue on the medial surface of the pinna as possible, but avoid damaging the major branches of the great auricular artery. Use curved Mayo scissors to dissect around the proximal and medial aspects of the vertical canal. During dissection, stay as close as possible to the cartilage of the ear canal to avoid inadvertently damaging the facial nerve. Free the entire vertical canal from all muscular and fascial attachments (see Fig. 17.8C). Transect the vertical canal ventrally 1 to 2 cm dorsal to the horizontal canal and submit it for histologic examination (see Fig. 17.8D). Incise the remnant of the vertical canal cranially and caudally to create dorsal and ventral flaps (see Fig. 17.8E). Reflect the ventral flap downward, and suture it to the skin for a drainboard using absorbable or nonabsorbable monofilament sutures (2-0 to 4-0). Suture the dorsal flap to the skin and close the subcutaneous tissue with an absorbable suture material (2-0 or 3-0). Close the skin in a T-shaped configuration (see Fig. 17.8F).

Total Ear Canal Ablation

TECA is indicated in animals with chronic otitis externa that have failed to respond to appropriate medical management, in cases of severe calcification and ossification of the ear cartilage, or when severe epithelial hyperplasia extends beyond the pinna or vertical ear canal (Fig. 17.9). The procedure is commonly performed on animals in which lateral ear resections have failed, and it may benefit those animals with severely stenotic ear canals (Fig. 17.10). Neoplasia of the ear canal (e.g., ceruminous gland adenocarcinoma) is also treated by TECA.

Because of the potential for serious complications, this surgery should not be performed on animals with mild disease or by surgeons unfamiliar with the anatomy of the ear. Otherwise healthy dogs requiring bilateral procedures can have single-stage or staggered surgery based on surgeon preference, as there are no significant differences in anesthetic or perioperative complications between unilateral or single-stage bilateral procedures.[1] A high percentage of dogs that undergo TECA have associated skin disease such as seborrhea, atopy, or food or contact allergy dermatitis. The skin disease should be treated before surgery is planned because effective dermatologic therapy usually benefits the ears also. If the skin condition is unresponsive, TECA is preferred over lateral ear resection (see p. 308) and should be performed in conjunction with a lateral bulla osteotomy (LBO) (see p. 309).

NOTE Most animals with severe, chronic otitis externa have concurrent otitis media. Removing the avenue for drainage of exudative material by performing a TECA without treating the otitis media is disastrous. Therefore always perform a bulla osteotomy in conjunction with a TECA for otitis externa and media.

Position the animal in lateral recumbency with the head elevated with a towel. Prepare the pinna and surrounding skin for aseptic surgery. Make a T-shaped incision with the horizontal component parallel and just below the upper edge of the tragus (Fig. 17.11A). From the midpoint of the horizontal incision, make a vertical incision that extends to just past the level of the horizontal canal (see Fig. 17.11A). Retract the skin flaps, reflect loose connective tissue, and expose the lateral aspect of the vertical canal. Continue the horizontal incision around the opening of the vertical ear canal with a scalpel blade (see Fig. 17.11B). Dissect around the proximal and medial aspects of the vertical canal using curved Mayo or Metzenbaum scissors or electrosurgery (see Fig. 17.11C). During dissection, stay as close as possible to the cartilage of the ear canal to avoid inadvertent damage to the facial nerve. Avoid damaging the major branches of the great auricular artery at the medial aspect of the vertical canal. Identify the facial nerve as it courses caudoventrally to the horizontal canal (gently retract it if necessary). If the facial nerve is trapped within thickened, calcified horizontal canal tissue, carefully dissect the nerve from the horizontal canal. Continue the dissection to the level of the external acoustic meatus

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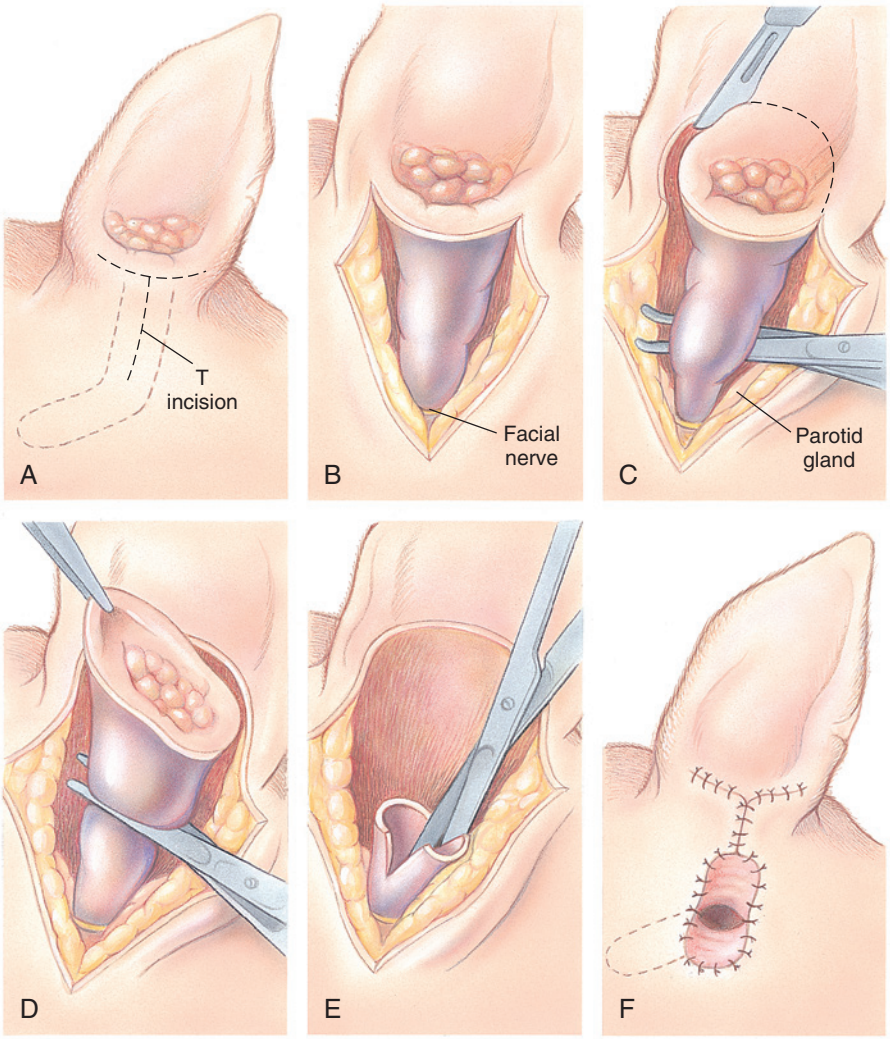


FIG. 17.8 Vertical ear canal ablation. (A) Make a T-shaped incision with the horizontal component parallel and just below the upper edge of the tragus. From the midpoint of the horizontal incision, make a vertical incision that extends to the level of the horizontal canal. (B) Retract the skin flaps, reflect loose connective tissue, and expose the lateral aspect of the vertical canal. (C) Continue the horizontal incision through the cartilage around the external auditory meatus with a scalpel blade. Use curved Mayo scissors to dissect around the proximal and medial aspects of the vertical canal. Free the entire vertical canal from all muscular and fascial attachments. (D) Transect the canal ventrally 1 to 2 cm dorsal to the horizontal canal and submit the canal for histologic examination. (E) Incise the remnant of the vertical canal cranially and caudally to create dorsal and ventral flaps. (F) Reflect the ventral flap downward and suture it to the skin for a drainboard. Suture the dorsal flap to the skin and close the subcutaneous tissue. Then close the skin in a T-shape.

(see Fig. 17.11D). Excise the horizontal canal attachment to the external acoustic meatus with a scalpel blade, rongeurs, or Mayo scissors, but again be mindful of the location of the facial nerve. Remove the entire ear canal, and obtain deep cultures around or just inside the external acoustic meatus. Submit the ear for histologic examination. Use a curette to carefully remove secretory tissue that is adherent to the rim of the external acoustic meatus (see Fig. 17.11E). Be sure to remove all epithelial tissue in this region or chronic fistulation will occur. Perform an LBO (see later discussion). Flush the area with sterile saline solution before closure. Close the subcutaneous tissue with absorbable suture (e.g., 3-0 poliglecaprone 25 or glycomer 631), and close the skin in a T-shaped configuration (see Fig. 17.11F).

Pinna deformity after TECA in cats or in erect-eared dogs can be a source of dissatisfaction for some owners. A technique using a single-pedicle advancement flap at the base of the pinna during a modified TECA may facilitate upright feline ear carriage and improve the cosmetic result and thus enhance owner satisfaction (Fig. 17.12).

Lateral Bulla Osteotomy

LBO exposes the tympanic cavity so that exudate and secretory epithelium can be removed. This procedure should be performed in conjunction with TECA in animals with chronic otitis externa and middle ear disease. Although an LBO affords less exposure to the tympanic cavity than a ventral bulla osteotomy, it does

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FIG. 17.9 A 7-year-old Pyrenees with severe epithelial hyperplasia and infection of the left external ear canal, an indication for total ear canal ablation.



FIG. 17.10 Ear canal excised from a dog with severe epithelial hyperplasia. The medial aspect of the canal has been incised, revealing the luminal surface. The horizontal ear canal is to the left of the image, and the vertical canal is held open by the hemostats and is largely unaffected.

not require that the animal be repositioned and is preferred when performed in conjunction with TECA.

Bluntly dissect the tissue from the lateral aspect of the bulla using a small periosteal elevator. Avoid damaging the external carotid artery and the maxillary vein, which travel just ventral to the bulla. Rongeur the lateral and ventral aspects of the bulla until the caudal aspect of the middle ear canal is exposed (Fig. 17.13). Extend the bony excision as needed to fully visualize the contents of the tympanic cavity, but avoid sharp dissection and curettage of the rostral aspect of the osseous ear canal to reduce the risk of retroauricular vein damage. Use a curette to remove infected material, but avoid curetting in the rostral (dorsal) or rostromedial area of the tympanic cavity so as not to damage the auditory ossicles or inner ear structures. A 1.9-mm rigid endoscope can be used to aid visualization of the inside of the tympanic cavity. *Gently irrigate the cavity with saline to remove all remaining debris.*

Ventral Bulla Osteotomy

Ventral bulla osteotomy allows increased exposure of the tympanic cavity and can be performed alone or in conjunction with lateral ear resection. It is the technique of choice for access to the epithelial lining of the bulla in cats with inflammatory polyps (see p. 327). This technique provides better access to the bulla than an LBO and allows for bilateral procedures to be performed without the need to reposition the animal.

Place the patient in dorsal recumbency and prepare a generous area surrounding the angle of the mandible for aseptic surgery. Palpate the bulla immediately caudal and medial to the vertical ramus of the mandible. Draw an imaginary line connecting the mandibular rami and a second imaginary line along the long axis of the ventral aspect of the head (Fig. 17.14A). In dogs, make a 7- to 10-cm incision (3–5 cm in cats) parallel to the midline of the animal and centered 2 cm toward the affected side from where these imaginary lines intersect (see Fig. 17.14A). Incise the platysma muscle, retract the linguofacial vein if necessary, and deepen the incision by bluntly dissecting the digastricus muscle (lateral) from the hyoglossus and styloglossus muscles (medial). Avoid damaging the hypoglossal nerve, located on the lateral aspect of the hyoglossus muscle. Confirm the location of the bulla and use self-retaining retractors (e.g., Gelpi, Weitlaner) to spread the digastric and glossal muscles and retract them from the bulla (see Fig. 17.14B). Palpate the bulla craniomedial to the cornu process of the hyoid bone and caudomedial to the angle of the mandible. Bluntly dissect tissues from the ventral surface of the bulla and use a Steinmann pin to make a hole in its ventral aspect. Enlarge the opening with a small rongeur (e.g., Lempert). Examine the interior of the bulla for inflammatory debris, neoplastic tissue, or foreign bodies, and obtain samples for culture, sensitivity, and histopathologic examination. In cats, be sure to examine both compartments of the bulla (see p. 305 and Fig. 17.3). Gently flush the cavity with warm saline and close the incision routinely.

HEALING AND THE EAR

Hyperkeratinization of the epidermis and hyperplasia of the dermis and epidermis of the ear canals occur secondary to chronic infection or inflammation. Sebaceous glands become less numerous and active, and apocrine tubular glands distend and increase secretion. Healing after the surgical techniques previously described is routine unless incisional infection develops.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Electrosurgery is useful for ear surgery because numerous small vessels are encountered. Small curettes simplify removal of epithelial tissue on the bony rim at the external acoustic meatus. An assortment of rongeurs of various sizes (e.g., Cleveland, Lempert, Kerrison, Ruskin) or an air-driven burr is needed to remove the lateral and caudal aspects of the bulla when a TECA is performed. A Freer elevator is useful for reflecting soft tissue attached around the ventrolateral wall of the bulla. Retractors, such as Senn or Army-Navy, are helpful in visualization of deep structures. A Steinmann pin, a hand chuck, rongeurs, or a

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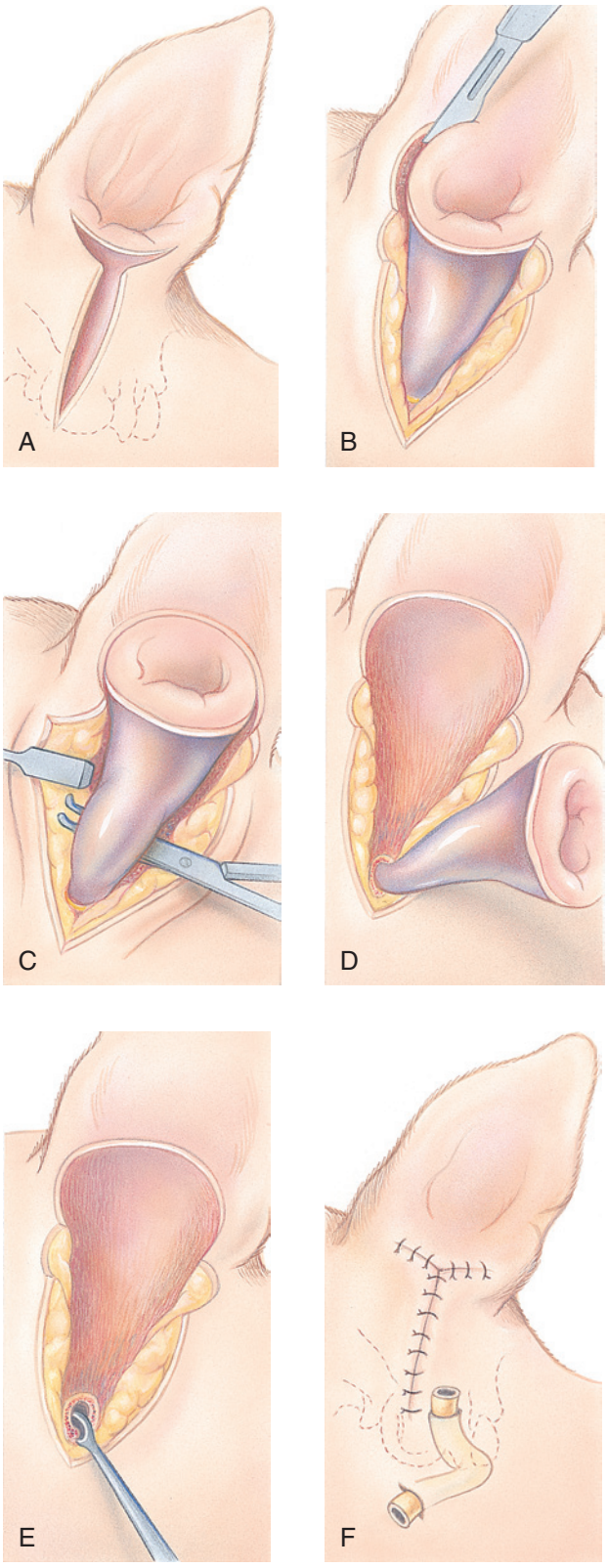


FIG. 17.11 Total ear canal ablation. (A) Make a T-shaped incision with the horizontal component parallel and just below the upper edge of the tragus. From the midpoint of the horizontal incision, make a vertical incision that extends to just past the level of the horizontal canal. (B) Retract the skin flaps, reflect loose connective tissue, and expose the lateral aspect of the vertical canal. Continue the horizontal incision around the opening of the vertical ear canal with a scalpel blade. (C) Dissect around the proximal and medial aspects of the vertical canal. (D) Continue the dissection to the level of the external acoustic meatus. (E) Excise the horizontal canal attachment to the external acoustic meatus with a scalpel blade, rongeur, or Mayo scissors, and use a curette to carefully remove secretory tissue that is adherent to the rim of the external acoustic meatus. (F) If desired, place a Penrose drain. Close the subcutaneous tissue and skin.

high-speed drill with burrs is necessary for access to the tympanic cavity during ventral bulla osteotomy (unless the bone has been eroded by infection or neoplasia). Self-retaining retractors are useful when a ventral bulla osteotomy is performed to allow retraction of the muscles superficial to the bulla. Culture swabs (both aerobic and anaerobic) should be available whenever ear surgery is performed.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative analgesics should be given after ear canal resection or ablation (see the discussion on anesthetics on p. 302); tranquilizers may be administered if the animal appears dysphoric or anxious (see p. 303). An Elizabethan collar should be used to prevent the animal from scratching at the incision site. If swelling is excessive, a cold pack can be applied to the side of the face several times a day for the first 24 to 36 hours after surgery. Antibiotics should be based on culture results and continued for 3 to 4 weeks. Sutures can be removed in 10 to 14 days.

NOTE Use caution when deciding to perform bilateral procedures, particularly ventral bulla osteotomies in cats. Significant postoperative swelling can lead to upper airway obstruction and respiratory distress. Avoid using head bandages as pharyngeal constriction may result.

COMPLICATIONS

Complications other than inadequate drainage and continued otitis externa are uncommon after lateral ear resection or vertical ear canal ablation. Clinical signs associated with otitis externa may not be relieved in dogs with underlying dermatologic disease that cannot be effectively managed. If the opening of the horizontal canal is insufficient for drainage, or if these techniques are performed in animals with concurrent middle ear disease without treating the middle ear infection, the result is persistent or recurrent signs of otitis externa. Facial nerve palsy is a rare complication of vertical ear canal ablation.

Potential complications of TECA are numerous and occasionally serious. These complications include superficial wound infection, facial nerve paralysis, vestibular dysfunction, Horner syndrome, deafness, chronic fistulation or abscessation, and avascular necrosis of the skin of the pinna (Fig. 17.15). Facial nerve paralysis is caused by stretching or retraction of the nerve; it usually resolves within a few weeks of surgery. Permanent damage may occur if the nerve is transected or is severely stretched. Temporary facial paralysis has been reported to occur

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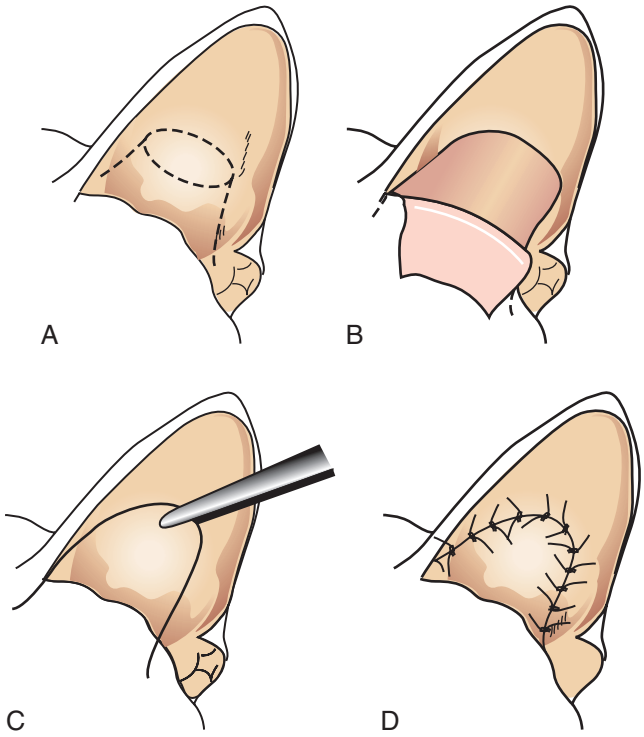


FIG. 17.12 To perform a modified total ear canal ablation in a cat, (A) make a vertical incision at the cranial and caudal ends of an elliptic incision centered around the external auditory meatus. (B) Dissect the single-pedicle advancement flap ventrally, allowing exposure of the subcutaneous tissue over the vertical ear canal. (C) After lateral bulla osteotomy and excision of the ear canal, pull the top of the advancement flap to the base of the pinna to determine whether further release of the flap is necessary to reduce tension on the pinna. (D) Suture the flap to the base of the pinna.

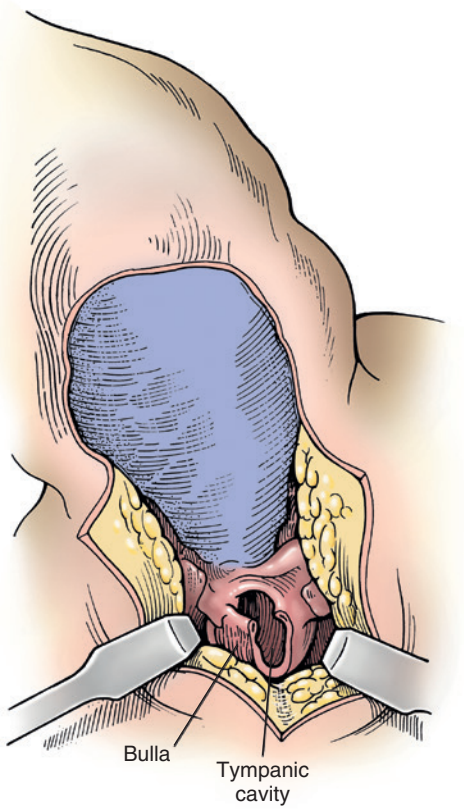


FIG. 17.13 Lateral bulla osteotomy.

in 3% to 27% of dogs undergoing TECA with concurrent LBO.[1] In contrast, facial paralysis in cats undergoing the same procedure can occur in up to 74%, with up to 47% having permanent damage. Horner syndrome following TECA and LBO is uncommon in dogs but frequent in cats. A 2013 study found Horner syndrome occurred in 3.3% of dogs and 58.3% of cats following TECA with LBO.[2]

Facial nerve damage may result in loss of the blink response and parasympathetic innervation to the lacrimal glands. The eye should be kept moistened with artificial tears or an ophthalmic lubricant to prevent corneal ulceration. If normal lid function does not return within 4 to 6 weeks, or if ulceration of the eye occurs as the result of chronic drying, enucleation may be indicated; however, this is seldom necessary, especially if normal tear flow is present.

Postoperative ipsilateral head tilt has been reported to occur in 3% to 11% of dogs, but it is rare in cats. Vestibular signs and a significant head tilt indicate inner ear damage that likely occurred due to curettage of the epitympanic recess and promontory of the tympanic cavity. These signs are transient in most cases, lasting 2 to 3 weeks.

Facial nerve paralysis, vestibular dysfunction, and Horner syndrome may all be caused by otitis media or interna or by surgery. The presence of these abnormalities before surgery must be noted to avoid having them considered as surgical complications.

Following ventral bulla osteotomy, transient Horner syndrome, facial nerve paralysis, and vestibular signs are common, particularly in cats.

SPECIAL AGE CONSIDERATIONS

Young cats with middle or inner ear signs or a previous history of respiratory disease should be examined for nasopharyngeal polyps (see p. 327).

SPECIFIC DISEASES

OTITIS EXTERNA

DEFINITIONS

Otitis externa is an inflammation of the epithelium of the horizontal and vertical ear canals and surrounding structures (i.e., external auditory meatus and pinna).

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Otitis externa is common in dogs (up to 20% of hospital admissions; up to 7% in cats), and between 50% and 89% of affected dogs have concurrent otitis media. It may be associated with other dermatologic conditions, particularly allergic or immune-mediated skin disease (e.g., food allergy dermatitis, atopy, and

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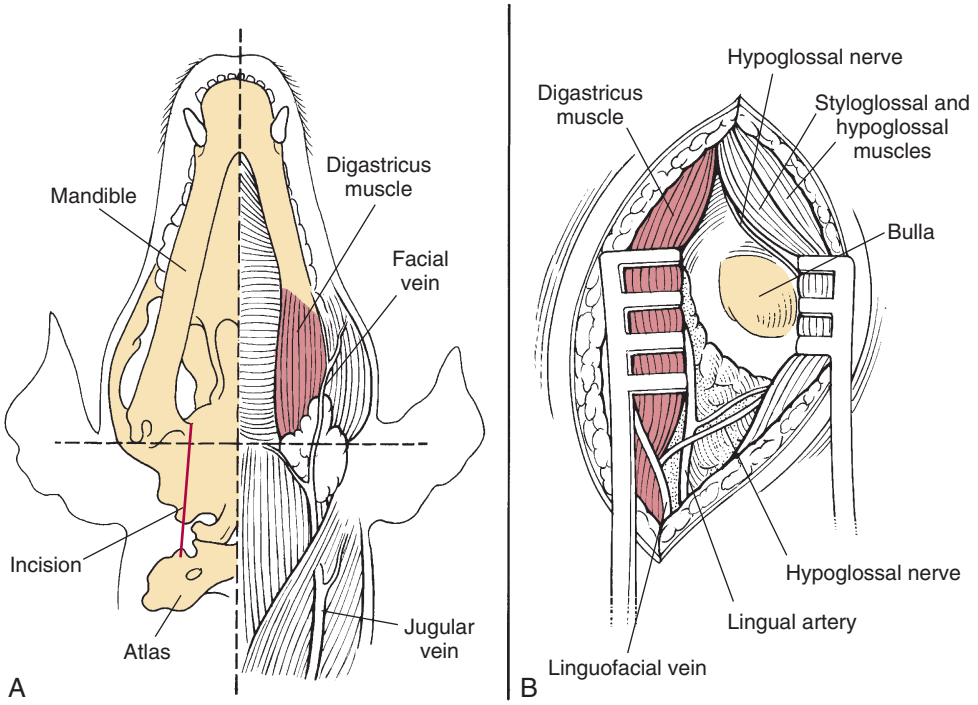


FIG. 17.14 Ventral bulla osteotomy. (A) For ventral bulla osteotomy, draw an imaginary line connecting the mandibular rami and a second imaginary line along the long axis of the ventral aspect of the head. Make a 4- to 10-cm incision (depending on the size of the animal) parallel with the midline of the neck and centered 2 cm toward the affected side from where these imaginary lines intersect. (B) Incise the platysma muscle, retract the linguofacial vein if necessary, and deepen the incision by bluntly dissecting the digastricus muscle (lateral) from the hyoglossal and styloglossal muscles (medial). Confirm the location of the bulla; use self-retaining retractors to spread the digastricus and glossal muscles and retract them from the bulla.

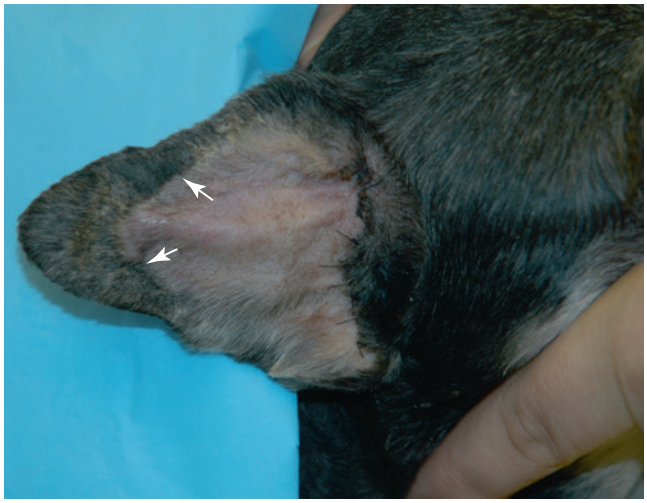


FIG. 17.15 Marginal necrosis of the pinna 2 weeks after total ear canal ablation/lateral bulla osteotomy in a dog. Arrows delineate the line of necrosis, and the suture line is visible on the right side of the image.

contact dermatitis) or systemic disease (e.g., endocrinopathies such as hypothyroidism). Bacterial infection, foreign bodies (e.g., foxtails), parasites (e.g., *Otodectes cynotis, Demodex canis, Sarcoptes scabiei, Notoedres cati,* ticks), fungi, yeasts (e.g., *Malassezia pachydermatis*), or neoplasia may also be the cause. *O. cynotis* is responsible for more than 50% of cases of otitis externa in cats.

Predisposing conditions for otitis externa include excessive moisture or increased humidity in the ear canal, a narrow canal conformation, and obstruction of the canal. The normal ear canal is inhabited by bacteria (e.g., *Staphylococcus* and β-*Streptococcus* spp.). High humidity and temperature promote moisture retention in the ear; this allows maceration of the epithelial lining and fosters secondary bacterial colonization. It has been proposed that in chronic otitis externa the apocrine glands increase in size, number, and secretory activity, whereas the sebaceous glands decrease in number and become less active. However, the dog may not have a common physiologic pathway for progressive changes, as was previously thought. Although the density and distribution of sebaceous and ceruminous gland tissue vary markedly among individuals, in general sebaceous tissue increases gradually from proximal to distal parts of the ear canal, whereas ceruminous gland tissue decreases. In dogs with otitis externa, the distribution of sebaceous and ceruminous glandular tissue is similar to that of normal ears, but the glands become larger and hyperplastic. Hair follicles also become hyperplastic in dogs with otitis externa. Cocker spaniels with otitis externa have been shown to have distinct differences in pathologic characteristics of the horizontal ear canal compared with other breeds. They more commonly have ceruminous tissue responses, whereas other breeds have predominantly fibrosis.

The bacteria most often isolated from the ears of dogs with chronic otitis externa are *Corynebacterium* spp., *Escherichia coli,*

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Proteus mirabilis, P. aeruginosa, and *S. pseudintermedius. Pasteurella multocida* and *S. pseudintermedius* are commonly isolated from the ears of cats. In some animals, chronic otitis externa may cause secondary changes in the ear canal (i.e., epithelial hyperplasia and ossification of chronically inflamed tissue) that perpetuate the infection and make medical management difficult because of constriction of the external ear canal lumen. In addition, ulceration and secondary infection with pyogenic bacteria, yeast, and/or fungi commonly occur.

DIAGNOSIS

Clinical Presentation

Signalment

Dogs and cats of any breed or age may develop otitis externa, but some groups are at higher risk. Dogs with long, pendulous ears (e.g., spaniels, basset hounds) and those with abundant hair in the ear canal (e.g., poodles) are commonly affected. Among erect-eared dogs, German shepherd dogs are most frequently affected. Spaniel breeds, particularly cocker spaniels, may have abnormal keratinization and increased sebaceous gland secretion of the pinna or ear canal or both. Chronic bacterial infection and hyperplastic changes in the sebaceous glands and the epithelial lining of the ear often lead to scarring and ear canal obstruction.

History

Animals with otitis externa may be presented for evaluation of acute or chronic signs. If a foreign body is lodged in the ear, head shaking and scratching at or near the ear are typical. Head shaking and ear scratching are also common among animals with parasitic infection and acute bacterial infection. A purulent, odoriferous discharge may be noted with chronic infection. The animal may constantly rub its head on objects and may seem to be in pain when the head or ear is touched.

Physical Examination Findings

Palpation of the ear may suggest thickening or calcification of the ear canal. A thorough otoscopic examination should be performed even if it requires tranquilization. Examination of the ear canal is often difficult if hyperplasia or exudation are present; general anesthesia may be necessary to allow meticulous inspection. The extent of involvement of the vertical and horizontal ear canals and the status of the tympanic membrane should be determined. Purulent yellow or cream-colored exudates may be associated with gram-negative infection, particularly *Pseudomonas* and *Proteus* spp. Dark brown or black exudates are more commonly associated with yeast infection or infection caused by *Staphylococcus* or *Streptococcus* spp. A bloody exudate may be suggestive of neoplasia. Definitive diagnosis requires examination of exudate collected during the procedure by placing sterile swabs into the canal through the otoscope cone. The exudate should be examined for parasites, bacteria, fungi, and yeast; bacterial and fungal cultures should be performed if indicated. The ear should be flushed with a bulb syringe or a soft catheter, and alligator forceps should be available to remove foreign bodies and debris. Biopsy of the external ear canal may allow diagnosis of neoplasia and some allergic conditions.

NOTE Perform a complete dermatologic examination in all animals with otitis externa unless an obvious cause is found, such as a foreign body.

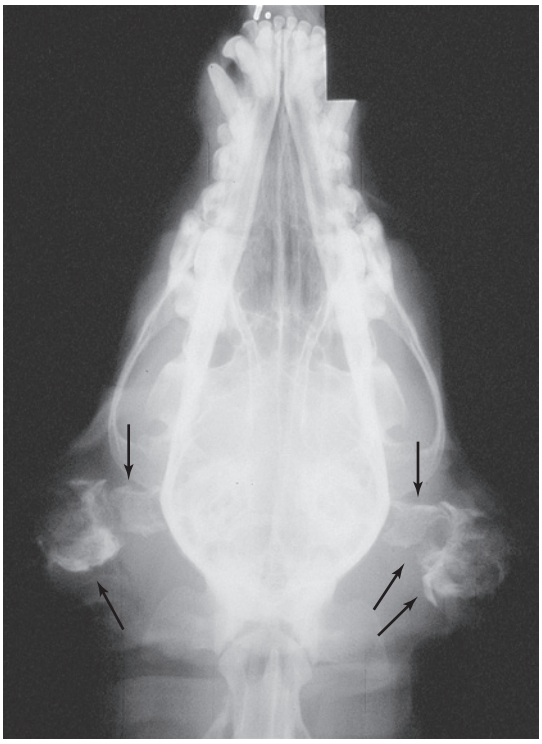


FIG. 17.16 Calcification of the external ear canals *(arrows)* in a dog with chronic otitis externa. (Courtesy L. Homco, Ithaca, NY.)

Diagnostic Imaging

Skull radiography or CT imaging should be performed to determine whether concurrent otitis media is present; CT imaging is preferred because of its lack of superimposition and increased sensitivity in detecting abnormalities (see p. 318). Because improper obliquity or angulation of the skull for radiographs or malpositioning of the tongue can result in an inadequate study, false-negatives or underestimation of the extent of middle ear disease is common with radiographs. Calcification of the external auditory canal is commonly noted with chronic otitis externa (Fig. 17.16); this finding may influence the choice of surgical techniques. Occasionally, imaging signs suggestive of neoplasia are found, such as bony lysis of the petrous temporal bone. CT findings with otitis externa include mineralization of the ear canal, narrowing of the lumen of the external ear canal, and soft tissue attenuating material within the lumen of the ear canal. CT may also be useful for evaluation of abscess formation following a TECA. Uptake of contrast may be noted in areas of infection (abscessation, fistulous tracts). Canalographic evaluation of the external ear canal, although uncommonly performed, may help delineate the status of the tympanic membrane before medical or surgical therapy in dogs. Preferably, video otoscopy can be used.

Laboratory Findings

Specific laboratory abnormalities are not found. Thyroid function tests should be performed if hypothyroidism is possible.

DIFFERENTIAL DIAGNOSIS

The diagnosis of otitis externa is usually simple; however, differentiation of the various causes may be difficult. It is important

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to identify treatable underlying causes of otitis externa before considering surgical intervention; optimal results require appropriate treatment of underlying diseases. In some cases, surgery is unnecessary if the underlying cause is treated. Concurrent otitis media should be identified in animals that are to undergo surgery for otitis externa.

NOTE Lateral ear canal resection and vertical canal ablation often fail if concurrent dermatologic or middle ear disease is not resolved.

MEDICAL MANAGEMENT

Treatment of otitis externa involves identifying the underlying or perpetuating causes, cleaning and drying the ear, and using appropriate topical medications (see p. 304). Because ceruminous material impairs the ability of topical medications to reach the infection and may inactivate some drugs, the ears must be thoroughly cleaned before treatment. The status of the tympanic membrane (intact vs. ruptured) should be ascertained. Many topical agents are available for treatment of otitis externa; most contain various combinations of antibiotics and parasiticidal, antiinflammatory, and/or antifungal agents. The reader is referred to a dermatology or medicine text for an in-depth discussion of the use of these various agents. Medications that can be used in animals with a ruptured tympanic membrane are listed in Box 17.1. The combination of tris-EDTA and an antimicrobial has a synergistic effect against some bacteria implicated in otitis externa (Box 17.3). An ear rinse containing tromethamine, EDTA, and benzyl alcohol was found to significantly decrease bacterial growth in vitro of *P. aeruginosa* and β-hemolytic streptococcal organisms within 15 minutes, *Proteus* spp. within 30 minutes, and *Staphylococcus* spp. within 60 minutes. Addition of benzyl alcohol enhanced antiseptic activity against β-hemolytic streptococci and *Proteus* spp., compared with a solution of tromethamine and EDTA alone. Ototoxicity of the various agents should be taken into account before they are used, particularly if the tympanic membrane has been ruptured. Silver sulfadiazine solution (0.1%; see Box 17.3) can be used without an intact tympanic membrane. Persistence of clinical signs after treatment of otitis externa may suggest concurrent otitis media. Targeted systemic antibiotics, administered for 6 to 8 weeks, are indicated for treatment of otitis media (see p. 319).

BOX 17.3 Otic Preparations

Tris-EDTA Solution[a] (0.05 mol/L Tris, 0.003 mol/L EDTA)

24.2 g Tris (base)
4.8 g EDTA (disodium salt)
3900 mL distilled water
100 mL white vinegar (5% acetic acid)
Adjust pH to 8 with additional vinegar (30–50 mL)
Autoclave and store sterile

Silver Sulfadiazine Solution (0.1%)

0.1 g silver sulfadiazine powder
100 mL distilled water
Mix to dissolve

[a]Commercially available (e.g., TrizEDTA [DermaPet], T8 solution [DVM Pharmaceuticals]).
EDTA, Ethylenediaminetetraacetic acid.

Surgical Treatment

Surgical therapy of otitis externa should be considered when medical management fails or in cases involving proliferative growth or stenotic canals. Surgical alternatives in animals with otitis externa that do not have middle ear involvement include lateral ear canal resection (see p. 306), vertical ear canal ablation (see p. 307), and TECA (see p. 308). If concurrent otitis media exists, a lateral ear canal resection in conjunction with a ventral bulla osteotomy (see p. 310) or a TECA with LBO (see p. 309) can be performed.

Preoperative Management

Preoperative antibiotic therapy is recommended. Bacterial cultures should be performed if purulent discharge is present, and appropriate antibiotics should be initiated before surgery is performed. If no discharge is present, perioperative antibiotics (see p. 86) may be given intravenously immediately before the surgical procedure or may be administered during surgery but after intraoperative cultures have been obtained. Based on published culture and susceptibility testing, cefazolin may be a poor choice for perioperative prophylaxis in dogs undergoing TECA and bulla osteotomy. Cefazolin, tetracycline, and trimethoprim-sulfamethoxazole have susceptibilities of less than 50%. Amoxicillin-clavulanic acid or cephalothin may be a reasonable choice for prophylaxis as most *S. pseudintermedius* organisms are susceptible to these (see Chapter 9), and *S. pseudintermedius* is a common isolate of ear samples. Culture and susceptibility testing is strongly advised in cases of chronic otitis externa or those warranting surgical treatment.

Anesthesia

See p. 302 for anesthetic recommendations for animals with ear disease.

Surgical Anatomy

See p. 305 for a description of the surgical anatomy of the ear canal.

Positioning

See pp. 306 to 310 for details on positioning for the various surgical procedures.

SURGICAL TECHNIQUE

The choice of surgical techniques depends on the severity and extent of the disease. See pp. 306 to 310 for a description of the surgical techniques used in animals with ear disease. A discussion is provided of the indications and contraindications for each procedure.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

See p. 310 for a discussion of appropriate suture materials and instruments for surgery of the ear.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative pain is common in animals that have undergone ear surgery. See p. 302 for a discussion of analgesic therapy in these patients. After administration of adequate analgesics, tranquilizers may be given if the animal appears dysphoric or

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anxious (see p. 303). Animals often shake their heads excessively after ear surgery and may try to paw or scratch at the bandage. Close supervision in the early postoperative period and use of an Elizabethan collar or sidebar are recommended.

PROGNOSIS

Chronic otitis externa is a difficult disease to treat with medical therapy or surgery. A poor surgical outcome may be the result of technical failure (e.g., not making the horizontal canal opening large enough with lateral ear canal resection or vertical ear canal ablation), lack of owner compliance in continuing to treat the ear (with lateral ear canal resection or vertical ear canal ablation), unrealistic expectations on the part of the owner, unrecognized middle ear disease, faulty diagnoses (e.g., not recognizing neoplasia as the underlying cause), or failure to treat the underlying disease or perpetuating cause. Surgical procedures designed to increase drainage (i.e., lateral ear canal resection and vertical ear canal ablation) often fail in animals with untreated dermatologic disease or unrecognized middle ear disease. A Zepp procedure results in a satisfactory outcome in less than half of patients; however, this may be related to the fact that the procedure is often done in dogs with chronic otitis externa, in which TECA would have been the preferred technique. A properly performed TECA combined with an LBO resolves clinical signs in most animals.

COMPLICATIONS

Partial or complete facial nerve paralysis occurs in many dogs after TECA with LBO. Other complications include persistent infection (dissecting cellulitis, prolonged wound drainage, incisional dehiscence, periauricular abscess formation), nystagmus, head tilt, postural abnormalities, and loss of hearing (see p. 311).

OTITIS MEDIA AND INTERNA

DEFINITIONS

Otitis media is inflammation of the middle ear; **otitis interna** is inflammation of the inner ear. **Myringotomy** is a surgical puncture of the tympanic membrane to relieve pressure or obtain samples for analysis. **Otoliths** are mineral opacities within the tympanic bullae. **Aural cholesteatomas** are an abnormal accumulation of keratin-producing squamous epithelium in the middle ear. **Primary secretory otitis media** (PSOM), also known as *glue-ear*, is a unique condition seen in Cavalier King Charles spaniels.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Otitis media may occur secondary to infection (i.e., bacterial, yeast, fungal), neoplasia, trauma, or foreign body. Otoliths have also been reported within the tympanic cavity of dogs and may or may not be associated with clinical signs. In cats, inflammatory and nasopharyngeal polyps (see p. 327) are additional causes of otitis media. Congenital palatine defects may also be associated with middle ear disease in dogs and cats. The most common cause in both dogs and cats is bacterial infection; more than half of animals with chronic end-stage otitis externa have documented evidence of otitis media at surgery. Consequently, pathogens cultured from the middle ear are similar to those cultured from the ears of animals with otitis externa (i.e., *Staphylococcus* spp., *Streptococcus* spp., *Pseudomonas* spp., *E. coli*, and *P. mirabilis*). In addition to spreading across the tympanic membrane to the middle ear, infections may ascend from the pharynx via the auditory tube or may reach the inner ear via the bloodstream. Bilateral otitis media is usually indicative of bacterial infection. Otitis media may lead to otitis interna (Box 17.4).

! BOX 17.4 Clinical Signs Associated With Otitis Interna (Vestibular Dysfunction)

- Head tilt to affected side
- Circling to affected side
- Falling to affected side
- Rolling to affected side
- Nystagmus (horizontal or rotary) with fast component away from affected side
- Asymmetric ataxia with strength preserved
- Positional or vestibular strabismus with the eyeball ipsilateral to the lesion deviated ventrally
- Postural reactions (except for the righting reflex)

Inflammatory or nasopharyngeal polyps are benign masses that may be located in the nasopharynx, auditory tube, or tympanic cavity, or all three. In rare cases they may rupture the tympanic membrane and protrude into the external ear canal. When located in the tympanic cavity, they often cause signs of unilateral otitis media. These polyps may occur as a result of ascending infection from the pharynx or may arise from chronic otitis media. Polyps thought to be congenital in origin have also been reported in kittens. Aural cholesteatomas are abnormal accumulations of keratin-producing squamous epithelium in the middle ear or tympanic recesses that may be congenital or acquired. Congenital cholesteatomas, although rare, are thought to arise from embryonal inclusions or nests of epithelial cells and are found behind an intact tympanic membrane and in the absence of causative factors such as a history of ear infections. PSOM in Cavalier King Charles spaniels is recognized by a bulging opaque tympanic membrane with an accumulation of mucus filling the entire middle ear. These dogs commonly present with signs of head and neck pain.[3]

Neoplasia originating in the middle ear is uncommon in both dogs and cats. In dogs, tumors that originate in the external ear canal and then extend into the tympanic cavity are more common than primary middle ear tumors. Benign tumors of the middle ear cavity of dogs (e.g., papillary adenomas and fibromas) have been more commonly reported than malignant tumors. Epidermoid cysts (cholesteatomas) occur in the canine middle ear. These cysts are often associated with chronic otitis media and must be differentiated from neoplastic lesions. Lesions associated with the trigeminal nerve can result in ipsilateral middle ear effusion.[4] In cats, squamous cell carcinoma is the most common tumor of the middle and inner ear. Other tumors found in the feline middle ear have included fibrosarcoma, anaplastic carcinoma, lymphoblastic lymphosarcoma, and ceruminous gland adenocarcinoma.

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DIAGNOSIS

Clinical Presentation

Signalment

Most animals that develop otitis media secondary to otitis externa are middle-aged. Older animals more commonly develop neoplasia of the middle ear, and young cats are more apt to have nasopharyngeal polyps. There is no known breed or gender predisposition in cats to nasopharyngeal polyps, or in either dogs or cats to neoplastic middle ear disease, aside from the previously mentioned Cavalier King Charles spaniels with PSOM. Canine breeds predisposed to otitis externa (see p. 314) also have a higher incidence of otitis media.

History

Many cats with nonneoplastic middle ear disease do not have associated clinical signs. The history and clinical signs of symptomatic animals with otitis media do not differ substantially from those of animals with otitis externa alone (see p. 314). Affected animals commonly scratch or paw at their ears, and they may shake their heads excessively. The ear may have an odor, and animals often appear to be in pain on manipulation or palpation of the ears or adjacent skull. A history of chronic, poorly responsive otitis externa is common. Some animals come for evaluation of vestibular signs caused by otitis interna (see Box 17.4). Pain during eating or when the mouth is opened may be noted, especially in cats with neoplastic middle ear disease. Ipsilateral facial nerve paralysis is also common in animals with middle ear neoplasia. In rare cases, neoplastic lesions of the middle ear may extend into the nasopharynx, causing gagging, retching, or dyspnea, or all three. Cats with nasopharyngeal polyps often have nasal discharge, sneezing, or stridor (see p. 327). If a nasopharyngeal polyp is present concurrently, dysphagia and/or dyspnea may be noted. Some nasopharyngeal polyps become so large that they protrude to the edge of the soft palate and cause gagging. Cats that have sinonasal disease with nasal exudate often have concurrent otitis media (which may be clinically silent). Evaluation of the external and middle ear in such cats is warranted. Deafness may be reported with bilateral disease, but loss of hearing is seldom evident with unilateral lesions.

Physical Examination Findings

Discharge from the external auditory canal, hyperplasia, and ulceration of the aural epithelial tissue are often obvious on physical examination in animals with otitis media and externa. Neurologic abnormalities referable to the inner ear and facial nerve paralysis (Box 17.5) are not found in most animals with otitis media. Horner syndrome may occur as a result of damage to the sympathetic trunk as it courses through the middle ear. Clinical signs associated with the syndrome are ptosis, miosis, enophthalmos, and protrusion of the third eyelid.

Otoscopic examination of these patients often requires general anesthesia, but it can be helpful in diagnosing otitis media, particularly as an alternative to advanced imaging. In one study, video otoscopy was 91% sensitive, 98% specific, and 97% accurate in diagnosing otitis media in dogs.[5] The tympanic membrane may be ruptured, or it may bulge outward because of purulent material, blood, mucus, or serum. However, an intact tympanic membrane does not rule out middle ear disease. Abundant mucus in the middle ear cavity has been associated with adenomatous lesions. The normal tympanic membrane appears shiny and is gray or white; it becomes opaque with infection. If the tympanic membrane has ruptured, samples for cytologic studies and culture can be taken directly from the middle ear. Otherwise, samples can be obtained using a 20-gauge, 3.5-inch (9 cm) spinal needle inserted through the ventral half of the tympanum (myringotomy). If the opening is too small, it may be enlarged with a small cannula.

Inflammatory polyps (see p. 327) usually appear as pedunculated, smooth, shiny, light-pink masses. The oral cavity of animals with inflammatory polyps or neoplastic masses should be carefully examined because extension into the nasopharynx may occur. Neoplastic lesions may be friable, but they are often difficult to differentiate grossly from chronically infected tissue.

! BOX 17.5 Clinical Signs Associated With Facial Nerve Paralysis

- Diminished palpebral reflex
- Widened palpebral fissure
- Drooping of the ear and lip
- Excessive drooling
- Blepharospasm
- Elevation and wrinkling of the lip
- Caudal displacement of the labial commissure
- Elevation of the ear on the affected side

Diagnostic Imaging

Radiography is not a sensitive indicator of middle ear disease, as nearly one-fourth of animals with middle ear disease will have normal appearing bullae on radiographs. If performed, the most valuable radiographic view in animals suspected of having otitis media is the frontal open-mouth projection (also known as the *rostrocaudal open-mouth view*) in which the animal is placed in ventrodorsal recumbency with the head flexed 80 to 90 degrees to the table (Fig. 17.17). The mouth is held open with gauze strips hooked on the upper and lower canine teeth, and the x-ray beam is centered on the temporomandibular joints. The tongue and endotracheal tube must be retracted from the field of view with gauze secured to the lower mandible. The tympanic bullae and their contents are demonstrated with this view. The most common findings with middle ear disease are opacification of the air-filled tympanic cavities and thickening and sclerosis of the walls of the bullae (Fig. 17.18). Lateral oblique

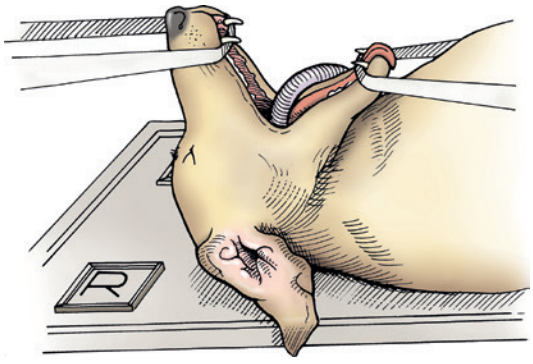


FIG. 17.17 Positioning for a frontal open-mouth projection.

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FIG. 17.18 Frontal open-mouth projection showing opacification, thickening, and sclerosis of the bullae *(arrowheads)*. (Courtesy L. Homco, Ithaca, NY.)

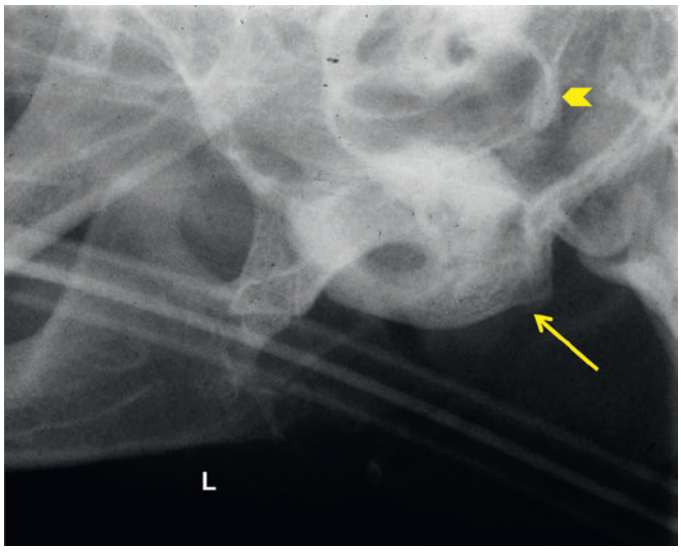


FIG. 17.19 Left lateral oblique radiograph of a dog with unilateral otitis media. Note the thickened wall of the left bulla *(arrow)* compared with the normal, thin-walled right bulla *(arrowhead)*.

views (Fig. 17.19) with the head tilted 10 to 15 degrees show individual bullae, whereas ventrodorsal or dorsoventral views show the external ear canals and the architecture of the petrous temporal bones. Lysis or periosteal reaction of the bullae and petrous temporal bone should increase suspicion of neoplasia. A soft tissue mass in the pharyngeal region on the lateral view may be noted in cats with inflammatory polyps. Ultrasound has been suggested as an adjunct to radiographic imaging of the tympanic bullae because it can be performed in dogs without the need for sedation or general anesthesia; however, in one study the sensitivity for detecting otitis media was low (21%).[5]

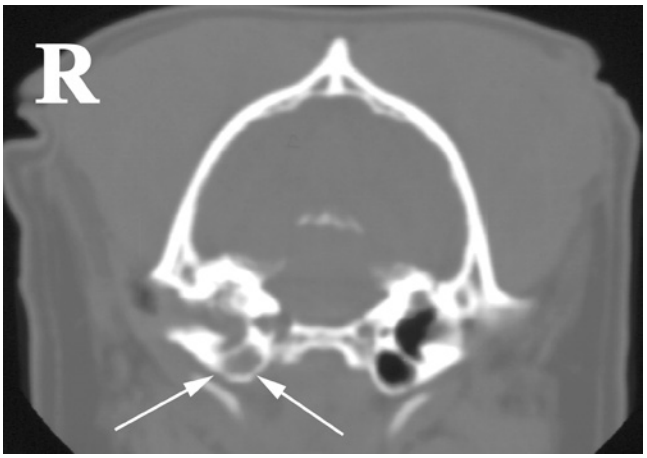


FIG. 17.20 Computed tomography axial image of a dog with otitis media. Soft tissue attenuating material is increased within the right tympanic bulla *(arrows)* without derangement of the bony aspect of the bulla.

CT is particularly useful for diagnosis of otitis media, evaluation of nasopharyngeal polyps, and determination of the extent of otitis externa or neoplasia (Fig. 17.20). Owing to collapse of the pharynx when the animal is under anesthesia, it may be difficult to identify a nasopharyngeal polyp using cross-sectional imaging techniques; reformatted sagittal images may be better. Direct visualization is always recommended after cross-sectional imaging examinations to better evaluate for the possibility of a nasopharyngeal polyp. On a well-positioned study, both bullae should appear symmetric. The tympanic bullae and external ear canal lumens should be filled with air, and the wall of the tympanic bulla should be thin and well defined. In animals with otitis media, thickening and irregularity of the tympanic bullae are often seen. Other findings may include lysis of the tympanic bulla, a soft tissue attenuating material within its lumen, and signs of otitis externa. Evaluation of the bone of the bullae is more difficult using magnetic resonance imaging, but it can be used to identify soft tissue changes that occur with otitis media and has the additional advantage of allowing evaluation of the brain in animals with signs of otitis interna. Middle ear disease may be an incidental finding on diagnostic imaging in cats that do not have clinical evidence of otitis media.

Laboratory Findings

Specific laboratory abnormalities are uncommon.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for disease of the middle and inner ear cavity include bacterial, fungal, or yeast infection; inflammatory polyps; neoplasms; foreign body; trauma; sebaceous gland and ceruminous gland hyperplasia; paraoral abscess; trigeminal nerve disease; and idiopathic vestibular disease. Otitis media is common in animals with otitis externa, but because the clinical signs are similar, the former often goes unrecognized.

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MEDICAL MANAGEMENT

The most important aspect of the treatment of otitis media is removal of infected tissues or exudate, neoplasms, polyps, or foreign bodies from the tympanic cavity. The external ear canal should be cleaned, and concurrent otitis externa must be treated (see p. 303). Medical management of animals with acute otitis media consists of myringotomy, cultures of middle ear contents, irrigation of the tympanic cavity, and topical and systemic antibiotics. Many veterinary dermatologists recommend starting oral fluoroquinolone pending culture and susceptibility results. If no improvement is seen in 3 to 4 weeks, ventral bulla osteotomy should be considered.

PSOM is a disease affecting predominantly Cavalier King Charles spaniels. Current treatment recommendations include repeated manual removal of the mucoid effusion from the tympanic cavity through a myringotomy incision and topical or systemic corticosteroids. Tympanostomy tubes have been placed by myringotomy incision in the pars tensa with the aid of an operating microscope. Clinical signs have resolved rapidly following this procedure, and it may be an acceptable alternative to repeated myringotomy for the treatment of PSOM.

SURGICAL TREATMENT

Surgery is often necessary to distinguish among the various possible causes of middle ear disease. Surgical treatment of otitis media caused by infection includes ventral bulla osteotomy, culture of affected tissue or exudate, drainage, and long-term antibiotics. Benign neoplastic or inflammatory lesions can usually be removed by a bulla osteotomy; however, Horner syndrome is a common, short-term complication of this procedure in cats (see p. 329). Neoplasia of the bulla warrants a poor prognosis.

NOTE If neurologic signs are present before surgery, be sure to warn the owner that they may persist after surgery.

Preoperative Management

Antibiotics may be given preoperatively; however, intraoperative cultures are often performed in animals with middle ear disease. In such patients, intravenous antibiotics should be given immediately after culture results have been obtained.

Anesthesia

See p. 302 and Table 17.1 for anesthetic recommendations for animals with ear disease. Chamber or mask induction should not be used in dyspneic animals (e.g., some cats with nasopharyngeal polyps).

Surgical Anatomy

The middle ear is composed of the tympanic cavity and its contents and the auditory tube. See p. 305 for a description of the surgical anatomy of the ear.

Positioning

The animal is positioned in lateral recumbency for an LBO and in dorsal recumbency for a ventral bulla osteotomy.

SURGICAL TECHNIQUE

Approach the middle ear by means of an LBO (see p. 309) in conjunction with TECA (see p. 308) or through a ventral bulla osteotomy (see p. 310).

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

See p. 310 for a description of surgical supplies necessary for a bulla osteotomy.

POSTOPERATIVE CARE AND ASSESSMENT

Cats with concurrent upper airway disease (e.g., nasopharyngeal polyps) may have respiratory distress after extubation and may require supplementary oxygen. Oxygen may be given by mask or nasal insufflation in these animals. Bilateral ventral bulla osteotomies, particularly in cats, may be associated with postoperative respiratory distress.

PROGNOSIS

Animals with bacterial otitis media may have persistent neurologic signs despite surgical treatment. Many cats with otitis interna before surgery will have permanent head tilts after surgery; however, these cats often have a normal level of activity despite their neurologic dysfunction. The prognosis for benign tumors is good, but surgical cure is rare with malignant tumors because of their extensive nature at the time of diagnosis. Inflammatory polyps may recur if they are simply removed from the external ear using traction (see p. 327). Recurrence is less likely if traction and bulla osteotomy are performed.

Although surgery can be curative, approximately half of dogs with aural cholesteatomas will have persistent or recurrent clinical signs. Dogs with chronic disease, temporal bone involvement, and neurologic signs tend to have a worse prognosis but can still be medically managed long-term with palliation of clinical signs.[6]

AURAL HEMATOMAS

DEFINITION

An **aural (auricular) hematoma** is a collection of blood within the cartilage plate of the ear.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Aural hematomas may occur in dogs or cats and are usually characterized as fluctuant swellings filled with hemorrhagic fluid, affecting part or all of the concave surface of the pinna. The cause of aural hematoma is not well understood; however, in many cases, it appears to be the result of head shaking or scratching at the ear caused by pain or irritation associated with otitis externa. The latter is usually bacterial in dogs and is due to *O. cynotis* infestation in cats. Head shaking may cause sinusoidal wave motions in the ear, resulting in fracture of the cartilage. The exact location of the source of hemorrhage is not known but is thought to come from branches of the great auricular arteries and veins within, under, or between the cartilage layers.

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These vessels penetrate the scapha to supply the concave surface of the ear.

Some animals that develop aural hematomas do not have evidence of concurrent ear disease; hematoma formation in some patients may be associated with increased capillary fragility (e.g., Cushing disease). An additional theory with regard to etiology involves underlying autoimmune disease. Some dogs and cats with aural hematomas have been found to have a positive Coombs test in the serum and fluid retrieved from the pinna, whereas a small percentage had positive antinuclear antibody tests and identification of immunoglobulin G deposition at the dermo-epidermal junction.

DIAGNOSIS

Clinical Presentation

Signalment

Dogs and cats with otitis externa have a greater risk of developing aural hematoma.

History

A history of violent head shaking and/or acute or chronic otitis externa (see p. 312) may be noted; some animals may have no history of previous ear disease.

Physical Examination Findings

Hematomas initially appear fluid filled, soft, and fluctuant, but eventually may become firm and thickened as a result of fibrosis. The ear may develop a "cauliflower" appearance.

Diagnostic Imaging

Skull radiography or CT may be indicated if underlying otitis externa or media (or both) has predisposed the animal to aural hematoma.

Laboratory Findings

Specific laboratory abnormalities are uncommon.

DIFFERENTIAL DIAGNOSIS

An aural hematoma is diagnosed during the physical examination; however, the underlying ear disease must be diagnosed and treated to reduce the likelihood of recurrence.

MEDICAL MANAGEMENT

Simple needle aspiration to drain the hematoma can be performed, but recurrence is likely. When this pathway is chosen, daily drainage of the hematoma has been advocated to prevent early recurrence. The concave surface of the pinna should be clipped and prepped before a large (16–20 g) hypodermic needle is inserted in the most dependent part of the ear. Flushing with sterile saline can be performed to facilitate removal of clots and fibrin. Other methods of drainage have been described with variable outcomes (Box 17.6). Classically, drains are inserted and secured on the concave (inner) surface of the pinna.

The use of corticosteroids administered by a variety of routes has been described to treat aural hematomas. Daily intravenous administration of dexamethasone (0.5–2.0 mg/kg) results in resolution in over 85% of cases. Oral administration of antiinflammatory doses of prednisone can help decrease head shaking and scratching and may help treat inflammation associated with ear disease. Injection of dexamethasone, methylprednisolone, and triamcinolone (Box 17.7) directly into the hematoma cavity following drainage of fluid has successful resolution in over 90% of cases.

BOX 17.6 Methods of Aural Hematoma Drainage

Teat cannula
Silastic tubing
Penrose drain
Butterfly catheter (closed-suction drainage)

BOX 17.7 Reported Dosages of Intralesional Corticosteroids

Dexamethasone 0.2–0.4 mg in saline q24h for 1–5 days
Methylprednisolone 0.5–1.0 mL q7d for 1–3 weeks
Triamcinolone 0.1–1.0 mL q7d for 1–3 weeks

SURGICAL TREATMENT

Numerous techniques have been described for surgical treatment of aural hematomas. The goals of surgery are to remove the hematoma, prevent recurrence, and retain the natural appearance of the ear (i.e., minimize thickening and scarring). The most commonly used procedure involves incising the tissue overlying the hematoma, evacuating blood clots and fibrin, and holding the cartilage in apposition with sutures until scar tissue can form. To prevent enlargement or fibrosis, hematomas should be treated soon after they occur, preferably within several days.

Preoperative Management

Concurrent otitis externa (see p. 315) should be treated simultaneously. Appropriate cultures should be submitted and the ear canal cleaned and flushed.

Anesthesia

Animals with aural hematomas are usually healthy, and a variety of anesthetic protocols can be used. Tranquilization may be necessary on recovery from anesthesia once sufficient analgesics have been provided (see p. 303). See p. 302 for information on the anesthetic management of animals with ear disease.

Surgical Anatomy

Structural support of the pinna is provided by cartilage interposed between the two skin surfaces. Branches of the great auricular arteries and veins supply the pinna. These main vessels are located along the convex surface of the ear, and small branches penetrate the scapha to supply the concave surface. Sensory innervation to the ear is supplied by the second cervical nerve (convex surface) and by the auriculotemporal branches of the trigeminal nerve (concave surface).

Positioning

Patients are generally placed in lateral recumbency for aural hematoma and laceration repair.

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SURGICAL TECHNIQUES

Aural Hematomas

Make an S-shaped incision on the concave surface of the ear and expose the hematoma and its contents from end to end (Fig. 17.21). Remove the fibrin clot and irrigate the cavity. Using 2-0 to 3-0 monofilament nonabsorbable suture (e.g., polypropylene, polybutester), place 0.75- to 1-cm long sutures through the skin on the concave surface of the ear and underlying cartilage. Place the sutures parallel to the major vessels (vertical rather than horizontal). They may be placed through the cartilage without incorporating the skin on the convex surface of the ear, or they may be full thickness. Place an ample number of sutures so that no pockets are left in which fluid can accumulate. Do not ligate the branches of the great auricular artery, visible on the convex surface of the ear. Do not suture the incision closed; it should gap slightly to allow for continued drainage. Place a light protective bandage over the ear, and support the ear over the animal's head (see p. 311, "Postoperative Care and Assessment"). Remove the bandage and sutures in 14 to 21 days.

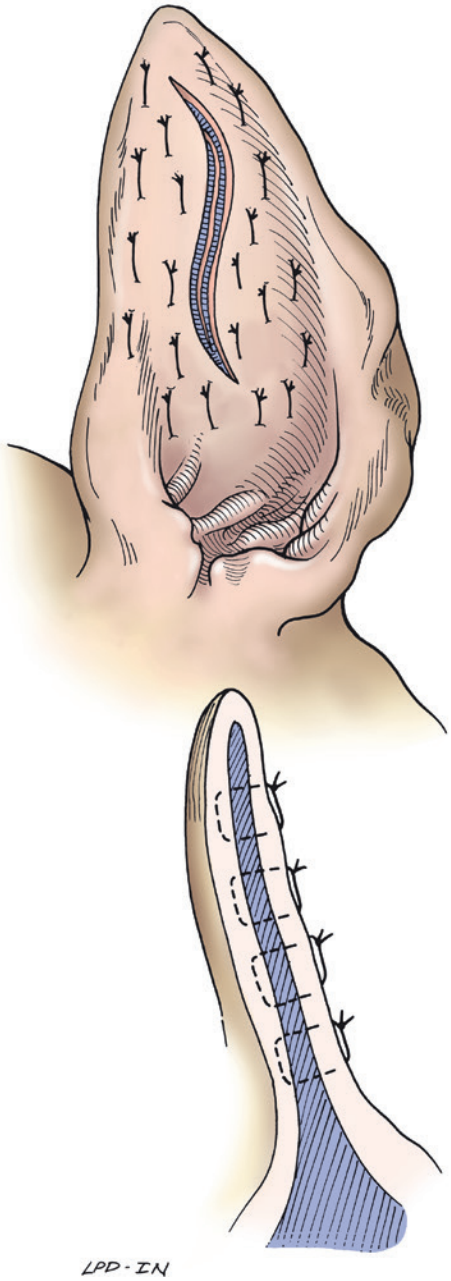


FIG. 17.21 Sutures should be placed vertically rather than horizontally for aural hematoma repair. They may be placed through the cartilage without incorporating the skin on the convex surface of the ear, or they may be full thickness.

An alternative surgical technique has recently been described in 23 dogs (Fig. 17.22).[7] No recurrence was documented in any of the dogs, and 91% had no auricular deformity. The advantages of this technique proposed by the authors include minimal need for aftercare, absence of discomfort or irritation caused by external sutures, and sutures that do not need to be removed.

Create a longitudinal incision on the concave surface of the pinna. Place several parallel suture lines intradermally on either side of the incision using monofilament absorbable suture (3-0 or 4-0 glycomer 631 or poliglecaprone 25).

Other surgical techniques described for treatment of aural hematomas involve the creation of circular fenestrations using either a 4-mm dermal biopsy punch or a CO_2 laser. When using a dermal punch, multiple partial-thickness circular plugs of tissue are removed approximately 0.5 to 1.0 cm apart from each other over the entire area of the hematoma. The skin edge of each incision is tacked through the cartilage using a 3-0 or 4-0 monofilament suture material in a simple interrupted pattern.

When using a CO_2 laser, a 1-cm circular partial-thickness incision is made in the most dependent part of the hematoma to facilitate drainage and flushing of the hematoma cavity. Multiple 1- to 2-mm full-thickness circular incisions are then made over the entire surface of the hematoma, with the goal being to stimulate adhesion formation between the cartilage layers due to fibrosis during cartilage healing. Outcomes and cosmetic appearance are reasonable, with no reports of hematoma recurrence.

A sutureless surgical technique for addressing aural hematomas is also available (Hematoma Repair System). Following the creation of the longitudinal incision on the concave surface of the pinna, preshaped silicon pads are placed on either side of

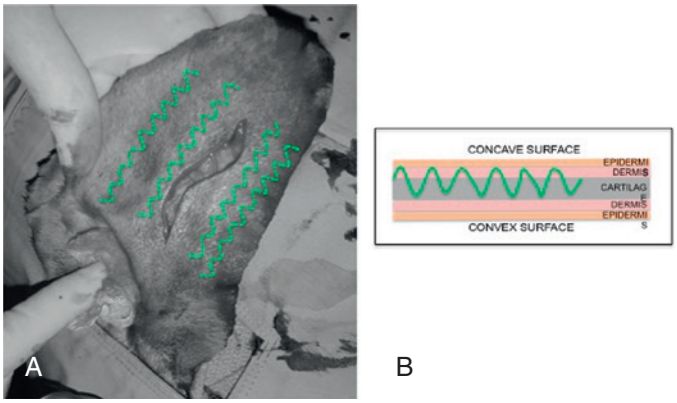


FIG. 17.22 Alterative internal suturing technique for aural hematomas. (A) One to three intradermal suture lines are placed parallel to the incision made in the concave surface of the pinna. (B) Longitudinal cross section of the pinna; the continuous suture line is placed from the concave dermis through the cartilage but not through to the convex surface.

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the pinna. A needle is passed through the pad from medial to lateral, facilitating placement of locking clips and rings over the entire surface of the ear. Pads are left on for up to 3 weeks, but daily inspection is required to observe for evidence of pressure necrosis.

POSTOPERATIVE CARE AND ASSESSMENT

A bandage can be used to protect the ear from contamination and self-inflicted trauma after hematoma repair. Maintaining bandages on the head can be difficult. One method is to place short strips of tape on the rostral and caudal margin of the convex surface of the pinna (Fig. 17.23A). The tape should extend beyond the ear border. Longer pieces of tape are placed on the concave surface of the pinna so that these tape pieces contact the tape on the convex surface (see Fig. 17.23B). The ear is placed over the top of the head (cotton can be placed between the ear and the top of the head to support the ear), and a nonadherent pad is placed over the incision (see Fig. 17.23C). The long pieces of tape are applied to the skin. Cast padding and Kling are applied over the ear (the unaffected ear on the other side is not incorporated into the bandage), and Vetrap (3M) or stockinette (cut a hole for the unaffected ear) can be placed as the external layer (see Fig. 17.23D). The bandage can then be secured to the head cranially or caudally with Elastikon (Johnson & Johnson) or 1-inch (2.5 cm) tape that is applied to both the hair and the bandage.

Alternatively, the ear can be secured using a hematoma pad. The pad is secured to the ear using additional horizontal mattress sutures. Hematoma pads are available commercially (Buster Othaematoma Compress), but they can also be made using simple materials found in most veterinary hospitals (Box 17.8; Fig. 17.24).

NOTE Be sure to check head bandages periodically to ensure that they are not too tight and are not restricting breathing.

PROGNOSIS

Aural hematomas seldom recur if they are properly addressed and if any underlying ear disease is recognized and appropriately treated. There can be changes in the cosmetic appearance of the

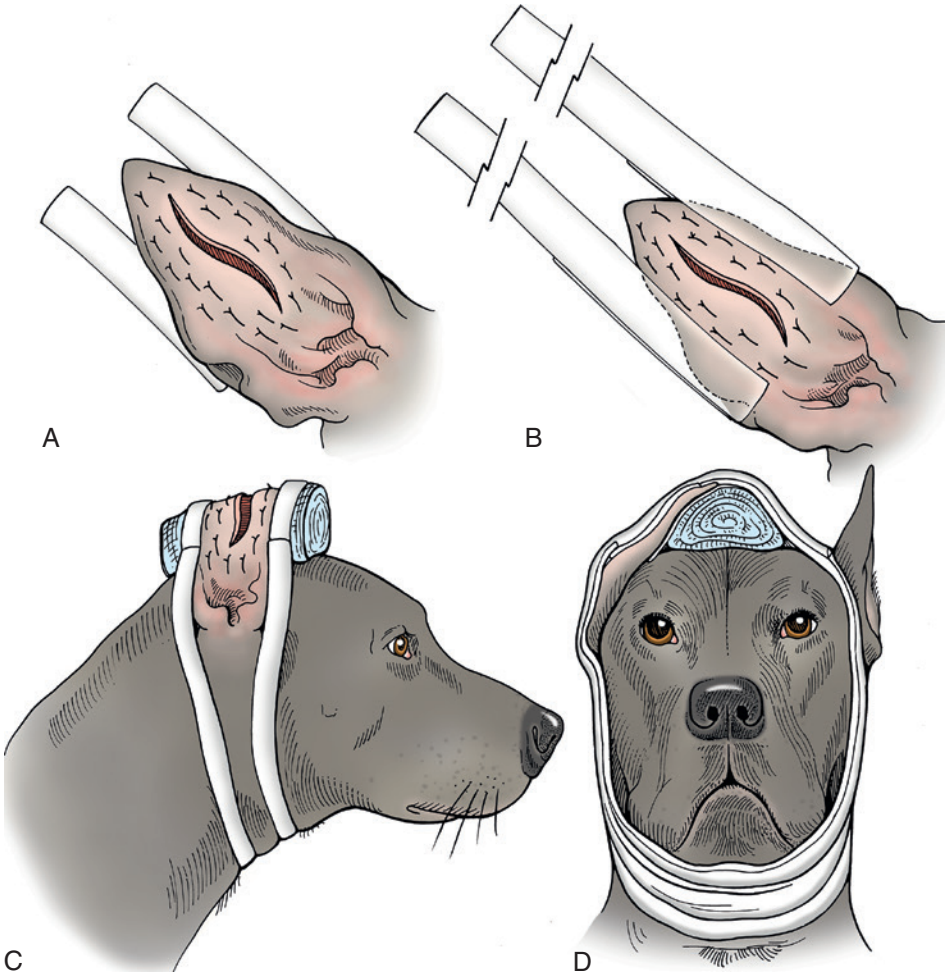


FIG. 17.23 Bandaging the ear after a surgical procedure. (A) Place short strips of tape on the rostral and caudal margins of the convex surface of the pinna. (B) Use longer pieces of tape on the concave surface of the pinna so that these tape pieces contact the tape on the convex surface. (C) Place the ear over the top of the head and place a nonadherent pad over the incision. (D) Apply cast padding and Kling over the ear, then use Vetrap or stockinette as an external layer.

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BOX 17.8 Aural Hematoma Pad

Materials

- Piece of used radiographic film
- Used surgical scrub brushes
- Household cyanoacrylate (e.g., Super Glue)

Technique

1. Remove the sponge from the scrub brush and split it in half to decrease the thickness of the sponge.
2. Trace the outline of the pinna on the piece of radiographic film.
3. Glue the sponges to the radiographic film to completely cover the outline of the pinna.
4. Once the glue has dried, cut along the trace of the pinna outline.
5. Attach the aural hematoma pad to the concave aspect of the pinna with three or four full-thickness mattress sutures (see Fig. 17.24B–C).
6. The pad is removed in 3 days. The remaining sutures are removed in 3 weeks (see Fig. 17.24D).

pinna; cats or dogs with erect or semierect ears may lose carriage of the pinna.

TRAUMATIC LESIONS OF THE PINNA AND EAR CARTILAGE

The ear may be lacerated as a result of fighting or other trauma. These wounds may be superficial, involving the skin on one surface of the ear only, or may perforate the cartilage and involve both skin surfaces. Depending on the severity of the wounds, some may be left to heal by secondary intention, whereas others will have a more cosmetic appearance if sutures are placed. In rare cases, a portion of the ear may be avulsed, resulting in an unacceptable cosmetic deformity.

Traumatic separation of the annular and auricular cartilages may occur in dogs and cats; however, it is rare. Treatments have included TECA and LBO or vertical canal ablation. Primary

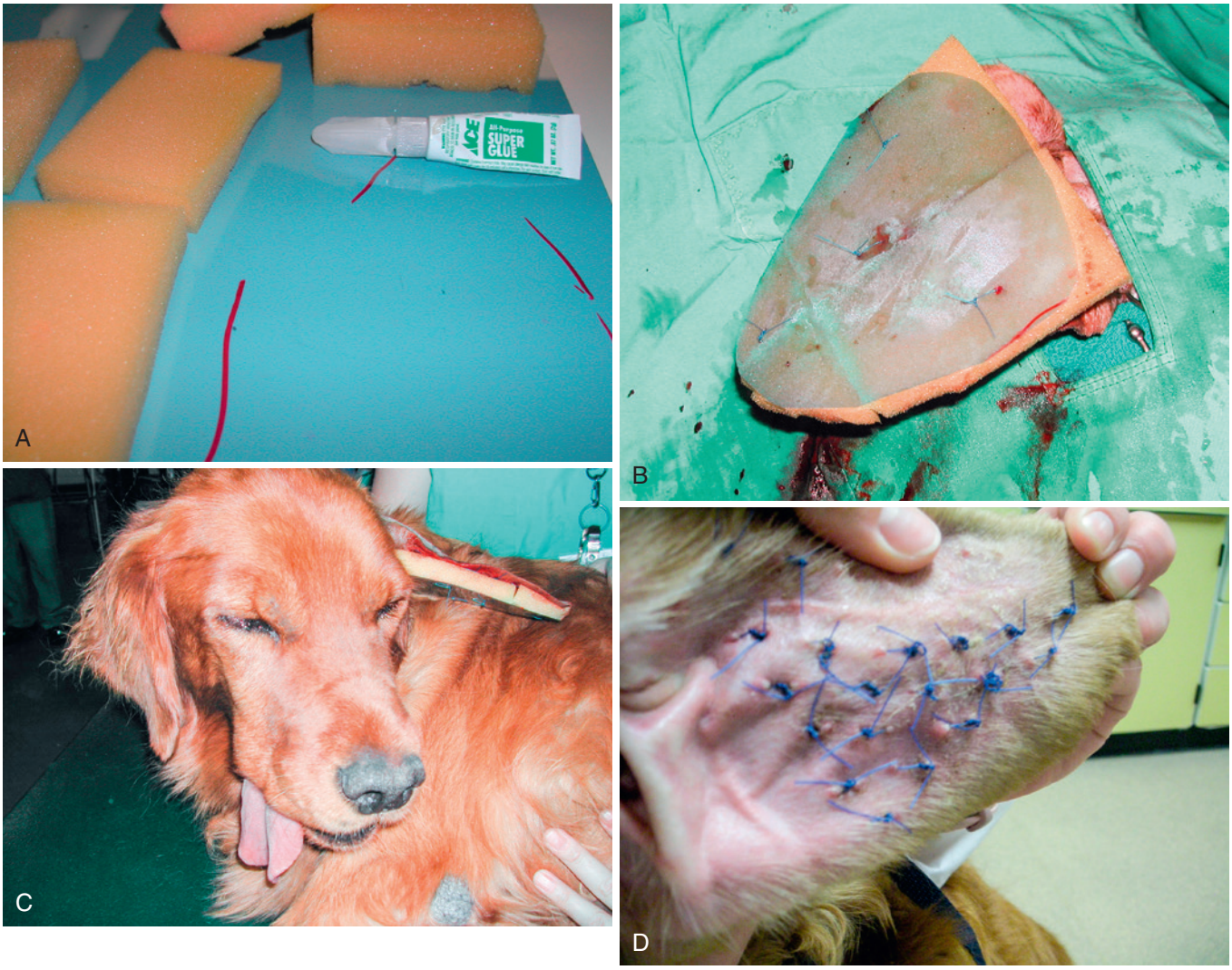


FIG. 17.24 The makings of an aural hematoma pad. (A) Materials needed include used surgical sponges, household cyanoacrylate glue, and used radiographic film. (B) The aural hematoma pad is attached directly to the concave aspect of the pinna with three to four full-thickness sutures. (C) The postoperative appearance following placement of an aural hematoma pad. (D) Appearance of the pinna following removal of the aural hematoma pad.

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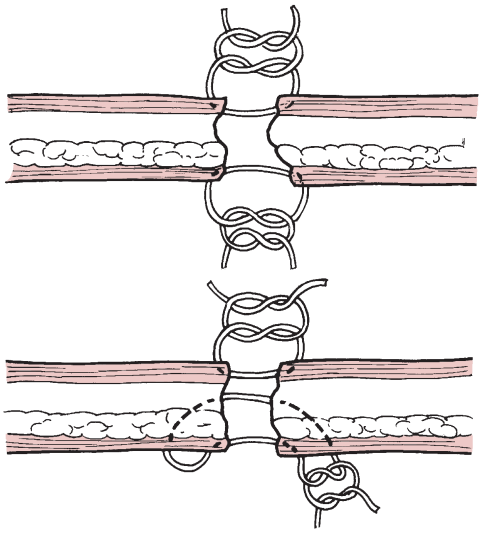


FIG. 17.25 Suture placement for repair of pinna lacerations.

repair of the traumatic injury using a caudal approach can be attempted.

Linear lacerations that involve only one skin surface may be left to heal by secondary intention or can be sutured. The laceration should be cleaned and the edges debrided if necrotic tissue is present. The skin margins can be apposed with simple interrupted sutures. If a flap of tissue has been elevated away from the cartilage, it should be sutured. Sutures are placed through the skin at the margins of the wound. Sutures also should be placed through the skin and cartilage at the center of the flap to obliterate any dead space where fluid might collect. Full-thickness injuries through the ear margin should be sutured. The skin on both sides of the defect may be sutured with simple interrupted sutures, or, as an alternative, a vertical mattress suture can be used to appose the skin and cartilage on one side of the ear and simple interrupted sutures used to appose the skin on the opposite side of the ear (Fig. 17.25).

Avulsions of the Ear Margin

Small avulsions of the ear margin may be treated by resecting surrounding tissue to restore a normal ear contour. The skin edges are sutured over the cartilage using a continuous suture pattern. Larger defects of the ear may be repaired using a pedicle flap obtained from the side of the neck in dogs with pendulous ears or from the dorsum of the head in dogs with erect ears.

NOTE Defects of the ear margins can be repaired for cosmetic reasons, but repair should be delayed after excision of neoplasms until it has been determined that recurrence is unlikely.

Prepare the ear and donor site for aseptic surgery. Debride the margins of the ear defect. Place the ear on the donor site and incise the skin, extending the limbs of the incision 0.5 to 1 cm longer than the defect (Fig. 17.26A). Suture the flap to the skin on the convex surface of the ear (see Fig. 17.26B). Place a nonadherent dressing over the wound and leave the ear bandaged for 10 to 14 days. Then, sever the flap from the donor site in the shape of the defect on the concave side of the ear (see Fig. 17.26C). Gently fold the flap over the ear margin and suture it to the skin (see Fig. 17.26D). Remove skin sutures in 10 to 14 days.

NEOPLASIA OF THE PINNA AND EXTERNAL EAR CANAL

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Neoplasms of the external ear canal are relatively uncommon in dogs and cats; however, they may arise from any structure that lines or supports the ear canal. Aural tumors in the dog and cat can be histologically benign or malignant. The most common malignant tumors of the external ear canal are ceruminous gland adenocarcinomas, squamous cell carcinomas, and carcinomas of undetermined origin. Benign aural conditions include inflammatory polyps (see p. 327), aural cholesteatomas (epidermoid cysts), basal cell tumors, papillomas, histiocytomas, fibromas, and ceruminous gland adenomas, among many others. Aural tumors tend to be more aggressive in cats than in dogs. Most canine ceruminous gland tumors are benign, but such tumors in cats are usually malignant.

Although tumors of the external ear canal are more common than those arising in the internal or middle ear cavity, clinical signs of middle or inner ear disease may predominate if these tumors extend through the tympanic membrane (see p. 317). Squamous cell carcinoma in the bullae with bone destruction may cause severe pain in the region of the temporomandibular joint. Neoplasms of the external ear are frequently associated with concurrent bacterial and yeast infections. It has been hypothesized that chronic otitis causes hyperplasia, which may eventually induce dysplastic and neoplastic changes. The mere presence of a tumor in the ear canal often obstructs drainage, resulting in otitis externa.

Any tumor that affects the skin may arise on the pinna, but the most common tumor of the pinna in cats is squamous cell carcinoma. These tumors are most often diagnosed in older cats, particularly white ones (Figs. 17.27 and 17.28). The association between lack of protective pigmentation and occurrence of these tumors suggests that solar radiation is a causative factor. Although these tumors are highly invasive, metastasis is uncommon. If metastasis does occur, it is usually seen in the regional lymph nodes and lungs. Tumors may be noted on the nares and eyelids.

Other tumors of the pinna of dogs and cats include melanoma, fibrosarcoma, basal cell tumor, fibroma, lymphoma, histiocytoma, papilloma, and mast cell tumor. A recent study found that dogs with mast cell tumors on the pinna, without evidence of gross metastasis, have a similar prognosis to mast cell tumors in other haired areas of the body following treatment with surgical excision, unlike mast cell tumors on other parts of the head.[8]

DIAGNOSIS

Clinical Presentation

Signalment

Most neoplastic lesions of the external ear are found in middle-aged or older animals. Older male cats may be at increased risk of developing ceruminous gland tumors of the ear canal. Squamous cell carcinoma of the pinna occurs almost exclusively in older white-eared cats or in multicolored cats with little pigmentation of the pinna.

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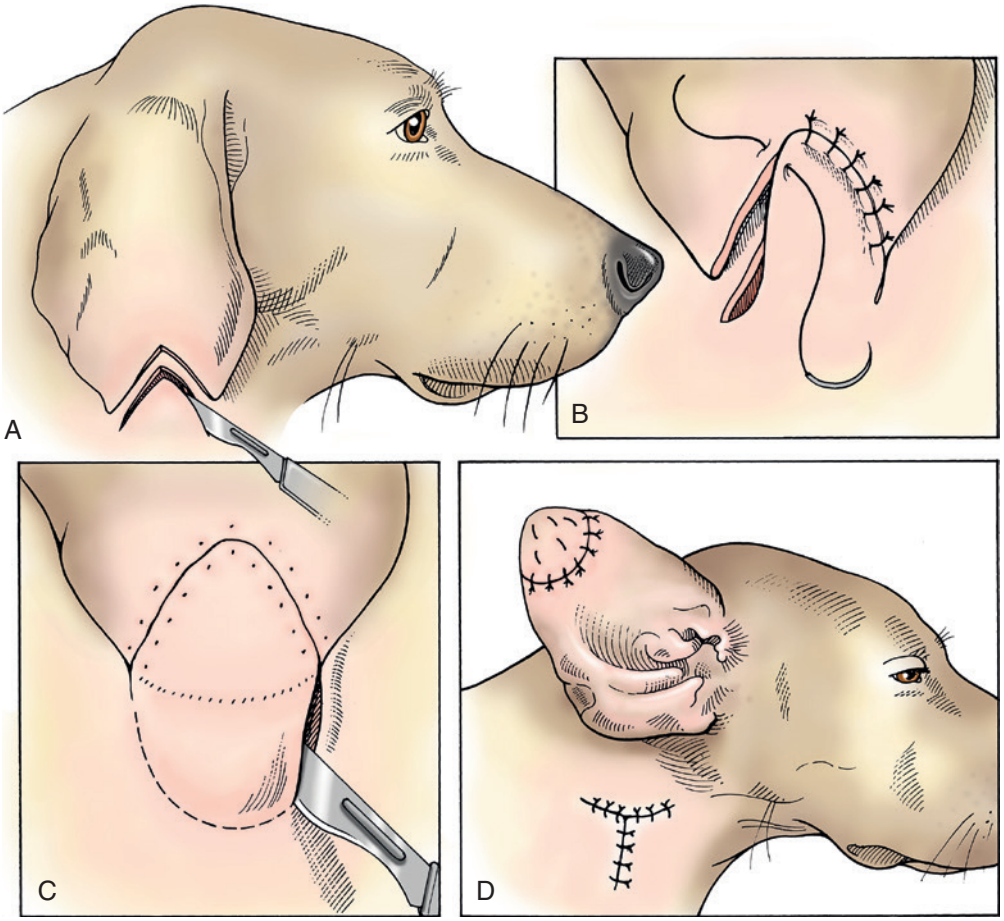


FIG. 17.26 Repair of pinnal defects. (A) Place the ear on the donor site and incise the skin, extending the limbs of the incision 0.5 to 1 cm longer than the defect. (B) Suture the flap to the skin on the convex surface of the ear. (C) After 10 to 14 days, sever the flap from the donor site in the shape of the defect on the concave side of the ear. (D) Gently fold the flap over the ear margin and suture it to the skin.

History

The history of a patient with a tumor arising from the external ear canal usually differs minimally from that of a patient with primary bacterial otitis externa (see p. 317). The history of cats with squamous cell carcinoma is often insidious and begins with the owner intermittently noticing crusty, eczematous lesions at the edge of the ear.

Physical Examination Findings

Small, pedunculated masses of the external ear canal suggest ceruminous gland hyperplasia or adenomas, papillomas, or inflammatory polyps. Infiltrative masses suggest ceruminous gland adenocarcinoma (Fig. 17.29). Squamous cell carcinoma usually originates on the tips of the ears, where little hair is present, and initially may appear as hyperemic skin. As the lesions progress, erosion, ulceration, crusting, and thickening become noticeable. The ear may bleed with mild trauma.

Diagnostic Imaging

Radiographic signs of neoplasia (i.e., bony lysis of the petrous temporal bone) may be noted on skull radiographs of animals with neoplasia of the external ear canal. CT is more sensitive for detection and evaluation of the extensiveness of lesions and is recommended (Fig. 17.30). Although metastasis usually occurs late in the course of the disease, pulmonary metastasis may be noted with some ear tumors; therefore thoracic radiographs are also recommended.

Laboratory Findings

Definitive diagnosis of ear neoplasia requires cytology or histopathology. Fine needle aspirate and cytopathology of external ear masses in the cat accurately distinguish inflammatory polyps from neoplasia. For differentiation of benign proliferation and malignant neoplasia, however, histopathology is recommended.

DIFFERENTIAL DIAGNOSIS

Neoplastic lesions of the ear pinna must be differentiated from nonneoplastic lesions, such as dermatitis caused by insect bites or immune-mediated lesions. Suspicious lesions should be biopsied to improve chances of early, complete resection.

MEDICAL MANAGEMENT

Squamous cell carcinoma may be prevented or reduced by applying sunscreen to nonpigmented areas of the ear and

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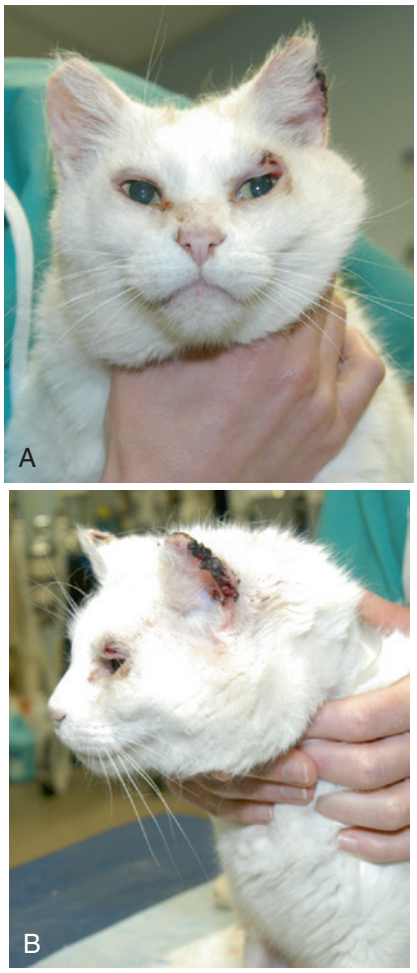


FIG. 17.27 Adult cat with squamous cell carcinoma of the pinnae and periorbital regions. (A) Rostral and (B) left lateral views.

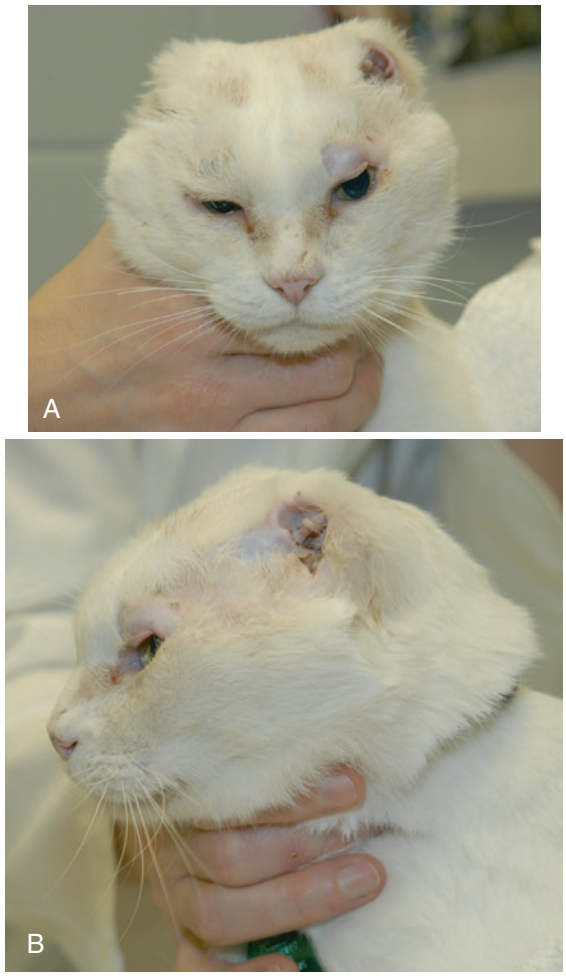


FIG. 17.28 Same cat in Fig. 17.27 1 month after bilateral pinnectomy and strontium therapy for squamous cell carcinoma. (A) Rostral and (B) left lateral views.



FIG. 17.29 Ceruminous gland adenocarcinoma on the pinna of a 6-year-old dog presented for treatment of chronic otitis externa.

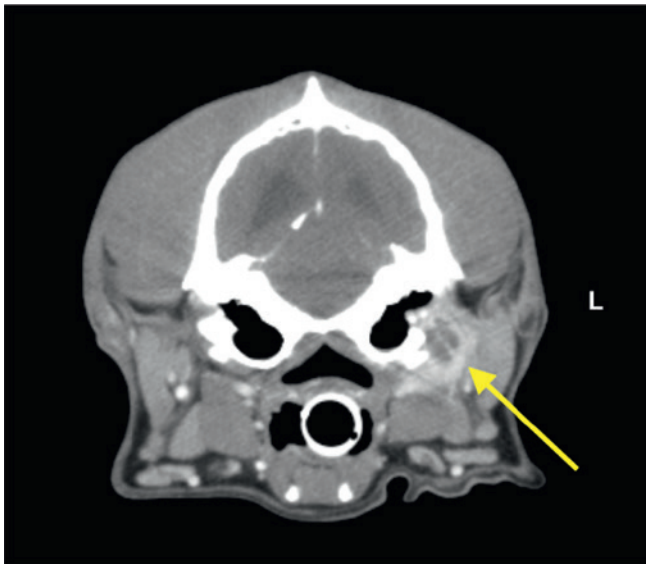


FIG. 17.30 Contrast-enhanced computed tomography axial image of a dog with an expansile neoplastic mass of the external and middle ear *(arrow)*.

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preventing physical exposure to ultraviolet radiation. Cryotherapy and radiation are alternatives to surgical removal of the pinna. Cryotherapy may be curative in small, superficial tumors, but local recurrence is common. Radiation therapy is less disfiguring than surgical removal of lesions and is a viable alternative for small, superficial tumors and preneoplastic lesions.

SURGICAL TREATMENT

For neoplasms of the external ear canal, vertical ear canal ablation or TECA with LBO is usually required (see pp. 307–309). Local treatment options for squamous cell carcinoma of the pinna include surgical resection, cryosurgery, and photodynamic therapy. For cats with more aggressive or advanced disease, systemic chemotherapy may provide limited improvement in survival times. The aim of surgical treatment of malignancies of the pinna is to remove the neoplasm with a wide margin of normal surrounding skin. This may require pinnectomy alone or a vertical ear canal ablation and removal of the pinna. The owner should be prepared for the resulting cosmetic deformity.

Preoperative Management

If concurrent otitis externa is present, perioperative antibiotics based on culture results should be given. Preoperative cytologic studies can help determine whether radical resection is necessary when neoplasia is suspected.

Anesthesia

See p. 302 for anesthetic recommendations for animals with ear disease.

Surgical Anatomy

See p. 305 for a description of the surgical anatomy of the ear canal and pinna.

Positioning

Positioning for ear surgery is described in the discussion of surgical techniques on pp. 306 to 310.

SURGICAL TECHNIQUE

The most important aspect of surgery for ear neoplasms is achieving wide margins to prevent local recurrence; this may require removal of the entire pinna and ear canal. If aggressive surgical therapy cannot provide clean margins, adjunctive therapy (e.g., radiation) should be considered. See pp. 306 to 310 for a description of surgical techniques commonly used for diseased or neoplastic ears.

NOTE Malignant ear tumors must be excised with wide margins of normal tissue. The owner should be advised of the resulting cosmetic defect before surgery is planned.

For pinnectomy, remove the affected portion of the ear and suture the remaining skin over the exposed cartilage. For small tumors on the central portion of the convex surface of the pinna, resect the neoplasm and mobilize the skin around the defect by undermining between the cartilage and the skin. Suture the skin margins or, if necessary, leave the defect open to heal by secondary intention under a light bandage. For small tumors on the concave surface of the ear, repair the skin defect by elevating a flap from surrounding skin and rotating it into the defect. Suture the flap to the wound margins. After 10 to 14 days, transect the flap and suture the edge to the defect. Close the donor site primarily.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

See p. 310 for a discussion of suture materials and surgical instruments for ear surgery.

POSTOPERATIVE CARE AND ASSESSMENT

An Elizabethan collar or sidebar should be used to prevent the animal from scratching the ear after surgery. Ear surgery is painful, and perioperative analgesics such as hydromorphone or fentanyl or FLK CRI (see p. 144) should be provided (see also Table 17.1). If the animal appears dysphoric or anxious, tranquilizers may be given once the patient has received adequate postoperative analgesics (see p. 303 and Table 17.1).

PROGNOSIS

For malignant ceruminous gland tumors of the external ear, TECA with LBO is seldom curative, and adjunctive therapy (radiation therapy) should be considered. Local recurrence of squamous cell carcinoma is common if wide margins are not obtained at surgery. The prognosis is poor with squamous cell carcinoma of the middle and inner ear; however, amputation of the pinna for squamous cell carcinoma of the ear margin may be curative.

INFLAMMATORY POLYPS

DEFINITION

Inflammatory polyps are benign, fibrous, pedunculated masses that may be found in the oropharynx, middle ear, or external ear canal. They are also known as *nasopharyngeal, middle ear, otopharyngeal,* or *aural polyps.*

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Feline inflammatory polyps are benign growths that originate from the middle ear or the auditory tubes of young cats. True nasopharyngeal polyps extend into the pharyngeal cavity causing signs of upper airway obstruction, including stertor, nasal discharge, gagging, dysphagia, and dyspnea. Aural polyps extend from the middle ear through the tympanic membrane and into the external ear canal, resulting in symptoms of otitis externa. More severe signs include evidence of middle ear disease such as Horner syndrome or facial nerve paresis, or inner ear disease such as head tilting, ataxia, and nystagmus.

The exact origin and cause are unknown; however, it is thought that inflammatory polyps arise as a result of a prolonged inflammatory process. It is unclear whether this inflammation initiates or potentiates the development and growth of inflammatory polyps. Bacterial cultures are generally negative, and organisms are not usually seen on histopathology. A viral cause has been proposed as affected cats often have a history of upper respiratory infection.

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DIAGNOSIS

Clinical Presentation

Signalment

Inflammatory polyps predominantly affect young cats; the reported mean age ranges from 14 months to 3 years. However, polyps have also been reported in older cats. No breed or sex predisposition has been identified. Although less commonly diagnosed, inflammatory polyps of the ear canal may occur in dogs.

History

Most cats present for evaluation of dysphagia or upper respiratory signs, such as stertorous respiration, nasal discharge, sneezing, voice change, and/or dyspnea. They may present for signs of otitis externa (head shaking and scratching at the ear). Occasionally, the animal may be seen for acute onset of head tilt, nystagmus, and/or vestibular imbalance. Clinical signs may be present for months before evaluation.

Physical Examination Findings

Most inflammatory polyps are unilateral, although they may be bilateral. Concurrent otic and nasopharyngeal polyps may be present. These masses tend to be pink and pedunculated but may be ulcerated to varying degrees. Polyp extension into the external canal may appear as a dark ceruminous mass on otoscopic examination, or may be red, pink, or white. These masses are often covered by mucus and/or blood. The tympanic membrane may appear distorted and discolored if the mass has not extended through it. Evidence of upper respiratory obstruction is present in some cats, and secondary infection, such as nasal discharge, rhinitis, and/or sinusitis, may also be noted.

The area dorsal to the soft palate can be evaluated by digital palpation of the soft palate and using a dental mirror and a spay hook, or by using a small-diameter flexible endoscopy tube that can be retroflexed over the soft palate (Fig. 17.31). The latter technique has the advantage of allowing excellent visualization and provides an opportunity to obtain biopsies and brush samples for cytology and culture if a mass is not present. Care should be taken when anesthetizing cats with possible upper airway obstruction due to the potential for respiratory decompensation.

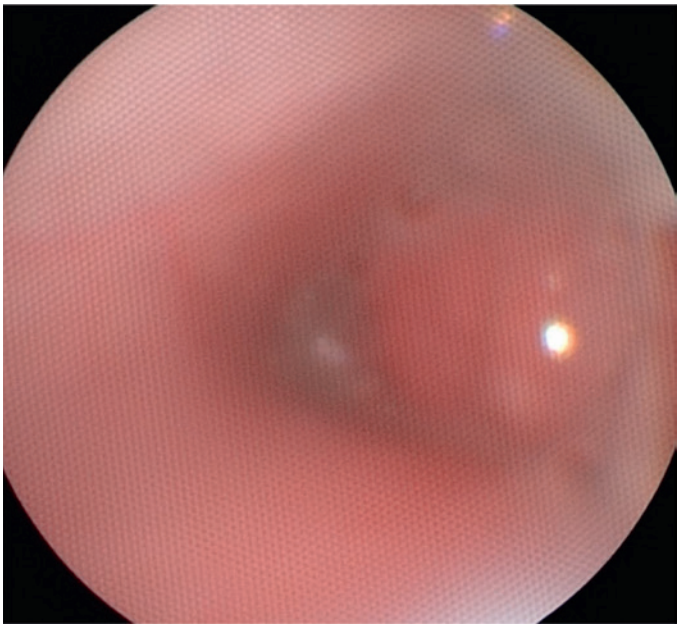


FIG. 17.31 Retroflexed endoscopic image of a nasopharyngeal inflammatory polyp in a 3-year-old cat.

Diagnostic Imaging

Well-positioned lateral radiographic images of the pharyngeal region are most useful for diagnosing nasopharyngeal polyps, yet CT is the gold standard for determining the extent of involvement and whether the condition is unilateral or bilateral before surgery (Fig. 17.32). On CT, a soft tissue attenuating mass may be seen extending from the middle ear into the lumen of the external ear canal. Alternatively, a soft tissue mass may be seen in the nasopharyngeal region (although collapse of the pharyngeal region during anesthesia can hinder this evaluation). Nasopharyngeal polyps, rather than neoplasia, should be suspected in young animals with a soft tissue mass in the nasopharyngeal region.

Thoracic radiographs are indicated to look for signs of lower airway disease and to rule out metastatic neoplasia in older cats. Echocardiography could be considered in cases of long-standing severe upper airway obstruction that could result in pulmonary hypertension.

Laboratory Findings

Specific laboratory abnormalities are uncommon.

DIFFERENTIAL DIAGNOSIS

Polyps should be differentiated from neoplastic masses, particularly in older cats. Other differentials include upper respiratory tract infections, nasal foreign bodies, otitis externa/media, and fungal diseases.

MEDICAL MANAGEMENT

Treatment of concurrent infection should be instituted after appropriate microbial cultures are performed. Surgical removal of the masses is indicated because medical management has been uniformly unsuccessful in eliminating these polyps. If traction-avulsion alone is used, concurrent treatment with an antiinflammatory (e.g., prednisolone, 1–2 mg/kg per day for 2 weeks followed by a tapered dose for an additional 2–4 weeks) may reduce the recurrence rate.

SURGICAL TREATMENT

Surgical procedures advocated for removal of polyps include traction-avulsion of the mass, traction-avulsion with direct endoscopic visualization, ventral bulla osteotomy, lateral ear canal resection, TECA combined with LBO, and myringotomy. Traction-avulsion is used as a first-line treatment to remove masses when there is no evidence of otitis media. Ventral bulla osteotomy should be performed if middle ear disease is evident from clinical signs or on radiography or CT. For cats with deep aural polyps, a lateral approach to the ear canal has been described.[9]

Preoperative Management

If severe respiratory obstruction is present, a temporary tracheostomy may be necessary; however, this is rarely required.

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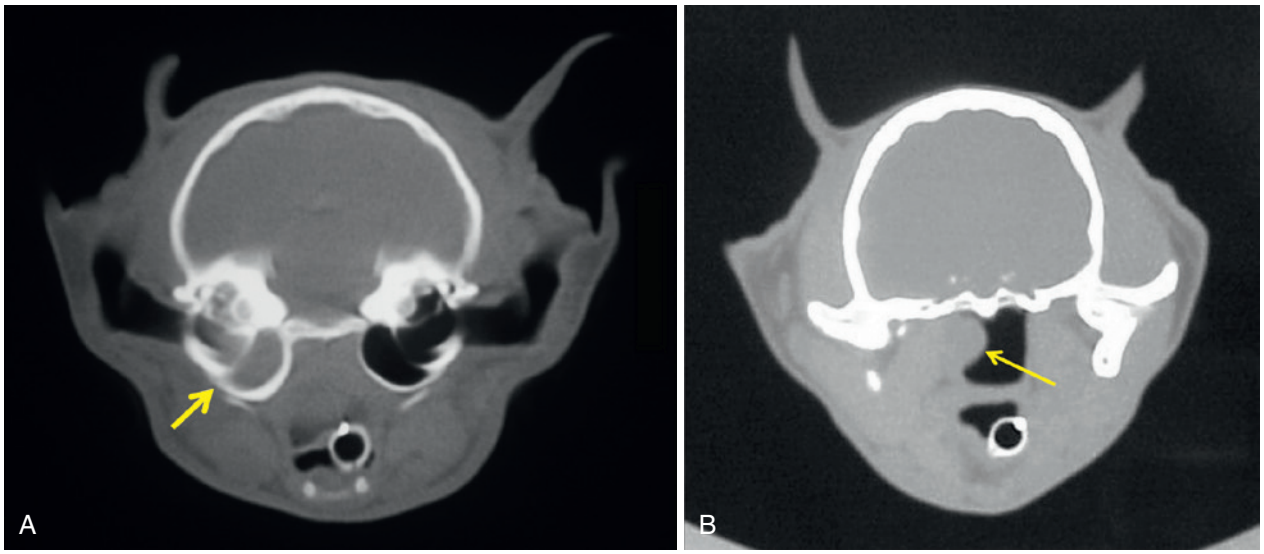


FIG. 17.32 Computed tomography axial image of the cat in Fig. 17.31 with soft tissue opacity within the tympanic bulla (*arrow*, A) and a soft tissue mass in the nasopharynx (*arrow*, B), consistent with an inflammatory polyp.

Antimicrobial therapy should await results of microbial cultures.

Anesthesia

Anesthetic management of animals undergoing ear surgery is provided on p. 302. If the patient is in respiratory distress, be prepared with monitors, airway devices, an anesthesia machine, and induction/emergency drugs before inducing anesthesia. Preoxygenating these patients for 3 to 5 minutes before induction can be helpful. When a patient resists preoxygenation with a mask, holding the anesthesia machine circuit with the oxygen at a high flow rate in front of the patient's nose and mouth will increase the inspired oxygen concentration enough to allow extra time to intubate the animal. Care must be used when intubating animals that have oropharyngeal masses; occasionally a smaller than expected endotracheal tube may be necessary. The cuff on the tube should be carefully checked to ensure its function and to prevent blood and other debris from entering the trachea during surgery.

Surgical Anatomy

Anatomy of the ear is detailed on p. 305.

Positioning

The animal is positioned in dorsal recumbency for ventral bulla osteotomy (see p. 310).

SURGICAL TECHNIQUES

Ventral bulla osteotomy is described on p. 310. In cats, the bullae should be entered through the larger ventromedial compartment. Most polyps are found in the dorsolateral compartment. Removal of the septum at its most lateral aspect may help prevent damage to the sympathetic trunk. Cultures should be taken of the bullae and the polyp. The polyp, epithelial lining, and associated exudate should be carefully removed with thumb forceps. The polyp should be submitted for histopathology.

Traction-avulsion of aural or nasopharyngeal polyps is the simplest technique and can be associated with limited recurrence rates if the entire polyp and stalk are removed, with or without administration of postprocedural corticosteroids.

For aural polyps, position the cat in lateral recumbency. Grasp the polyp using toothed grasping forceps such as Allis tissue forceps or alligator forceps. If necessary, use a small rigid endoscope with a biopsy channel to facilitate visualization.[10] *Alternatively, with a No. 11 scalpel blade, make a small stab incision into the vertical canal distal to the junction with the horizontal canal.*[9] *Place stay sutures on either side of the incision to improve visualization. Insert small curved hemostatic forceps into the canal and advance over the polyp. Grasp the polyp as close as possible to the external auditory meatus. Twist and pull with the forceps to avulse the polyp in its entirety. Close the canal using several simple interrupted sutures using 4-0 monofilament absorbable suture material. Close the rest of the incision routinely.*

For nasopharyngeal polyps, place the cat in dorsal or sternal recumbency. Retract the soft palate rostrally using a spay hook or stay sutures. It is rarely indicated to incise the soft palate on the midline to gain access to the nasopharynx (staphylotomy). *Grasp the polyp using toothed grasping forceps or a small towel clamp (e.g., 3.5 inch [9 cm] Backhaus towel clamp). Expect mild bleeding, but excessive hemorrhage is uncommon.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Ventral bulla osteotomy can be performed using a small Steinmann pin and a hand chuck or an air drill. The opening is typically enlarged with a rongeur (e.g., Kerrison, Lempert).

POSTOPERATIVE CARE AND ASSESSMENT

The animal should be evaluated for seroma formation after surgery. These seromas rarely require drainage. Surgical curettage of the tympanic cavity frequently causes transient Horner syndrome in cats, which usually resolves in 2 to 3 weeks (see

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discussion on surgical anatomy, p. 305). Horner syndrome is rare in dogs after this procedure, probably reflecting the difference in the anatomy of the tympanic cavity between the two species.

PROGNOSIS

The prognosis is excellent with complete removal of the polyp. Nasopharyngeal polyps may be less likely to recur than aural ones. Horner syndrome typically resolves within a few weeks. Rarely, temporary or permanent vestibular signs (e.g., nystagmus, head tilt) may occur. Transient facial nerve paralysis is uncommon but may occur after bulla osteotomy (see p. 311).

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18

Surgery of the Digestive System

Surgery of the Oral Cavity and Oropharynx

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Maxillectomy is removal of a portion of the maxilla, and **mandibulectomy** is removal of a portion of the mandible. **Tonsillectomy** is excision of one or both tonsils. **Glossectomy** is excision of a portion of the tongue. **Cheiloplasty** is performed to alter the shape of the lip, generally to reduce drooling. **Mucoceles** are subcutaneous collections of saliva or mucus (or both). **Ranulas** are collections of fluid from the mandibular or sublingual salivary glands that occur beneath the tongue on either side of the frenulum.

PREOPERATIVE CONCERNS

Surgical diseases of the oral cavity and oropharynx are common in dogs and cats. They include congenital and traumatic abnormalities, foreign bodies, neoplasia, salivary gland disease, and dental disease. Patients with oral cavity or oropharyngeal disease may have drooling, dysphagia, anorexia, bleeding from the mouth, and/or fetid breath. Some animals are asymptomatic until the lesions become large or are discovered on routine physical examination. Others are presented for treatment of a mass, oral hemorrhage, oral pain, difficulty eating, nasal regurgitation, chronic rhinitis, and/or dyspnea. Oropharyngeal disorders often present with a combination of upper airway and upper gastrointestinal (GI) tract signs, including cough, sneezing, dysphagia, and dyspnea if the laryngopharynx is involved. They may have a history of dental disease, weight loss, or trauma. The diagnosis is based on history, clinical signs, physical examination, cytologic studies, radiographs, ultrasonography, computed tomography (CT), magnetic resonance imaging (MRI), and/or biopsy.

Before major surgery is performed, a thorough physical examination, complete blood cell count (CBC), and serum biochemical profile should be performed; urinalysis and electrocardiogram (ECG) may be appropriate also. Animals undergoing maxillectomy or mandibulectomy and those predisposed to coagulopathies should have the coagulation system checked (i.e., platelet count and mucosal bleeding time) and blood cross matching done before surgery. Skull radiography, or preferably MRI or CT images, are indicated to determine the extent of the lesion. Thoracic radiographs or CT are indicated to evaluate for metastasis, cardiac size, and pulmonary disease. The teeth of animals with periodontal disease should be cleaned several days before major reconstructive surgery to improve tissue health and reduce oral bacterial numbers. Nutrition should be maintained by tube feeding if necessary (see Chapter 10). Animals with oronasal fistulae can be fed via feeding tubes to reduce the chances of rhinitis and inhalation pneumonia before surgery. Metabolic abnormalities should be corrected. Mature animals should be fasted for 12 to 18 hours before induction of anesthesia (pediatric patients are fasted for 4–8 hours). After induction and orotracheal intubation, the mouth should be flushed with dilute Betadine or chlorhexidine solution to reduce bacterial numbers.

ANESTHETIC CONSIDERATIONS

Preemptive analgesics and local nerve blocks (infraorbital, maxillary, mandibular, mental, or palatine nerves; bupivacaine 0.5%, 0.25–1 mL per site; see also Chapter 13) should be used to minimize pain associated with major oral reconstructive surgery. An orally placed endotracheal tube can sometimes hinder oral cavity and oropharyngeal surgery; in these cases, endotracheal intubation can be performed through a pharyngotomy (see p. 1044) or tracheotomy incision (see p. 842). It is important that the endotracheal tube and its cuff prevent blood and fluid from entering the lower airways. One or two gauze sponges can be placed in the oropharynx around the endotracheal tube to help absorb fluids; however, be sure to remove these sponges prior to waking the patient. Postoperative swelling of the oral mucous membranes may obstruct the glottis; this can be minimized by pretreatment with glucocorticoid (e.g., dexamethasone, 0.5–1 mg/kg given subcutaneously or intramuscularly before induction of anesthesia or intravenously at induction). Most animals with oral disease are healthy, and numerous anesthetic protocols can be used (Table 18.1). Because the oral cavity is highly vascular and hemostasis can sometimes be difficult, blood, hypertonic saline, and/or colloids should be available in the event of severe hemorrhage (see p. 32). Preoperative placement of two large cephalic catheters provides better venous access if volume replacement becomes necessary. Evaluation of blood pressure during surgery is warranted because of the risk of bleeding and subsequent hypotension.

ANTIBIOTICS

The oral cavity and oropharynx are contaminated (aerobic, facultative, and anaerobic bacteria); but saliva is antimicrobial

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ABSTRACT

Complex surgery of the oral cavity is required to repair oronasal communications, including those that are congenital (cleft palate) and those acquired due to trauma or dental disease. Mass lesions of the oral cavity may be benign or malignant in dogs; most are malignant in cats. Salivary mucoceles are a common condition in dogs and are typically corrected with surgery. Surgery of the gastrointestinal tract requires adherence to a number of general principles to avoid complications. Surgery of the esophagus (e.g., for foreign bodies) is difficult because the esophagus heals poorly due to the lack of a serosa. The most common indication for surgery of the stomach and small intestine is ingested foreign material. Large intestinal surgery presents a challenge; the most common indications are for neoplasia and megacolon (in cats). Surgery of perineal and rectal masses, perineal hernias, and anal sac disease require attention to detail and carry the risk of incontinence.

KEYWORDS

mandible
maxilla
salivary
esophagus
gastric
stomach
gastrotomy
gastropexy
intestine
enterotomy
resection anastomosis
obstruction
foreign body
mucocele
intussusception
peritonitis
neoplasia
hernia
gastric dilatation volvulus

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TABLE 18.1 Anesthetic Considerations in the Healthy Patient Undergoing Oral or Rectal Surgery

Preoperative Considerations

Associated conditions	• Minimal; usually healthy if young patient • May have other comorbidities if older patient
Bloodwork	• HCT • TP • In patients older than 5–7 years, consider electrolytes, liver enzymes, BUN, and Cr • Calcium if rectal mass
Physical exam	• Depends on diagnosis

Premedications	• If patient is anxious give:
	  • Diazepam (0.2 mg/kg IV), *or*
	  • Midazolam (0.2 mg/kg IV, IM), *plus*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM in dogs; 0.05–0.1 mg/kg IV, IM in cats), *or*
	  • Oxymorphone (0.05–0.2 mg/kg IV PRN), *or*
	  • Morphine[b] (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM)

Intraoperative Considerations

Induction	• If premedicated, give:
	  • Propofol (2–4 mg/kg IV), *or*
	  • Alfaxalone (2–3 mg/kg IV)
	• If no premeds given, give:
	  • Propofol (4–8 mg/kg) IV), *or*
	  • Alfaxalone (2–5 mg/kg IV), *or*
	• Ketamine (5 mg/kg IV with diazepam or midazolam as above)

Maintenance	• Isoflurane or sevoflurane, *plus*
	  • Fentanyl (2–10 µg/kg IV PRN in dogs; 1–4 µg/kg IV PRN in cats) for short-term pain relief, *plus*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Morphine[b] (0.1–1 mg/kg IV PRN in dogs; 0.05–0.2 mg/kg IV PRN in cats), *or*
	  • Oxymorphone (0.05–0.2 mg/kg IV PRN), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV PRN), *plus*
	  • Dexmedetomidine[d] (low dose) (0.5–1 µg/kg IV) *or*
	  • Medetomidine[d] (low dose) (1–2 µg/kg IV), *plus*
	  • Ketamine (low dose) (0.5–1 mg/kg IV), *or*
	  • Ketamine CRI (0.5 mg/kg IV loading dose then 10 µg/kg/min IV)

Fluid needs	• 5–10 mL/kg/h plus 3 × EBL • Two large IV catheters if potential for large EBL • Have blood or colloids available
Monitoring	• Blood pressure • HR • Respiratory rate • SpO_2 • Temperature

Blocks	• If oral surgery, 0.25–1.0 mL local anesthetic for infraorbital, maxillary, mandibular, or mental blocks[e]
	• If epidural for rectal surgery, then:
	  • Morphine (0.1 mg/kg preservative free), *or*
	  • Buprenorphine (0.003–0.005 mg/kg diluted in saline)

Postoperative Considerations

Analgesia	• Fentanyl CRI (1–10 µg/kg IV loading dose, then 2–20 µg/kg/h IV), *or* • Hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs), *or* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q3–4h in dogs 0.05–0.1 mg/kg IV, IM q3–4h in cats), *or* • Morphine[b] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.05–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h in cats), *or* • Oxymorphone (0.05–0.2 mg/kg IV PRN), *or* • Buprenorphine[c] (5–20 µg/kg IV, IM, SC q4–8h), *plus* • ± Ketamine CRI (2 µg/kg/min IV; if no previous loading dose, give 0.5 mg/kg IV prior to CRI), *plus* In dogs: • Carprofen (2.2 mg/kg q12h PO), *or* • Deracoxib (3–4 mg/kg q24h for <7 days PO), *or* • Meloxicam (0.1–0.2 mg/kg once SC, PO, then 0.1 mg/kg PO q24h) In cats: • Meloxicam[f] (0.15 mg/kg SC once; approved for doses up to 0.3 mg/kg SC; see also Table 34.3), *or* • Buprenorphine[c] (0.01–0.02 mg/kg OTM q6–12h)

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TABLE 18.1 Anesthetic Considerations in the Healthy Patient Undergoing Oral or Rectal Surgery—cont'd

Monitoring	• SpO_2 • Blood pressure • HR • Respiratory rate • Temperature
Bloodwork	• HCT and TP if large blood loss
Estimated pain score	• Depends on procedure

[a]Monitor for hyperthermia in cats.
[b]Give slowly to prevent histamine release.
[c]Buprenorphine is a better analgesic than morphine in cats.
[d]Use only in young, healthy animals.
[e]Do not exceed maximum local anesthetic dose for very small dogs and cats.
[f]Black box warning added by the Food and Drug Administration in October 2010 identified cases of renal failure and death in cats with repeated uses of meloxicam. Meloxicam is approved for single use only in cats in the United States.
BUN, Blood urea nitrogen; *Cr,* creatinine; *CRI,* constant-rate infusion; *EBL,* estimated blood loss; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *OTM,* oral transmucosal; *PO,* orally; *PRN,* as needed; *SC,* subcutaneous; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein.

BOX 18.1 Prophylactic and Therapeutic Antibiotics for Oral Surgery

Cefazolin (Ancef, Kefzol)
22 mg/kg IV q8–12h

Amoxicillin (Amoxi-Tabs, Amoxi-Drops, Amoxi-Inject)
22 mg/kg PO, IM or SC q8–12h

Clindamycin (Antirobe)
11–33 mg/kg PO q8–12h or 11 mg/kg IV q8–12h; when giving IV dilute and administer slowly

Metronidazole (Flagyl)
10–15 mg/kg PO q12h

IM, Intramuscular; *IV,* intravenous; *PO,* orally; *SC,* subcutaneous.

and blood supply to this region is excellent. For these reasons, infections after oral surgery are rare. One dose of a prophylactic antibiotic effective against gram-positive aerobes and anaerobes (e.g., ampicillin, clindamycin) may be given at anesthetic induction (Box 18.1). Therapeutic antibiotics (e.g., cefazolin plus metronidazole, amoxicillin plus clavulanic acid, or clindamycin; see Box 18.1) are indicated in debilitated or immunosuppressed patients and those with severe periodontal disease.

SURGICAL ANATOMY

The oral cavity is divided into the vestibule and the oral cavity proper. The vestibule is the cavity lying outside the teeth and gums but inside the lips and cheeks. The ducts of the parotid and zygomatic salivary glands open in the dorsocaudal part of the vestibule. The oral cavity proper is the area bounded by the hard palate and a small part of the soft palate dorsally, by the dental arches laterally and rostrally, and by the tongue and adjacent mucosa ventrally. The tongue is attached to the floor of the oral cavity by the lingual frenulum. The oropharynx extends from the level of the palatoglossal arches to the caudal border of the soft palate and the base of the epiglottis. The oropharynx is bounded dorsally by the soft palate and ventrally by the root of the tongue. The palatine tonsils are found in the lateral wall of the oropharynx.

The blood supply to this region originates from branches of the common carotid arteries. The paired major and minor palatine arteries are important (Fig. 18.1). Two or three vessels emerge from the major palatine foramen at the caudal edge of the fourth upper premolar and course rostrally, midway between the midline and the dental arcade. The right and left major palatine arteries anastomose caudal to the incisors. The minor palatine arteries enter the palate caudal to the last molar and lateral to the major palatine artery, then course caudomedially to ramify in the caudal hard palate and soft palate. The major blood supply to the mandible is via the mandibular alveolar artery, which enters the mandibular canal on the medial surface of the mandible (see Fig. 18.1). The entry point is where an oblique line connecting the last molar tooth and the angular (muscular) process (which is hidden beneath the pterygoid muscle) meet. The mandibular alveolar artery ends at the middle mental foramen, where it branches to form the caudal, middle, and rostral mental arteries and exits via the mental foramina. The mandibular canal also transmits the mandibular vein and the mandibular alveolar nerve.

NOTE Infection is rare and healing is rapid after surgery of the oral cavity because of its excellent blood supply.

SURGICAL TECHNIQUES

Atraumatic surgical technique is important for reducing tissue damage and swelling and encouraging rapid healing (Box 18.2). Hemorrhage is expected and should be controlled with pressure and vessel ligation. Electrosurgery should be used sparingly because excessive use delays healing and may cause dehiscence. Electrocoagulation should be applied only to discrete, isolated areas. The possibility exists that flammable gases (i.e., oxygen) leaking around the endotracheal tube could be ignited when electrosurgery or laser surgery is used.

Tension-free closure is imperative to prevent wound dehiscence and a subsequent oronasal fistula. Flaps created during reconstructive procedures should be approximately 2 to 4 mm larger than the defect, and major vessels entering these flaps must be preserved. Tension on these flaps should be minimized by adequate mobilization. Flaps should be manipulated with skin hooks or stay sutures to minimize trauma. Suture lines should be placed over connective tissue or bone, rather than the defect, to help support mucosal flaps. Cleanly incised edges of tissue should be apposed with a two-layer closure in an appositional suture pattern (simple interrupted, simple continuous, cruciate, or vertical

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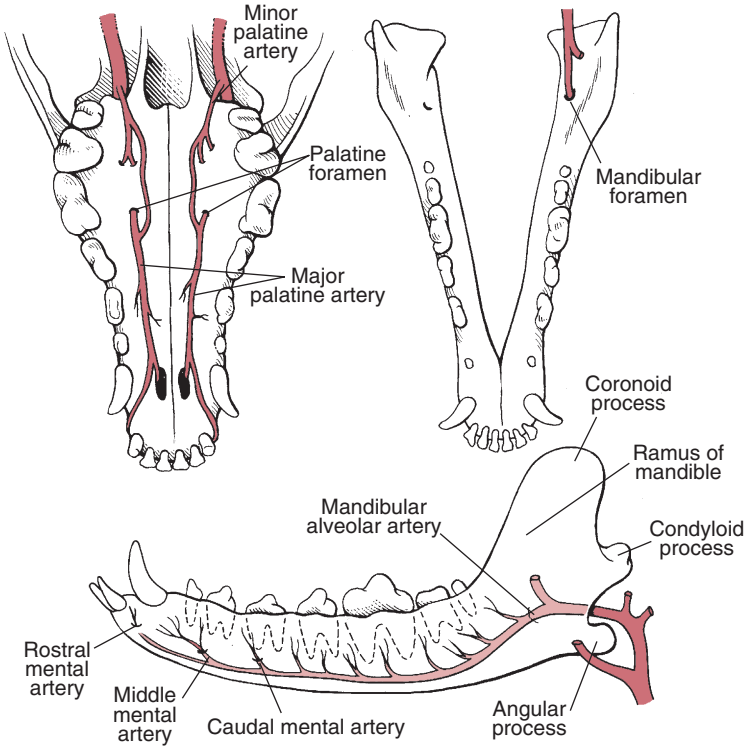


FIG. 18.1 The major blood supply to the maxilla is via the major and minor palatine arteries and to the mandible via the mandibular alveolar artery.

BOX 18.2 Principles of Oral Surgery

- Use atraumatic technique.
- Control hemorrhage using pressure and ligation.
- Avoid tension; make flaps 2–4 mm larger than the defect.
- Support flaps; do not suture over defects.
- Use appositional sutures (e.g., simple interrupted, simple continuous, cruciate, vertical mattress patterns).

mattress patterns). Monofilament suture material (i.e., 4-0 or 3-0 poliglecaprone 25, polyglyconate, polydioxanone, or polypropylene) minimizes wicking and tissue reaction. A temporary acrylic obturator can be cemented or wired to the teeth to protect healing tissues in the occasional case in which tongue or pharyngeal muscle activity is apt to break down the suture line.

Biopsy Techniques

Impression smears or aspirates should be obtained from oral lesions before incisional or excisional biopsy. Cytologic studies may allow a tentative diagnosis that will help in planning further diagnostics; however, it must be realized that most oral lesions have inflamed surfaces secondary to oral bacterial flora. Therefore one should routinely obtain deep samples, especially if inflamed, necrotic tissue is present. An incisional biopsy using a core needle or wedge biopsy technique should be performed if the definitive diagnosis will change the course of therapy. A Tru-Cut or Vim-Silverman needle can be used to obtain small cores from several areas of the mass, whereas wedge biopsies should be performed from nonnecrotic areas of the mass when larger pieces of tissue are needed. A loop or needle electrode of an electrosurgical unit is useful in obtaining oral biopsies. However, the specimen will be nondiagnostic if too much current is applied, especially to a small sample. Tissue coagulation can be prevented by keeping the power setting on the electrosurgical unit as low as possible. Diseased tissue is often friable and difficult to appose with sutures after a biopsy; however, pressure over the cut area is usually sufficient to control hemorrhage. Silver nitrate cautery can be used if necessary. Wide excision (e.g., partial maxillectomy, mandibulectomy, tonsillectomy, glossectomy, or lip resection) should be performed and the area reconstructed if the definitive histologic diagnosis will not alter the course of therapy. All specimens should be submitted for histologic evaluation.

NOTE Avoid areas of superficial necrosis when obtaining biopsies; sample deeper, viable tissues instead.

Temporary Carotid Artery Ligation

Some surgeons perform temporary carotid artery occlusion before maxillectomy to minimize blood loss.

To do so, place the animal in dorsal recumbency and prepare the ventral cervical area for surgery. Expose the trachea through a 5- to 8-cm ventral cervical midline incision. Palpate the carotid pulse and exteriorize the carotid sheath. Separate the common carotid artery from the vagosympathetic trunk and internal jugular vein. Temporarily occlude the carotid artery with a vascular clamp or tie. Repeat the procedure on the opposite carotid artery. Temporarily appose skin with a continuous suture pattern or staples during the maxillectomy procedure. After the maxillectomy, reopen the cervical wound and remove the vascular clamps or ties. Lavage the area thoroughly and appose the sternohyoid muscles, subcutaneous tissue, and skin in separate layers.

NOTE Temporary carotid artery ligation may not be safe in cats or in severely anemic dogs. Perform with caution!

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Partial Maxillectomy

Maxillectomy is most often performed to resect an oral neoplasm (Fig. 18.2). Varying amounts of the maxilla and hard palate may be excised, depending on the gross and radiographic, CT, or MRI extent of the lesion. Depending on the area being resected, partial maxillectomies may be classified as hemimaxillectomies (rostral, central, or caudal) or premaxillectomies (bilateral rostral) (Figs. 18.3 and 18.4). Hemimaxillectomy, without a definition of site, usually refers to removal of one entire maxilla. Partial maxillectomies can be combined with nasal planectomy and dorsolateral approaches through the skin. Partial maxillectomy is limited by the surgeon's ability to reconstruct the oronasal defect; lesions that cross the midline of the palate are difficult to reconstruct.



FIG. 18.2 An odontogenic tumor involving the maxillary dental arcade, palate, and maxilla.

Clip and aseptically prepare the maxillary and nasal skin. Flush the mouth with antiseptic solution. Place the patient in dorsal recumbency for lesions of the premaxilla and open the mouth to its maximum extent by placing a mouth speculum or by taping the mouth open. Place the patient in lateral or dorsal recumbency for lesions caudal to the premaxilla. Determine the extent of resection based on the size of the soft tissue lesion and the radiographic, CT, or MRI degree of bony involvement. Generally, excise the mass and a minimum of 1 to 2 cm of normal soft tissue and bone on all borders. Remove the mass en bloc by first making mucosal incisions (buccal, gingival, and hard palate) around the tissue to be resected. Avoid rectangular excision because the corners are susceptible to dehiscence. Using a periosteal elevator, undermine and reflect the gingival and palatal mucosa.

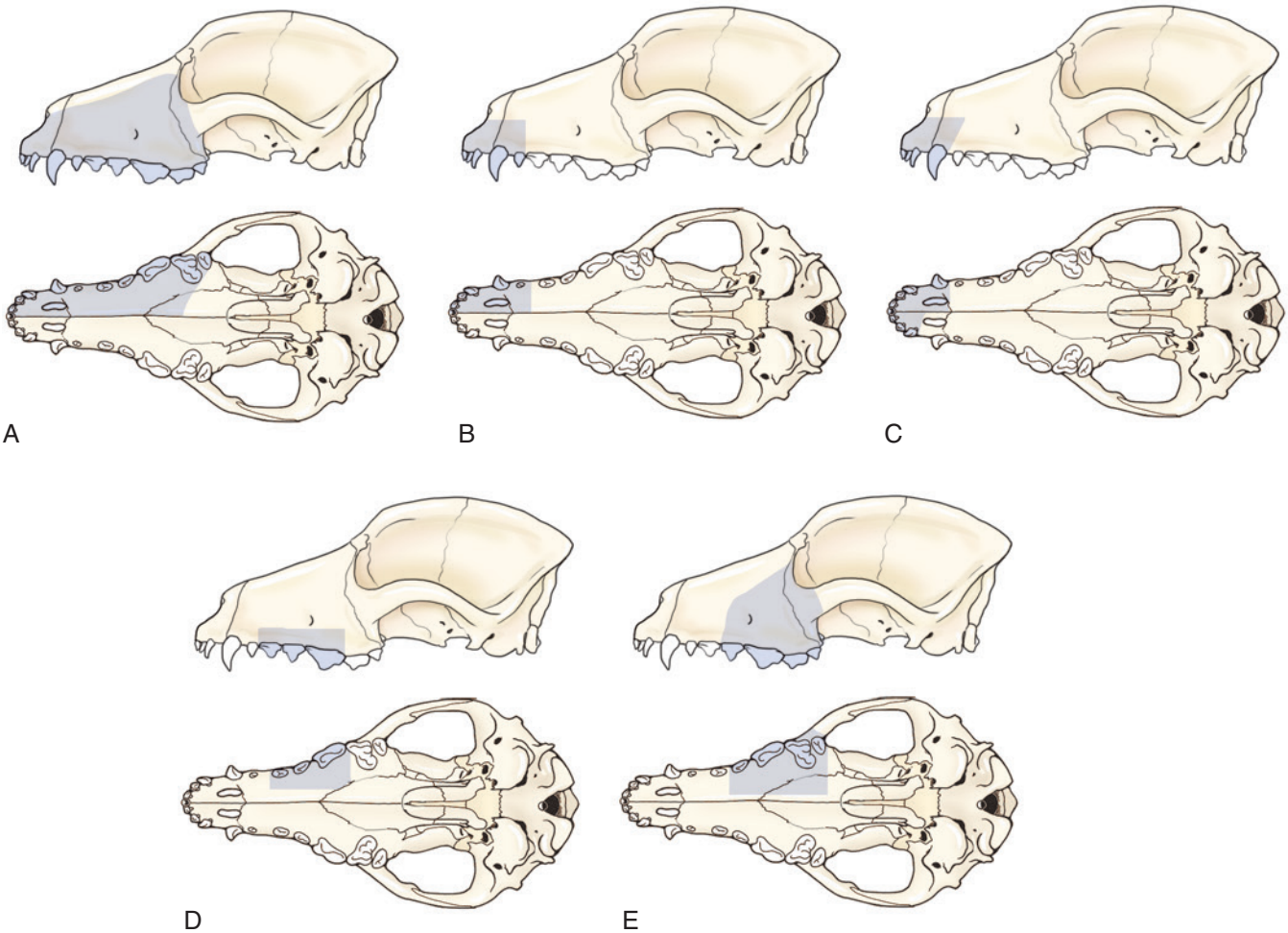


FIG. 18.3 Areas resected with partial maxillectomy techniques. (A) Hemimaxillectomy. (B) Rostral hemimaxillectomy. (C) Premaxillectomy (bilateral rostral hemimaxillectomy). (D) Central hemimaxillectomy. (E) Caudal hemimaxillectomy.

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FIG. 18.4 Appearance of the oral cavity of a dog after a rostral hemimaxillectomy. A buccal flap has been advanced across the defect and secured with apposition sutures.



FIG. 18.6 A buccal mucosal flap was advanced over the defect evident in Fig. 18.5 and secured with approximating sutures.

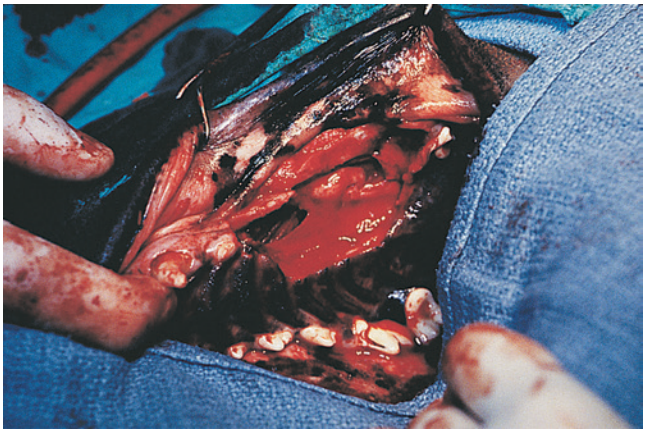


FIG. 18.5 A central hemimaxillectomy was performed to remove the lesion shown in Fig. 18.2 and an additional 1 to 2 cm of normal tissue. Note that the nasal cavity is exposed.



FIG. 18.7 Postoperative appearance after rostral mandibulectomy. Note that the tongue is barely apparent despite the short mandible.

Use an oscillating saw or an osteotome and mallet to cut the maxilla, incisive bone, and/or palate. Resect all premolar and molar teeth for lesions extending to the third premolar because of the outward turn of the dental arch. When performing a caudal maxillectomy, remove a portion of the zygomatic arch and orbit if necessary to obtain clean borders. Elevate the tissue block and sever any remaining soft tissue attachments to complete the resection and expose the nasal cavity (Fig. 18.5). Remove involved nasal turbinates with rongeurs and hemostats if disease extends into the nasal cavity. Control hemorrhage by ligating identifiable vessels and applying pressure to other areas. Isolate and ligate the major palatine and infraorbital artery and vein if they are included in the resection site. Use bone wax or electrofulguration to help control bone hemorrhage. Lavage and inspect the defect to ensure that all grossly diseased tissue has been excised. Close the defect by elevating a buccal mucosal flap from the adjacent cheek or lip (Fig. 18.6). Elevate enough buccal mucosa and submucosa to allow a tension-free approximation with the gingival and palatal mucosa. Place the first layer of simple interrupted sutures in the submucosa, with the knot directed toward the nasal cavity. Place a second layer of interrupted approximating sutures (i.e., simple, cruciate, vertical mattress) to accurately appose buccal mucosa to the palatal and gingival mucosa. A double-flap technique may be used to close premaxillectomy defects to provide mucosa on both the nasal and oral surfaces. However, an epithelial surface on the nasal aspect of the flap is not necessary because the connective tissue surface of the flap is covered with respiratory epithelium within 1 to 2 weeks. *If carotid artery occlusion was performed, release the occlusion after the defect has been closed.*

Partial Mandibulectomy

Mandibulectomy is most often performed to resect an oral neoplasm. Occasionally mandibular fractures are also treated by partial mandibulectomy. Varying amounts of mandible may be excised, depending on the extent of the tumor or lesion (Fig. 18.7). Depending on the extent of resection, hemimandibulectomies may be classified as rostral, rostral-bilateral, central, caudal, or total (Fig. 18.8). These techniques may be combined when more extensive resection is necessary. After mandibulectomy, cheiloplasty (commissuroplasty) may be performed to minimize excessive drooling and lateral protrusion of the tongue (Fig. 18.9). It is accomplished by removing the mucocutaneous junction of the upper and lower lip to the level of the second premolar or canine tooth. The commissure is advanced rostrally during closure. The upper and lower lip

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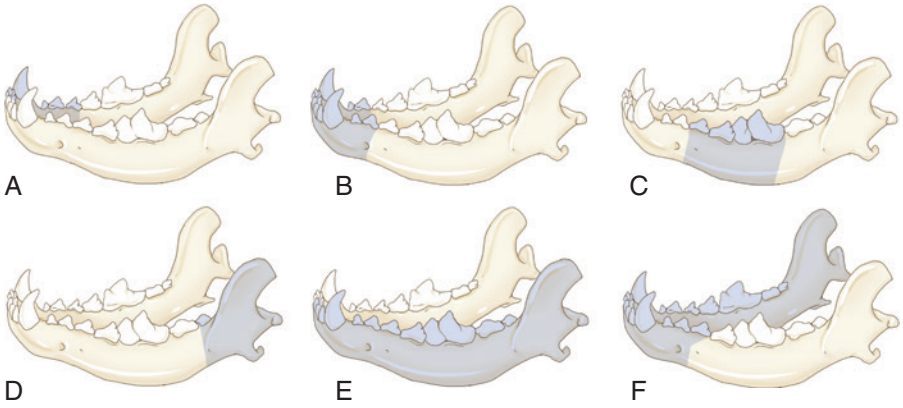


FIG. 18.8 Areas resected with partial mandibulectomy techniques. (A) Rostral hemimandibulectomy (unilateral rostral hemimandibulectomy). (B) Rostral mandibulectomy (bilateral rostral hemimandibulectomy). (C) Central hemimandibulectomy. (D) Caudal hemimandibulectomy. (E) Total hemimandibulectomy. (F) Three-quarter mandibulectomy.



FIG. 18.9 Postoperative appearance after hemimandibulectomy in which cheiloplasty was not performed. Although the tongue is movable, it protrudes from the oral cavity.

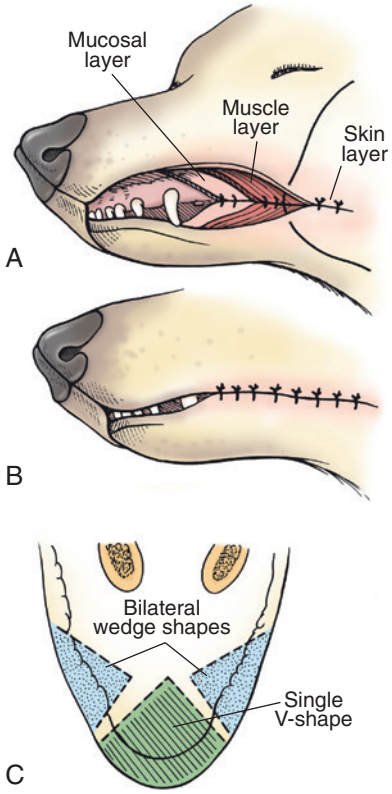


FIG. 18.10 Cheiloplasty. (A) Excise the lip margins to the level of the second premolar. (A–B) Appose the incised lip margins in three layers (oral mucosa, muscle and connective tissue, and skin). (C) To improve cosmesis after rostral mandibulectomy, excise redundant skin from one or more of the indicated sites.

margins are apposed in three layers (oral mucosa, muscle and connective tissue, and skin) (Fig. 18.10). Opening the mouth fully during the first 2 weeks may cause dehiscence. Tension-relieving button sutures or a loose tape muzzle may be used to help prevent this. During rostral mandibulectomies, redundant skin and mucosa may be eliminated by excising and apposing V-shaped wedges. The base of the V is along the mucocutaneous junction (see Fig. 18.10).

Position the patient in lateral, sternal, or dorsal recumbency with the neck extended (Figs. 18.11A and 18.12A). Clip and aseptically prepare the skin of the lateral face and ventral mandible. Flush the mouth with antiseptic solution. Determine the amount to be resected based on the size of the soft tissue lesion and the radiographic, CT, or MRI evidence of bony involvement. Generally, excise the mass and a minimum of 1 to 2 cm of normal soft tissue and bone on all borders. Retract the commissure and lip to give maximum exposure. If necessary, improve visualization by incising the commissure to the level of the mandibular angle (see Fig. 18.12B). Begin en bloc resection by first incising mucosa (buccal, gingival, and sublingual) around the diseased area (see Figs. 18.11B and 18.12B). Using a periosteal elevator, undermine and reflect the gingival mucosa to expose the lateral and ventral aspects of the mandible. Transect or elevate and retract muscles (mentalis, orbicularis oris, buccinator, mylohyoideus, geniohyoideus, genioglossus, masseter, digastricus, temporalis, and pterygoideus) attached to the portion of the mandible being resected (see Figs. 18.11B and 18.12C–D). Use an oscillating saw or an osteotome and mallet to

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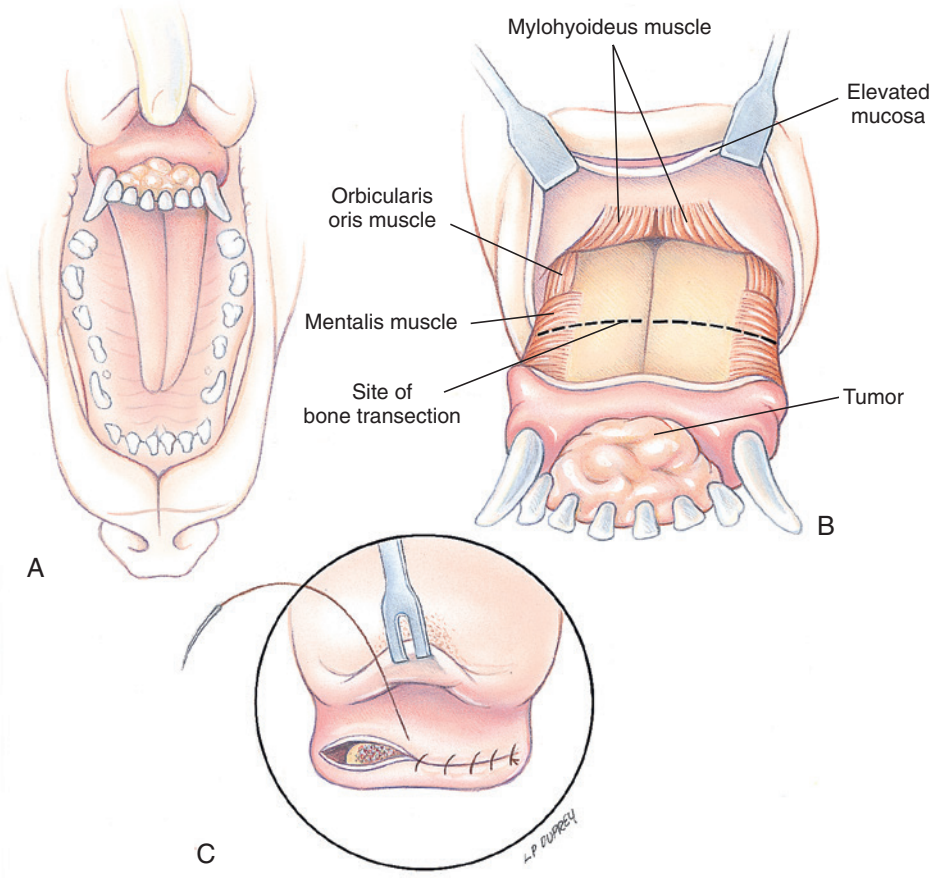


FIG. 18.11 Rostral mandibulectomy. (A) Position the patient in dorsal recumbency. (B) Incise the mucosa 1–2 cm from the lesion, exposing the underlying muscles. Elevate the muscles and transect the bone *(dashed line)*. (C) Appose the labial and sublingual mucosa after en bloc excision.

transect the mandible and separate the symphysis. As an alternative, use a Gigli wire to transect the mandible. Complete a total hemimandibulectomy by incising the joint capsule and disarticulating the temporomandibular joint (see Fig. 18.12C). Locate the temporomandibular joint by rotating the mandible and palpating the articulation. Ligate or cauterize the mandibular artery (see Fig. 18.12D). Sever any remaining soft tissue attachments to complete the resection. Avoid traumatizing the lingual frenulum or sublingual and mandibular salivary ducts. Contour the ostectomy sites with bone rongeurs, removing sharp bone and tapering the edges to facilitate closure. Close the defect by elevating a mucosal flap from the adjacent lip or cheek (see Figs. 18.11C and 18.12E). Elevate enough mucosa and submucosa to allow a tension-free approximation with the gingival and sublingual mucosa. Place the first layer of simple interrupted sutures in the submucosa with the knots buried. Place a second layer of interrupted approximating sutures (simple, cruciate, or vertical mattress) to accurately appose the labial, sublingual, and gingival mucosa. As an alternative, use a single-layer, simple continuous or interrupted suture pattern.

NOTE It is not necessary to stabilize the remaining mandible after mandibulectomy. However, cortical bone grafts have been used to improve the animal's appearance.

Mandibular Rim Excision

Mandibular rim excision may be considered for small, minimally invasive tumors of the mandible in large dogs in which the tooth roots do not approach the mandibular canal. Advanced imaging is strongly recommended to help ensure complete excision of mass lesions using this technique. *Incise the mucoperiosteum in a curvilinear pattern, 1 cm around the mass and associated teeth. Elevate the attached gingiva and excise the mandibular segment with a handpiece that irrigates but does not insufflate. Be certain that no tooth roots remain. Smooth the osteotomy site and reattach the mucosa over the bony defect with monofilament absorbable suture.*

Tonsillectomy

When neoplasia of the palatine tonsils is suspected, they should be biopsied or removed. Squamous cell carcinoma (SCC) and lymphosarcoma are the most common tumors of the tonsils, and neither are generally cured. Enlarged tonsils are occasionally removed if they contribute to airway obstruction or dysphagia and in cases of unresponsive chronic tonsillitis. However, tonsillitis is usually secondary to other diseases (e.g., esophageal dysfunction), and removing the tonsils rarely effects a cure.

Administer dexamethasone (0.1–0.2 mg/kg IV [intravenous]) at the time of induction to minimize postoperative swelling and

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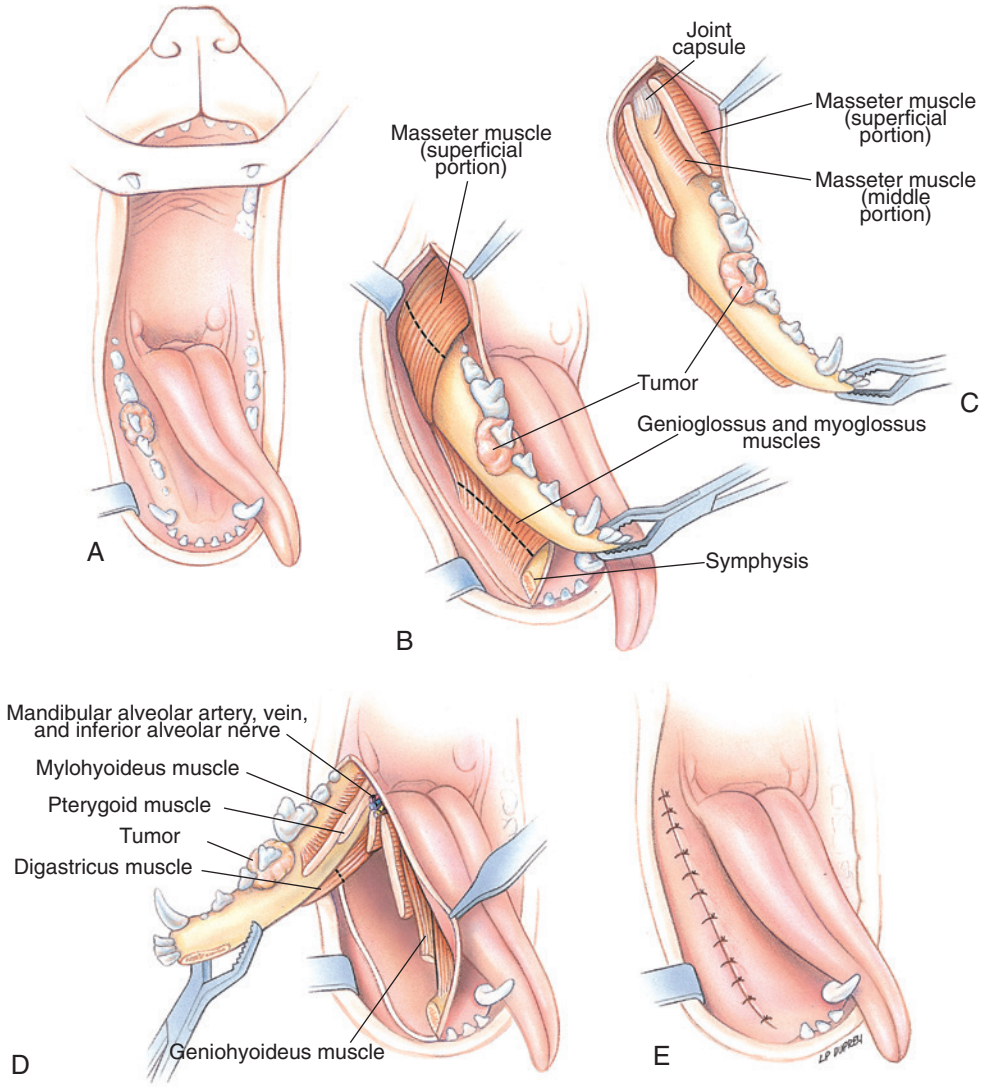


FIG. 18.12 Total hemimandibulectomy. (A) Position the patient in ventral recumbency. (B) Incise the mucosa 1 to 2 cm from the lesion. Incise the commissure to allow better exposure of the caudal mandible. Separate the mandibular symphysis and identify and transect *(dashed line)* the muscles. (C) Dissect and transect the lateral mandibular muscles and expose the temporomandibular joint. (D) Dissect and transect the medial muscles of the mandible and identify the mandibular artery entering the mandibular foramen. Ligate the mandibular vessels, disarticulate, and remove the mandible. (E) Appose the buccal and sublingual mucosa with approximating sutures.

edema. Position the animal in ventral recumbency with the maxilla suspended from an IV stand or similar device. Open the mouth maximally and secure it open with tape or gauze. Locate the tonsil in the tonsillar fossa or crypt on the dorsolateral wall of the oropharynx just caudal to the palatoglossal arch (Fig. 18.13). Retract the edge of the tonsillar crypt caudodorsally to expose the tonsil. Grasp the tonsil at its base with an Allis tissue forceps or hemostat and retract it from the crypt. Transect the hilar mucosa at the base of the tonsil with Metzenbaum scissors or a tonsillectomy snare. Ligate the tonsillar artery as it enters the caudal aspect of the tonsil. Some surgeons excise the tonsil using electrosurgery or laser surgery. *Appose the edges of the tonsillar crypt with a simple continuous suture pattern of 3-0 or 4-0 monofilament absorbable suture to minimize hemorrhage.*

Glossectomy

The primary reason for partial tongue amputation is neoplasia, which usually occurs on the margin or base of the tongue. The most common lingual tumor is SCC (Fig. 18.14), but others include malignant melanoma, granular cell myeloblastoma, and mast cell tumor. Most lingual lacerations are amenable to repair with a one- or two-layer closure rather than amputation. Other reasons for tongue surgery include abscess drainage, glossitis, severe trauma, and congenital ankyloglossia (limited tongue movement). Tongue lesions are associated with signs of drooling, malodor, dysphagia, and sometimes dyspnea.

Glossectomies are classified as partial (involving only the free tongue), subtotal (entire free tongue and part of the genioglossus and geniohyoideus muscles), near total (greater than 75% of

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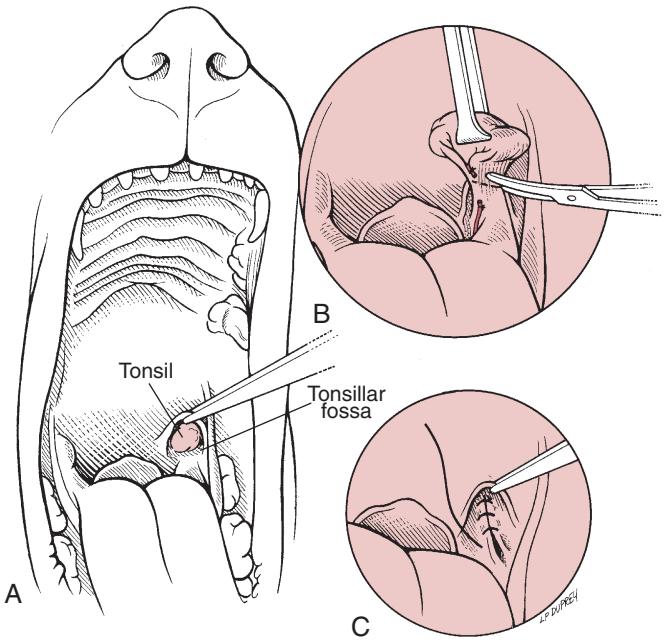


FIG. 18.13 Tonsillectomy. (A) The palatine tonsils are located in the dorsolateral pharynx. (B) During tonsillectomy evert the tonsil from the crypt, ligate the tonsillar vessels, and begin transection along the base. (C) Close the crypt with a simple continuous pattern to help control hemorrhage.



FIG. 18.14 Appearance of a squamous cell carcinoma of the tongue in a dog.

tongue), and total (100% of tongue). Glossectomy is considered major if more than the free portion of the tongue is removed. Amputation of 40% to 60% of the rostral tongue is usually well tolerated. Amputation at the base of the tongue or total glossectomy makes eating and drinking difficult; however, the animal can learn to suck in food and water, or chunks of food can be tossed to the base of the tongue. Ptyalism following major glossectomy occasionally occurs; improvement may be seen with bilateral sialoadenectomy. When total glossectomy is performed, a clamp is not used. *Wedge the incision so that slightly more tongue muscle than dorsal or ventral mucosa is excised. Control hemorrhage by ligation, pressure, or electrosurgery. Use through-and-through horizontal mattress sutures as needed to control hemorrhage.*

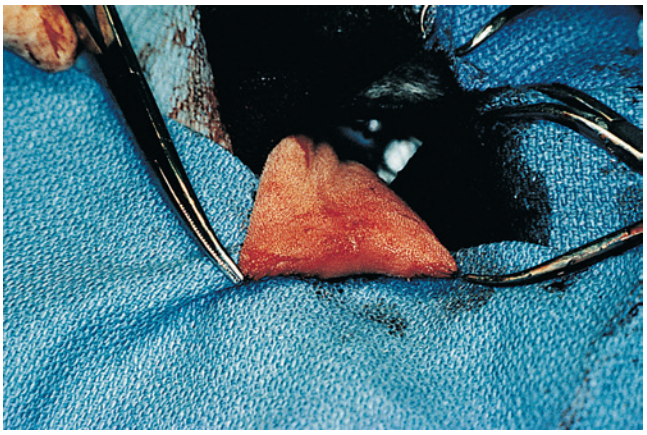


FIG. 18.15 Postoperative appearance of the tongue in Fig. 18.14 after partial glossectomy. The glossectomy was completed by apposing epithelial edges with a simple continuous suture pattern.

Appose the epithelial edges with a simple continuous suture pattern using 3-0 or 4-0 monofilament absorbable suture (Fig. 18.15). Place a feeding tube when major glossectomies are performed (see pp. 94–102).

NOTE Dogs seem to adapt better than cats to tongue amputation. A feeding tube may be required until the animal learns to eat after the procedure.

Pharyngotomy

Pharyngotomy may be performed to allow endotracheal intubation if the tube is in the surgeon's way when placed via the oral cavity. For a description of the technique, please see p. 1044.

Salivary Gland Excision

The salivary glands are most often removed to treat salivary mucoceles or neoplasia, and the mandibular and sublingual salivary glands are most often removed to treat cervical, sublingual, and pharyngeal salivary mucoceles. Neoplasms (usually adenocarcinomas or carcinomas) occur most frequently in the parotid (~50% dogs, ~20% cats) and the mandibular (~30% dogs, ~60% cats) salivary glands. Salivary gland excision is described on pp. 361 to 363.

HEALING OF THE ORAL CAVITY AND OROPHARYNX

The oral cavity and oropharyngeal mucosa heal more rapidly than skin because phagocytic activity (primarily monocytes rather than polymorphonuclear leukocytes) and epithelialization are more extensive and occur earlier in mucosa. An excellent mucosal blood supply, warmer temperatures, higher metabolic activity, and a higher mitotic rate contribute to rapid healing of mucosa. Apposed wounds reepithelialize within a few days, and defects heal by second intention.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Special instruments that may facilitate surgical procedures of the oral cavity include periosteal elevators, an oscillating saw or osteotome and mallet, Gigli wire, rongeurs or bone cutters,

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vascular bulldog clamps, vascular ties, tissue hooks, Metzenbaum scissors, noncrushing clamps (Doyen intestinal forceps), and Penrose drains. Piezoelectric bone surgery has been reported for maxillectomy and mandibulectomy, as well as dental surgery.[1] Proposed advantages include decreased soft tissue trauma and blood loss, improved precision of osteotomies, and decreased thermal bone damage, edema, and inflammation. Disadvantages include the cost of the equipment, learning curve, and slower osteotomy speed. Although many suture materials may be used in the oral cavity and oropharynx, 3-0 or 4-0 polydioxanone, polyglyconate, or poliglecaprone 25 (monofilament absorbable) and 3-0 or 4-0 polypropylene or nylon (monofilament nonabsorbable) are often suggested; however, many surgeons utilize polyglactin 910 (multifilament absorbable) because of its softness and presumed decreased postoperative irritation.

POSTOPERATIVE CARE AND ASSESSMENT

After oral surgery, gauze sponges should be removed from the caudal pharynx and the nasopharynx should be suctioned. Extubation should be delayed until a well-developed swallowing reflex is present. Patients should be recovered in a slightly head-down position, and the tube should be removed with the cuff slightly inflated to help ensure blood clots are expelled through the mouth rather than being aspirated or swallowed. These patients should be monitored for airway obstruction or pain, and analgesics should be provided as needed (for dosages of opioid analgesics, see Tables 12.1 and 13.2; for dosages of nonsteroidal antiinflammatory drugs [NSAIDs], see Table 13.2). Elizabethan collars or similar restraining devices should be used on some animals to prevent disruption of the surgical site. Occasionally an acrylic oral splint is used to protect the surgical site.

NOTE Be sure to suction the oral cavity and oropharynx before extubation, extubate with the cuff inflated, and monitor for airway obstruction during recovery.

Oral intake should not be allowed for the first 8 to 12 hours after surgery (except in pediatric patients at risk for hypoglycemia); hydration should be maintained with IV fluids. Water should be offered after 12 hours and the animal observed for signs of dysphagia, pain, or regurgitation. If no serious problems are identified, soft food may be offered between 12 and 24 hours after surgery. Gruel is unnecessary and may seep between sutures, inhibiting healing. Hand feeding (tossing meatballs) and watering may be necessary for 1 to 2 weeks following major glossectomy, until the animal is trained to suck water and prehend food without a tongue. Feeding through a gastrostomy or esophagostomy tube is occasionally necessary for animals with severe wounds or those unwilling to eat within 3 days of surgery (see Chapter 10). Soft food should be fed and the animal prevented from chewing on sticks, toys, or other hard items until the wound has healed. Healing should be evaluated at 3 to 5 days, 2 weeks, and 4 weeks after surgery. Additional reconstruction may be necessary if areas of partial dehiscence do not heal by second intention or if an oronasal fistula remains after 4 to 6 weeks. Animals with neoplasia should be evaluated every 3 to 6 months for tumor recurrence.

Epistaxis, serous to mucoid nasal discharge, and pain are expected after maxillectomy. Crusting of the nares and epiphora may occur when the nasolacrimal duct is transected. Subcutaneous emphysema occasionally occurs when a large portion of the nasal cavity is exposed. Emphysema, crusting, and nasal discharge are short-term sequelae that usually resolve in days to weeks. Cosmesis is usually good, with a slight facial concavity and lip elevation after lateral maxillectomy. Removal of a canine tooth causes more noticeable concavity. Premaxillectomies involving all incisors and one or both canine teeth produce ventral drooping of the nose and displacement of the maxillary lip caudal to the mandibular canine teeth unless further reconstruction (e.g., cantilever technique) is performed. Extensive premaxillectomies (caudal to the first premolar) shorten the nose and may cause an obvious prognathic appearance (protrusion of the lower jaw).

NOTE Dogs do better after hemimandibulectomies than do cats. Warn owners that some cats refuse to eat after this procedure and may require a feeding tube.

Cosmesis and function after partial mandibulectomy are good (see Fig. 18.7). Mandibular "drift" and instability occur more often when the osteotomy is caudal to the second premolar. The remaining hemimandible may deviate medially, producing malocclusion, clicking of the teeth, and/or trauma to the palatal or gingival mucosa. Most animals adapt and serious problems seldom occur. The involved canine tooth may be pulled or shortened if erosion or ulceration develops. Alternatively, use of orthodontic elastic chain attached to the ipsilateral canine and maxillary fourth premolar has been shown to decrease mandibular drift in a small number of patients. Significant client compliance is required in changing the chain weekly for 4 to 6 weeks, but indefinite use has been considered for prevention of mandibular drift. Rostral mandibulectomy (bilateral) caudal to the third or fourth premolar may cause difficulty with prehension and is less cosmetic. Aggressive rostral mandibulectomy may be combined with reconstruction using a plate and matrix combination; however, advanced imaging and 3D printing of the skull are required and dehiscence may require reoperation.[2] Tongue protrusion may occur, but most patients are able to keep the tongue retracted. Cheiloplasty may decrease drooling and lateral tongue protrusion in animals after partial mandibulectomies or maxillectomies.

COMPLICATIONS

Minor swelling of the skin and mucous membranes should be expected after partial maxillectomy or mandibulectomy but should resolve within 2 to 3 days. Infection is possible because the oral cavity is contaminated. However, infection is rare if the blood supply is maintained and good surgical technique is used. Partial dehiscence may occur 3 to 5 days after surgery if tissue is severely traumatized, blood supply is inadequate, or excessive motion or tension affects any area of the repair. Sometimes appearances that are unacceptable to owners may be modified with further cosmetic reconstruction following maxillectomy or mandibulectomy. Zygomatic mucocele has been reported after caudal hemimaxillectomy. The most common postoperative complication after tonsillectomy is hemorrhage. Regrowth of tonsillar tissue may occur if excision is incomplete. Apparent hyperptyalism following major glossectomy occasionally occurs;

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improvement may be seen with bilateral sialoadenectomy. Tumor recurrence or metastasis is always possible after any oncologic surgery.

SPECIAL AGE CONSIDERATIONS

Pediatric patients with congenital cleft palate should be fed with a tube until they are 8 to 12 weeks old and may be more safely anesthetized. Refer to the anesthesia section in Chapter 26 (see p. 722) for discussion of special concerns with pediatric patients. Pediatric patients are at higher risk of hypothermia and hypoglycemia; therefore they should not be fasted for more than 4 to 8 hours. Neoplasia is more common in geriatric patients; they should be thoroughly evaluated before surgery for concurrent disease and metastasis.

SPECIFIC DISEASES

CONGENITAL ORONASAL FISTULA (CLEFT PALATE)

DEFINITIONS

A **congenital oronasal fistula** is an abnormal communication between the oral and nasal cavities, involving the soft palate, hard palate, premaxilla, and/or lip. The **primary palate** consists of the lip and premaxilla. Incomplete closure of the primary palate is a **primary cleft** or **cleft lip** (harelip). The **secondary palate** consists of the hard and soft palates. Incomplete closure of either of these structures is a **secondary cleft** or **cleft palate.** Animals may also have unilateral or bilateral failure of the soft palate to fuse with the nasopharyngeal wall; this is termed a **lateral cleft of the soft palate** or **hypoplastic soft palate**.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Congenital palatal defects result when the two palatine shelves fail to fuse during fetal development (Fig. 18.16). The most critical time for development and closure of the fetal palate appears to be 25 to 28 days of gestation in dogs. Incomplete closure of either the primary or secondary palate is attributed to inherited (recessive or irregular dominant, polygenic traits), nutritional (inadequate folic acid), hormonal (steroids), mechanical (in utero trauma), and toxic (including viral) factors. Primary cleft palate alone is rare (Fig. 18.17); however, secondary cleft palate may occur alone or in combination with primary clefts. Some affected neonates are unable to nurse effectively and die soon after birth. Others contaminate their nasal cavity with saliva and food. Signs of rhinitis and other respiratory infections are common. Middle ear disease that is often clinically unrecognized is sometimes associated with congenital clefts.

DIAGNOSIS

Clinical Presentation

Signalment

Dogs, particularly brachycephalic breeds, are more commonly affected with cleft palate than cats. Purebred dogs have a higher incidence than mixed breeds. Breeds at high risk for cleft palate include Boston terriers, Pekingese, bulldogs, miniature schnauzers, beagles, cocker spaniels, and dachshunds. Siamese cats have a higher incidence than other cat breeds. Females are more commonly affected than males. The cleft is present at birth, although it is not always recognized immediately.

History

A history of difficulty nursing, nasal regurgitation, nasal discharge, and failure to thrive are common problems. Signs related to incomplete separation of the oral and nasal cavity include drainage of milk from the nares during or after nursing; gagging, coughing, or sneezing (or all three) while eating; poor growth; and respiratory infection (i.e., rhinitis, aspiration pneumonia).

Physical Examination Findings

All puppies and kittens should be checked on initial presentation for evidence of a cleft palate. Diagnosis of congenital oronasal fistula is made by visual examination. Incomplete closure of the lip is easily recognized when the patient is first examined; however, a thorough oral examination is required to identify incomplete closure of the premaxilla, hard palate, or soft palate. Anesthesia may be necessary to thoroughly assess the soft palate. A secondary cleft may occur without a primary cleft. Patients may be thin and stunted. Abnormal respiratory sounds are auscultated if aspiration pneumonia is present.



FIG. 18.16 A puppy with a cleft extending through the primary and secondary palate.

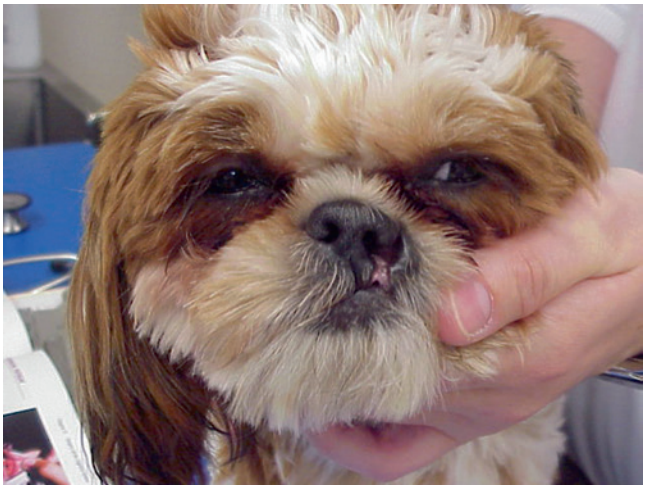


FIG. 18.17 Primary cleft palate in a young shih tzu involving the lip, premaxilla, and nostril.

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NOTE Be sure to thoroughly evaluate affected neonates for concurrent congenital anomalies.

Diagnostic Imaging

Radiographic examination of the skull is not necessary; however, thoracic radiographs are useful in evaluating for aspiration pneumonia, typically characterized by interstitial to alveolar infiltrates within the ventral aspect of the lungs. It should be noted that middle ear disease can be seen in association with defects in the palate; thus imaging of the tympanic bullae may be indicated. Advanced imaging may also be considered for dramatic palatal abnormalities and for acquired defects.

Laboratory Findings

Laboratory abnormalities are not present unless the animal has aspiration pneumonia or is cachectic.

DIFFERENTIAL DIAGNOSIS

Traumatic or acquired clefts, rhinitis, nasal foreign body, and aspiration pneumonia are potential differential or concurrent diagnoses.

MEDICAL MANAGEMENT

Affected patients should be tube fed to maintain an adequate nutritional status and reduce the incidence of aspiration pneumonia until they are old enough for surgery. Aspiration pneumonia may be treated with antibiotics, fluids, oxygen, bronchodilators, and/or expectorants. Use of glucocorticoids for acute aspiration is controversial and clearly has no place in chronic aspiration. A tracheal wash or brushing for culture and sensitivity testing should be performed if aspiration pneumonia is severe. Broad-spectrum antibiotics with efficacy against anaerobes (Box 18.3) are indicated for severe aspiration or purulent rhinitis. Animals with severe rhinitis may benefit from having the nasal infection treated before surgical closure of the defect. Cultures should be obtained from the nasal cavity after anesthetizing the patient and the nose and oral cavity flushed of debris and exudate. To prevent recontamination of the nasal cavity with food, the animal should be given nothing by mouth for 10 to 14 days. Nutrition can be provided via tube feeding (e.g., esophagostomy or gastrostomy tube; see pp. 95 and 97, respectively) during this time.

SURGICAL TREATMENT

Most animals with defects of the primary and secondary palate are euthanized or die. Surgical treatment is generally delayed until the patient is at least 8 to 12 weeks of age to allow growth and easier access to the palate. Older patients seem to have less friable tissue that holds suture better. Palatoplasty performed before 16 weeks of age may hinder maxillofacial growth and development. Although rare, a narrower maxilla and occlusal problems may result. The primary goal of repairing a cleft palate is to reconstruct the nasal floor. Several procedures may be necessary before the entire cleft is permanently reconstructed. This is a hereditary defect; affected patients should be neutered.

Preoperative Management

Pediatric patients should not be fasted longer than 4 to 8 hours. After induction of anesthesia and placement of a cuffed endotracheal tube, the nasal and oral cavities should be flushed with saline and a dilute antiseptic solution. Perioperative antibiotics may be given intravenously at anesthetic induction if the animal is not already receiving them. Poorly nourished animals should be fed through a gastrostomy or esophagostomy tube for several days before surgery.

BOX 18.3 Antibiotic Treatment of Aspiration Pneumonia

Chloramphenicol (Chloromycetin)
Dogs: 50 mg/kg PO; 10–15 mg/kg IV q8h
Cats: 20–40 mg/kg PO, IM, SC, or IV q12h

Cefazolin (Ancef, Kefzol)
22 mg/kg IV q8h

Enrofloxacin (Baytril)
Dogs: 7–20 mg/kg PO or IV q24h (must be given diluted and slowly over 30 min)
Cats: 5 mg/kg PO or IV q24h (must be given diluted and slowly over 30 min)[a]

Ampicillin
22 mg/kg PO, IV, IM, or SC q6–8h

Amikacin (Amiglyde-V)
Dogs: 15–22 mg/kg IV q24h
Cats: 10–14 mg/kg IV q24h

Clindamycin (Antirobe, Cleocin)
Dogs: 11–33 mg/kg PO q8–12h, *or* 11 mg/kg IV q8–12h; when giving IV dilute and administer slowly
Cats: 5–33 mg/kg PO or SC q12–24h

Ampicillin Sodium & Sulbactam Sodium (Unasyn)
15–30 mg/kg IV q6–8h (off-label use)

[a]Can cause blindness in cats receiving high doses.
IM, Intramuscular; *IV,* intravenous; *PO,* orally; *SC,* subcutaneous.

Anesthesia

Puppies and kittens are better able to metabolize drugs after 8 weeks of age, making them better anesthetic risks. Special precautions should be taken to prevent hypothermia and hypoglycemia in young patients. Refer to the anesthesia section in Chapter 26 (see p. 722) for discussion of special concerns with pediatric patients. Intubation through a pharyngotomy (see p. 1044) or tracheotomy incision (see p. 842) may facilitate repair of secondary clefts. Pharyngotomy is generally preferred if it allows adequate visualization of the defect. General anesthetic recommendations for animals undergoing oral surgery are provided on p. 331. Guarded tracheostomy tubes should be used to prevent kinking during the procedure. Care should be taken to prevent and recognize dislodgment of the anesthetic tubing from the endotracheal tube during oral manipulations.

Surgical Anatomy

The major palatine arteries emerge from the major palatine foramen midway between the midline and caudal edge of the upper fourth premolar (see Fig. 18.1). The main artery courses rostrally, equidistant between the lingual border of the teeth and the palatal midline, to anastomose with the major palatine artery of the contralateral side caudal to the incisors. The minor palatine arteries enter the palate at the level of the last molar, caudal and

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slightly lateral to the major palatine foramen. The minor palatine arteries course caudomedially and ramify in the caudal hard palate and soft palate. The soft palate is also supplied by branches of the ascending pharyngeal artery.

POSITIONING

To facilitate repair of a secondary palate defect, the animal should be positioned in dorsal recumbency with the mouth maximally opened. Ventral or dorsal recumbency may be used for a primary palate repair.

SURGICAL TECHNIQUES

The first step in the repair of combined primary and secondary clefts is to separate the oral and nasal cavities by reconstructing the nasal floor. After separation, the lip defect is reconstructed. Areas where flaps were harvested for closure of primary or secondary clefts are allowed to heal by second intention. Granulation and epithelialization are usually complete within 2 to 3 weeks.

Closure of Hard Palate Defects

AP The two procedures most often used to repair secondary clefts are sliding bipedicle flaps and overlapping flap techniques. Sliding bipedicle flaps (von Langenbeck technique) are created to close hard palate defects. The disadvantage of this technique is that the repair is unsupported and directly over the defect. Staged repair can be done to increase coverage of palatal defects.[3] In a study of six dogs, dental extractions with repair of the alveolar defects was done first, followed by double flap closure over the defect 4 weeks later. Successful repair was reported in three dogs; two dogs required surgical revision, and in one failure, further surgery was declined by the owner.

Incise the margins of the defect and make bilateral releasing incisions along the margins of the dental arcade (Fig. 18.18A). Elevate the mucoperiosteal layer on both sides of the defect with a periosteal elevator (see Fig. 18.18B). Avoid damaging the major palatine arteries. Control hemorrhage with pressure and suction. Appose the nasal mucosal edges or periosteum at the margin of the defect with buried interrupted sutures (knots within the nasal cavity) if possible. Slide the elevated mucoperiosteal flaps across the defect and appose with simple interrupted sutures (see Fig. 18.18C–D). Allow the denuded hard palate near the dental arcades to heal by secondary intention. A variation of this technique, in which a partial-thickness rather than a full-thickness incision of the mucoperiosteum is made without exposure of the palatal bone, may reduce maxillofacial deformity.

An alternate technique for repair of hard palate defects is the overlapping "sandwich" technique (Figs. 18.19 and 18.20). This

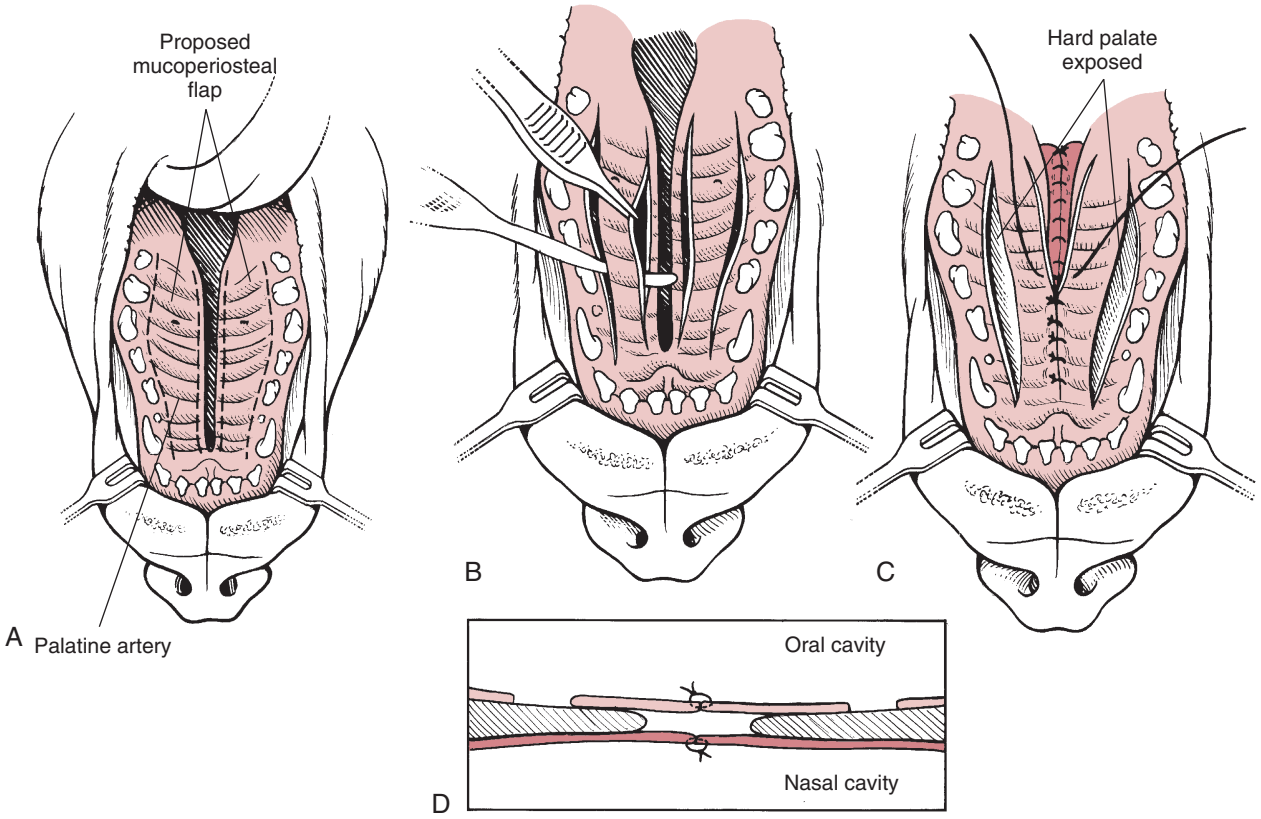


FIG. 18.18 A sliding bipedicle flap repair may be used to repair a congenital oronasal fistula. (A) The *dashed lines* represent the mucoperiosteal incisions necessary to create two sliding flaps. (B) The mucoperiosteum is elevated from the hard palate with the major palatine artery. (C) The nasal mucosa and mucoperiosteum are apposed in two layers over the defect in the hard palate. (D) Cross-sectional view of the repair.

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FIG. 18.19 The secondary palate of the puppy in Fig. 18.16 was repaired with an overlapping flap technique. The defect over the hard palate was allowed to heal by second intention.

technique is advantageous because it does not place the repair over the palate defect. *Incise one margin of the defect separating the oral and nasal mucosa (see Fig. 18.20A). Elevate the mucoperiosteum at this edge by approximately 5 mm. At the opposite side of the defect, create a mucoperiosteal rotational flap large enough to cover the defect with its base hinged at the margin of the palatal defect (see Fig. 18.20B). Begin the incision near and parallel to the dental arcade, creating a flap 2 to 4 mm larger than the defect. Make perpendicular incisions at the rostral and caudal end of the incision extending to the cleft. Elevate this mucoperiosteal flap, taking care not to disrupt the margin of the defect (see Fig. 18.20B). Dissect carefully around the palatine artery to release it from fibrous tissue. Rotate the flap across the defect (see Fig. 18.20C). Place the edge of the flap under the mucoperiosteal flap on the opposite side. Preplace and then tie a series of horizontal mattress sutures to secure the flaps in position (see Fig. 18.20D).*

NOTE Warn owners that several operations may be needed to completely close large hard palate defects.

Closure of Soft Palate Defects

Incise the margins of the cleft to separate the oral and nasal mucosae. Continue incisions made in the margins of hard palate clefts caudally into the soft palate (see Fig. 18.20D). Isolate the nasal mucosa, palatal muscles, and oral mucosa. Appose the palatal edges in three layers, beginning caudally and working rostrally to a point adjacent to the caudal or midpoint of the tonsil. First appose the nasal mucosa using a series of simple interrupted sutures with nasally oriented knots or use a simple continuous pattern. Then appose the palatal muscle and connective tissue with a simple continuous suture pattern. Last, appose the oral mucosa with a simple continuous or interrupted suture pattern. Make tension-relieving incisions in the oral mucosa from the lingual aspect of the last molar to near the tip of the soft palate.

An overlapping flap technique, rotational flaps from the hard or soft palate, and nasal or nasopharyngeal mucosal flaps can also be used to repair soft palate defects. Correction of lateral soft palate clefts generally includes an oropharyngeal/nasopharyngeal mucosal flap. Some surgeons fracture the pterygoid hamulus (where the palatal muscles attach) with an osteotome to reduce tension on the soft palate.

Reconstruction of Soft Palate Hypoplasia

Bilateral pharyngeal mucosal flaps may be used to reconstruct a hypoplastic soft palate. *Create two random-pattern flaps from the right and left pharyngeal walls that are based laterally and extend into the nasopharynx.* The base of each flap is at the caudal aspect of the last molar and extends caudally to the rostral margin of the tonsillar crypt. *Create a flap of hard palate mucoperiosteum, extending from the lateral aspects of the hard palate medial to the dental arcades, taking care to preserve the palatine arteries and attachment at the caudal border of the hard palate.* Caudal rotation of the mucoperiosteum results in the mucosa facing the new floor of the soft palate. *Suture the edges of the flap to the lateral walls of the pharynx. Suture the ends of the lateral pharyngeal flaps together on the midline to form the oral mucosal surface of the soft palate. Suture the rostral border of the lateral pharyngeal flaps to the base of the hard palate and the caudal aspect of the three flaps in separate rows of simple continuous patterns.*

Closure of Primary Clefts

AP Cosmetic repair of primary clefts can be very complicated, requiring elaborate planning to achieve a successful outcome (Fig. 18.21A).

Create a mucosal flap to separate the nasal cavity from the oral cavity (see Fig. 18.21B). If the cleft extends into the premaxilla, evaluate the gingival mucosa of the deciduous incisors and pull them if necessary. Suture the buccal or gingival mucosal flap to the nasal mucosa. Use a freehand modified Z-plasty for reconstruction of the lip defect (see Fig. 18.21C). Close the lip defect so that the distance from the ventral nostril to the free ventral edge of the lip is the same on both sides. Make multiple small flaps, if necessary, for a cosmetic closure. Place a layer of sutures in the fibromuscular layer (orbicularis oris muscle and connective tissue) before skin closure.

POSTOPERATIVE CARE AND ASSESSMENT

Soft food should be fed for a minimum of 2 weeks after surgery, and chewing on hard objects (e.g., bones, sticks, chew toys) should be prevented. Esophagostomy feeding for 7 to 14 days may facilitate healing. Reevaluation at 2 weeks may require anesthesia to identify small defects.

COMPLICATIONS

Dehiscence and subsequent incomplete healing of oronasal fistulae are the most common complications. Dehiscence usually occurs within 3 to 5 days of surgery but may occur later. Early dehiscence may occur if tension is excessive, blood supply is poor, or the tissue is traumatized. Motion of the tongue against the repair and particulate matter in the surgical site can also lead to dehiscence. Early dehiscence of the lip occurs if the orbicularis oris muscle has not been apposed. Contraction of the unapposed muscle during lip movement causes excess tension on the suture line. Tissues are friable after dehiscence; therefore repair of early dehiscence should be delayed for 4 to 6 weeks to allow tissues to revascularize and regain strength. Late dehiscence is a result of growth-induced stress on the repair and is best treated after the patient matures.

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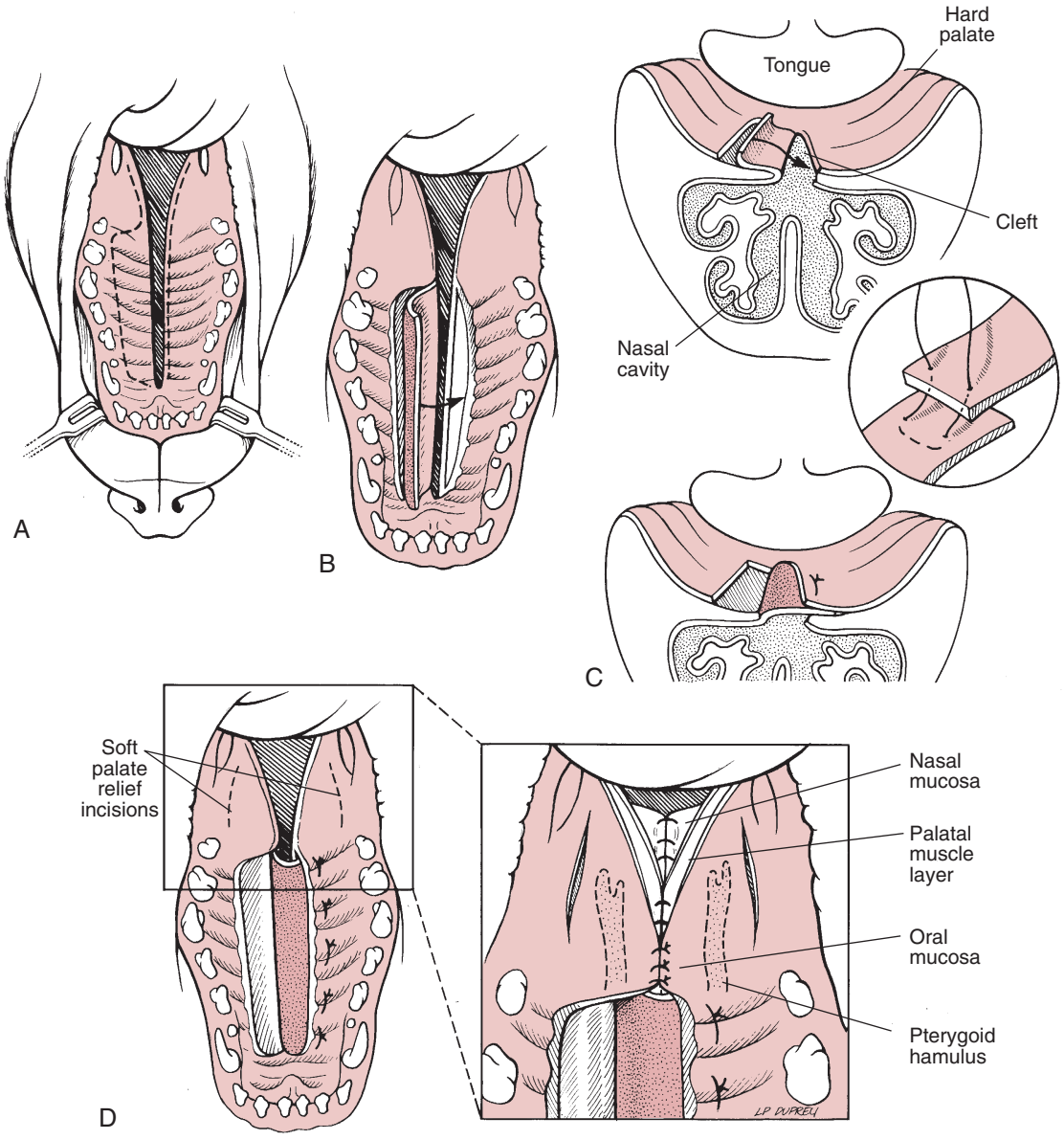


FIG. 18.20 A congenital oronasal fistula may be repaired with an overlapping flap technique. (A) The *dotted lines* represent the incisions necessary to allow soft tissue closure. (B) Elevate the mucoperiosteal flap and rotate it medially to cover the hard palate defect. (C) Insert the edge of this flap between the hard palate and the mucoperiosteum on the opposite side of the defect. Secure the flaps in position with horizontal mattress sutures *(insert)*. (D) Complete the repair by apposing the incised edges of the cleft soft palate in three layers. Make lateral relief incisions *(broken lines)* to reduce tension on the repair.

PROGNOSIS

The prognosis is good for animals with successful cleft palate repair; however, several operations may be required. Chronic rhinitis and aspiration pneumonia persist if large defects are not repaired. Untreated patients with small clefts may have few clinical signs.

ACQUIRED ORONASAL FISTULAE

DEFINITION/SYNONYMS

Acquired oronasal fistulae (traumatic cleft palate, palatal defect) are abnormal communications between the nasal and oral cavities caused by trauma or disease.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Acquired palatal defects are most often caused by dental disease (Fig. 18.22). An oronasal fistula results when a deep maxillary periodontal pocket progresses to the apex of the tooth, lysing bone between the apex of the alveolus and the nasal cavity or maxillary sinus. A fistula may also result from trauma (i.e., bite wounds, gunshot wounds, blunt trauma to the head, electrical burns) or may be a complication of surgery (e.g., mass excision or ventral rhinotomy), radiation, or hyperthermic treatment of oral lesions. Foreign bodies lodged between the dental arcades may cause pressure necrosis of the hard palate and subsequent development of an oronasal fistula (Fig. 18.23). Ingested food

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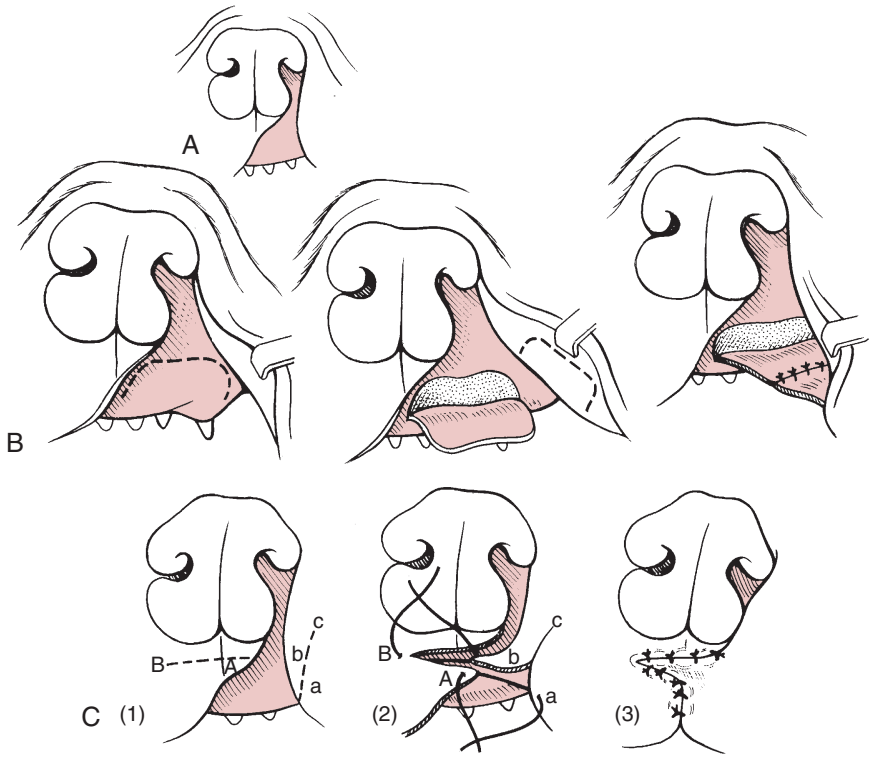


FIG. 18.21 (A) Schematic drawing of a repair of a primary cleft palate involving the lip, premaxilla, and nostril. (B) Create a flap from the nasal wall and suture it to a labial mucosal flap to separate the nasal cavity from the oral cavity. (C) Repair the cleft lip with one or a series of Z-plasties: *(1)* make incisions from A to B and a to c; *(2)* place a suture between A and a and B and b to transpose the flaps; *(3)* place additional sutures as needed.



FIG. 18.22 An oronasal fistula that occurred from loss of the canine tooth.



FIG. 18.23 An acquired oronasal fistula caused by a foreign body that lodged between the dental arcades.

that passes through the fistula into the nasal cavity may be expelled from the nostril by sneezing. Chronic rhinitis is common.

DIAGNOSIS

Clinical Presentation

Signalment

Any breed or sex may acquire an oronasal fistula. Oronasal fistulae that occur secondary to dental disease or tumors are seen more often in middle-aged and older animals. Oronasal fistulae that develop secondary to trauma may occur at any age.

History

An oronasal fistula should be suspected in patients with chronic rhinitis and a history of dental disease, trauma, or previously treated oral tumors. Common clinical signs are sneezing and chronic unilateral serous or mucopurulent nasal discharge.

Physical Examination Findings

The diagnosis of an oronasal fistula can be made by identifying an abnormal communication between the oral and nasal cavities

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(see Figs. 18.22 and 18.23). Small fistulae associated with periodontal disease are not easily identified unless the area around the involved tooth is explored with a narrow dental probe. If passing the probe into the gingival pocket causes epistaxis, a fistula is present. The palatal aspect of the maxillary canine tooth is a common site for an oronasal fistula. Anesthesia is generally required to probe periodontal pockets.

Diagnostic Imaging

Skull radiography or cross-sectional imaging may identify underlying causes of fistulae, such as periapical abscesses, advanced periodontal disease, maxillary neoplasia (see p. 352), or broken or retained tooth roots. Lysis, especially of the laminae dura around tooth roots, is indicative of periapical abscesses.

Laboratory Findings

Inflammatory changes on a CBC may be seen secondary to rhinitis or aspiration pneumonia.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include any disease that causes chronic rhinitis (e.g., fungal disease, nasal foreign body, congenital oronasal fistula, invasive oral neoplasia) and congenital clefts. These conditions can generally be differentiated based on physical examination, imaging, and/or histopathologic examination. Histopathologic evaluation should be performed to distinguish fistulae that occur secondary to neoplasia from those associated with infection or trauma.

MEDICAL MANAGEMENT

Broad-spectrum antibiotics effective against anaerobes (e.g., chloramphenicol, amoxicillin plus clavulanic acid, clindamycin) should be given if severe purulent rhinitis is present (see Box 18.3). Such animals may benefit from having the nasal infection treated before closure of the defect. With the patient under general anesthesia, cultures should be obtained from the nasal cavity, and the nose and oral cavity flushed of debris and exudate. To prevent recontamination of the nasal cavity with food, the animal should be given nothing by mouth for 10 to 14 days. Nutrition can be provided via tube feeding (e.g., tube gastrostomy, p. 97, or esophagostomy, p. 95) during this time.

SURGICAL TREATMENT

Most oronasal fistulae require surgical reconstruction, although small or traumatic fistulae occasionally heal spontaneously. A variety of surgical techniques have been described for repair, including simple suturing of the fistula edges, mucosal flaps, mucoperiosteal flaps, double reposition flaps, orbicularis oris axial pattern flaps, palatal island flaps, and two-staged tongue flaps. Successful repair of oronasal fistulae requires a well-supported, air-tight, tension-free closure. Flap techniques are more successful than direct apposition of the fistula edges because there is less tension and increased support for the repair. Teeth involved in the fistula should be extracted several weeks before reconstruction of the defect. Central lesions may require that normal teeth be extracted to allow creation of adequate mucosal flaps. If the fistula is of dental origin, it may be necessary to perform a limited maxillectomy (at least 5 mm from each margin) to remove necrotic or diseased bone. Traumatic oronasal fistulae may require stabilization of the maxilla and hard palate with small pins or wire. Interdental wiring (see p. 1045) using the carnassial teeth or the canine teeth (or both) can help bring bone edges into apposition. Areas where flaps were harvested heal by second intention in 2 to 3 weeks. Acrylic, Silastic, or metal obturators may also be fitted over the defect.

Preoperative Management

Pediatric patients should not be fasted for longer than 4 to 8 hours. After anesthetic induction, the nasal and oral cavities should be flushed with saline-diluted antiseptic solution. Aggressive management of rhinitis (see Medical Management, presented earlier) may reduce infection and improve the tissues' ability to hold suture.

Anesthesia

General anesthetic recommendations for animals undergoing oral surgery are provided on p. 331 (see Table 18.1). Intubation through a pharyngotomy (see p. 1044) or tracheotomy incision (see p. 842) may facilitate repair of large or centrally located oronasal fistulae. Pharyngotomy is generally preferred if it allows adequate visualization of the defect. Care should be taken to prevent and recognize dislodgment of the anesthetic tubing from the endotracheal tube during oral manipulations.

> **NOTE** Use guarded tracheostomy tubes to prevent kinking during oral procedures.

Surgical Anatomy

The surgical anatomy of the hard palate is discussed on p. 343.

Positioning

The patient should be positioned in lateral recumbency for repair of oronasal fistulae associated with the dental arcade. Dorsal recumbency with the mouth opened maximally facilitates repair of more centrally located fistulae involving the secondary palate.

SURGICAL TECHNIQUES

Direct Apposition

Direct apposition of the fistula should be performed only if the fistula is very small. *Debride the fistula to healthy, bleeding mucosal edges (Fig. 18.24A–B). Incise or debride the margin of the fistula and elevate the edges enough to allow approximation without excess tension. Appose the mucosa with interrupted appositional sutures (i.e., simple, cruciate, or vertical mattress pattern) (see Fig. 18.24C).*

Single-Layer Flap Repair

AP *Debride the epithelial margin of the fistula (Fig. 18.25A). Incise the gingival and buccal mucosa to outline a flap 2 to 4 mm larger than the debrided fistula (see Fig. 18.25B). Make these incisions perpendicular to the dental arcade. Elevate the gingival mucosa with a periosteal elevator. Then undermine the buccal mucosa until the flap can be advanced across the defect without tension (see Fig. 18.25C). Using a rongeur, remove infected alveolar and maxillary bone. Expose approximately 1 to 2 mm of the hard palate at the medial aspect of the fistula by excising 1 to 2 mm of mucoperiosteum. Lavage the surgical site with saline. Suture the gingival-buccal flap to the mucoperiosteum of the hard palate in an interrupted approximating pattern (i.e., simple, cruciate, or vertical mattress) using monofilament, absorbable (3-0 to 4-0)*

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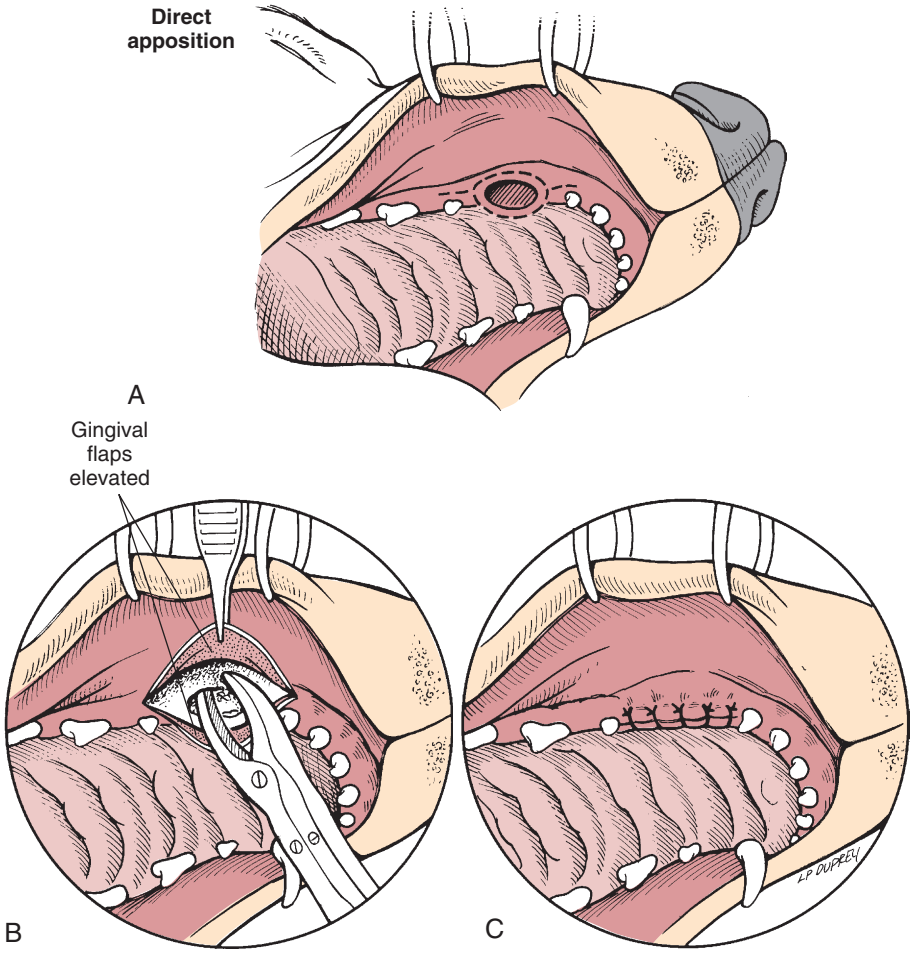


FIG. 18.24 Small fistulae may be repaired with a direct appositional technique. (A) Incise the mucosa around the fistula. (B) Elevate the gingival flaps and debride the edges of the fistula. (C) Appose the mucosa over the defect.

sutures. Inclusion of the angularis oris artery, which arises from the facial artery caudal to the commissure of the lips, during flap creation yields a mobile, strong flap with a good blood supply that may extend to the ipsilateral canine or across the palate to the opposite dental arcade.

Rotational Flap Repair

A rotational or advancement flap may be created from the hard or soft palate (see Fig. 18.25D–G), or an overlapping technique similar to that described for repair of congenital oronasal fistulae may be used (see p. 346). *Create a flap 2 to 4 mm larger than the debrided fistula. To ensure a good blood supply, incorporate the major or minor palatine artery in palatal flaps. Transpose and suture the flap over the defect (see Fig. 18.25D–G).*

NOTE Granulation tissue fills the defect over the hard palate, and the area reepithelializes within a few weeks.

Double-Flap Repair

Double-flap techniques may be used with large dental fistulae and fistulae located in more central areas of the palate. Double-flap techniques provide a mucosal surface on both the oral and nasal sides of the fistula. If buccal flaps are planned to close large central defects, extraction of teeth may be necessary. The extraction sites should be allowed to heal before reconstruction. Do not debride the palatal epithelial margin during debridement of the fistula, because this edge serves as the base of the mucoperiosteal flap and must remain continuous with the nasal mucosa to be effective.

Create a flap in the mucoperiosteum 2 to 4 mm larger than the debrided fistula (Figs. 18.26–18.28). Elevate the flap without disrupting the palatal margin of the fistula. Fold the flap over the defect and suture it to the gingival mucosa with interrupted, approximating, monofilament, absorbable sutures for the first layer of closure (see Fig. 18.26). This flap provides "nasal" mucosa. *Create a rotational mucoperiosteal flap 2 to 4 mm larger than the defect for the second layer of closure (see Fig. 18.26). Appose this flap to the gingival mucosa with approximating 3-0 to 4-0 sutures. To ensure a good blood supply, incorporate the major palatine artery in palatal flaps. As an alternative, create one or two mucoperiosteal flaps 2 to 4 mm larger than the defect (see Fig. 18.28A). Transpose and suture the flap in place for the first layer of the closure (see Fig. 18.28B) to provide the "nasal" mucosa. Cover this layer with a mucosal flap (gingival and buccal) to provide the "oral" mucosal layer of the closure (see Fig. 18.28C–D). Allow the denuded hard palate to heal by second intention.*

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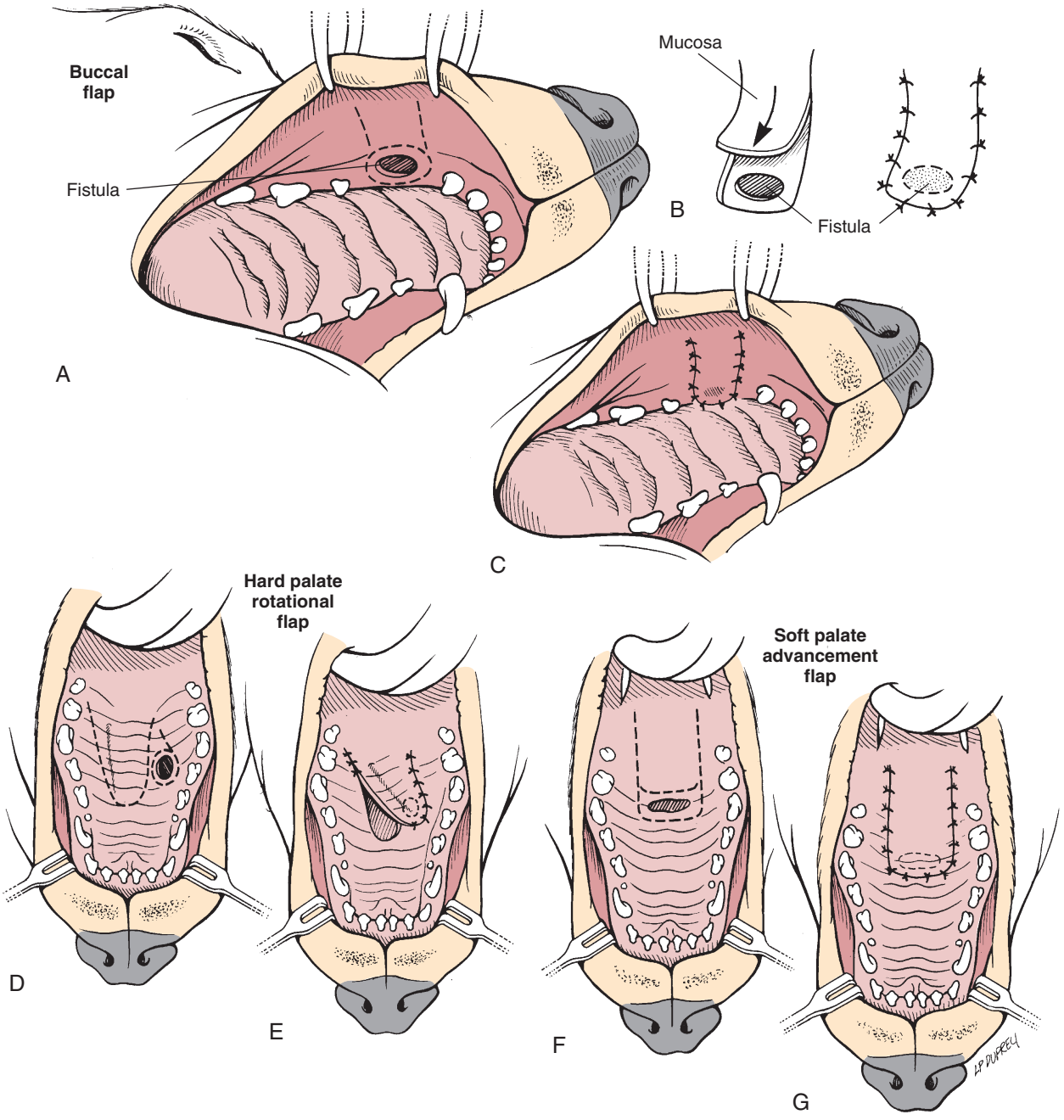


FIG. 18.25 Single-flap technique for fistula repair. (A) Incise mucosa around the fistula to create the buccal flap *(dashed line),* then debride the fistula. (B–C) Advance a buccal flap over the defect and suture into place. (D–E) After debriding the fistula, create a hard palate rotational flap *(dashed line)* and rotate the mucoperiosteal hard palate flap over the defect. Suture the flap to surrounding mucoperiosteum. (F–G) To repair lesions at the junction of the hard and soft palates, debride the defect, then create and close the defect with a soft palate advancement flap *(caudal dashed line).*

Conchal Cartilage Graft Repair

Repair of oronasal fistulae may be accomplished using auricular cartilage in cats with small, central defects of the hard palate. *Incise the skin over the caudoventral surface of the pinna, excise a round piece of the scapha of the pinna leaving the rostral skin intact (Fig. 18.29). Elevate the hard palate mucoperiosteum approximately 2 to 3 mm around the oronasal fistula to accommodate the graft. Insert the graft and suture it to the surrounding palate with simple interrupted sutures rostrally and caudally.*

Vertical Ear Canal Graft Repair

Excise the lateral two-thirds of the vertical ear canal and suture the skin to the mucosa of the horizontal ear canal (Fig. 18.30).

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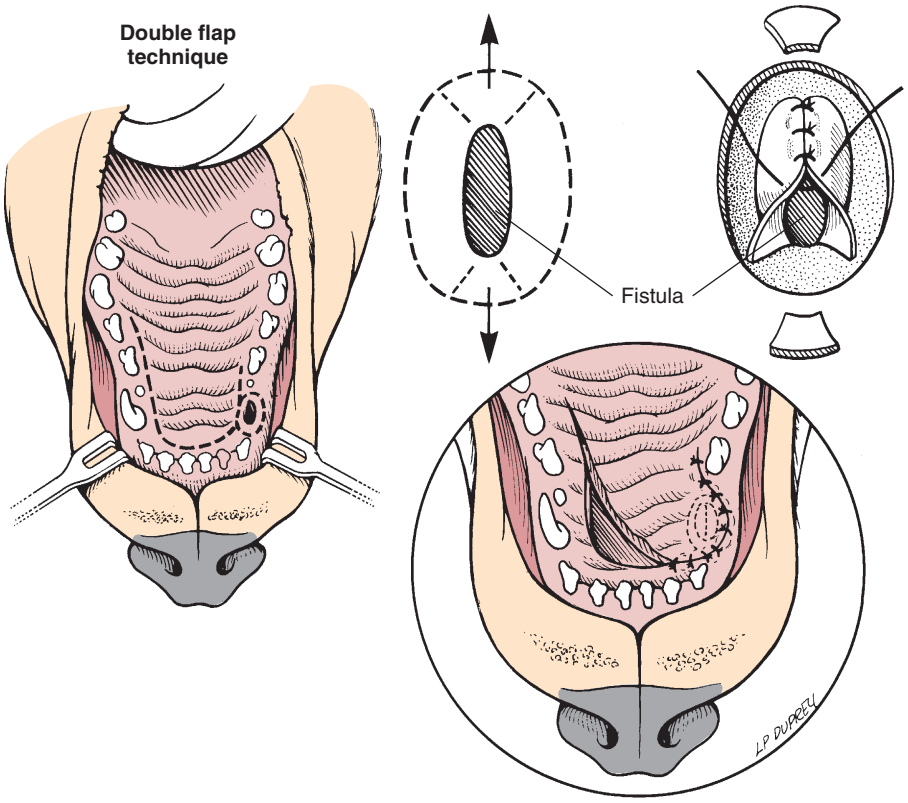


FIG. 18.26 A double-layer flap technique may be performed using tissue surrounding the fistula and a flap from the mucoperiosteum of the hard palate. Create the first flap *(gingival dashed line)* by rotating the gingival margins of the fistula medially and apposing with sutures *(top inset)*. Cover this flap *(bottom insert)* with a rotational mucoperiosteal hard palate flap *(palatal dashed line)*.

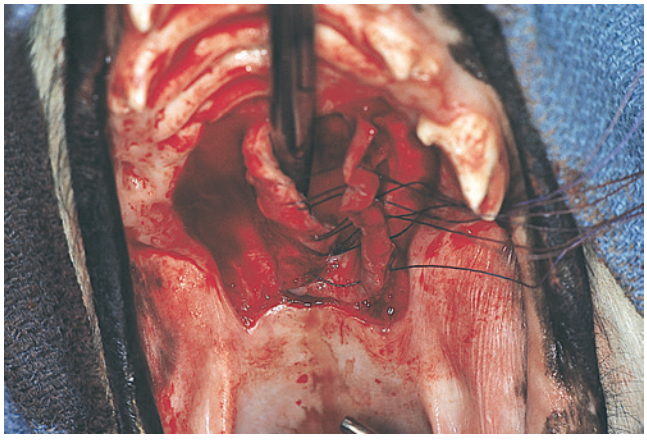


FIG. 18.27 Intraoperative image showing repair of an oronasal fistula using a double-layer flap technique. The margins of the fistula have been elevated and rotated medially to cover the defect. This flap was later covered with a flap created from the buccal mucosa.

Unfold the harvested cartilage, sharply excise the mucosa, and position the mucosal surface dorsally into the nasal cavity. Elevate the mucoperiosteum of the hard palate approximately 2 to 3 mm around the fistula to create a pocket for the graft and to create mucosal flaps to cover as much of the cartilage graft as possible. Secure the graft to the palate with 3-0 polydioxanone (PDS) vertical mattress sutures at each corner and midway between corners.

POSTOPERATIVE CARE AND ASSESSMENT

IV fluids should be provided until the animal begins eating and drinking (usually within 24 hours of surgery). Soft food should be fed for 2 to 3 weeks, and chewing on hard objects (i.e., toys, sticks) must be prevented to avoid dehiscence or perforation of the flap separating the oral and nasal cavities. If the animal paws at the mouth, an Elizabethan collar should be used. Severe rhinitis should be treated with antibiotics (see Box 18.3). Healing should be evaluated 2 and 4 weeks after surgery.

COMPLICATIONS

Most oronasal fistulae are successfully repaired if flaps can be apposed without tension and with a good blood supply. Dehiscence and recurrence of the oronasal fistula are expected if conditions for healing are not ideal. Motion of the tongue against the repair and particulate matter in the surgical site can also lead to dehiscence. Tension, poor blood supply, infection, poor debridement, lack of flap support, and traumatic technique may inhibit healing. Additional attempts to repair recurring fistulae should be delayed by 4 to 6 weeks to allow sites of previous flap harvesting to heal, revascularize, and mature before additional flaps are created. Rhinitis should resolve after the fistula has healed if irreversible mucosal changes have not occurred.

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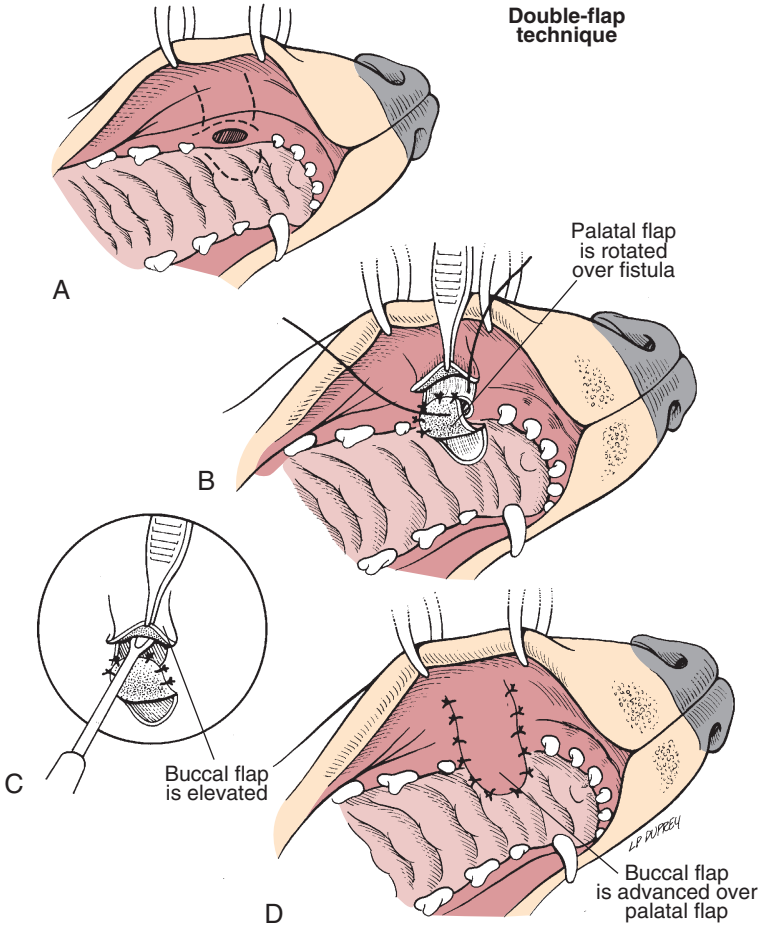


FIG. 18.28 A double-layer flap technique for fistula repair may be performed using a mucoperiosteal hard palate flap *(palatal dashed line)* and a buccal flap *(buccal dashed lines).* (A–B) Create a flap from the mucoperiosteum of the hard palate *(palatal and gingival dashed lines)* rotate and suture to gingival margin. (C–D) Cover it with a second flap created from the buccal mucosa *(inset),* then advanced and sutured over the first flap.

PROGNOSIS

Traumatic clefts may heal spontaneously in 2 to 4 weeks. Signs of rhinitis caused by regurgitation of food into the nasal cavity may be controlled with chronic antibiotic therapy. The long-term prognosis for most patients with nontraumatic fistulae is poor when surgical correction is not possible because fistulae do not heal without surgical reconstruction.

ORAL TUMORS

DEFINITIONS

Oral tumors encompass those neoplasms that arise from the gingiva, buccal mucosa, labial mucosa, tongue, tonsils, or dental elements. Malignant melanoma is also known as *melanosarcoma;* ameloblastoma is also referred to as *adamantinoma.*

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The oral cavity is the fourth most common site of neoplasia in dogs and cats; oral tumors account for 5% (dogs) to 7% (cats) of all malignant tumors in these species. Oral tumors occur 2.6 times more often in dogs than cats. Malignant oral tumors have a higher relative risk of occurring in male than in female dogs. Oral tumors originate from the mucosa, tongue, periodontium, odontogenic tissue, mandible, maxilla, tonsils, and lips, spreading by direct extension or invasion of adjacent bone and cartilaginous tissue. Metastasis occurs by lymphatics or blood to the regional lymph nodes and lungs. All tumors should be clinically staged according to the TNM classification system (primary tumor, regional lymph nodes, and metastasis) described by the World Health Organization (Table 18.2).

The most common malignant canine tumors are malignant melanoma, SCC, and fibrosarcoma, whereas SCC is the most common malignant oral tumor in cats. Other malignancies are listed in Box 18.4. Benign oral tumors are rare in cats. The most common benign oral neoplasms in dogs are epulides; other benign oral lesions are listed in Box 18.4.

Malignant melanomas are the most common malignant oral tumor in dogs; they rarely occur in cats (Box 18.5). Melanomas are rapidly growing, white-gray or brown-black tumors that are firm and vascular. They usually occur on the gingiva and are characterized by early local invasion. Some types are easily mistaken histologically for fibrosarcoma. Metastasis to the regional

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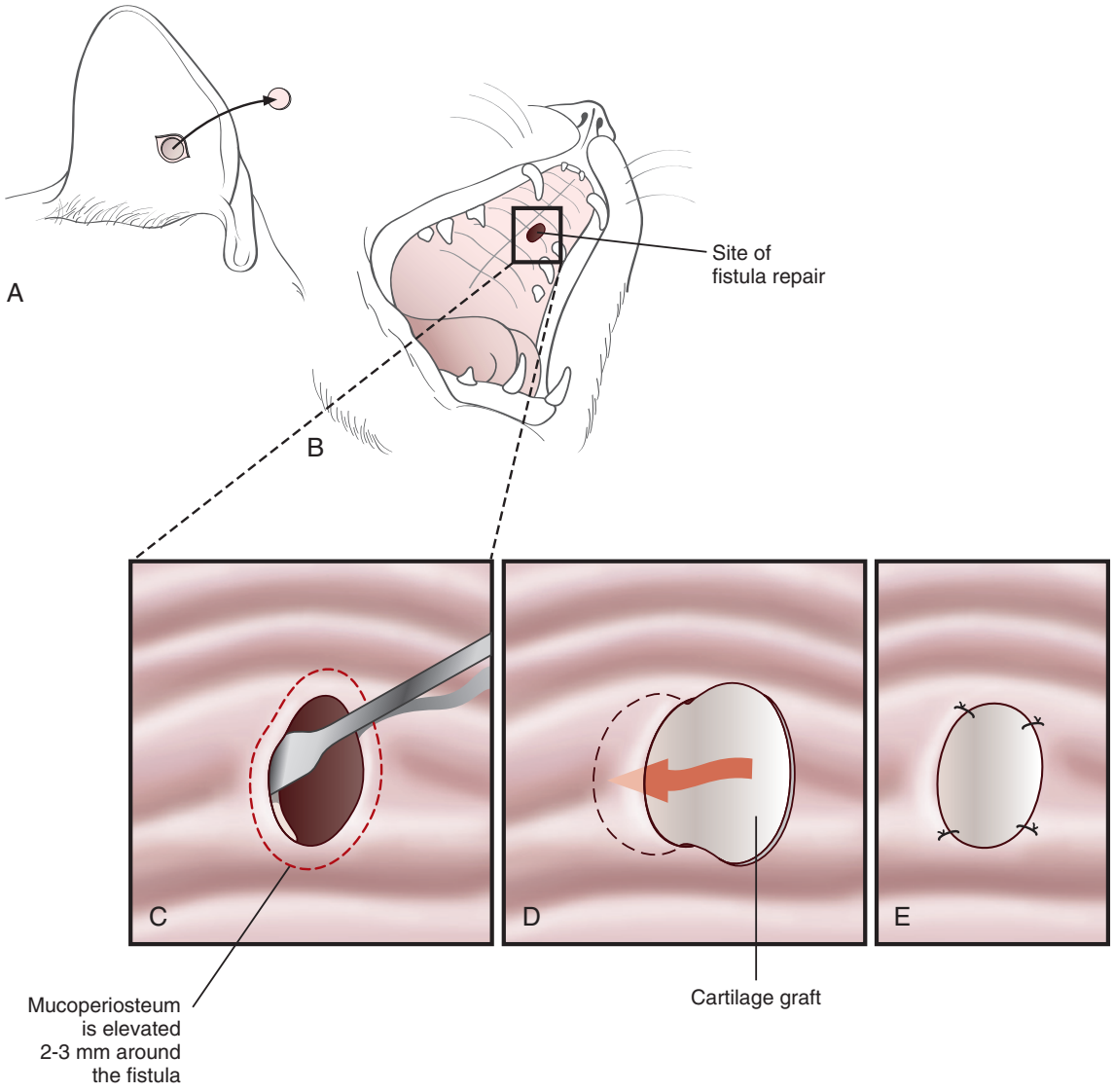


FIG. 18.29 Conchal cartilage graft repair. (A) Excise a round piece of the scapha of the pinna. (B) and (C) Elevate the mucoperiosteum. (D) Insert the cartilage. (E) Suture it to the surrounding tissues.

lymph nodes and lungs occurs in 80% of cases. Wide surgical excision using partial maxillectomy, mandibulectomy, tonsillectomy, or glossectomy is recommended. Radiation therapy may be used, but there is a high rate of recurrence. The response to radiotherapy may be better if concurrent hyperthermia is used, but the high rate of metastasis makes long-term survival unlikely. Administering cisplatin or carboplatin prior to radiation therapy provides better local control than radiation therapy alone. Chemotherapy and immunotherapy are of minimal benefit in treating malignant melanomas. A vaccine for canine melanoma is available, but its efficacy is uncertain at the time of this writing.

SCC is the most common malignant oral tumor in cats and the second most common malignant oral tumor in dogs (Box 18.6). Increased risk of SCC in cats has been associated with flea control products, diet, and environmental tobacco smoke. These tumors occur on the gingiva, lip, tongue, or tonsil. The masses are red, friable, vascular, and sometimes ulcerated. Most tumors in the rostral oropharynx are locally invasive, often invading bone and having a low metastatic potential, whereas those in the caudal oropharynx tend to be more infiltrative and metastasize more rapidly. Canine gingival SCC tends to be highly invasive and osteolytic but has a low rate of metastasis. Feline gingival SCC has a poorer prognosis than canine SCC. Wide resection is recommended for gingival tumors. They are radiosensitive, and combining radiotherapy with hyperthermia has been effective in palliating these tumors. Investigations into the use of chemotherapy alone or as an adjunct to surgery continue. Piroxicam may be helpful in controlling some SCCs. Tonsillar SCC grows rapidly and is associated with early local invasion and a high rate of lymph node and lung metastasis. It can also spread to the contralateral tonsil. These tumors occur most often in male urban dogs; tonsillar SCC has rarely been reported in cats. The prognosis for these tumors is guarded. SCCs may also occur on the tongue.

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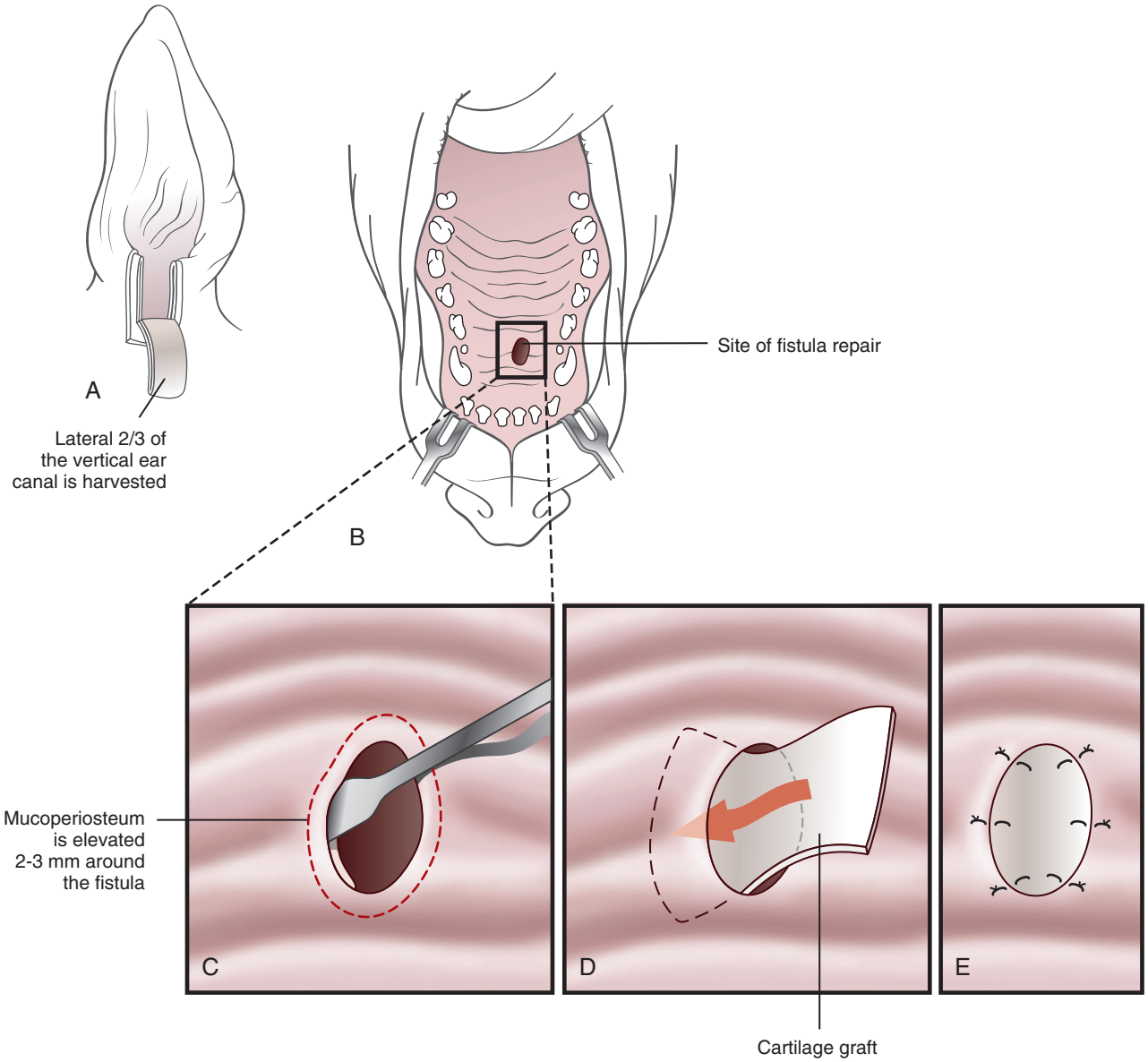


FIG. 18.30 Vertical ear canal graft repair. (A) Excise the lateral two-thirds of the vertical ear canal. (B) and (C) Elevate the mucoperiosteum around the fistula. (D–E) Position the mucosal surface of the cartilage dorsally into the nasal cavity. (E) Suture it to the surrounding tissues.

Fibrosarcomas are found primarily in dogs (Box 18.7). They most commonly occur on the maxillary gingiva and hard palate and appear as pink-red, firm, smooth, multilobulated masses that are often attached to underlying tissue. Local infiltration with bony involvement is common, but distant metastasis is uncommon. Local recurrence is high with any treatment; wide surgical resection is recommended. Most fibrosarcomas are poorly responsive to chemotherapy. Fibrosarcomas are generally radioresistant, although the median tumor control time with radiation alone may be as long as 12 months. Aggressive postoperative radiation therapy has improved survival times, but cryotherapy is believed to stimulate recurrence.

Osteosarcomas account for approximately 10% of canine mandibular and maxillary tumors. They are locally aggressive and have a high metastatic potential. The response to conventional therapies (i.e., surgery, radiation, chemotherapy) is poor, although survival is longer than with appendicular osteosarcoma.

The epulides are the most common benign oral neoplasms, accounting for 30% of all canine oral neoplasms (Box 18.8). They are rare in cats. These tumors are firm gingival masses that arise from the periodontal ligament. There are three types of epulides: fibromatous, ossifying, and acanthomatous. *Fibromatous epulides* are noninvasive, firm, smooth, pink masses that originate at the gingival sulcus and may be single or multiple, and pedunculated or sessile (Fig. 18.31). The primary cell type is periodontal ligament stroma. *Ossifying epulides* are similar to fibromatous epulides except that they have large amounts of osteoid matrix in the stroma of the periodontal ligament. They are firm and difficult to cut. Malignant transformation to osteosarcoma has been reported. *Acanthomatous epulides* are classified as benign

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TABLE 18.2 Clinical Stage Classification System for Canine and Feline Tumors of the Oral Cavity[a]

Minimum Requirements for Assessing T, N, and M Categories (If These Cannot Be Met, Tx, Nx, and Mx Should Be Used)

T categories: Clinical and surgical examination
N categories: Clinical and surgical examination
M categories: Clinical and surgical examination, radiography of the thorax
T: Primary tumor
Tis—Preinvasive carcinoma (carcinoma in situ)
T0—No evidence of tumor
T1—Tumor <2 cm maximum diameter
T1a—Without bone invasion
T1b—With bone invasion
T2—Tumor 2–4 cm maximum diameter
T2a—Without bone invasion
T2b—With bone invasion
T3—Tumor >4 cm maximum diameter
T3a—Without bone invasion
T3b—With bone invasion
The symbol m added to the appropriate T category indicates multiple tumors.
N: Regional lymph nodes (RLN)[b]
N0—No evidence of regional lymph node involvement
N1—Movable ipsilateral nodes
N1a—Nodes not considered to contain growth[c]
N1b—Nodes considered to contain growth[c]
N2—Movable contralateral or bilateral nodes
N2a—Nodes not considered to contain growth[c]
N2b—Nodes considered to contain growth[c]
N3—Fixed nodes
M: Distant metastasis
M0—No evidence of metastasis
M1—Distant metastasis (including distant nodes) detected; specify site or sites

Stage Grouping

I	T	N	M
	T1	N0, N1a, or N2a	M0
II	T2	N0, N1a, or N2a	M0
III[d]	T3	N0, N1a, or N2a	M0
	Any T	N1b	
IV	Any T	Any N2b or N3	M0
	Any T	Any N	M1

[a]Modified from document VPH/CMO/80.20, World Health Organization, 1980.
[b]Regional lymph nodes are the cervical, submandibular, and parotid nodes.
[c](–), Histologically negative; (+), histologically positive.
[d]Any bone involvement.



FIG. 18.31 Appearance of a fibromatous epulis surrounding the maxillary incisors in a dog.

BOX 18.4 Types of Oral Tumors

Malignant

- Malignant melanoma
- Squamous cell carcinoma
- Fibrosarcoma
- Osteosarcoma
- Lymphosarcoma
- Mast cell tumor
- Gingival hemangiosarcoma
- Neurofibrosarcoma
- Anaplastic sarcoma
- Chondrosarcoma
- Myxosarcoma
- Invasive nasal tumors
- Transmissible venereal tumor
- Histiocytic neoplasia

Benign

- Epulides
- Viral papillomatosis
- Odontogenic tumors: adamantinoma[a] (ameloblastoma)
- Fibroma
- Peripheral giant cell granuloma
- Chondroma
- Lipoma
- Hemangioma
- Plasma cell tumor

[a]Biological behavior is unpredictable.

BOX 18.5 Characteristics of Oral Malignant Melanomas

- Most common malignant oral tumor in dogs (approximately 20%)
- Rare in cats
- Most common on gingiva
- More common in male dogs
- Mean age of affected animals is 9–11 years (average, 10.3 years)
- Breeds with pigmented oral mucosa, cocker spaniels, and German shepherds may be predisposed
- Metastasis is common
- Prognosis is poor; median survival is 8–9 months

BOX 18.6 Characteristics of Oral Squamous Cell Carcinomas

- Most common tumor in cats (approximately 70%)
- Second most common tumor in dogs (with fibrosarcoma) (approximately 15%)
- Occur on the gingiva, lip, tongue, or tonsil
- Biological behavior varies with location and species; regional lymph node involvement is common with tongue and tonsillar squamous cell carcinomas

BOX 18.7 Characteristics of Oral Fibrosarcomas

- Second most common malignant oral tumors in dogs (with squamous cell carcinoma)
- Occur most commonly on the gingiva and hard palate
- More common in large breeds (>20 kg) and male dogs
- Younger dogs may be affected (mean age <7 years)
- Locally invasive; high metastatic potential in dogs <2 years of age

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BOX 18.8 Characteristics of Epulides

- Most common oral tumor in dogs (approximately 30%)
- Mean age approximately 8.2 years
- More common in large-breed dogs (>20 kg)
- Do not metastasize
- Acanthomatous epulis is most common form

masses, but they are often locally aggressive and sometimes difficult to differentiate histologically from SCC. They are the most common type of epulis and frequently infiltrate bone, causing lysis. Acanthomatous epulides most often occur rostral to the mandibular canine teeth. They are composed primarily of epithelial cells arranged in sheets and cords, intimately associated with the underlying stroma and invading bone. Wide surgical excision is recommended even though they are radiosensitive. They recur locally if not treated adequately.

Ameloblastomas (adamantinoma) are benign tumors that arise from the dental lamina. They usually occur in younger dogs and involve the rostral mandible. Ameloblastomas develop as intraosseous tumors and are locally invasive and nonmetastasizing. *Odontomas* are rare, benign odontogenic tumors that arise from the dental follicle and cause induction of both enamel and dentin in the lesion. *Dentigerous cysts* appear as closed cavities or sacs with one or more teeth embedded in the cyst wall. They arise within islands of odontogenic epithelium and are described as benign, nonneoplastic lesions; however, they may represent an early stage of malignant epithelial tumor development.

Oral papillomas are benign tumors caused by a papillomavirus or papovavirus in young dogs. They occur primarily on the buccal and gingival mucosa, appearing as multiple gray-white pedunculated lesions. Papillomas spontaneously regress within 2 months in most dogs as immunity to the viral agent develops. Surgical resection is necessary only to confirm the diagnosis or in dogs that are dysphagic as a result of many large papillomas. Some animals have been treated with autogenous vaccines, but this is not recommended because malignant skin tumors may develop at the site of inoculation.

DIAGNOSIS

Clinical Presentation

Signalment

Breeds that appear to be predisposed to oral tumors include boxers, German shepherds, golden retrievers, cocker spaniels, poodles, German shorthaired pointers, collies, old English sheepdogs, and Weimaraners. Oral tumors are generally observed in middle-aged or older animals (older than 7–10 years). Exceptions to this include oral papillomatosis (typically occurring in dogs 1 year old or younger) and fibrosarcoma (mean age of occurrence of approximately 5 years).

Melanomas are more common in males (the reported male to female ratio has been as high as 4:1), with an average age of onset of 9 to 11 years. Breeds with pigmented oral mucosa, cocker spaniels, and German shepherds appear to have an increased incidence. SCCs usually occur in cats of either sex older than 10 years of age. Nontonsillar SCCs are most common in small-breed dogs of either sex between 8 and 10 years of age. Fibrosarcomas occur more commonly in large-breed dogs, particularly Doberman pinschers and golden retrievers. Males are affected more often than females (2:1). For fibrosarcomas the age of onset is younger (4–5 years) in large-breed dogs (more than 25 kg) than in smaller dogs (8 years or older). Oral osteosarcomas most often affect the mandible and occur in females more often than males (1.8:1). Fibromatous epulides are common in boxers.

NOTE Remember, not all melanomas are pigmented; amelanotic melanomas may be easily confused with other oral tumors.

History

Oral tumors are often large when recognized by an owner; however, some are found during yearly examinations or routine dentistry. Neoplasia should be suspected during dentistry if teeth are excessively mobile. Affected animals are frequently presented for evaluation of a visible mass, oral bleeding, difficulty eating, or halitosis. Anorexia, weight loss, loose or displaced teeth, salivation, facial deformity, and/or nasal discharge may also be noted. A history of recent tooth extraction may precede rapid growth of a mass at the extraction site. Clinical signs in dogs with tonsillar SCC may be related to oropharyngeal obstruction (i.e., dyspnea, anorexia, cough, or drooling), and a large ventral cervical swelling may be associated with lymph node metastasis.

Physical Examination Findings

Oral tumors arising from the rostral portion of the oral cavity are generally easily visualized; however, examination for tonsillar or caudal oropharyngeal tumors may require sedation or anesthesia. General anesthesia is often necessary to define the extent of disease. The surface of growing neoplasms may appear ulcerated, infected, and necrotic. Regional lymph nodes should be evaluated for evidence of enlargement, nodularity, and adherence to surrounding tissue.

NOTE When you first observe the tumor, measure it, record its location, and photograph it, particularly if treatment is to be delayed.

Diagnostic Imaging

Three-view thoracic radiographs or thoracic CT should be performed to look for pulmonary metastasis and concurrent pulmonary or cardiovascular disease. Ventrodorsal and both lateral radiographic views should be taken because tumors may be missed on a single lateral view due to recumbent atelectasis. Further therapy may not be indicated if metastasis is noted. Skull radiographs, CT, or MRI are performed with the patient under general anesthesia and are used to assess the extent of the lesion and bony involvement. Malignant tumors show a tendency for irregular, destructive, or aggressive bone loss, whereas bone production predominates in benign tumors. Contrast enhancement with adjacent osteolysis is common on CT studies of cats with oral SCC.

Laboratory Findings

Laboratory examination of animals with oral tumors should include a CBC, chemistry profile, and bleeding time. In older dogs and those with evidence of renal or cardiac disease, urinalysis and ECG are appropriate. Other than anemia of chronic blood loss, abnormalities related to the tumor are uncommon.

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DIFFERENTIAL DIAGNOSIS

Granulation tissue secondary to foreign body, trauma, or infection; eosinophilic granuloma complex; and gingival hyperplasia and dental disease are primary differential diagnoses. Fluctuant swellings in the sublingual and pharyngeal area may be salivary mucoceles (see p. 358) or congenital cysts. Other differential diagnoses include nasopharyngeal polyp, osteomyelitis, and feline plasma cell gingivitis-pharyngitis. Cytologic or histologic analysis of masses may be necessary to differentiate neoplastic from some nonneoplastic oral lesions.

MEDICAL MANAGEMENT

Cytologic analysis of the tumor and draining lymph nodes is indicated before surgery. Excisional or incisional biopsy (see p. 633) is usually necessary to determine the prognosis and treatment. Treatment modalities other than surgery that have been used alone or in combination for oral tumors include radiotherapy, hyperthermia, chemotherapy, cryosurgery, immunotherapy, and photodynamic therapy. SCCs are radiosensitive and are successfully treated by radiotherapy. Piroxicam is sometimes helpful in treating SCC. Fibrosarcomas are radioresistant. Melanomas may be sensitive to radiotherapy, but distant metastasis frequently renders it ineffective. Radiation-induced tumors occur in up to 20% of the irradiated sites. A vaccine for canine melanoma is available, but of uncertain efficacy. Refer to an oncology text for additional information about these techniques.

SURGICAL TREATMENT

Treatment protocols must be based on the tumor type, site, extent, and stage; the patient's age and health; and treatment limitations. Early, aggressive therapy offers the best chance of success in treating oral malignancies. Aggressive surgical excision (e.g., mandibulectomy, maxillectomy) may be curative for gingival tumors if resection is completed before metastasis occurs. Because most gingival tumors invade bone, mandibulectomy or maxillectomy is usually necessary. Shaving the tumor down to bone invariably results in recurrence. Caudal tumors and those crossing the midline may be unresectable, or the area may be difficult to reconstruct successfully with flaps. Excision of the middle and caudal maxilla is limited by the size of mucosal flap that can be created. Mandibulectomy is limited by the medial and caudal extent of the tumor (see p. 336). Tumors invading the sublingual musculature and caudal pharynx may not be resectable. Tumor extension into the lip necessitates full-thickness lip resection with partial maxillectomy (see p. 335) or mandibulectomy.

NOTE Owners often think that their pets will be unacceptably disfigured after major oral resections. It may help to show them pictures of animals that have had procedures similar to the one you are recommending for their pet.

Preoperative Management

Perioperative antibiotics are indicated for oral tumors, which often have focal areas of necrosis and infection (see p. 331). Debilitated animals may require IV fluids and enteral or parenteral hyperalimentation before surgery.

Anesthesia

General anesthetic protocols for animals undergoing oral surgery are given on p. 331 (see Table 18.1). Sedation or general anesthesia may be required for fine needle aspiration, depending on the location of the tumor and the animal's disposition. General anesthesia is generally recommended for biopsy because of subsequent bleeding. Surgical excision of tumors requires general anesthesia with inhalant anesthetics. Because most affected animals are old, isoflurane is the inhalant anesthetic of choice. Cuffed endotracheal tubes, preferably guarded to prevent kinking, should be used. Intubation may be accomplished through a pharyngotomy (see p. 1044) or tracheotomy (see p. 842) if necessary to facilitate surgery. Nerve blocks provide analgesia and reduce the amount of other anesthetic drugs needed during surgery (see Chapter 13).

NOTE Sterile sponges should be placed in the caudal oropharynx to prevent aspiration of blood; however, remember to remove these sponges prior to waking the patient.

Surgical Anatomy

The surgical anatomy of the oropharynx is discussed on p. 333.

Positioning

Mandibular lesions are usually resected with the patient in lateral recumbency. Maxillary lesions may be resected with the patient in lateral or ventral recumbency.

SURGICAL TECHNIQUE

AP Identify the soft tissue and/or bone to be resected and remove it according to the techniques for maxillectomy, mandibulectomy, glossectomy, and tonsillectomy described on pp. 335 to 340. Radiographing the excised segment before wound closure may help determine whether adequate bone was removed; however, tumor growth up the mandibular foramen may necessitate wider margins than radiographic evaluation of bone destruction might predict. Intraoperative cytologic evaluation is often more beneficial in determining the adequacy of resection. Submit excised tissues for histologic analysis. If additional bone is excised, mark the caudal border to allow determination of whether additional resection is needed (i.e., if this margin contains tumor). Concurrent or prior excision of regional lymph nodes (mandibular, parotid, and medial retropharyngeal) is important in staging the disease.

POSTOPERATIVE CARE AND ASSESSMENT

Sponges should be removed from the caudal oropharynx, and the oral cavity and nasopharynx suctioned before anesthetic recovery. Analgesics (i.e., hydromorphone, butorphanol, or buprenorphine; see Table 13.2) should be provided postoperatively. Soft food and water may be offered the day after surgery. IV fluids may be discontinued when the animal maintains hydration by drinking. Dogs are seldom reluctant to eat after surgery; however, cats may require 2 to 3 days to adapt. The patient should be reevaluated 1 and 2 weeks after surgery to assess healing. Sutures are usually extruded or sloughed 2 to 4 weeks postoperatively. Facial swelling usually resolves within 3 to 7 days after surgery. Thoracic radiographs may be taken at 3, 6, and 12 months postoperatively to evaluate for metastases, and oral examinations should be performed regularly to look for tumor recurrence. After partial maxillectomy or

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mandibulectomy, excessive dental tartar may accumulate on the teeth of the opposite dental arch. Owner satisfaction after partial mandibulectomy or maxillectomy is usually good.

COMPLICATIONS

Tumor recurrence and dehiscence are the most common complications after major oral reconstruction. Overall, tumor recurrence after resection with tumor-free margins is less than 40%. Recurrence in these cases is due to the presence of a multifocal tumor, development of a new tumor, or inadequate pathologic assessment. Dehiscence occurs in less than one-third of cases with major reconstruction. Suture line tension, excessive use of electrocautery, ischemic necrosis of a mucosal flap, excessive flap movement, infection, and tumor recurrence are major causes of dehiscence. Dehiscence occurs more often if surgery is combined with radiotherapy or chemotherapy because these adjuvant therapies may inhibit wound healing.

PROGNOSIS

The prognosis for oral tumors depends on the type of tumor, its biological behavior, and the stage of disease. The prognosis is good for benign oral tumors, whereas the prognosis for malignant oral tumors is poor. The best chance for cure or control of malignant or benign oral tumors is surgical resection and reconstruction. Elimination of local disease is essential. Overall, the 1-year survival rate is approximately 50% with a median survival time of 8 months for dogs with malignant maxillary tumors. For malignant mandibular tumors, the overall 1-year survival rate is 45% with a median survival time of 11 months. Dogs with tumors rostral to the maxillary canine or the first mandibular premolar teeth have a better prognosis. This may be because of earlier recognition, altered tumor behavior based on location, or prevalence of tumor type.

SCCs respond best to surgery because they are localized and have not usually metastasized. In dogs, more than 50% of SCCs are controlled locally for a year or more, and the mean survival time is approximately 12 to 18 months. Cats with oral SCC have a poor prognosis, with a median survival time of approximately 2 months regardless of the form of therapy. Fibrosarcomas are localized but locally aggressive and difficult to resect completely. Most recur within the first year after resection, and 1-year survival rates vary from 20% to 50%. Melanomas have the poorest prognosis because they metastasize early. Fewer than 20% of affected animals are disease-free 1 year after surgery. Median survival times vary from 8 to 10 months. Tumors arising from the tongue have a poor prognosis. They are controlled locally in only approximately one-fourth of animals 1 year after resection or radiation therapy. Osteosarcoma of the mandible treated with surgical excision and in some cases chemotherapy was associated with a 58% metastatic rate, most often to the lungs (69%) and also to the lymph nodes, spinal column, long bones, and skin.[4] Median survival time was 525 days, with 50% surviving 1 year. Higher tumor grade was associated with a greater metastatic rate and lower percentage surviving 1 year.

SALIVARY MUCOCELES

DEFINITIONS

A **salivary mucocele** *(sialocele, salivary cyst, honey cyst)* is a collection of saliva that has leaked from a damaged salivary gland or duct and is surrounded by granulation tissue. A **cervical mucocele** is a collection of saliva in the deeper structures of the intermandibular space, the angle of the jaw, or the upper cervical region. **A sublingual mucocele (ranula)** is a collection of saliva in the sublingual tissue caudal to the openings of the sublingual and mandibular ducts, and a **pharyngeal mucocele** is a collection of saliva in the tissues adjacent to the pharynx. A **zygomatic mucocele** is a collection of saliva ventral to the globe. **Complex mucoceles**, consisting of two or more types, occur in some animals. **Marsupialization** is the process of incising a mucocele and suturing the edges to the mucosa. The interior of the mucocele suppurates and gradually closes by granulation.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Tearing of a salivary gland or duct results in leakage of saliva into the surrounding tissue. Salivary mucoceles are not cysts. Cysts are cavities lined by epithelium, whereas the granulation tissue lining of a mucocele is produced secondary to inflammation caused by free saliva in the tissues. The cause of salivary mucoceles is rarely identified, although blunt trauma (choke chains), foreign bodies, and sialoliths have been suggested. The sublingual salivary gland is most commonly involved. Saliva takes the path of least resistance, most commonly accumulating in the cranial cervical or intermandibular tissue (Fig. 18.32), sublingual tissue (Fig. 18.33), or pharyngeal tissue (Fig. 18.34). Saliva irritates the tissue and causes inflammation. The swelling may be firm and painful initially, but the animal is usually asymptomatic. Granulation tissue forms in response to the inflammation and prevents saliva from migrating further. The diagnosis of salivary mucocele is based primarily on the history, clinical signs, and cytologic findings. Radiographs may determine which gland is involved, and histopathologic examination is diagnostic.

DIAGNOSIS

Clinical Presentation

Signalment

Dogs are more frequently affected than cats. All breeds are susceptible, but some reports indicate that poodles, German shepherds, dachshunds, and Australian silky terriers are more commonly affected. There is a slight predisposition for males to be affected. An animal of any age may develop a mucocele.

History

Clinical signs depend on the location of the mucocele. Most dogs have cervical or intermandibular mucoceles and are asymptomatic. These animals are usually presented for treatment with a history of a gradually developing, fluctuant, painless mass. Patients with a sublingual mucocele (i.e., ranula) may have abnormal prehension and oral bleeding, which is caused by trauma during chewing. Respiratory distress and dysphagia are common in patients with pharyngeal mucoceles due to the sublingual salivary gland. Swelling in the oropharyngeal area may cause abnormal tongue movements and may interfere with eating or breathing, and swelling in the orbital area with zygomatic mucoceles may cause enophthalmos and divergent strabismus. Dogs with pharyngeal mucoceles can have concurrent cervical mucoceles.[5]

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FIG. 18.32 Cervical mucocele in a dog. (A) Appearance of the mucocele on physical examination. (B) Viscosity of material aspirated from a cervical mucocele.



FIG. 18.33 Intraoperative appearance of a sublingual mucocele (ranula) located lateral to the tongue in the sublingual tissue. Note the ulcer from mucosal entrapment between teeth.

NOTE Ask the owners where they first noticed the swelling, because this may help you determine the affected side.

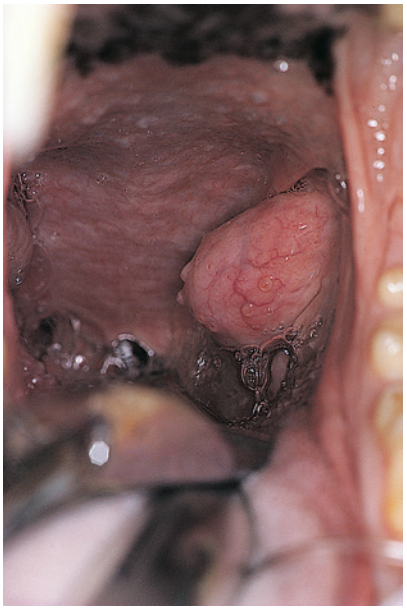


FIG. 18.34 Pharyngeal mucocele in a dog.

Physical Examination Findings

The parotid and mandibular glands are easily palpated. The sublingual gland is occasionally palpable in a cooperative or sedated patient. Palpation of the glands is expected to be normal and without discomfort. Most mucoceles are soft and fluctuant, whereas tumors and abscesses are generally firm. Mucoceles are painless except during the acute phase of swelling. It is sometimes difficult to identify the affected side when mucoceles are located on the ventral midline or intermandibular space. Examining these animals in dorsal recumbency often allows the mucocele

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to gravitate to the affected side. Palpation of some cervical mucoceles causes the sublingual tissues to bulge on the affected side. Concurrent sublingual and cervical mucoceles originate from the side where the sublingual mucocele is found. Blood-tinged saliva may occur in patients with sublingual mucoceles because teeth often traumatize the mucocele. Periorbital facial swelling, enophthalmos, and periocular pain are signs of a zygomatic mucocele. Optic neuropathy may occur secondary to pressure from zygomatic mucoceles.

NOTE Animals with pharyngeal mucoceles are often presented in acute respiratory distress. Institute appropriate therapy rapidly or they may die!

Diagnostic Imaging

Survey radiographs rarely help, except in cases involving sialoliths, some foreign bodies, or neoplasia. Thoracic radiographs are indicated to evaluate for metastasis if neoplasia is suspected. Sialography (i.e., injecting iodinated, water-soluble contrast agent into a salivary duct) to confirm the diagnosis or determine the site of origin is difficult and usually unnecessary. In some cases, particularly pharyngeal mucoceles, cross-sectional imaging can be used to better evaluate the location and extensiveness of the lesion. Advanced imaging is important for diagnosing zygomatic salivary disease.[6] Fluid can be differentiated from tissue, and aspirates can be taken for cytologic evaluation to differentiate between infection and mucocele.

Laboratory Findings

Laboratory abnormalities are rare. Salivary gland function and duct patency can be evaluated by placing a drop of topical ophthalmic atropine solution on the tongue to stimulate saliva flow; however, it may be difficult to distinguish flow from individual ducts. Paracentesis should be performed under aseptic conditions to prevent infection of the mucocele. Aspiration of a clear, yellowish, or blood-tinged, ropy, mucoid fluid with a low cell count is consistent with saliva (see Fig. 28.32B). Staining a smear with a mucus-specific stain, such as periodic acid-Schiff, confirms the presence of saliva. An elevated white blood cell count may indicate concurrent sialoadenitis.

DIFFERENTIAL DIAGNOSIS

Sialoadenitis, sialadenosis, salivary neoplasia, sialolith (calcium phosphate or carbonate), cervical abscess, foreign body, hematoma, cystic or neoplastic lymph nodes, tonsil cyst, thyroglossal cyst, cystic Rathke's pouch, or branchial cysts may cause swellings in the same region as mucoceles. Occasionally mucoceles may be difficult to distinguish from cysts or tumors. Histopathologic examination is necessary to diagnose salivary gland tumors and to differentiate a congenital cyst from a mucocele. Congenital cysts have an epithelial lining, whereas mucoceles are lined by granulation tissue.

Salivary neoplasia presents as a mass in older animals and may be associated with halitosis, weight loss, dysphagia, exophthalmos, Horner's syndrome, sneezing, and dysphonia. The mandibular gland is most commonly affected in cats and the parotid in dogs, and simple adenocarcinoma is the most common histologic type.

Animals presenting with chronic oropharyngeal abscesses typically have draining tracts, cervical swelling, dysphagia, and oral pain. A history of fighting, penetrating trauma, or chewing on sticks is common with abscesses.

MEDICAL MANAGEMENT

Emergency aspiration of the mucocele may be necessary for animals in respiratory distress (especially pharyngeal mucoceles). Repeated drainage or injection of cauterizing or antiinflammatory agents does not eliminate mucoceles; however, it complicates subsequent surgery by leading to abscessation or fibrosis. Use of a sclerosing agent injected into the mucocele has been suggested, but this procedure is rarely performed.[7] Mucoceles rarely resolve without surgery.

SURGICAL TREATMENT

Complete excision of the involved gland-duct complex and drainage of the mucocele are curative. The side of origin of the mucocele may be determined by oral examination, palpation, sialography, or exploration of the mucocele.

NOTE If you are having trouble identifying the affected side in an animal with a cervical mucocele, place the animal on its back. The contents of the mucocele often gravitate to the side of the affected gland.

Preoperative Management

Animals with pharyngeal mucoceles may be presented for treatment in acute respiratory distress and rapid intubation may be necessary. Intubation may not be possible through the mouth, and a temporary tracheostomy may be required. These animals are generally stable once they have been intubated, and surgical excision of the salivary glands and drainage of the mucocele can be delayed while further diagnostics are performed if necessary. IV antibiotics may be given at induction of anesthesia but are not essential.

Anesthesia

Most animals undergoing salivary gland excision for mucoceles are healthy, and a variety of anesthetic protocols can be used (see Table 18.1). General anesthetic recommendations for animals undergoing oral surgery (i.e., for ranulas or pharyngeal mucoceles) are provided on p. 331.

Open-mouth breathing, abducted forelimbs, labored breathing, and restlessness indicate moderate to severe respiratory distress that may require emergency therapy. Minimal restraint should be used with severely dyspneic patients, and they should be allowed to maintain the position in which they feel most comfortable. Premedication with any drug that causes hypoventilation is contraindicated (see Box 28.2 on p. 834). Additionally, every attempt should be made to minimize stress to the patient prior to induction. Oxygen support, even while placing an IV catheter, may be necessary. Pulse oximetry with a sensor that can be secured to the tail can be especially helpful with perioperative monitoring of patients in respiratory distress. Prior to induction, the anesthesia provider should be prepared with airway devices, anesthesia machine, and monitors, as well as induction and emergency drugs. Preoxygenating the patient for 3 to 5 minutes prior to induction with flow-by or a loose face mask should be followed with a rapid induction and intubation. Propofol is recommended

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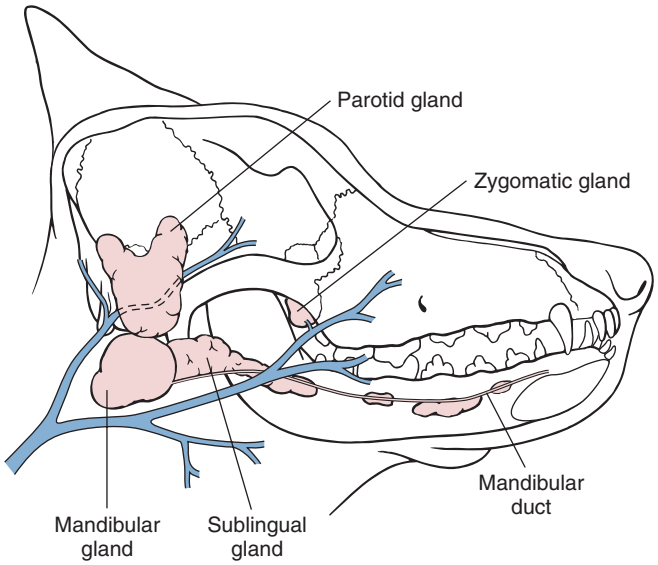


FIG. 18.35 Surgical anatomy of the salivary glands. The parotid gland lies ventral to the ear canal. The mandibular gland is ventral to the parotid gland, lying between the maxillary and linguofacial veins. The sublingual gland follows the rostral course of the mandibular duct toward the oral cavity. The zygomatic gland is protected by the zygomatic arch.

for induction because it has a rapid onset of action and is short-acting. Oral intubation may be difficult in patients with large pharyngeal mucoceles (discussed previously), and placement of the endotracheal tube by means of tracheostomy often is necessary to allow adequate visualization of the lesion. Once intubation has been accomplished, a more thorough exam may be done.

Surgical Anatomy

Dogs and cats have four major pairs of salivary glands of surgical significance (Fig. 18.35). They are the parotid, mandibular, sublingual, and zygomatic glands. The parotid gland is a triangular-shaped serous gland located ventral to the horizontal ear canal. Numerous arteries, veins, and nerves are closely associated with the medial aspect of the gland. The parotid duct papilla is located on the mucosal surface of the cheek at the level of the upper carnassial tooth (fourth premolar). The mandibular gland is large and ovoid and lies within a fibrous capsule, caudal and ventral to the parotid gland. It is located between the linguofacial and maxillary veins as they merge to join the external jugular vein. The mandibular duct runs with the sublingual gland toward the floor of the mouth and opens on a small papilla lateral to the rostral border of the frenulum (Box 18.9). The sublingual gland is divided into monostomatic and polystomatic portions. The monostomatic portion originates on the rostroventral border of the mandibular gland. The ducts from this portion of the sublingual gland course with the mandibular duct but often open on separate papillae. The polystomatic portion is divided into several loosely connected lobules that surround the mandibular duct and lie immediately beneath the oral mucosa, secreting directly into the oral cavity. The zygomatic gland, an irregularly ovoid gland, is located on the floor of the orbit ventrocaudal to the eye and medial to the zygomatic arch. The zygomatic gland has several ducts that run ventrally and open on a fold of mucosa lateral to the last upper molar tooth. The major zygomatic duct can usually be identified at this location, approximately 1 cm caudal to the parotid papilla.

BOX 18.9 Oral Openings of the Salivary Gland Ducts

Parotid Duct
- Labial mucosa at level of upper carnassial tooth

Mandibular Duct
- Papilla lateral to the rostral border of the frenulum

Sublingual Duct
- Opens with the mandibular duct near the lingual frenulum

Zygomatic Gland
- Lateral to the last upper molar tooth

Positioning

Salivary gland excision is performed with the animal in lateral recumbency. Ventral recumbency and maximum opening of the mouth facilitate marsupialization of pharyngeal mucoceles and ranulas.

SURGICAL TECHNIQUES

Mandibular and Sublingual Salivary Gland Excision

The mandibular and sublingual salivary glands are excised together because the sublingual gland is intimately associated with the mandibular salivary gland duct; removal of one would traumatize the other. Removal of glands on the involved side is all that is necessary for mucocele resolution; however, both pairs of mandibular and sublingual glands may be resected without risk of xerostomia. If it is not clear from which side the mucocele originated, make a stab incision in the mucocele and digitally palpate the lumen. The unaffected side is rounded and smooth. The affected side has a tract or tunnel toward the site of leakage.

Position the patient in lateral recumbency. Place a pad under the neck to rotate the ventral aspect dorsally and fix the neck in an extended position. Locate the mandibular salivary gland between the linguofacial and maxillary veins as they join the external jugular vein (Fig. 18.36A). Incise the skin, subcutaneous tissue, and platysma muscle from the angle of the mandible caudally to the external jugular vein to expose the fibrous capsule of the mandibular gland (see Fig. 18.36B). Avoiding the branch of the second cervical nerve that crosses the capsule, incise the capsule and dissect it away from the mandibular and monostomatic sublingual salivary glands. Ligate the artery (branch of the great auricular artery) and vein as they are encountered on the dorsomedial aspect of the gland. Continue dissecting cranially, following the mandibular duct, sublingual duct, and polystomatic sublingual glands toward the mouth (see Fig. 18.36B). Incise the fascia between the masseter and digastricus muscles. Expose the entire mandibular and sublingual salivary gland complex by retracting the digastricus muscle and applying caudal traction on the mandibular gland. If necessary, perform digastricus muscle myotomy or tunnel the caudal sublingual gland duct complex under the digastricus muscle to improve visualization. Dissect (digital and sharp) rostrally until the lingual branch of the trigeminal nerve is identified and only ducts remain in the complex. Avoid traumatizing the lingual or hypoglossal nerves. Try to identify the gland-duct defect causing the mucocele, because failure to identify

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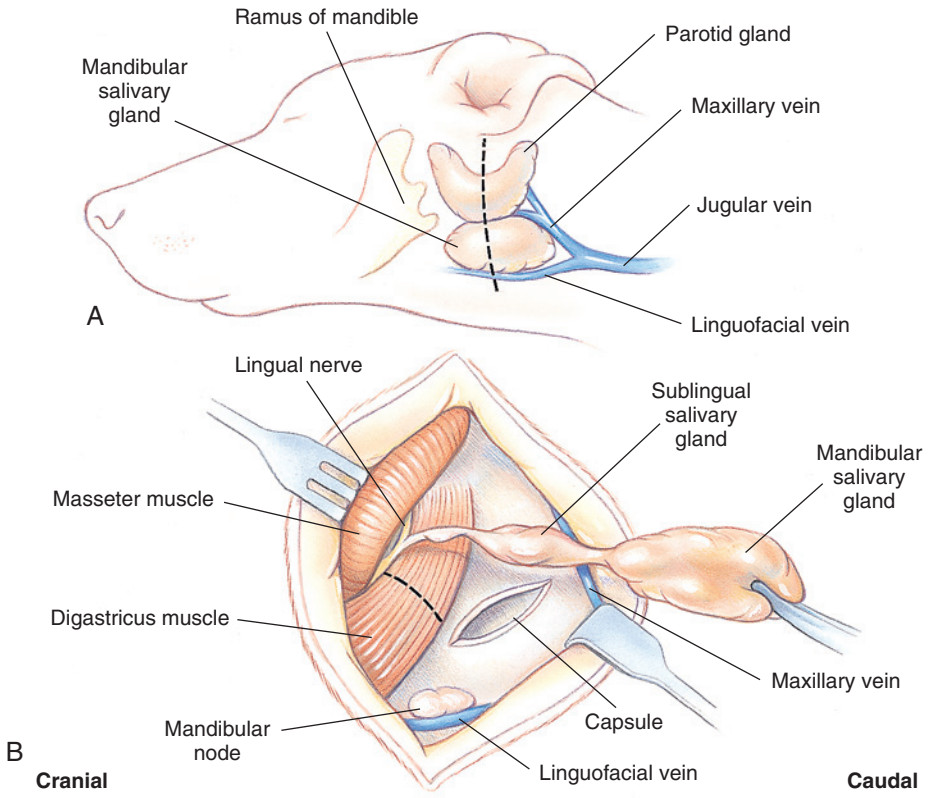


FIG. 18.36 Excision of the mandibular and sublingual salivary glands. (A) Make an incision ventral to the external ear canal and over the mandibular salivary gland at the junction of the linguofacial and maxillary veins. (B) Dissect the mandibular salivary gland after incising the capsule. Apply caudal traction on the mandibular gland and dissect the duct and sublingual gland until the lingual nerve has been identified. Transect the digastricus muscle *(dashed line)* to aid dissection if necessary.



FIG. 18.37 Mandibular and sublingual glandular tissue removed from the dog in Fig. 18.32.

this defect may indicate that the mucocele originated from the contralateral gland-duct complex (Fig. 18.37). Ligate and transect the mandibular sublingual gland-duct complex just caudal to the lingual nerve.

Traction on the gland-duct complex may cause the ducts to tear. If this occurs near the point of proposed transection or on the oral aspect of the gland-duct defect, no further dissection is needed. However, if the tear occurs before the gland-duct defect or when the defect has not yet been identified and glandular tissue is identified oral to the tear, further resection of glandular tissue is recommended to prevent recurrence.

Lavage the surgical site before closure. Appose the digastricus muscle if it has been incised with horizontal mattress or cruciate sutures. Close the dead space with a few sutures in the capsule and deep tissue. Routinely appose superficial muscles, subcutaneous tissue, and skin. Following excision, submit the glands and ducts to rule out neoplasia and submit a portion of the mucocele wall to rule out congenital cysts.

Drain cervical mucoceles by making a stab incision at the most dependent point; place a closed suction or Penrose drain if desired. Protect the drain with an absorbent bandage. Change the bandage and cleanse discharge from the neck as needed to prevent excoriation of the skin. Maintain the drain for 1 to 5 days, removing it when there is minimal discharge. Allow the stab incision to heal by second intention. Redundant skin resumes its normal appearance within several weeks. *Drain sublingual mucoceles (ranulas) by excising an elliptical, full-thickness section of the mucocele wall. Suture the granulation tissue lining to the sublingual mucosa (marsupialization) to encourage drainage for several days (Fig. 18.38). Drain pharyngeal mucoceles by aspiration or marsupialization. Excise redundant pharyngeal tissue to prevent airway obstruction after the mucocele has been evacuated.*

Marsupialized ranulas contract and heal quickly by second intention. After bilateral mandibular and sublingual salivary

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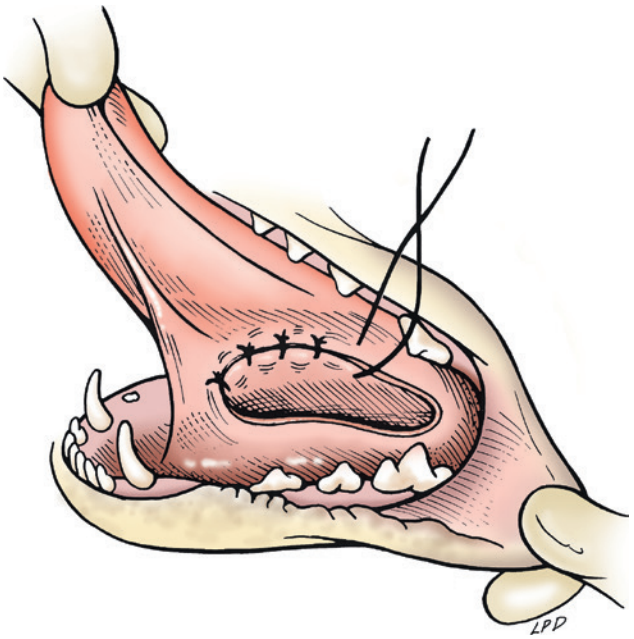


FIG. 18.38 Marsupialization of a ranula. After excising an elliptical piece of mucosa and granulation tissue from the mucocele wall, suture the mucosa to the lining of the mucocele.

gland excision, dogs still have sufficient saliva to adequately moisten their food.

Zygomatic Gland Excision

AP Zygomatic gland excision is required for zygomatic mucoceles, unresponsive infections, inflammatory conditions, and neoplasia.

Position the patient in lateral or ventral recumbency. Protect the animal's eye from irritants with ophthalmic ointment. Incise the skin and subcutaneous tissues over the dorsal rim of the zygomatic arch. Incise the palpebral fascia, retractor anguli oculi muscle, and orbital ligament, and elevate them dorsally with the skin and globe. Further expose the gland by partly removing the zygomatic arch with rongeurs or by osteotomy. Retract the globe dorsally to expose the periorbital fat and underlying zygomatic gland. Remove the gland by blunt dissection (the gland is friable). Avoid the ventrally located anastomotic branch between the deep facial and external ophthalmic veins. Drain the mucocele if present. If possible, replace the zygomatic arch by securing the bone with suture placed through predrilled holes. Lavage the area and appose the palpebral fascia to the zygomatic periosteum with sutures. Close subcutaneous tissues and skin.

Parotid Gland Excision

AP Parotid gland excision is occasionally performed for neoplasia, fistula, chronic infection, or mucocele. The triangular-shaped gland is located at the base of the auricular cartilage.

Position the patient in lateral recumbency. Incise the skin from 1 to 2 cm ventral to the external acoustic meatus to a point midway between the ramus of the mandible and the bifurcation of the jugular vein. Incise the platysma muscle to expose the parotidoauricularis muscle, vertical ear canal, and parotid salivary gland. Sever and retract the parotidoauricularis muscle from its vertical ear canal attachment. Ligate and divide the caudal auricular vein. Begin dissection of the parotid gland at its dorsocaudal angle. Separate the parotid from the mandibular gland ventrally. Continue dissection between the gland and the vertical ear canal. Avoid traumatizing the facial nerve at the base of the horizontal ear canal. Ligate and divide the superficial temporal vein (a branch of the maxillary vein) coursing through the gland. Cauterize or ligate small vessels on the gland's medial surface. Ligate and transect the parotid duct as it leaves the gland. Lavage the area. Reappose the parotidoauricularis muscle. Complete the closure by apposing subcutaneous tissues and skin. Ligation of the parotid duct may be an alternative treatment for parotid mucoceles or fistulae. Ligating the duct proximal to the disruption, near the body of the gland, causes glandular atrophy.

POSTOPERATIVE CARE AND ASSESSMENT

Histologic assessment of the excised gland rules out neoplasia as a cause of the mucocele. Change bandages daily if a Penrose drain has been placed. Depending on the amount of drainage, remove the drain 24 to 72 hours after surgery or when drainage is minimal. Allow the drain site to heal by second intention. Soft food should be fed for 3 to 5 days after ranula marsupialization or drainage of pharyngeal mucoceles.

COMPLICATIONS

Postoperative complications after salivary gland resection are uncommon but may include seroma formation, infection, and mucocele recurrence. Seromas can form in the dead space created by removal of the glands; they typically resorb and do not need aspiration or drainage. Infection is rare if aseptic technique is used. Mucoceles recur if the side of mucocele origin was misdiagnosed or if an inadequate portion of the gland was excised. Regional lymph nodes are sometimes mistaken for salivary glands. Dissection may be difficult if the mucocele was previously infected or injected. Attention to anatomic detail during surgery should minimize recurrence and complications associated with salivary gland excision.

PROGNOSIS

In rare cases a mucocele resolves without surgery. The prognosis is excellent if the disease is accurately diagnosed and excision is complete.

OROPHARYNGEAL PENETRATING TRAUMA

DEFINITION

Oropharyngeal penetrating trauma (OPT) is an injury of the oropharynx, palate, and adjacent cervical structures caused by foreign material.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Sticks are the most common material associated with OPT in dogs, likely because of play behavior. Direct trauma to the pharyngeal structures with bacterial inoculation of associated cervical structures results in local signs of inflammation and infection, with possible subsequent abscessation. Airway compromise resulting from local inflammation or systemic signs of septic shock can

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occur, depending on the duration of time between injury and presentation and the severity of the associated infection.

DIAGNOSIS

Clinical Presentation

Signalment

Dogs are more likely to have OPT due to indiscriminate eating and play behaviors, compared with cats. Any breed of dog can be affected; however, commonly reported breeds include the border collie, mixed breed, English springer spaniel, and Labrador retriever. Cats, having a tendency to play and hunt, more commonly have string or needle foreign bodies than bones. Foreign bodies may occur in an animal of any age but are most common during the first 3 years of life.

History

Known trauma results in acute vocalization, gagging, and pawing at the mouth.

Physical Exam Findings

The most commonly observed clinical signs are ptyalism, depression, and pain with cervical flexion or opening the mouth; bloody saliva and collapse are less commonly seen. Mediastinal involvement may result in fever, dyspnea, and tachycardia.

Diagnostic Imaging

Emphysema and soft tissue swelling may be present on radiographs (Figs. 18.39 and 18.40). Wood material may not be readily identified radiographically, but ultrasound may show hyperechoic linear material with shadowing.

Laboratory Findings

Penetrating trauma and associated infection may result in neutrophilic leukocytosis. Hypoglycemia may be seen in young patients that are unable to eat or that have systemic inflammatory response syndrome (SIRS).

DIFFERENTIAL DIAGNOSIS

Mass lesions, strictures, esophagitis, cricopharyngeal dysfunction, and other possibilities of ptyalism and dysphagia should be ruled out. Thoracic radiographs should be performed to rule out pneumomediastinum (see Fig. 18.40), pneumothorax, and/or pleural effusion if concurrent esophageal trauma is suspected.

SURGICAL TREATMENT

Surgical exploration of the oropharynx and neck are done via an oral and ventral cervical approach, respectively. One study used rigid endoscopy for minimally invasive removal of foreign material via the original entry tract.[8] Fluid irrigation was used to dilate the original wound and tract during foreign body extraction using flexible instruments via the endoscope instrument channel. Care should be taken to avoid pressurization of the irrigation solution; cervical swelling occurred in one of nine dogs, and the outcome was successful with no recurrences in any dog.

Preoperative Management

Therapy to correct significant dehydration and electrolyte and acid-base imbalances should be initiated before surgery. Prophylactic antibiotics should be given (see p. 331).



FIG. 18.39 Radiographs of a dog with known oropharyngeal penetrating trauma. (A) Lateral image showing emphysema surrounding the pharynx and esophagus. (B) Ventrodorsal image showing emphysema on the left side of the pharynx and esophagus *(arrows)*.

Anesthesia

Deep general anesthesia and/or muscle relaxants should be considered, as oropharyngeal stimulation results in vigorous responses and motion. Opioids (e.g., fentanyl, hydromorphone) obtund oropharyngeal reflexes and may also be administered.

Surgical Anatomy

The surgical anatomy of the pharynx, tongue, and esophagus is described on pp. 333 and 369.

SURGICAL TECHNIQUE

Oral wounds should be examined and foreign material removed if present. Subsequent debridement, lavage, and closure are done for pharyngeal wounds; however, not all palatal wounds require immediate closure (Fig. 18.41). Lingual abscessation has been reported following presumptive foreign body penetration of the

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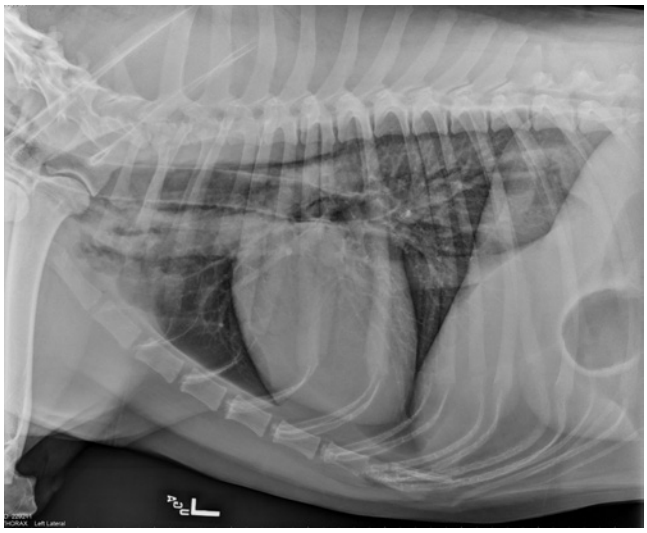


FIG. 18.40 Lateral thoracic radiograph of the dog in Fig. 18.39 showing cranial mediastinal emphysema.



FIG. 18.41 Left-sided palatal trauma of the dog in Figs. 18.39 and 18.40.

tongue, and drainage, debridement, and lavage should be done with consideration of drain placement in large, sublingual abscesses. *Explore subcutaneous cervical emphysema via a ventral midline approach, as described for esophageal foreign bodies (p. 378); take care to closely evaluate the retropharyngeal region. Place a closed suction drain if dead space cannot be closed because of anatomic considerations of neurovascular structures. Consider concurrent gastrostomy or esophagostomy tube placement for postoperative nutritional support.*

POSTOPERATIVE CARE AND ASSESSMENT

All patients should be observed carefully for 2 to 3 days for signs of pain and dehiscence. Analgesia with opioids and NSAIDs may be beneficial to reduce pain and encourage oral intake of food and water if a gastrostomy tube has not been placed. In such cases, small balls of canned food that require minimal prehension (representing preformed food boluses) are recommended. Repeat thoracic radiographs should be performed in animals with preoperative mediastinal widening or emphysema, pneumomediastinum, pneumothorax, and/or pleural effusion and in cases that fail to improve following surgery.

BOX 18.10 Possible Surgical Diseases of the Esophagus

- Foreign bodies
- Tumors
- Perforation
- Hiatal hernia
- Fistulae[a]
- Gastroesophageal intussusception[a]
- Diverticula[a]
- Cricopharyngeal achalasia[a]
- Strictures

[a]Rare.

COMPLICATIONS

Draining tracts, abscessation, and extension into the mediastinum can result from persistent infection or unidentified foreign material. Persistent clinical signs may signify remaining foreign material and the need for repeated evaluation and possible surgery.

PROGNOSIS

Early diagnosis and removal of a foreign material is essential, resulting in a good prognosis. Involvement of the mediastinum may cause significant inflammation and can be fatal.

Surgery of the Esophagus

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Esophagotomy is an incision into the esophageal lumen; **esophagectomy** is partial resection of the esophagus. **Esophagostomy** is the creation of an opening in the esophagus for placement of a feeding tube. **Regurgitation** is the passive expulsion of undigested food or fluid from the pharynx or esophagus. **Vomiting** is a centrally mediated reflex that causes expulsion of food or fluid from the stomach or intestines or both.

PREOPERATIVE CONCERNS

The esophagus carries food, water, and saliva from the pharynx to the stomach. Although less common than intestinal obstruction, esophageal obstruction associated with foreign bodies, strictures, or masses may occur in dogs and cats. Esophageal surgery may be indicated if function is interrupted by obstruction or perforation (Box 18.10).

Diagnosis of esophageal disorders is based on history, clinical signs, imaging, and/or endoscopy. The predominant clinical signs of esophageal pathology are typically regurgitation or dysphagia (Box 18.11). The history can be used to try to distinguish vomiting from regurgitation, but this is not always easy to do. Regurgitation is classically a passive process, whereas vomiting is usually preceded by salivation, vigorous retching, and abdominal contractions. Regurgitated material generally consists of undigested food

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BOX 18.11 Clinical Signs of Esophageal Disease

- Regurgitation
- Coughing
- Dysphagia
- Dyspnea
- Ptyalism
- Fever
- Altered appetite
- Weight loss

and saliva, whereas vomited material may contain bile or digested blood. However, some animals vomit material that looks just like regurgitated material, and not all vomiting animals have the previously listed signs. If a patient has the classic signs of vomiting, then it is usually vomiting. However, if it does not have the classic signs of vomiting, it can be hard to know whether it is vomiting or regurgitating.

Some patients with esophageal disease present with respiratory signs (especially coughing, but also pulmonary crackles, mucopurulent nasal discharge, and/or fever suggestive of aspiration pneumonia) without any history of regurgitation or spitting up. Anytime a dog presents with pneumonia, aspiration must be a differential even if there is no history of throwing up. A patient with esophageal disease may have a normal, ravenous, or depressed appetite, and some patients lose weight.

Distention of the cervical esophagus may occur in patients with severe esophageal weakness. Some patients with severe weakness of the cervical esophagus will have a bellows-like effect; expirations will be associated with a ballooning in the neck when the cervical esophagus fills with air that is expelled from the thoracic esophagus. The same signs can sometimes be accomplished by compressing the thorax while the nostrils are occluded (this was an old way of diagnosing megaesophagus). Abnormalities of prehension and swallowing may sometimes be noted by observing the animal while it is eating. Masses and foreign bodies may sometimes be palpated in the cervical esophagus. Rarely, esophageal perforations may cause septic mediastinitis, producing fever, mediastinal or pleural effusion, respiratory distress, and eventual death.

Definitively diagnosing esophageal disease and then defining the type may require a variety of techniques. Thoracic and cervical radiographs, including the esophagus from the caudal portion of the oral cavity to the stomach, should be assessed for foreign bodies, esophageal dilatation with gas or liquid, periesophageal fluid or gas opacities, and aspiration pneumonia. The esophagus is not radiographically visible in most normal dogs and cats; however, small amounts of swallowed air may sometimes be seen in the cranial cervical and cranial thoracic esophagus of normal animals. Mediastinitis, pneumomediastinum, and/or pleural effusion suggest esophageal perforation. Barium contrast radiographs of the esophagus are often helpful if survey radiographs are nondiagnostic; a reasonable number of dogs with esophageal weakness do not have obvious abnormalities on plain radiographic studies. Contrast fluoroscopic examination of the esophagus allows evaluation of swallowing, motility, and gastroesophageal sphincter function. Fluoroscopy is needed to best assess motility. Barium sulfate paste may cause complications if aspirated. Liquid barium sulfate is the preferred preparation when performing esophagrams and is relatively safe if aspirated (unless it is aspirated in large quantities or if there is preexisting pneumonia). Food mixed with barium may reveal some partial obstructions that would be missed with liquid or paste barium sulfate.

BOX 18.12 Preoperative Management of Patients With Esophageal Disorders

- Withhold food: mature animals, 12–18 hours; pediatric animals, 4–8 hours
- Correct fluid, electrolyte, and acid-base imbalances
- Give prophylactic antibiotics (i.e., ampicillin, cephalosporins), if appropriate
- Support nutrition
- Treat esophagitis and aspiration pneumonia

BOX 18.13 Treatment of Esophagitis

Omeprazole (Prilosec)
1–2 mg/kg PO q12–24h

Esomeprazole (Nexium)
1 mg/kg IV q12–24h[a]

Pantoprazole (Protonix)
1 mg/kg IV q12–24h[a]

Metoclopramide (Reglan)
0.2–0.4 mg/kg PO, SC, or IV q8h

Cisapride (Pharmacy Compounded)
0.1–0.5 mg/kg PO q8–12h

Sucralfate[b] (Carafate Suspension, Not Tablets)
0.5–1 g PO q6–8h

[a]Dose is anecdotal.
[b]Carafate impairs absorption and/or reduces bioavailability of some drugs; give at different intervals.
IV, Intravenous; *PO,* orally; *SC,* subcutaneous.

NOTE Use aqueous iodinated contrast if esophageal perforation is suspected. Do not use barium sulfate in such cases. Nonionic iodinated contrast agents (such as iohexol or iopamidol) are the safest. Hypertonic iodinated contrast can cause fulminant pulmonary edema if aspirated.

Lateral body position typically used for restraint during esophagography prolongs transit time in the cervical esophagus and decreases the percentage of boluses producing primary esophageal peristaltic waves. Secondary waves are not affected by body position. Functional abnormalities of the esophagus are best evaluated with dogs in sternal recumbency during fluoroscopic examination of barium swallows.

Esophagoscopy is indicated if plain or contrast radiographs reveal what may be a mass or foreign body or if esophageal obstruction, tumor, hiatal hernia, or inflammation is suspected. During esophagoscopy, mucosal lesions may be identified and biopsied, and foreign bodies can be removed or advanced into the stomach. Esophagotomy may be performed as a diagnostic and therapeutic procedure if a definitive diagnosis cannot be made by other means (see p. 370). Esophagectomy and esophageal resection/anastomosis should be avoided, if possible.

Treatment for aspiration pneumonia, esophagitis, and nutritional debilitation should be initiated before surgery (Box 18.12). For mild esophagitis, antacids (e.g., proton pump inhibitors; Box 18.13) with or without gastric prokinetics (e.g., metoclopramide; see Box 18.13) are administered, and food and water

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are withheld for 24 to 48 hours to reduce esophageal irritation. Water should be offered first. If there is no regurgitation, a low-fat, high-protein gruel that will enhance lower esophageal high-pressure zone tone, speed gastric emptying, and reduce reflux should be fed for 3 to 4 days. Soft food should be fed for an additional 5 to 7 days, and then the animal should be gradually returned to its normal diet. For moderate to severe esophagitis, a proton pump inhibitor (see Box 18.13) and more potent gastric prokinetic therapy (e.g., cisapride; see Box 18.13) are often necessary. Oral intake of food and water may need to be withheld for days in the case of very severe esophagitis. Hydration should be maintained with IV fluids; nutritional support may require a gastrostomy tube (see p. 97). Oral feeding with a low-fat gruel should be initiated as described for mild esophagitis and continued for 10 to 14 days. Oral administration of sucralfate suspension may help alleviate discomfort in patients with reflux esophagitis (see Box 18.13). If oral pain is so severe that the patient refuses even to swallow saliva, oral administration of viscous lidocaine (over the counter) may help. Antibiotics effective against oral contaminants (e.g., ampicillin, amoxicillin, clindamycin; Box 18.14) are of dubious effectiveness. Concurrent glucocorticoid therapy (e.g., prednisolone, 1.1 mg/kg given orally once daily) might reduce the risk of obstruction due to cicatrix secondary to severe esophagitis; however, the benefit of this therapy is dubious.

Treatment of aspiration pneumonia should be started before esophageal surgery, unless there is an esophageal foreign object, in which case the foreign object should be removed as soon as possible. If aspiration is observed while the animal is anesthetized, the airway should be suctioned to remove irritants. Fluid therapy is indicated for animals that are severely dyspneic or in shock. Nasal oxygen supplementation should be provided to dyspneic animals, and positive pressure ventilation may be needed in unresponsive patients. Bronchodilators (i.e., theophylline, aminophylline, terbutaline sulfate) may reduce bronchospasms and ventilatory muscle fatigue in these patients (see Box 18.14). Glucocorticoids such as dexamethasone (0.02 mg/kg IV) are rarely used and should only be considered if the animal acutely aspirates and is severely dyspneic. Expectorants (e.g., guaifenesin) are occasionally used in animals with productive coughs. Systemic antibiotic therapy is indicated in animals with pulmonary infection or sepsis. Broad-spectrum antibiotics (or combinations of antibiotics) effective against gram-negative and anaerobic bacteria should be used (e.g., clindamycin or ampicillin plus either enrofloxacin or amikacin; see Box 18.14). Culture and sensitivity should be done, if possible. Aerosol therapy and coupage facilitate both delivery of antibiotics to the respiratory tree and elimination of excessive respiratory secretions.

BOX 18.14 Treatment of Aspiration Pneumonia

Bronchodilators

Aminophylline

Dogs: 10 mg/kg PO, IM, IV (if given IV, give slowly over 5 min) q6–8h
Cats: 5 mg/kg PO q12h

Theophylline (Extended Release)

Dogs: 10 mg/kg PO q12h, dose may vary with product
Cats: 20–25 mg/kg PO q24–48h, dose may vary with product

Terbutaline (Brethine, Bricanyl)

Dogs: 1.25–5 mg/dog PO q8–12h
Cats: 100–125 mg/cat PO q12h

Antimicrobials

Ampicillin

22 mg/kg IV, IM, SC q6–12h

Clindamycin (Antirobe, Cleocin)

Dogs: 11–33 mg/kg PO q8–12h, *or* 11 mg/kg IV q8–12h; when giving IV dilute and administer slowly
Cats: 11–33 mg/kg PO or SC q12–24h

Enrofloxacin (Baytril)

Dogs: 7–20 mg/kg PO, IV q24h (diluted and given slowly over 30 min)
Cats: 5 mg/kg PO or IM q24h

Amikacin (Amiglyde-V)

Dogs: 15–2 mg/kg IV or SC q24h
Cats: 10–14 mg/kg IV or SC q24h

IM, Intramuscular; *IV,* intravenous; *PO,* orally; *SC,* subcutaneous.

ANESTHETIC CONSIDERATIONS

Fluid, electrolyte, and acid-base imbalances should be corrected before induction of anesthesia. If feasible, mature animals should be fasted for 12 to 18 hours before esophageal surgery; however, young puppies and kittens may be fasted for shorter periods (4–8 hours) to prevent hypoglycemia. Refer to the anesthesia section in Chapter 26 (see p. 722) for discussion of special concerns with pediatric patients. Selected anesthetic protocols for patients in a stable condition undergoing cervical esophageal surgery are listed in Table 18.3.

Procedures on the thoracic esophagus require modification of general anesthetic protocols to accommodate compromised function of the respiratory and cardiovascular systems. General recommendations for anesthesia in patients undergoing thoracic surgery are provided in the anesthesia section of Chapter 29 on p. 884, Table 29.1, and Table 30.3. Aspiration at induction is of primary concern (see Table 18.3). Although this is an uncommon complication in dogs and cats, esophageal disease puts the patient at increased risk. Many of these patients have previously aspirated. Inducing general anesthesia with the subsequent obtunding of airway reflexes enhances the risk of aspiration. Have suction turned on and immediately available. Have an assistant elevate the patient's head to aid in keeping esophageal secretions out of the oropharynx until the endotracheal tube has been secured, the cuff inflated, and the anesthesia circuit attached. The patient should be preoxygenated for 3 to 5 minutes prior to induction, followed by rapid induction and intubation. Propofol is recommended for induction because of its rapid onset of action and short duration. Once intubation is complete, the head can be lowered. A mechanical ventilator is recommended. If the patient has aspiration pneumonia, positive pressure ventilation should be performed with lower tidal volumes, normal peak ventilation pressures, and higher respiratory rates. The tidal volume (6–10 mL/kg) must be adequate to expand the lungs during thoracotomy without overdistending alveoli. The inspiratory time should be kept between 1 and 1.5 seconds because a prolonged inspiratory time may collapse alveolar capillaries and impede venous return. The respiratory rate should be 10 to 20 breaths per minute. Nitrous oxide (rarely used) is comparatively less soluble in plasma than are oxygen and other inhalation anesthetics; therefore it rapidly diffuses into the alveoli when it is discontinued, resulting in diffusion hypoxia if the nitrous oxide

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TABLE 18.3 Anesthetic Considerations in the Patient With Esophageal Disease

Preoperative Considerations

Associated conditions	• ± Aspiration pneumonia
Bloodwork	• CBC • Electrolytes • BUN • Cr
Physical exam	• Dysphagia • Regurgitation • Crackles or areas of dullness with auscultation of lungs and fever if aspiration pneumonia present
Other diagnostics	• Blood pressure • ECG • Radiographs (cervical, thoracic, abdominal) • ± Fluoroscopy • ± Barium
Premedications	• Avoid moderate to heavy sedation. Patient needs to maintain strong laryngeal reflexes until intubated

Intraoperative Considerations

Induction	• Preoxygenate 3–5 minutes
	• Be prepared for regurgitation; have suction turned on and immediately available; induce with patient sternal and head up; head can be lowered after intubation, cuff inflation, and circuit attachment
	• Rapidly induce so that endotracheal tube can be quickly placed:
	  • Propofol (4–8 mg/kg) IV, *or*
	  • Alfaxalone (2–5 mg/kg IV), *or*
	  • Midazolam (0.2 mg/kg) IV followed by propofol (2–6 mg/kg IV), *or*
	  • Fentanyl (2–10 µg/kg) IV followed by propofol (2–6 mg/kg IV)

Maintenance	• Isoflurane or sevoflurane, *plus*
	  • Fentanyl (2–10 µg/kg IV PRN in dogs; 1–4 µg/kg IV PRN in cats) for short-term pain relief, *plus*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Morphine[b] (0.1–1 mg/kg IV PRN in dogs; 0.05–0.2 mg/kg IV PRN in cats), *or*
	  • Oxymorphone (0.05–0.2 mg/kg IV PRN), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV PRN), *plus*
	  • Ketamine (low dose) (0.5–1 mg/kg IV), *or*
	  • Ketamine CRI (0.5 mg/kg IV loading dose then 10 µg/kg/min IV)
	  • For hypotension (to keep MAP 60–80 mm Hg) give phenylephrine, ephedrine, norepinephrine, or dopamine as needed

Fluid needs	• 10 mL/kg/h plus 3 × EBL
Monitoring	• Blood pressure • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature • U/O

Blocks	If thoracic surgery, consider:
	• Epidural:
	  • Morphine (0.1 mg/kg preservative free), *or*
	  • Buprenorphine (0.003–0.005 mg/kg diluted in saline), *plus*
	• Local anesthetics (see Box 30.2 and Chapter 13)
	  • Intercostal nerve blocks with local anesthetics (see Box 30.2 and Chapter 13), *plus*
	  • Incisional block with local anesthetic (see Box 30.2 and Chapter 13)

Postoperative Considerations

Analgesia	• Fentanyl CRI (1–10 µg/kg IV loading dose then 2–20 µg/kg/h IV), *or* • Morphine[b] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.05–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h in cats), *or* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats), *or* • Hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs), *or* • Oxymorphone (0.05–0.2 mg/kg IV PRN), *or* • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM q4–8h; 0.01–0.02 mg/kg OTM q6–12h in cats), *plus* • ± Ketamine CRI (2 µg/kg/min IV. If no previous loading dose, give 0.5 mg/kg IV prior to CRI)
Monitoring	• Blood pressure • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature
Bloodwork	• HCT if significant blood loss • Repeat abnormal preoperative bloodwork
Estimated pain score	• Moderate to severe if open thoracic surgery

[a]Monitor for hyperthermia in cats.
[b]Give slowly to prevent histamine release.
[c]Buprenorphine is a better analgesic than morphine in cats.
BUN, Blood urea nitrogen; *CBC,* complete blood cell count; *Cr,* creatinine; *CRI,* constant-rate infusion; *EBL,* estimated blood loss; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *IM,* intramuscular; *IV,* intravenous; *MAP,* mean arterial pressure; *OTM,* oral transmucosal; *PRN,* as needed; *SpO_2,* hemoglobin saturation with oxygen; *U/O,* urine output.

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is not turned off 5 to 10 minutes prior to extubation. Once surgery is completed, extubation should not be rushed. Suction the oropharynx well. Make sure the patient is awake and not overly sedated, respirations are adequate, and the patient is comfortable prior to extubation.

See Table 29.1 for recommendations of analgesics for patients undergoing thoracotomy. After induction of anesthesia, suctioning secretions and ingesta from the obstructed esophagus may help prevent aspiration and minimize contamination of the surgical site.

ANTIBIOTICS

Perioperative antibiotics may be given to prevent infection of periesophageal tissues. Prophylactic IV antibiotics should be given when anesthesia is induced and repeated 2 to 3 hours later. A third dose may be given 8 hours after the second dose. Broad-spectrum antibiotics effective against anaerobes are recommended (i.e., clindamycin, cefoxitin). Animals with preoperative perforation or severe esophageal trauma should be treated with therapeutic antibiotics (see Box 18.14). Specimens collected from the surgical site or perforation should be submitted for bacterial culture and susceptibility testing. The duration of antibiotic therapy varies according to the source of infection and the contaminating organisms, but they should generally be continued for a minimum of 2 weeks.

SURGICAL ANATOMY

The cervical and proximal thoracic portions of the esophagus lie to the left of the midline; however, the esophagus lies slightly to the right of the midline from the tracheal bifurcation to the stomach. The layers of the esophageal wall include the mucosa, submucosa, muscularis, and adventitia. The submucosa is the holding layer of the esophagus and must be incorporated with all sutures. The normal canine esophagus has linear mucosal striations throughout its length. The distal portion of the feline esophagus usually has circular mucosal folds that form a herringbone pattern with positive contrast.

NOTE Because the esophagus lacks a serosal layer, early fibrin sealing of esophagotomy sites may occur more slowly than in other areas of the gastrointestinal tract.

The vascular supply of the cervical esophagus is from branches of the thyroid and subclavian arteries. Bronchoesophageal arteries and segmental branches from the aorta supply the thoracic esophagus. The abdominal esophagus is supplied by branches from the left gastric and left phrenic arteries. Intramural branches ramify and anastomose in the submucosal layer. Collateral blood flow from the cervical and abdominal portions of the esophagus can provide the thoracic esophagus with adequate blood flow if the intramural esophageal vascular system is intact.

SURGICAL TECHNIQUES

Abnormalities of the cervical esophagus are approached using a ventral midline cervical incision. Thoracic esophageal abnormalities at the base of the heart are approached using a right lateral thoracotomy and those cranial or caudal to the heart with a left cranial or caudal thoracotomy. The abdominal esophagus is approached through a ventral midline celiotomy. Hair should be clipped from the entire ventral cervical area for surgery of the cervical esophagus and from the entire hemithorax for approaches to the thoracic esophagus. The skin should be aseptically prepared for surgery. Atraumatic meticulous technique must be employed to ensure rapid healing without dehiscence or stricture (Box 18.15).

BOX 18.15 Principles of Esophageal Surgery

- Use atraumatic meticulous technique; healing is challenged by: no serosa, no omentum, segmental blood supply, constant motion and bolus distention, and intolerance of longitudinal stretching
- Choose the most advantageous approach
- Preserve vasculature; dissect sparingly
- Suction the lumen prior to incising
- Make incision through healthy tissue
- Make longitudinal esophagotomy incisions
- Inspect for contralateral perforations or necrosis
- Resect only 3 to 5 cm during esophagectomy
- Incorporate submucosa with all sutures
- One-layer closure—keep knots extraluminal
- Two-layer closure—inner layer: intraluminal knots; outer layer: extraluminal knots
- Tension-relieving techniques: circumferential myotomy, gastric advancement, phrenic nerve interruption, pexy sutures
- Seal and support with a harvested omental flap or muscle flap
- Treat esophagitis with proton pump inhibitors and/or gastric prokinetics

Approach to the Cervical Esophagus

Position the patient in dorsal recumbency (Fig. 18.42A). Incise the skin on the midline, beginning at the larynx and extending caudally to the manubrium. Incise and retract the platysma muscle and subcutaneous tissues. Separate the paired sternohyoid muscles along the midline to expose the underlying trachea (see Fig. 18.42B). Retract the thyroidea ima vein with the sternohyoid muscle or ligate it. If access to the caudal cervical esophagus is needed, separate and retract the sternocephalicus muscles. Retract the trachea to the right to expose the adjacent anatomic structures, including the esophagus, the thyroid gland, the cranial and caudal thyroid vessels, the recurrent laryngeal nerve, and the carotid sheath (vagosympathetic trunk, carotid artery, and internal jugular vein) (see Fig. 18.42C). Pass a stomach tube or esophageal stethoscope to facilitate identification of the esophagus and lesion. After completing the definitive procedure, lavage the surgical site with warm sterile saline and return the trachea to its normal position. Close the incision by apposing the sternohyoid muscles using absorbable suture (3-0 or 4-0) in a simple continuous pattern. Appose subcutaneous tissues in a simple continuous pattern with 3-0 or 4-0 absorbable suture. Use nonabsorbable suture (3-0 or 4-0 monofilament) and an appositional suture pattern to appose the skin.

Approach to the Cranial Thoracic Esophagus via a Lateral Intercostal Thoracotomy

Position the patient in right lateral recumbency over a rolled towel placed perpendicular to the long axis of the body (Fig. 18.43A). Choose the appropriate intercostal space incision based on the radiographic location of the abnormality (see Fig. 18.43B–D). Most abnormalities cranial to the base of the heart can be accessed through an incision in the left third or fourth intercostal space (the technique for thoracotomy is described on p. 889). *Identify*

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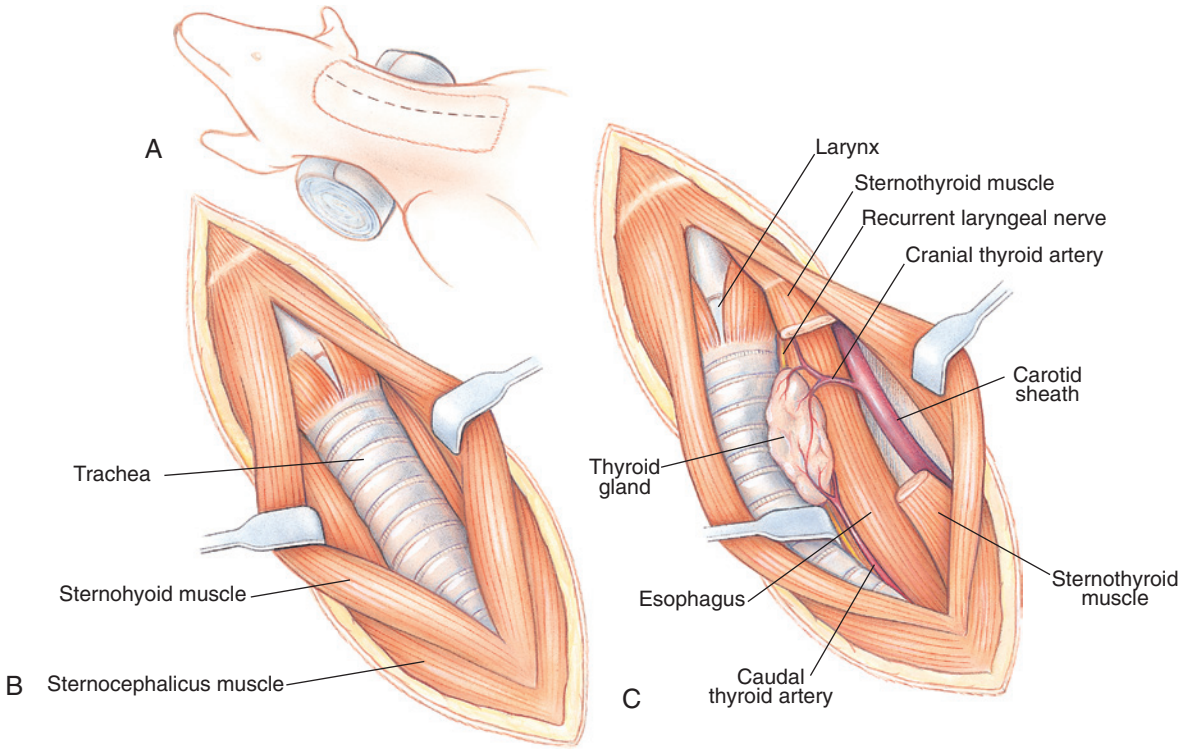


FIG. 18.42 Approach to the cervical esophagus. (A) Position the patient in dorsal recumbency with the neck resting on a rolled towel. (B) Incise the skin from the larynx to the manubrium, and separate the sternohyoid muscles to expose the trachea. (C) Retract the trachea to the right to expose the esophagus, thyroid, carotid sheath, and recurrent laryngeal nerve.

the esophagus in the mediastinum dorsal to the brachiocephalic trunk (see Fig. 18.43E). Identification may be aided by passage of a stomach tube or by palpating the abnormality. Dissect the mediastinal pleura overlapping the esophagus to just above and below the proposed surgical site. Preserve the branch of the internal thoracic vein and the costocervical vein, which cross the cranial esophagus.

Approach to the Esophagus at the Heart Base via a Right Lateral Thoracotomy

The approach is the same as that for the cranial esophagus except that the incision is made through the right fourth or fifth intercostal space (Fig. 18.44A–B). *Identify the esophagus, located just dorsal to the trachea in the mediastinum (see Fig. 18.44C). Dissect and retract the azygos vein from the esophagus to allow adequate exposure. Ligate the azygos vein if necessary to adequately expose the esophagus. Closure is the same as for cranial thoracotomy.*

Approach to the Caudal Esophagus via a Caudal Lateral Thoracotomy

Position the patient in lateral recumbency as described previously for cranial lateral thoracotomy. Perform a caudal lateral thoracotomy (Fig. 18.45A). Although the caudal esophagus can be approached through an incision in either the left or right eighth or ninth intercostal space, the left ninth space is preferred. *Expose the caudal esophagus by transecting the pulmonary ligament and packing the caudal lung lobes cranially. Identify the esophagus, which is just ventral to the aorta (see Fig. 18.45B). Identify the dorsal and ventral vagal nerve branches on the lateral aspect of the esophagus and protect them.*

Esophagotomy

Pack off the esophagus from the remainder of the field with moistened laparotomy pads. Suction material from the cranial esophagus before making the esophagotomy incision to minimize contamination of the surgical site. If ingesta and secretions have not been completely suctioned, occlude the lumen cranial and caudal to the proposed esophagotomy site with fingers or noncrushing forceps. Place stay sutures adjacent to the proposed incision site to stabilize, aid manipulation, and avoid trauma to the esophageal edges. Make a stab incision into the lumen of the esophagus, and extend the incision longitudinally as necessary to remove the foreign body or observe the lumen. Make the incision over the foreign body if the esophageal wall appears normal. If the wall appears compromised, make the incision caudal to the lesion or foreign body. Remove foreign bodies with forceps, taking care to avoid further esophageal trauma (tearing or perforation). Examine the esophageal lumen. Obtain culture specimens from necrotic and perforated areas. Debride and close perforations surrounded by healthy tissue that involve less than one-fourth the circumference of the esophagus. Identify large necrotic areas or extensive perforations and perform a resection and anastomosis (discussed later).

Esophagotomy incisions may be closed with a one- or two-layer closure. A two-layer simple interrupted closure results in greater immediate wound strength, better tissue apposition, and improved healing after esophagotomy but takes longer to perform than single-layer techniques. *Place each suture approximately 2 mm*

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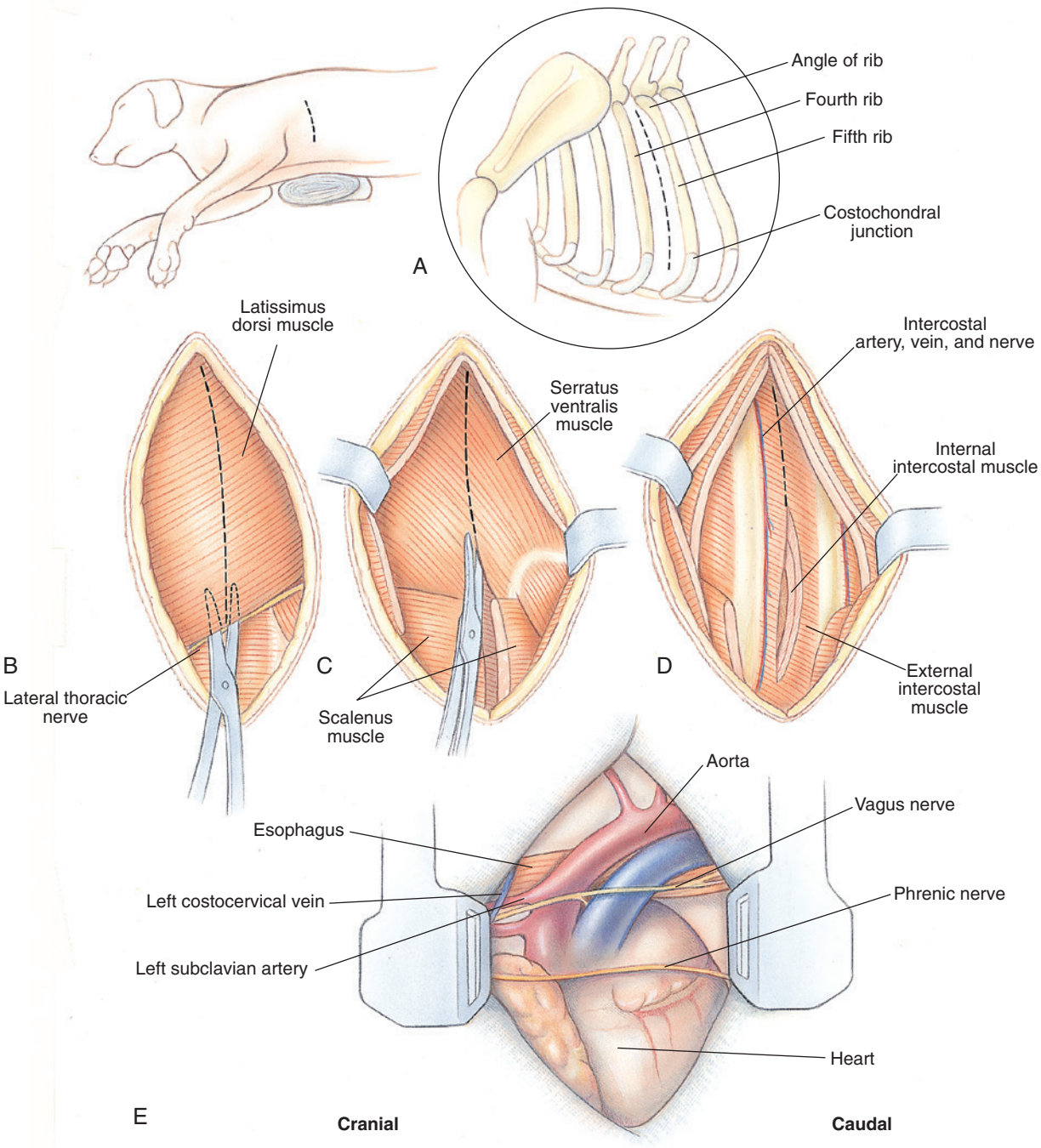


FIG. 18.43 Approach to the cranial thoracic esophagus. (A) Position the patient in right lateral recumbency over a rolled towel placed perpendicular to the long axis of the body. Select the appropriate incision site based on the radiographic location of the lesion *(inset).* (B) Identify and transect the latissimus dorsi muscle *(dashed line).* (C) Identify and transect or retract the serratus ventralis *(dashed line)* and scalenus muscles. (D) Expose and incise the intercostal muscles *(dashed line).* (E) Position rib retractors and identify the thoracic viscera.

from the edge and 2 mm apart. Incorporate the mucosa and submucosa in the first layer of a two-layer simple interrupted closure. Place sutures so that the knots are within the esophageal lumen (Fig. 18.46A–B). Incorporate the adventitia, muscularis, and submucosa in the second layer of sutures with the knots tied extraluminally (see Fig. 18.46C). When a one-layer closure is used, pass each suture through all layers of the esophageal wall, and tie the knots on the extraluminal surface. Check closure integrity by occluding the lumen, injecting saline, applying pressure, and observing for leakage between sutures.

Partial Esophagectomy

Esophagectomy is performed to remove devitalized or diseased esophageal segments. Benign strictures should be dilated

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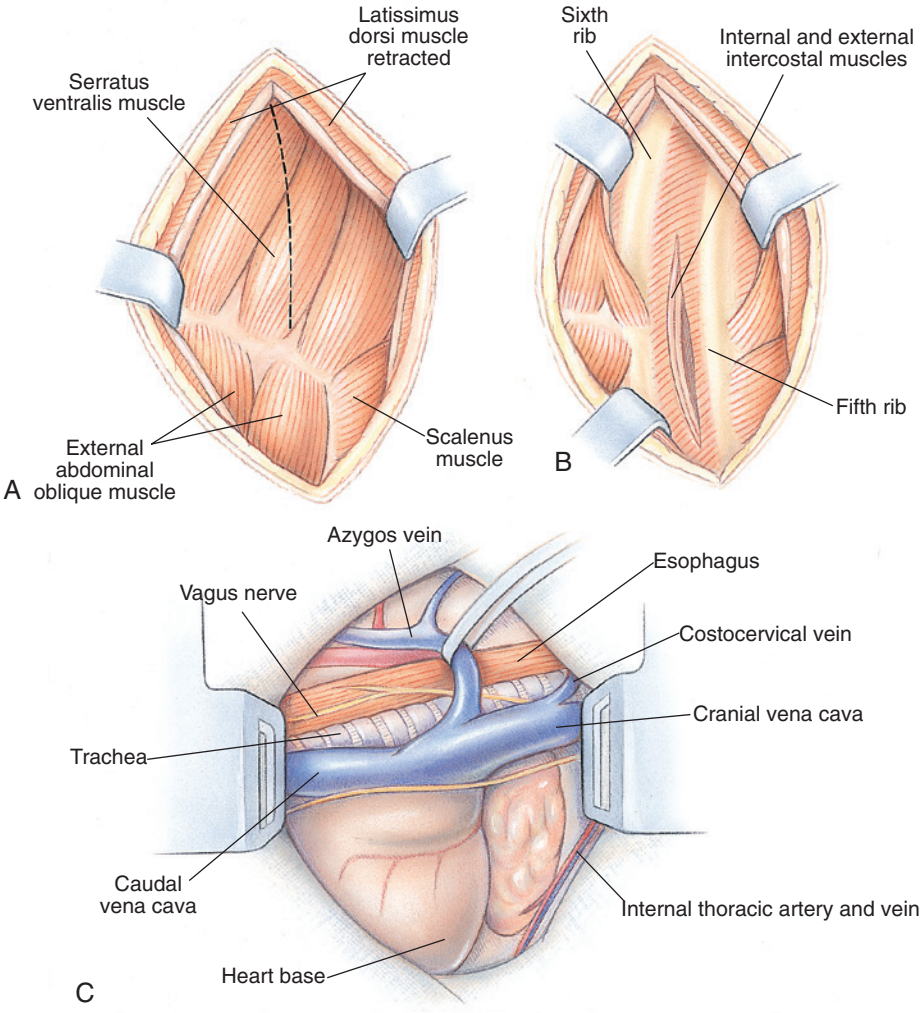


FIG. 18.44 Approach to the esophagus at the heart base. (A) Make an incision through the right fourth or fifth intercostal space. Identify and transect or retract the latissimus dorsi, serratus ventralis *(dashed line),* scalenus, and external abdominal oblique muscles. (B) Incise the intercostal muscles. (C) Expose the thoracic viscera.

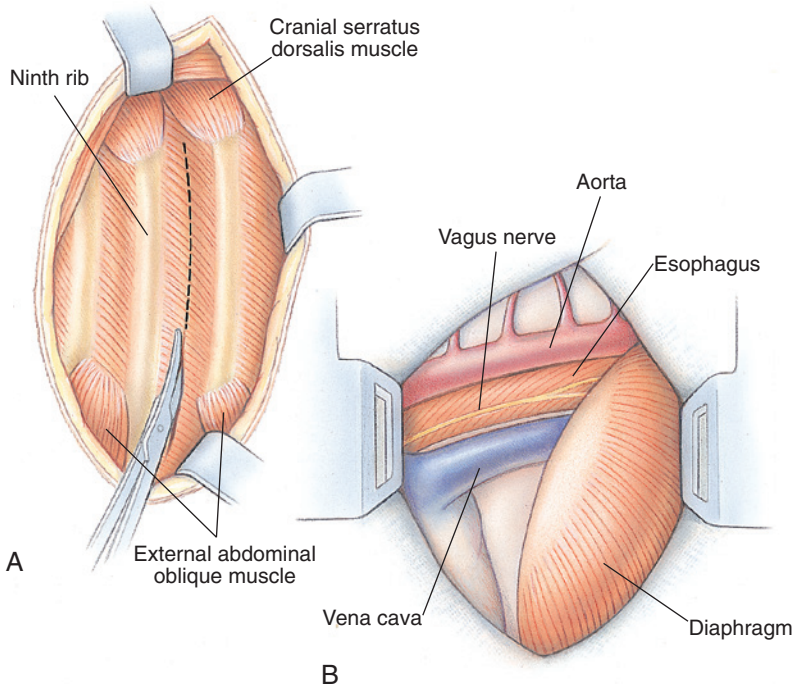


FIG. 18.45 To approach the caudal thoracic esophagus, position the animal in right lateral recumbency and make an eighth or ninth intercostal space incision. (A) Identify and transect or retract the latissimus dorsi, cranial serratus dorsalis, external abdominal oblique, and intercostal muscles *(dashed line).* (B) Identify the diaphragm and other thoracic viscera.

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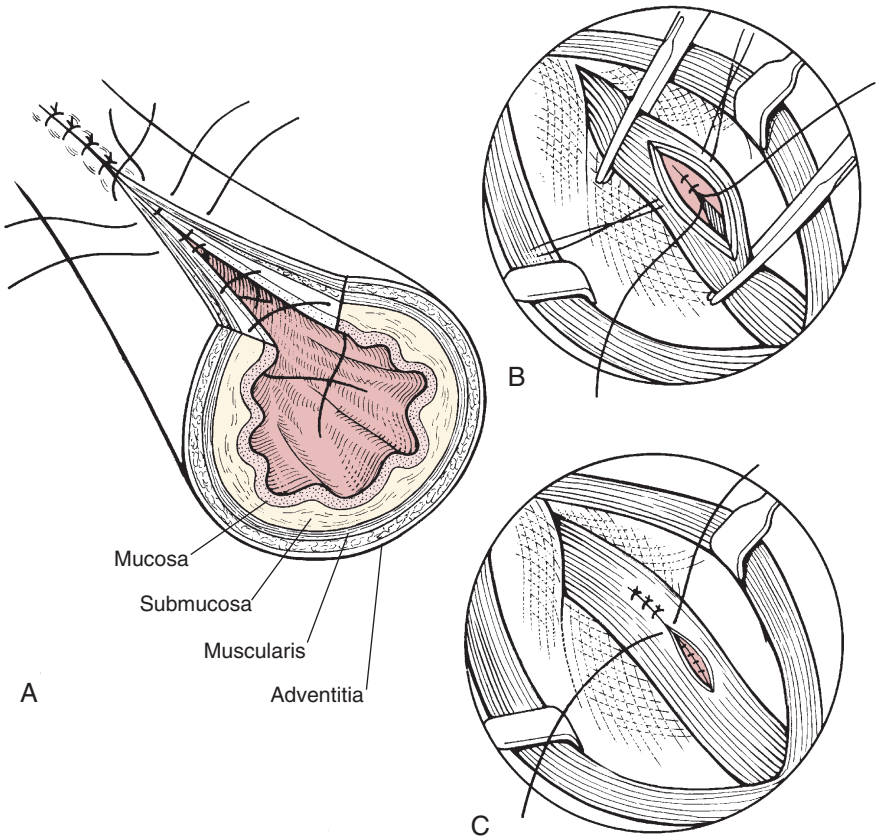


FIG. 18.46 Esophagotomy closure. (A–B) Close the mucosa and submucosa with simple interrupted sutures so that the knots are intraluminal. (A) and (C) Appose the adventitia and muscularis with a second layer of simple interrupted sutures oriented with extraluminal knots.

endoscopically if possible, with surgery reserved as a salvage procedure. Periesophageal tissues must be dissected from around the abnormal area to allow resection of diseased tissue and mobilization of normal esophagus; however, extensive dissection should be avoided to preserve vasculature. Excessive tension along the anastomosis may cause dehiscence. Although 20% to 50% of the esophagus has been resected and primarily anastomosed without tension-relieving techniques, resection of more than 3 to 5 cm risks anastomotic dehiscence. Partial myotomy is recommended to relieve anastomotic tension when resecting large segments of esophagus (Fig. 18.47). Circumferential myotomy is a partial-thickness myotomy through the longitudinal muscle layers, 2 to 3 cm cranial and caudal to the anastomosis. The inner circular muscle layers are not incised to avoid damaging the submucosal blood supply. Injection of saline into the muscularis may aid identification of the different muscle layers. The myotomy gap heals by second intention without stricture or dilatation. Mobilizing the stomach cranially through an enlarged esophageal hiatus can also help reduce tension across the anastomosis. Other tension-relieving techniques include interruption of the phrenic nerve and placement of "pexy" sutures between the esophagus and the prevertebral fascia. Esophageal replacement may be necessary if segments of more than 3 to 5 cm are resected. Many replacement techniques have been described, including microvascular anastomosis of the colon or small intestine to the esophagus, gastric tubes, skin tubes, and various prostheses. Replacement

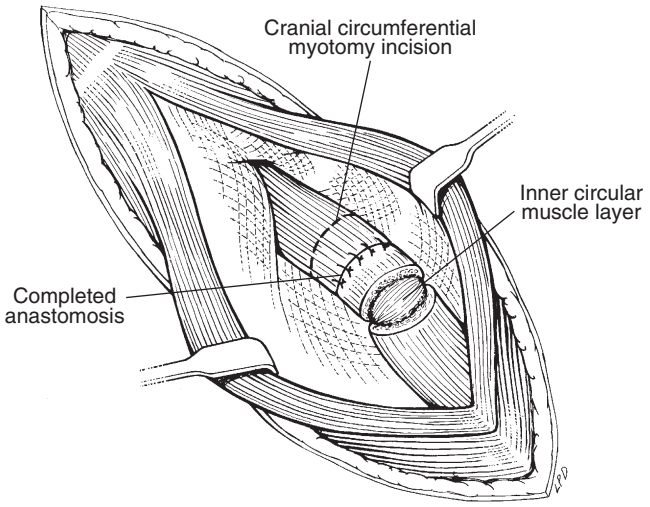


FIG. 18.47 Tension-relieving esophageal myotomy is performed 2 to 3 cm cranial and caudal to the anastomosis.

of the esophagus requires specialized training, techniques, and equipment.

For esophagectomy, occlude and stabilize the esophagus with fingers (scissor action of middle and index fingers) or noncrushing forceps. Resect the diseased portion of the esophagus (Fig. 18.48).

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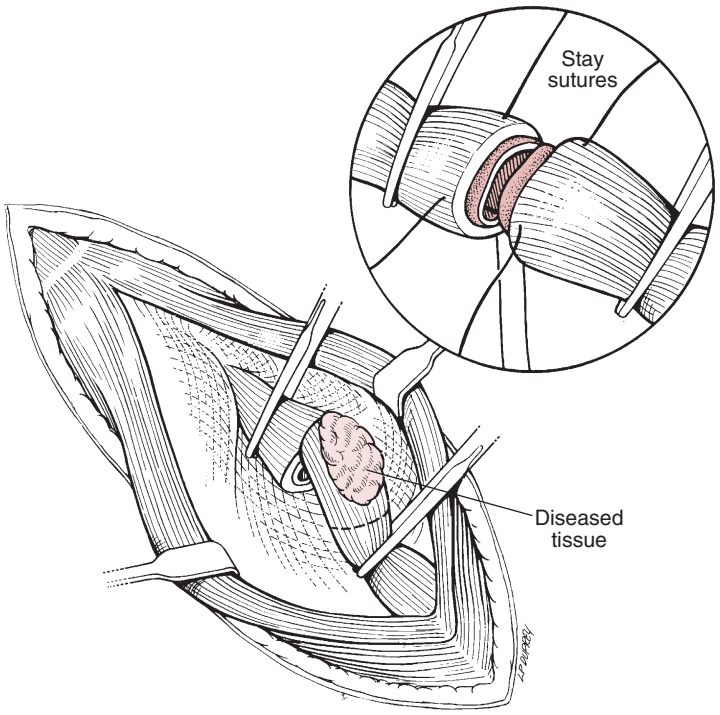


FIG. 18.48 For partial esophagectomy, occlude the esophageal lumen with noncrushing forceps and mobilize and resect the diseased esophagus *(dashed line)*. Place stay sutures to manipulate the esophageal ends *(inset)*. Anastomose the ends as shown in Fig. 18.46.

Suction debris from the lumen of the remaining esophagus. Place three equally spaced stay sutures at each end of the remaining esophagus to facilitate gentle handling of the esophagus and help maintain apposition and alignment of the transected ends (see Fig. 18.48). Bring the esophageal ends into apposition with the stay sutures and suture the ends together using a one- or two-layer closure, as described for esophagotomy. Place sutures in the contralateral (far) wall first and then in the more accessible ipsilateral (near) wall. When using a two-layer closure, appose the esophagus in the following four steps: (1) appose the adventitia and muscularis of the contralateral wall around approximately half of the esophageal circumference (Fig. 18.49A); (2) appose the mucosa and submucosa of the contralateral wall (see Fig. 18.49B); (3) appose the mucosa and submucosa of the ipsilateral wall (see Fig. 18.49C); and (4) appose the adventitia and muscularis of the ipsilateral wall (see Fig. 18.49D). Check the integrity of the closure by occluding the lumen, injecting saline, applying pressure, and observing for leakage between sutures.

An end-to-end circular stapling device may reduce operating time and contamination of the surgical field, but this technique may have a greater potential for stricture formation. *Place a purse-string suture in the cranial esophageal remnant. Insert the anvil and tie the purse string around the instrument shaft. Place a second purse-string suture around the distal resection site and secure it. Appose the esophageal ends by tightening the stapler's wing nut. Activate the instrument and then remove it with a rotating motion. Close the access incision for placement of the stapling device with a linear stapler (p. 445).*

Support or Patching Techniques

Augmentation of esophagotomy or esophagectomy sites with omentum or muscle can aid healing by supporting, sealing, and revascularizing the surgical site. Muscle pedicles from the sternohyoid, sternothyroid, intercostal, diaphragm, or epaxial muscles can be mobilized and sutured over the primary repair or esophageal defect (Fig. 18.50A). As an alternative, omentum can be mobilized from the abdomen, brought through a rent in the diaphragm, and sutured over the esophageal site (see Fig. 18.50B). Pedicles from the gastric wall and pericardium have also been used.

Esophagostomy

Feeding tubes placed in the midcervical esophagus are associated with fewer complications than pharyngostomy or nasogastric feeding tubes. The technique for esophagostomy tube placement is described on p. 95. The distal end of the tube is positioned rostral to the gastroesophageal junction to reduce gastroesophageal reflux. Ostomy wounds typically heal by second intention after removal of the tube, without stricture or esophagocutaneous fistula formation.

HEALING OF THE ESOPHAGUS

The esophagus is subject to constant movement from swallowing and respiration. This continuous motion may interfere with healing and must be overcome with good surgical technique. Although large segments of the esophagus have been resected successfully, the esophagus does not tolerate longitudinal stretching well and may dehisce if tension is excessive. Complications (especially dehiscence, stricture, fistulization) are common after esophageal surgery. The high complication rate has been blamed on the lack of serosal covering, lack of omentum, segmental blood supply, constant motion, and distention with passage of food boluses. Careful surgical technique and patient management can minimize most of the possible complicating factors.

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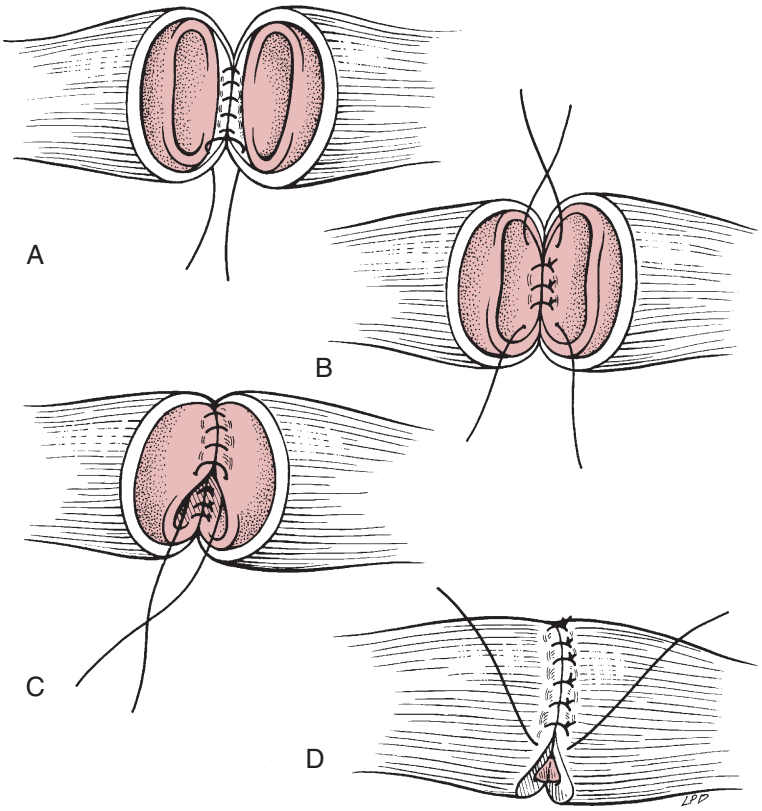


FIG. 18.49 During partial esophagectomy, appose the ends with two layers of sutures in a four-step procedure. (A) First, appose the adventitia and muscularis on the far side with simple interrupted sutures and extraluminal knots. (B) Second, appose the submucosa and mucosa on the far side with simple interrupted sutures using intraluminal knots. (C) Third, appose the nearside submucosa and mucosa. (D) Last, appose the nearside muscularis and adventitia.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

In addition to a general pack, other requirements may include special forceps (i.e., noncrushing Doyen forceps), delicate hemostats (i.e., Adson), Metzenbaum scissors, retractors (Gelpi retractors for the cervical approach; Finochietto retractors for the thoracic approach; malleable retractors), and tubes (chest tube, gastrostomy tube, stomach tube). A suction unit and laparotomy pads should also be available. Surgical stapling equipment (e.g., linear stapler, circular end-to-end stapler, ligate-and-divide vascular stapler, skin stapler) is beneficial but optional. Monofilament absorbable suture (polydioxanone, polyglyconate, or poliglecaprone 25 with a swaged-on taper point needle) and nonabsorbable suture (polypropylene, nylon with a reverse cutting needle) (3-0 or 4-0) are recommended in the esophagus.

POSTOPERATIVE CARE AND ASSESSMENT

After esophageal surgery, analgesics should be provided as described for thoracotomy patients on p. 886. Air and fluid must be evacuated via a thoracostomy tube or needle thoracentesis after thoracic procedures. Unless periesophageal or thoracic infection is anticipated, thoracostomy tubes can generally be removed within 8 to 12 hours after esophageal surgery. Nasal oxygen may benefit animals after thoracotomy (see Table 4.4).

Oral intake should be withheld for 24 to 48 hours. IV fluids should be continued until oral intake resumes. Water may be offered 24 hours postoperatively if the esophagus is in good condition and regurgitation or vomiting does not occur. Blenderized food (gruel) may be offered during the next 24 hours if no vomiting or regurgitation occurs after water consumption. Blenderized food should be continued for 5 to 7 days, then the animal should be gradually returned to its normal diet over the next week. If oral intake is not anticipated or possible within 48 to 72 hours after surgery, feeding should be performed via a gastrostomy tube (see p. 97).

Esophagitis and aspiration pneumonia should be treated as described in the discussion on Preoperative Concerns (p. 365). Patients undergoing esophageal surgery should be closely monitored for fever and neutrophilia, which may indicate infection secondary to leakage. Dysphagia and regurgitation that occur 3 to 6 weeks after surgery may indicate esophageal stricture formation. It is extremely important that clients be informed of possible complications before and after surgery (Box 18.16).

COMPLICATIONS

Infection, regurgitation, pneumonia, esophagitis, dehiscence, fistula, stricture, and recurrence of disease are possible complications of esophageal surgery. Common errors in treating esophageal disorders include delayed identification of esophageal foreign

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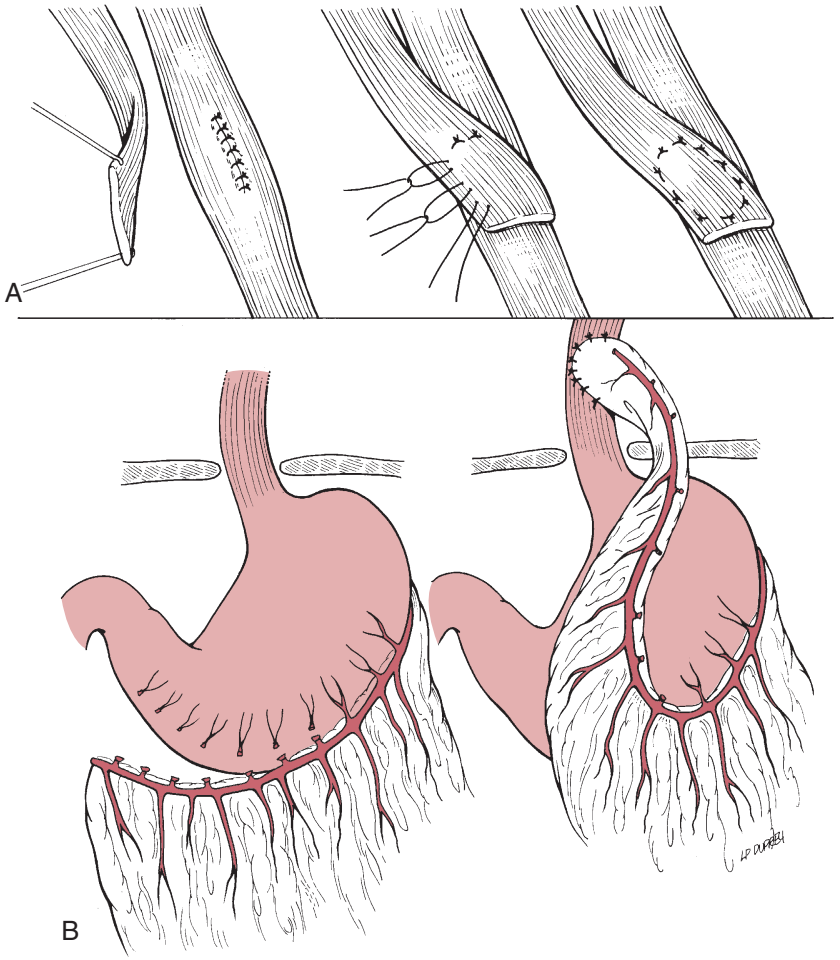


FIG. 18.50 Patching the esophagus. (A) Mobilize muscle adjacent to the esophagus and suture it over an esophageal incision to create an esophageal patch. (B) As an alternative, mobilize omentum from the greater curvature of the stomach, pass it through an incision in the diaphragm, and suture it over the esophageal closure to create an omental patch.

BOX 18.16 Client Education and Communication for Patients Undergoing Esophageal Surgery

- Regurgitating or vomiting can cause aspiration pneumonia, which can be fatal if not controlled.
- Surgery may not resolve all clinical signs of esophageal disease.
- Preventing oral intake, tube feeding, or feeding from an elevated platform such that the patient must be near vertical to the floor may be necessary.
- Esophageal healing is poor compared with other parts of the gastrointestinal tract; therefore leakage, infection, dehiscence, and stricture are more common.
- Foreign bodies may perforate the esophagus or great vessels during extraction, with fatal results.
- Dogs should not be fed bones.

bodies, perforation, or aspiration pneumonia; failure to control esophagitis; inappropriate surgical approach; and failure to patch or support the esophagus appropriately.

SPECIAL AGE CONSIDERATIONS

Care must be used in anesthetizing young animals for esophageal surgery. Surgery is often performed in animals with persistent right aortic arches (PRAAs; see p. 394) or hiatal hernias (see p. 386) at 8 to 16 weeks of age. Perioperative hypothermia and hypoglycemia are common problems in these pediatric patients and may be life-threatening.

SPECIFIC DISEASES

ESOPHAGEAL FOREIGN BODIES

DEFINITION

Foreign bodies are inanimate objects that may obstruct the esophageal lumen to varying degrees.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The most common foreign bodies found in the esophagi of dogs and cats are bones, although sharp metal objects (e.g., needles and fishhooks), rawhide chew toys, balls, string, and an assortment of other objects have lodged there. Foreign bodies lodge in the esophagus because they are too large to pass or they have sharp edges that become embedded in the esophageal mucosa. Foreign bodies are most commonly found at the thoracic inlet, the base of the heart, or in the epiphrenic (diaphragm) area because extraesophageal structures limit esophageal dilatation at these sites. The persistence of a foreign body (acting as a bolus) in the esophagus stimulates peristaltic activity. If the foreign body puts excessive pressure on the esophagus or if it remains at one location, pressure necrosis may occur and in some cases can cause

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perforation within 3 to 4 days. If esophagitis occurs, it may interfere with esophageal motility and lower esophageal high-pressure zone function. Food that does not pass the obstruction accumulates and may be regurgitated or may cause proximal esophageal distention. Prolonged distention disrupts normal neuromuscular function and reduces peristalsis. Esophageal perforation and aspiration pneumonia (see p. 367) are the major complications possible.

Sharp objects may abrade or lacerate the esophageal mucosa, causing irritation and inflammation of the underlying tissues (esophagitis) or hemorrhage. Sharp objects may also perforate the esophageal wall and allow bacteria, ingesta, and secretions to contaminate the periesophageal tissues. Occasionally sharp objects perforate the esophageal wall and one of the great vessels at the base of the heart, causing severe hemorrhage into the mediastinum. Foreign bodies may penetrate the esophageal wall and establish a fistula with the trachea, bronchi, pulmonary parenchyma, or skin (Box 18.17).

DIAGNOSIS

Clinical Presentation

Signalment

Indiscriminate eaters (dogs) are more commonly affected than more particular eaters (cats). Although any breed of dog or cat may have an esophageal foreign body, small-breed dogs are more frequently affected because their esophagus is smaller. Yorkshire terriers and West Highland white terriers may be overrepresented compared with hospital populations.[9] Cats, having a tendency to play and hunt, more commonly have string or needle foreign bodies than bones. Foreign bodies may occur in an animal of any age but are most common during the first 3 years of life.

History

Animals may be presented for treatment within minutes of foreign body ingestion (especially when it is seen, as commonly occurs with fishhooks) or days later. An acute onset of pawing at the mouth, oral or cervical pain, dysphagia, and/or regurgitation are the most common initial signs. Other signs may include gagging, excessive salivation, retching, hyporexia, restlessness, depression, dehydration, and respiratory distress. Clinical signs vary somewhat depending on the duration, location, and type of obstruction. Patients with acute obstructions generally show excess salivation and gag or regurgitate soon after eating. Weight loss and emaciation are sometimes seen in patients with long-term esophageal obstruction. Patients with complete obstruction regurgitate both solids and liquids, whereas those with partial obstruction may retain liquids. Esophageal pain may cause anorexia. Foreign bodies that impinge on the upper airways may cause acute respiratory distress. Sharp foreign bodies or those that have caused necrosis of the esophageal wall allow leakage of saliva and ingesta into the surrounding tissues, causing inflammation and infection. These patients are likely to be hyporexic, febrile, and/or dyspneic as a result of pleural effusion. Hypovolemic shock can occur if a foreign body penetrates a major vessel adjacent to the esophagus. A history of being fed bones, getting into garbage, or roaming is consistent with foreign body ingestion. Fishhook ingestion is typically obvious from the history.

NOTE Strive to differentiate between vomiting and regurgitation; acute-onset regurgitation strongly suggests the presence of an esophageal foreign body.

Physical Examination Findings

Patients may be normal or depressed and dehydrated on physical examination. If the foreign body is lodged in the cervical esophagus, it may sometimes be palpated. Poor body condition may be a factor if the patient has been anorexic or regurgitating for weeks. Abnormal lung sounds may be auscultated in patients with aspiration pneumonia. Animals that are in great pain may drool because they refuse to swallow.

Diagnostic Imaging

Most foreign bodies can be seen on good quality survey radiographs (Fig. 18.51) provided that they are radio-opaque, but it is not always obvious that they are in the esophagus. Esophageal masses and foreign bodies may be poorly demarcated densities that closely resemble pulmonary masses. Foreign bodies are usually found at or cranial to the thoracic inlet, the base of the heart (approximately 10%), and the diaphragm (approximately

BOX 18.17 Esophageal Foreign Bodies: Key Points

- History of roaming or garbage eating is a diagnostic clue
- Common location: thoracic inlet, heart base, or diaphragm
- Suspect perforation, pressure necrosis, aspiration pneumonia, fistula
- Most are identified or suspected from survey radiographs
- Most patients with esophageal perforations will have pleural effusion, pneumothorax, or pneumomediastinum; contrast radiographs are rarely necessary
- If you use contrast radiographs to find perforations, use iodinated contrast media
- Most can be removed during esophagoscopy; do not use force
- Fatal hemorrhage can occur if a sharp object penetrates the wall
- If the mass cannot be retrieved per mouth, then advance object into stomach if possible
- Radiograph the patient after endoscopic removal to look for signs of perforation (pneumothorax)
- Feeding tubes may be required
- Postoperative strictures may occur with or without surgery

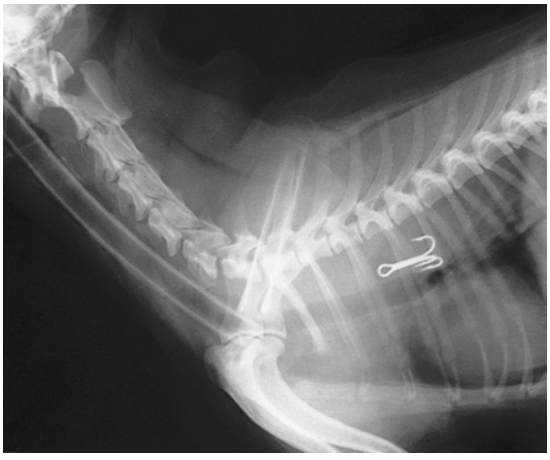


FIG. 18.51 Lateral thoracic radiograph showing a treble fishing hook esophageal foreign body at the base of the heart in a dog.

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85%). In addition to the foreign body, there may be an adjacent soft tissue opacity and cervical subcutaneous emphysema, and a dilated, air-filled cranial esophagus may be seen. Pneumonia and tracheal distortion may also be present. Radiographs should be closely examined for signs of pneumomediastinum, pneumothorax, and pleural effusion, each of which suggests esophageal perforation. If pleural effusion is present, it must be examined cytologically to see if there is evidence of inflammation or infection.

Esophagrams are rarely necessary to identify foreign bodies and perforations. If contrast radiographs will be performed to look for esophageal perforation, water-soluble, organic, iodine contrast materials or iohexol are recommended. Isotonic iodinated contrast agents are the safest as they will not cause pulmonary edema if aspirated or if there is a bronchoesophageal fistula. Hypertonic iodinated contrast agents should be avoided in these situations. However, one does not generally do a contrast radiograph to look for perforation (see the discussion on endoscopy presented later), and the presence of a foreign body may mask identification of a perforation during an esophagram. If a bronchoesophageal fistula is suspected, a hypertonic iodinated contrast agent should not be used because its hypertonicity may cause pulmonary edema. Foreign bodies can also be diagnosed endoscopically, which is usually the next step after finding a suggestive density on plain radiographs.

NOTE If available, endoscopy is generally of greater value than radiographic contrast studies in diagnosing esophageal foreign bodies because the foreign body can potentially be removed during the endoscopic procedure.

Laboratory Findings

Laboratory findings are normal with acute obstructions. Perforations usually cause neutrophilic leukocytosis. Hypoglycemia may be seen in young patients that are unable to eat or in SIRS (or both).

DIFFERENTIAL DIAGNOSIS

Vascular ring anomalies, extraluminal masses, esophageal neoplasia, strictures, esophagitis, gastroesophageal intussusception, esophageal diverticula, hiatal hernias, megaesophagus, and cricopharyngeal dysfunction are other possible causes of regurgitation that must be differentiated from esophageal foreign bodies. Radiographs of pulmonary tumors and esophageal masses can perfectly mimic radiographs of esophageal foreign bodies.

SURGICAL TREATMENT

It is very important to take a radiograph just before induction of anesthesia to ensure that the foreign body has not moved and that there is no sudden evidence of perforation; this is true for endoscopy as well as surgery.

Most esophageal foreign bodies can be successfully removed endoscopically (see also Chapter 14). Rigid endoscopes (i.e., colonoscopes) are preferred, assuming that they are long enough. Rigid endoscopes allow one to use rigid grasping forceps, which in turn allow better manipulation and grasping than is available with forceps inserted through flexible endoscopes. The rigid scopes also offer some degree of protection to the esophagus; the foreign body is partially drawn up into the scope during removal so that it does not traumatize the esophageal mucosa. The major advantage to flexible endoscopes is that some esophageal foreign bodies are out of reach of rigid scopes; but the retrieval devices used in flexible endoscopes do not offer the operator as much control or power as do the rigid endoscopic forceps. Surgical removal of esophageal foreign bodies should be elected if (1) there is pneumothorax or pleural exudate suggesting perforation, (2) endoscopy cannot retrieve the foreign body, or (3) if a fishhook is deeply embedded such that the point and barb are obviously completely through the wall of the esophagus and free to lacerate intrathoracic vessels. After removal of the foreign body, the esophagus must be carefully reevaluated endoscopically and the chest re-radiographed to look for evidence of perforation (i.e., pneumothorax). Be careful when insufflating air; it is possible to cause a tension pneumothorax if there is a perforation. Perforations should generally be managed with debridement and surgical closure (see p. 370); however, small perforations occasionally may be allowed to close on their own.

Preoperative Management

Therapy to correct significant dehydration and electrolyte and acid-base imbalances should be initiated before surgery. Prophylactic antibiotics should be given (see p. 369).

Anesthesia

Anesthetic management of patients undergoing esophageal surgery is described on p. 367 and in Table 18.3. Deep general anesthesia or muscle relaxants that reduce esophageal tone facilitate endoscopic manipulations. Nitrous oxide should not be used in these patients. Most animals should be preoxygenated before induction. If the patient has aspiration pneumonia, positive pressure ventilation should be performed with lower tidal volumes, normal peak ventilation pressures, and higher respiratory rates.

Surgical Anatomy

The surgical anatomy of the esophagus is described on p. 369.

SURGICAL TECHNIQUE

Foreign bodies may be removed by extracting them endoscopically with grasping instruments, pulling them out using a balloon-type catheter (e.g., Foley), advancing them into the stomach where they can dissolve or be removed by gastrotomy (see p. 401), or by performing an esophagotomy (see p. 370), or a partial esophagectomy (see p. 371).

Endoscopic Removal of Esophageal Foreign Bodies

Endoscopic removal of esophageal foreign bodies is generally preferred unless there is a good reason to the contrary; it is successful in removing foreign bodies in the majority of affected animals. *After examining the radiograph taken just before anesthetic induction, carefully advance the rigid endoscope down the esophagus to the level of the foreign body.* The larger the diameter of endoscope, the better the visualization and the more the esophageal lumen will be dilated (which can aid in removing the foreign object). *Position the patient such that the neck is extended so that the endoscope will not put undue pressure on the trachea or the nearby vessels (if a flexible scope is used, positioning of the neck is not as important). Only advance the scope while observing through it so as not to push the scope into the foreign object and cause further trauma. Insufflate with minimal amounts of air and only if necessary; insufflating too much air might rupture weakened*

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areas in the esophagus, cause a gastric dilatation that cannot be relieved by stomach tube because of obstruction of the esophagus, or cause tension pneumothorax. Once the foreign object is found, aspirate any fluid or debris surrounding it to enhance visualization. After examining the object, grasp it firmly and then carefully manipulate it to free it from the esophagus (there are typically points where the foreign object protrudes into esophageal mucosal defects). Once it is freed, draw it as far as possible up into the rigid scope, and then remove the scope and foreign object as one unit. If there is severe trauma to the esophageal mucosa, place a gastrostomy tube (see p. 97).

If the foreign object cannot be grasped, it can sometimes be pushed into the stomach *if* the endoscopist *knows* that there are no sharp objects or structures that could cause perforation. If necessary, a water-soluble lubricant can be applied around the foreign object to facilitate this maneuver. Forcefully pushing an object that is firmly embedded in the esophageal wall is contraindicated because doing so may cause perforation or enlargement of a preexisting perforation. Once in the stomach, the foreign object may be removed by gastrotomy (see p. 401) or, in the case of bones, allowed to dissolve. **However, do not administer antacids or the stomach will not be able to dissolve the bone**.

Fishhooks can usually be removed with rigid scopes, depending on the size of the barb (hooks with very large barbs often cannot be torn out of the mucosa) and whether the barb has been pushed all the way through the wall of the esophagus such that it could lacerate thoracic vessels when it is pulled back into the esophagus. The technique varies a little, depending on whether the point of the hook is pointed craniad or caudad. *If the point of the hook is pointed caudad, then firmly grasp the hook at the bend such that when it is pulled, the point of the hook is pulled straight out, parallel with the esophageal lumen.* The barb will tear through the esophageal wall, leaving a small defect. *If the point of the hook is pointed craniad, firmly grasp the eye of the hook with the rigid forceps. Next, advance the rigid endoscope until the rim of the rigid scope rests on the bend of the hook. Then, holding the eye of the hook so that the shaft remains parallel with the lumen of the esophagus and the rigid scope, advance the scope and the rigid grasping forceps as one unit, tearing the hook out of the mucosa.*

Balloon Catheter-Assisted Removal of Foreign Objects

An alternative to grasping the foreign body is to pass a balloon catheter distal to the object (Figs. 18.52 and 18.53). The esophageal lumen is then dilated beyond its normal size by inflating the balloon, and the object is disengaged from the esophageal wall by endoscopic manipulation if necessary and removed as the catheter is pulled out through the mouth. This procedure is advisable only for foreign bodies with a relatively smooth contour. After nonsurgical foreign body removal, radiographs may be taken to look for evidence of perforation (e.g., pneumomediastinum and pneumothorax).

Surgical Removal of Esophageal Foreign Objects

Foreign bodies can be surgically removed by performing an esophagotomy (see p. 370) or a partial esophagectomy (see p. 371). Distal esophageal foreign bodies are occasionally removed by a gastrotomy. *Make an incision midway between the greater and lesser curvature, direct a forceps into the distal esophagus and grasp the object, pull into the stomach, and remove it.* An esophagotomy or a partial esophagectomy is performed when foreign bodies cannot be removed by other means, when the risk of esophageal

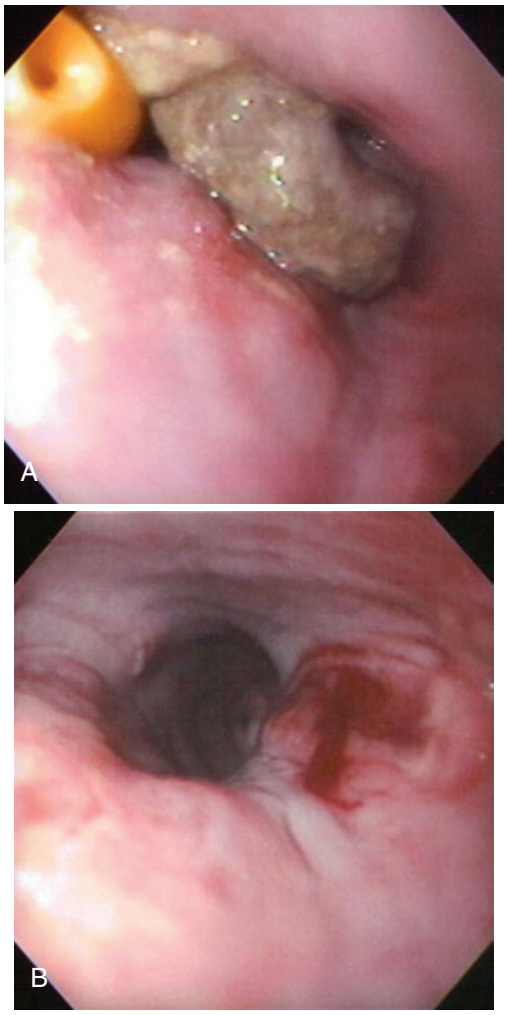


FIG. 18.52 (A) Endoscopic appearance of a piece of liver lodged in the esophagus. Note the tip of a Foley catheter, which is being passed to retrieve it. (B) After retrieval of the foreign body, ulceration of the mucosa was identified.

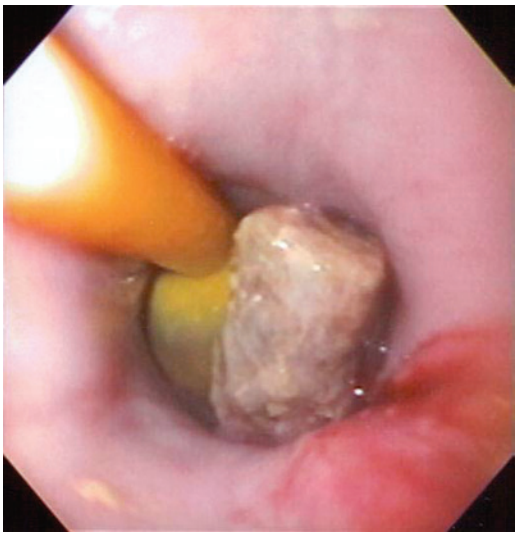


FIG. 18.53 Smooth esophageal foreign bodies can be removed by balloon retraction during endoscopy. Dilate the esophagus, pass the catheter distal to the foreign body, then inflate the balloon and withdraw the catheter and foreign body.

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perforation or laceration is high, or when there is evidence of mediastinitis, pleuritis, or esophageal necrosis. *Debride all esophageal disruptions if necessary and close in one or two layers as for esophagotomy.* Abnormal communications between the alimentary and respiratory tracts (i.e., bronchoesophageal or tracheoesophageal fistula) must be closed. Partial or complete pulmonary lobectomy (see p. 894) is required with some bronchoesophageal fistulae.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Endoscopes (both flexible and rigid, but preferably rigid), endoscopic grasping forceps, or balloon-tipped catheters (i.e., Foley catheter) are needed for nonsurgical foreign body removal. Thoracic retractors are needed for surgical removal of intrathoracic foreign bodies. Noncrushing intestinal forceps to occlude the esophageal lumen may be needed for esophagotomy or esophagectomy procedures.

POSTOPERATIVE CARE AND ASSESSMENT

All patients should be observed carefully for 2 to 3 days for signs of esophageal leakage and infection. Esophagitis and aspiration pneumonia should be treated as described on pp. 366–367. Antibiotics should be continued for several days if the esophageal mucosa is severely eroded or lacerated, especially if a fishhook was removed. IV fluids should be continued until feeding resumes. To avoid delays in healing, all oral intake (food, water, medications) should be withheld for a minimum of 24 hours after removal of foreign bodies causing substantial erosion or ulceration. If no regurgitation is observed, water and then a bland gruel should be introduced gradually. Animals with minimal esophageal trauma may be offered water within 24 to 48 hours, followed by small meals of gruel. After 3 to 7 days of gruel feeding, soft, moist food should then be offered for 5 to 7 days, followed by a gradual return to a normal diet. Animals with moderate to severe esophageal trauma should avoid oral intake for 3 to 7 days. In debilitated patients or those requiring no oral intake for longer than 3 days, gastrostomy feeding tubes should be placed (see p. 97). Severe esophagitis should be treated with proton pump inhibitors to reduce gastric acidity. Sucralfate or viscous lidocaine may be considered if pain relief is needed. Antibiotics effective against oral anaerobes (ampicillin, amoxicillin, clindamycin; see Box 18.14) are used, and glucocorticoids may help prevent cicatrix formation (controversial).

COMPLICATIONS

Complications of foreign body removal include esophagitis, ischemic necrosis, dehiscence, leakage, infection, fistulae, esophageal diverticula, and stricture formation. Esophageal perforation may lead to mediastinitis, pleuritis, and/or pyothorax. Prolonged duration of clinical signs and increased numbers of immature neutrophils may suggest esophageal perforation; however, perforation is best diagnosed radiographically. Complications are more common in small dogs (<10 kg) and cases in which foreign material is present for more than 3 days. Dogs with foreign bodies that are removed within 24 hours or less tend to have fewer complications.

PROGNOSIS

Removal of the foreign body is essential. The prognosis is good if perforation has not occurred; however, it is guarded if perforation has resulted in either mediastinitis or pyothorax (or both). Ischemic necrosis or perforation of the esophagus may occur after foreign body removal. Leakage of saliva and ingesta into the mediastinum or pleural cavity usually causes severe inflammation, infection, and death. Severe, circumferential damage to the mucosa may produce cicatrix.

ESOPHAGEAL STRICTURES

DEFINITION

Benign esophageal strictures (*stenosis, cicatrix*) are bands of intraluminal or intramural fibrous tissue that may completely or partly obstruct the esophagus.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Benign esophageal strictures may be caused by esophagitis secondary to esophageal foreign bodies, gastroesophageal reflux during anesthesia, surgery, trauma, or caustic agents (e.g., doxycycline, acid, clindamycin, ciprofloxacin). The risk of developing a stricture after an anesthetic episode is low (probably less than 1%), whereas more than half of animals presenting with strictures have recently undergone anesthesia. Most strictures are single, but occasionally they are multiple, and most occur near the lower esophageal high-pressure zone (Box 18.18). Strictures occur more commonly after circumferential esophageal trauma. To produce a stricture, esophageal damage must generally involve the muscular layers and affect most of the circumference in a focal area. The mucosal defect is then replaced by epithelial migration. The gap in the muscle is filled by fibrous connective tissue, and wound contraction and collagen remodeling reduce the width of the scar. This leads to narrowing of the esophageal lumen, which may cause obstruction. The degree of obstruction varies, depending on the severity of the original lesion. Peristaltic waves carrying food boluses are disrupted by the obstruction. In cases of partial obstruction, part of each bolus passes the obstruction and moves down the esophagus. The other portion of the bolus accumulates proximal to the obstruction, and the proximal esophagus may

BOX 18.18 Esophageal Strictures: Key Points

- Strictures form when the mucosa is traumatized
- Suspect if there has been an anesthetic episode during the previous month or the patient has been receiving tetracycline, doxycycline, nonsteroidal antiinflammatory drugs, or ciprofloxacin
- Signs may begin immediately after injury
- Often missed by survey radiography and sometimes by contrast radiography
- Esophagoscopy is diagnostic, but inexperienced endoscopists can miss lesions in larger animals or lesions near the lower esophageal sphincter
- Strictures may be multiple or single and may be very short or several centimeters long
- Most occur near the lower esophageal high-pressure zone
- Treat for esophagitis and pneumonia if indicated
- Balloon dilatation results in a satisfactory outcome in many patients, but some patients (especially those with long strictures) often need multiple balloonings and may never respond well
- Most can eat canned or dry food after treatment
- Strictures may recur

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eventually distend. If distention occurs and is severe, it may disrupt normal neuromuscular function and reduce peristalsis. The accumulated food and secretions are frequently regurgitated.

Factors related to the development and severity of esophagitis following reflux include the duration of esophageal contact, acidity and presence of enzymes such as pepsin, effectiveness of esophageal clearance, and mucosal resistance to injury. Once esophagitis develops, a vicious circle can occur in that esophageal inflammation decreases lower esophageal high-pressure zone tone predisposing to more reflux, which exacerbates the inflammation, and so on. Gastroesophageal reflux may occur during general anesthesia when protective mechanisms are diminished and the gastroesophageal sphincter loses tone. There is no consensus on what constitutes a major risk factor for anesthesia-associated reflux with subsequent cicatrix. Gastric acid can damage the esophagus severely if not neutralized by saliva or removed by peristalsis within a few minutes. Signs of regurgitation may become evident within a few days or weeks after surgery because of stricture formation.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, breed, or sex of dog or cat may be affected.

History

Regurgitation is the most common presenting sign, and stricture should be suspected in animals experiencing frequent regurgitation with a history of previous esophageal trauma, surgery, anesthetic episode, vomiting, or treatment with various drugs (i.e., tetracyclines, NSAIDs, ciprofloxacin, clindamycin). Some animals retain fluids but regurgitate solids. Pain may be experienced when solid food becomes lodged in the stricture by forceful esophageal peristaltic waves. Regurgitation or vomiting often begins within 2 weeks of an anesthetic episode. Vomiting and regurgitation are often associated with eating, ptyalism, gagging, and dysphagia. Weight loss, coughing, and aerophagia may also be recognized.

Physical Examination Findings

Although animals with esophageal strictures may be thin and depressed, physical examination is usually normal. Occasionally the cervical esophagus is dilated. There may be putrid breath secondary to decay of material retained in the esophagus. Patients with aspiration pneumonia may be febrile and have increased respiratory rate and effort, crackles, wheezes, and increased lung sounds.

Diagnostic Imaging

Care is required to identify esophageal strictures radiographically. Positive contrast esophagrams are required (Fig. 18.54). Partial strictures are more readily identified if barium is mixed with food and observed fluoroscopically. There may or may not be dilatation of the esophagus proximal to an abrupt narrowing. It may be difficult to determine the extent of a stricture radiographically.

Esophagoscopy

Esophagoscopy allows visualization of the lesion. The mucosa may be inflamed and eroded, or it may appear relatively normal but with a compromised diameter (Fig. 18.55). Occasionally the stricture is a ring of white fibrous tissue that narrows the esophageal lumen and fails to distend with insufflation. In severe cases, it may be impossible to advance the scope beyond the stricture, and gastric overdistention can be a significant complication of esophagoscopy if the scope cannot be passed into the stomach to suction air that was insufflated into the patient. Biopsy and histologic examination rule out stricture secondary to neoplasia.

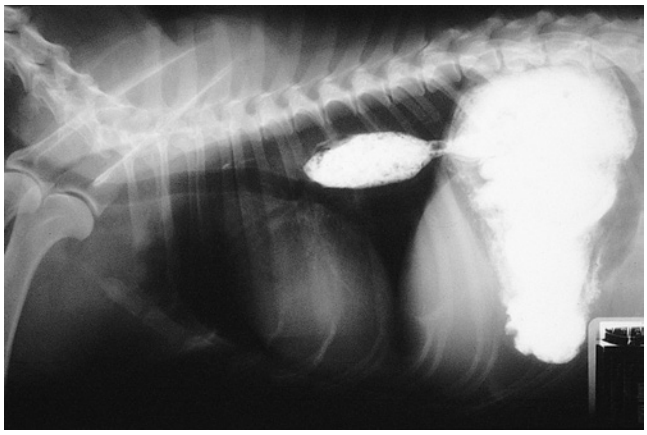


FIG. 18.54 Radiographic appearance of an esophageal stricture after the administration of barium. Notice the accumulation of contrast in the distal thoracic esophagus just cranial to the diaphragm.



FIG. 18.55 Endoscopic appearance of a distal esophageal stricture subsequent to anesthesia and surgery to remove mammary tumors.

Laboratory Findings

Animals with esophageal strictures show no specific laboratory abnormalities unless aspiration pneumonia is present.

DIFFERENTIAL DIAGNOSIS

Vascular ring anomalies, extraluminal masses, esophageal neoplasia, foreign bodies, esophagitis, gastroesophageal intussusception, esophageal diverticulum, hiatal hernias, megaesophagus,

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and cricopharyngeal dysfunction are other possible causes of regurgitation that must be differentiated from esophageal stricture.

MEDICAL TREATMENT

The goal of treatment is to resolve regurgitation and maintain nutrition and hydration by oral feeding. Treat esophagitis (see p. 366) and aspiration pneumonia (see p. 367) as previously described (see Boxes 18.13 and 18.14). Sometimes one can soften the food by adding water until it is liquid enough to pass through the obstruction. A gastrostomy tube will allow feeding if the stricture is severe. Alter the environment so foreign bodies, which may become lodged at the stricture, are avoided.

SURGICAL TREATMENT

Strictures are treated by correcting the cause and then expanding the narrowed segment with balloon catheter dilatation or bougienage. Balloon catheter dilatation is typically preferred because there is less chance of perforation, but clinicians trained in bougienage can be just as successful as those using balloons. Electrocautery or laser incision of the scar tissue at three or four equidistant sites around the circumference of the stricture, intralesional injections of repositol steroids, topical application of mitomycin C after dilatation, insertion of stents, and/or use of balloon-esophagoscopy tubes can promote successful dilatation in many patients. Partial esophagectomy is usually not necessary and may not be possible, depending on the length of the stricture. Resistant cervical strictures may be corrected by the creation of a traction diverticulum.

Preoperative Management

Animals should be fasted before esophageal dilatation. Treatment for esophagitis and aspiration pneumonia should be initiated as needed, before stricture treatment (see pp. 366–367). Dilatation of esophageal strictures is associated with bacteremia in people, and preprocedural antibiotics are recommended, although there are no data on their effect on outcome in dogs and cats.

Anesthesia

General anesthesia is required for esophageal dilatation or bougienage. Recommendations for anesthesia in patients with esophageal disorders are given on p. 367 and Table 18.3.

Surgical Anatomy

The surgical anatomy of the esophagus is discussed on p. 369.

Positioning

Balloon dilatation and bougienage are generally performed with the patient in lateral recumbency.

SURGICAL TECHNIQUE

Bougienage

Bougienage involves the dilatation of a stricture using blunt dilators that are graduated in size. A lubricated, tapered probe (dilator) is pushed through the stricture. The initial dilator is followed by a graduated series of successively larger probes until the desired lumen size is achieved or excess resistance is encountered. Some types of dilators are passed over guide wires. The esophagus should be as straight as possible during the procedure. This technique is best done under direct endoscopic visualization or with fluoroscopic guidance. Bougienage also exerts a longitudinal shearing force at the stricture site that can be more traumatic than is usual with balloon dilatation; however, success is more related to the training of the endoscopist than the type of equipment (i.e., bougienage vs. balloon dilatation) used.

> **NOTE** It is advised that you are trained in this technique before attempting it, as it is possible to perforate the esophagus if you do it improperly.

Balloon Dilatation

There are two main techniques: placing a balloon catheter through the biopsy channel of the endoscope, and running the balloon catheter down and over a previously placed guide wire.

Guide wires may be placed endoscopically or fluoroscopically. It is safer to use a guide wire than to try to advance the balloon catheter down the esophagus without a guide wire. *First, place a guide wire through the stricture site. Use a wire that is stiff at one end and floppy at the other. If an endoscope is used, insert the floppy end of the wire through the scope's biopsy channel and through the stricture. Then, withdraw the endoscope from the patient while continually feeding the wire into the patient, thus removing the endoscope from around the wire while the latter is kept in the stricture. Next, place the balloon in the stricture by running the balloon catheter over the wire while observing it endoscopically. Once the balloon is positioned so that the middle of it is near the center of the stricture, inflate the balloon with fluid or air (depending on the type of balloon) and deflate it after a minute.* If (1) the stricture is very mature and thick and the balloon cannot rupture the stricture, or (2) ballooning is typically associated with excessive trauma and reformation of the stricture, it may be advantageous to incise mature fibrotic strictures at three or four sites around the circumference of the esophagus using an electrosurgical or laser unit prior to dilatation. This hopefully causes the stricture to rupture more evenly at three or four sites as opposed to causing a major, deep rupture at just one site.

The balloon must stay in the stricture during this process. If the balloon is not correctly positioned, it will migrate out of the stricture as it is inflated and the stricture will not be dilated. Progressively larger balloons may be used until the desired degree of dilatation has been achieved. The amount of dilatation necessary is subjective and should not be overly ambitious. Most small animals will be functional if the esophageal lumen is opened up to 10 mm; larger animals may need to be opened up to 15 or even 20 mm. Repeat the dilatation as often as necessary to attain a satisfactory outcome. Many animals only require 1 dilation, but some require 15 or more. If multiple dilations are required, they are usually done two to three times weekly until the stricture no longer reforms.

> **NOTE** Cause as little trauma as possible during dilation to lessen the likelihood of stricture reformation. Try not to tear off or aspirate any more esophageal mucosa than is absolutely necessary.

To inhibit scarring and reformation of the stricture postdilatation, one may topically apply mitomycin C. *Reconstitute 5 mg*

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of mitomycin C in 10 mL of sterile water. Soak a gauze sponge in this solution, grasp it with endoscopic forceps that have already been placed through the endoscope, and then pass the scope, forceps, and sponge into the esophagus, taking care to minimize application of the mitomycin to other parts of the esophagus. Leave the gauze that is soaked in the mitomycin for 5 minutes on the sites where the mucosa is torn and on the submucosa. Then remove the sponge and wash the site with 50 mL of sterile water, which is then aspirated by the endoscope. Follow routine precautions for chemotherapeutic drugs.

Another technique is to inject repositol steroids (e.g., triamcinolone acetate). A needle catheter designed to be placed through the biopsy channel of the endoscope is used to place a total dose of 1 to 2 mL of the steroid in three to four spots around the circumference of the site. The steroids may be injected before or after dilatation. The author prefers to inject after the dilatation procedure.

The goal is to open the stricture just enough so that the patient is functional and can eat soft food, not just gruel. It is often not possible to completely eliminate the stricture. These patients are often at risk for esophageal foreign bodies at the site of the stricture, as usually some degree of lumen compromise remains.

Covered, nonbiodegradable stents have been used successfully in some dogs. Biodegradable, metallic, and plastic stents have been described in the treatment of refractory canine esophageal strictures.[10] Stenting followed attempts at treating with balloon dilatation and included polydioxanone, polyurethane, and nitinol stents. The majority were sutured in place to prevent migration, and complications included ptyalism, regurgitation, dysphagia, vomiting, megaesophagus, gagging, and stent migration in the short term. Despite improvement in dysphagia score, complications long term included restructure following stent dissolution, migration, vomiting, regurgitation, hyperplastic tissue growth, and tracheoesophageal fistula formation. Half of the dogs were euthanized due to stent-related complications. Failure of dilatory therapy may necessitate esophageal resection or replacement. A relatively new technique involves placing an esophagostomy tube that also has a balloon at the distal end. After initial dilation of the stricture, the tube is placed much like any esophagostomy tube but with care taken to ensure that the balloon is present in the stricture. The dog is then sent home, and the owner inflates the balloon once or twice daily for 4 to 6 weeks. Care must be taken to select clients that are able to perform the needed dilations and care for the tube.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

When dilating an esophageal stricture, balloons specifically designed for use in the esophagus should be used (e.g., MaxForce TTS High Performance Balloon Dilatation Catheter). Electrocautery snares and injection needles for use through endoscopes are available (e.g., Olympus Corp.). Stents custom built for specific animals may be ordered (e.g., Infinity Corp.). An electrosurgical unit adaptable to laparoscopic sites or a laser unit is advantageous with mature fibrotic strictures.

POSTOPERATIVE CARE AND ASSESSMENT

Continue to monitor for regurgitation and aspiration pneumonia. Superficial mucosal tears are expected following dilation. However, patients should be monitored closely for signs of perforation, which include dysphagia, subcutaneous emphysema, pneumothorax, pneumomediastinum, and mediastinitis. Nasal oxygen may benefit these animals postoperatively, and analgesics (see Chapter 13) should be provided if necessary (esophageal lesions are surprisingly painful). If perforation is suspected, one should verify with thoracic radiographs. A small rupture may be treated by placing a gastrostomy tube and administering antibiotics. A major rupture will require surgical closure. Monitor for hemorrhage, which, although rare, can require transfusion. Diverticulum formation is another complication that may develop (see later discussion).

If the procedure has caused a substantial mucosal tear, therapy for esophagitis (antacids, prokinetics, sucralfate, antibiotics) may be administered (see Boxes 18.13 and 18.14). Antibiotic therapy intuitively makes sense, but there are no data showing that it has an effect on outcome. Glucocorticoid (e.g., prednisolone, 1.1 mg/kg given orally once a day) may be administered to help prevent stricture reformation, but the effect of systemic treatment on outcome is unknown, whereas some data suggest intralesional steroids are effective. Preexisting esophagitis and aspiration pneumonia should be treated as described on pp. 366 to 367. Placement of a gastrostomy tube (see p. 97) may be beneficial in these patients so that oral feeding can be avoided for 7 to 10 days but the patient can be kept in a positive nitrogen balance during healing. When oral feeding is resumed, feed small, frequent meals of a soft (not gruel) consistency.

PROGNOSIS

Most patients (approximately 85%) with esophageal strictures can be helped by dilatation, but strictures may reform. Most are able to tolerate canned or dry food without regurgitation, but some require gruel. Thin stricture bands may require only a single dilatation; however, patients with severe or long strictures often require multiple dilatations. There is a more guarded prognosis if strictures are several centimeters long; if there is dense, thick, mature fibrous tissue; or if severe esophagitis is persistent. Prognosis is worse if perforation occurs. Resection of long strictures may result in dehiscence caused by excessive anastomotic tension. Bougienage is commonly associated with a good outcome in a majority of dogs and cats, but multiple sessions are typically required and one-quarter to one-third of animals treated with this technique may have a poor outcome.

ESOPHAGEAL DIVERTICULA

DEFINITIONS

Esophageal diverticula are saclike dilatations that produce pouches in the wall of the esophagus. A **pulsion diverticulum** is a herniation of the mucosa through the muscular layers of the esophagus. These diverticula are produced by exaggerated intraluminal pressure in association with abnormal regional peristalsis or when obstruction interferes with normal peristalsis. **Traction diverticula** are distortions, angulations, or funnel-shaped bulges of the full-thickness wall of the esophagus caused by adhesions resulting from an external lesion.

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GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Esophageal diverticula are rare. They may be acquired or congenital and are most commonly found in the distal cervical esophagus cranial to the thoracic inlet or in the distal thoracic esophagus just cranial to the diaphragm (epiphrenic). *Congenital diverticula* are believed to develop as a result of a congenital weakness of the esophageal wall, abnormal separation of tracheal and esophageal embryonic buds, or eccentric vacuole formation in the esophagus. Acquired forms are classified as pulsion or traction diverticula based on their cause. *Pulsion diverticula* are most common in the epiphrenic area but can form cranial to any diseased esophageal segment. Many conditions may initiate the formation of diverticula, including esophagitis, esophageal stenosis, foreign bodies, vascular ring anomalies, neuromuscular dysfunction, and hiatal hernias. The esophageal mucosa herniates secondary to increased intraluminal pressure, accumulation of food, and esophageal inflammation. The wall of a pulsion diverticulum consists of only esophageal epithelium and connective tissue.

Traction diverticula occur after an inflammatory process involving the trachea, bronchi, lymph nodes, or other extraesophageal structures. Inflammation causes fibrous tissue to form between the esophagus and the diseased structure. As the fibrous tissue matures, it contracts and pulls an area of the esophagus outward to form a pouch. Most traction diverticula occur in the cranial and midthoracic esophagus. The wall of a traction diverticulum consists of adventitia, muscle, submucosa, and mucosa.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, breed, or sex of dog or cat may be affected.

History

Small diverticula can be asymptomatic. Large, multilobulated diverticula are usually associated with clinical signs. Diverticula may result in esophageal impaction, chronic esophagitis, and rupture of the diverticulum wall, with resultant mediastinitis or the formation of an esophagotracheal-bronchial fistula. Clinical signs may include distress or gasping after eating, postprandial regurgitation, intermittent anorexia, fever, weight loss, thoracic or abdominal pain, and respiratory distress.

Physical Examination Findings

The physical examination is normal if the diverticulum is asymptomatic. Abnormal lung sounds may be auscultated if aspiration pneumonia has developed.

Diagnostic Imaging

Contrast radiographs are required. Be sure not to confuse the normal esophageal redundancy, seen in brachycephalic breeds and shar peis, with esophageal pathology. Diverticula appear as air-filled or food-filled masses in the area of the esophagus. An esophagram usually demonstrates a deviation or outpouching of the esophageal lumen that fills partly or completely with contrast material. Esophagoscopy is helpful in confirming the radiographic diagnosis and identifying associated esophagitis, strictures, or other abnormalities. The esophageal wall may be very thin, and esophagoscopy must be performed with care.

NOTE Dogs and cats with generalized megaesophagus tend to have greater outpouching of the esophageal wall cranial to the base of the heart. Brachycephalic dogs and shar peis often have a loop of redundant esophagus cranial to the base of the heart. Do not misdiagnose these animals as having diverticula.

Laboratory Findings

Esophageal diverticula do not produce any specific laboratory abnormalities. Laboratory findings consistent with pyothorax (see p. 949) may result if the diverticulum has ruptured. Neutrophilia may be present if aspiration pneumonia has developed.

DIFFERENTIAL DIAGNOSIS

Esophageal hiatal hernia, gastroesophageal intussusception, stricture, neoplasia, extraluminal masses, vascular ring anomalies, esophageal foreign body, esophagitis, and megaesophagus are other possible causes of regurgitation.

SURGICAL TREATMENT

Persistent underlying causes of the diverticula must be identified and treated. Asymptomatic, small diverticula may be treated by feeding a soft, bland diet with the animal in an upright position to avoid food accumulation in the pouch. Large diverticula should be surgically excised.

Preoperative Management

Esophagitis and aspiration pneumonia should be treated preoperatively as described on pp. 366 to 367. Prophylactic antibiotics are indicated if esophageal resection is considered likely.

Anesthesia

Anesthetic recommendations for animals undergoing cervical esophageal surgery are discussed on p. 367 and in Table 18.3. Anesthetic recommendations for thoracotomy are given on p. 884, Table 29.1, and Table 30.3.

Surgical Anatomy

The surgical anatomy of the esophagus is described on p. 369.

Positioning

Patients are positioned in dorsal or lateral recumbency, depending on the site of the diverticulum. Radiographs are important to help determine the best surgical approach. Diverticula in the cervical esophagus are approached via a ventral cervical midline incision with the animal in dorsal recumbency. Thoracic diverticula are usually approached via a lateral thoracotomy. Occasionally a median sternotomy or thoracic wall flap may be necessary to approach diverticula at the thoracic inlet or cranial mediastinum.

SURGICAL TECHNIQUE

After identifying the diverticulum, isolate it from surrounding structures with blunt and sharp dissection and then pack it off with laparotomy pads. Partial lung lobectomy may be necessary if adhesions cannot be easily separated. *Position a thoracoabdominal (TA; linear) or GIA (gastrointestinal anastomosis) stapling device along the base of the diverticulum and fire. Transect and*

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remove the diverticulum without contaminating the surgical site. If stapling equipment is not available, suction the esophageal lumen and place noncrushing forceps across the proposed transection site. Transect the diverticulum and appose the edges as for esophagotomy with a one- or two-layer simple appositional pattern (see p. 370). Lavage the surgical site and, if possible, mobilize and place omentum over the incision. Thoracostomy tubes may be placed in animals having thoracotomies to evacuate residual air and fluid.

POSTOPERATIVE CARE AND ASSESSMENT

After surgery, these patients should be monitored for esophagitis and aspiration pneumonia and treated appropriately (see pp. 366–367). Postoperative esophagoscopy and esophagrams may be indicated if problems are detected. Regurgitation that occurs secondary to persistent esophagitis is a possible problem. Infection as a result of contamination of the surrounding tissues during surgery, dehiscence, or leakage at the surgical site are other possible problems. Postoperative analgesics should be provided to control pain (see p. 886 for recommendations after thoracotomy).

PROGNOSIS

Asymptomatic patients often continue to do well without surgery; feeding them in an upright position to avoid food accumulation in the diverticula may help. Clinical signs may be difficult to control in symptomatic patients with medical therapy alone. If surgery is not possible, such patients should be treated for esophagitis and fed soft food in an upright position. The prognosis with surgical correction is good if thoracic contamination is avoided and good esophageal apposition is achieved.

ESOPHAGEAL NEOPLASIA

DEFINITION

Esophageal neoplasia is any abnormal, noninflammatory proliferation of cells in the esophagus.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Esophageal neoplasia is rare and usually malignant. Tumors are often locally invasive and metastasize through lymphatic and hematogenous routes. The most common types of tumors are sarcomas, SCCs, and leiomyomas. Leiomyomas have been reported at the lower esophageal high-pressure zone in aged beagles. These tumors are usually advanced by the time clinical signs are recognized. Primary esophageal carcinomas are of unknown etiology. Primary esophageal sarcomas (osteosarcoma, fibrosarcoma) are often located in the vicinity of parasitic granulomas caused by *Spirocerca lupi*. The life cycle of *S. lupi* involves a coprophagous beetle that the dog has eaten or a transport host that the dog subsequently eats. Tumors from the thyroid, thymus, heart base, or lung may invade the esophagus secondarily.

Esophageal tumors initially cause partial obstruction of the esophagus, which may interfere with motility and lead to dilatation of the proximal esophagus. As the tumor enlarges, signs of complete esophageal obstruction become apparent. Most esophageal tumors are locally invasive and metastasize to draining lymph nodes.

DIAGNOSIS

Clinical Presentation

Signalment

In cats, SCCs are usually seen in females in the middle third of the esophagus just caudal to the thoracic inlet. Most esophageal tumors occur in dogs and cats over 6 to 8 years of age.

History

Chronic progressive signs of obstructive esophageal disease in middle-aged and older animals suggest esophageal neoplasia. Animals with primary tumors may be asymptomatic until the mass becomes large enough to cause near complete obstruction. These animals may be presented for regurgitation, lethargy, depression, drooling, dysphagia, anorexia, weight loss, and/or fetid breath. In animals with secondary tumors, clinical signs may include regurgitation, dyspnea, palpable masses, and/or systemic and local tumor effects.

Physical Examination Findings

The physical examination is usually normal. Some animals are thin, and a dilated cervical esophagus may be identified. Hypertrophic osteopathy and spondylosis deformans may be noted, especially with *S. lupi*–induced sarcomas. Some will have pyrexia, submandibular swelling, pale mucous membranes, melena, salivation, cough, neurologic signs, and weight loss. Crackles from aspiration pneumonia may be detected.

Diagnostic Imaging

Aerophagia (swallowing of air), displacement of the esophagus, and megaesophagus may be signs of esophageal neoplasia. Survey thoracic radiographs may be normal or may reveal a soft tissue opacity in the plane of the esophagus or mediastinum (Fig. 18.56). This density may be impossible to distinguish from esophageal foreign body in some cases. The esophagus may retain air cranial to the tumor, or megaesophagus may be present.[11] The lungs should be evaluated for metastatic lesions. Contrast esophagrams may demonstrate an intraluminal mass (mucosal irregularities, filling defects, or stricture) with primary tumors, or an impinging

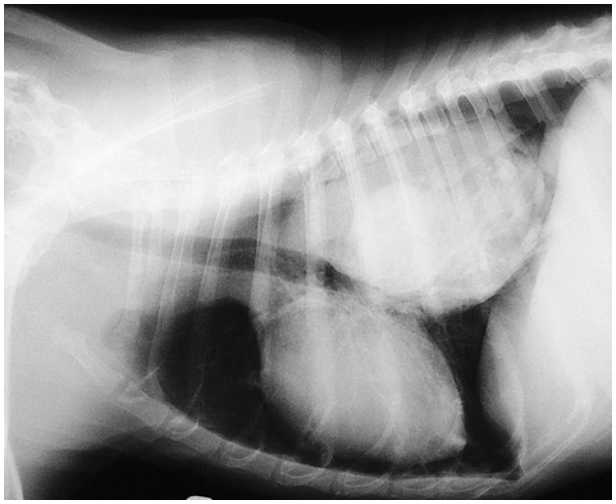


FIG. 18.56 Lateral thoracic radiograph of an 8-year-old dog with a large esophageal carcinoma. Notice the soft tissue opacity mass in the plane of the distal thoracic esophagus.

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FIG. 18.57 Appearance of an intraluminal mass on esophagoscopy. Esophagoscopy allows biopsy of such masses.

mural or extraluminal mass with smooth mucosal margins and no filling defect with secondary tumors. Fluoroscopic studies may reveal abnormal motility. Signs of spondylosis and spondylitis may be detected at the ventral aspect of the cervical and thoracic vertebrae. Dogs with *S. lupi* infestation commonly have spondylosis deformans or spondylitis of the cervical or thoracic vertebrae with or without hypertrophic osteopathy. CT scans may help determine the degree of esophageal wall involvement. Esophagoscopy allows direct visualization of intraluminal masses and biopsy for definitive diagnosis (Fig. 18.57). An adult *S. lupi* worm is occasionally seen protruding into the lumen from the mass. Identification of a granuloma with a nipple-like orifice is characteristic of spirocercosis. Extraluminal masses cannot be visualized unless they have eroded into the lumen. It is difficult to inflate the lumen if it is impinged on or has been invaded by extraluminal masses.

Laboratory Findings

Laboratory values may indicate chronic disease or paraneoplastic syndromes. Animals with spirocercosis-associated sarcomas may have neutrophilic leukocytosis (82%) and microcytic anemia (30%). *S. lupi* eggs may be detected on a fecal sedimentation test but are usually difficult to find. A histologic diagnosis of esophageal sarcoma in addition to detecting *S. lupi* eggs in feces, esophageal nodular granulomas, or spondylitis of the caudal thoracic vertebrae is considered diagnostic of spirocercosis-associated sarcomas.

DIFFERENTIAL DIAGNOSIS

Esophageal stricture, extraluminal masses, vascular ring anomalies, esophageal foreign body, esophagitis, gastroesophageal intussusception, esophageal diverticulum, hiatal hernia, and megaesophagus are other possible causes of regurgitation. The clinical signs of ptyalism, odynophagia, and sialadenopathy associated with *Spirocerca* lesions appear similar to the clinical signs in dogs with salivary gland necrosis and necrotizing sialometaplasia syndrome.

SURGICAL TREATMENT

Early diagnosis is important, before metastasis or extensive esophageal involvement has occurred. Partial esophagectomy with end-to-end anastomosis is indicated when approximation can be accomplished without excess tension. Endoscopic mass ablation has been described in a series of dogs.[12] Complications included esophageal perforation, excess mucosal resection, and esophageal hemorrhage. Short-term treatment of an esophageal tumor with a metallic stent has also been described.[13]

Preoperative Management

S. lupi–associated esophageal granulomas often resolve after treatment with doramectin (200 mg/kg given subcutaneously at 14-day intervals for three treatments). Complete resolution of the esophageal nodules is possible, and adverse drug effects are not expected. Chemotherapy, radiation therapy, or photodynamic therapy may be beneficial in some animals.

Anesthesia

Anesthetic recommendations for animals undergoing cervical esophageal surgery are provided on p. 367 and in Table 18.3. Selected anesthetic protocols for animals undergoing thoracic surgery are provided on p. 884, Table 29.1, and Table 30.3.

Surgical Anatomy

The surgical anatomy of the esophagus is described on p. 369.

SURGICAL TECHNIQUE

Esophagectomy is described on p. 371. Refer to p. 375 for a description of suture material and special instruments for esophageal surgery.

POSTOPERATIVE CARE AND ASSESSMENT

The patient should be monitored for esophagitis and aspiration pneumonia and treated as needed postoperatively (see pp. 366–367). Nasal oxygen may benefit these animals, and analgesics should be provided to control pain. See p. 375 for recommendations for postoperative care of animals undergoing esophageal surgery. Dehiscence caused by excessive tension at the anastomosis and tumor recurrence are possible complications of esophageal surgery. Continue doramectin treatment in animals with spirocercosis.

PROGNOSIS

Most esophageal tumors are advanced at the time of diagnosis and do not respond well to radiation therapy or chemotherapy. Radiation therapy may be palliative, but radiation-induced esophagitis or damage to the heart, lungs, and great vessels are possible sequelae that are poorly tolerated. Photodynamic therapy may palliate some nonresectable tumors. Resection may be curative for leiomyomas, but the prognosis for other malignancies is guarded for cure or palliation because resection is difficult due to the advanced nature of most tumors at the time of detection.

HIATAL HERNIA

DEFINITION

Hiatal hernias are protrusions of the abdominal esophagus, gastroesophageal junction, and sometimes a portion of the gastric fundus through the esophageal hiatus into the caudal mediastinum cranial to the diaphragm.

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BOX 18.19 Hiatal Hernia: Key Points

- Most are congenital rather than acquired
- Shar peis and English bulldogs are most commonly affected
- Some are asymptomatic
- Symptomatic animals often have regurgitation and esophagitis
- Herniation may be intermittent, making radiographic diagnosis challenging
- Herniorrhaphy includes hiatal reduction, esophagopexy, and left-sided gastropexy
- Surgery is usually most appropriate for younger symptomatic animals; older symptomatic animals are usually best treated medically
- Treat for esophagitis initially after surgery
- Prognosis is usually good after repair

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Hiatal hernias usually are caused by congenital abnormalities of the hiatus that allow cranial movement of the abdominal esophagus and stomach (Box 18.19). The phrenicoesophageal ligament is lax or stretched and allows the gastroesophageal junction to be displaced through the hiatus into the caudal mediastinum. Malpositioning or lack of support of the gastroesophageal sphincter reduces gastroesophageal sphincter pressure and leads to gastroesophageal reflux. Gastroesophageal reflux and subsequent esophagitis and megaesophagus are responsible for most of the clinical signs. Hiatal hernia occasionally occurs secondary to trauma and has occurred concurrently with severe respiratory distress. Trauma may damage diaphragmatic nerves and muscles, resulting in hiatal laxity and subsequent herniation. In patients with upper respiratory obstruction, reduced intrathoracic pressure during inspiration has been theorized to contribute to esophageal reflux and visceral herniation. Hiatal hernia has been reported with tetanus.

With hiatal hernias, the stomach commonly slides in and out of the thorax. If the hernia is large enough, other abdominal viscera may also be cranially displaced into the thorax. Various types of hiatal abnormalities have been described (Fig. 18.58). In patients with sliding or axial hiatal hernias, the gastroesophageal junction is located within the thoracic cavity. In patients with paraesophageal or rolling hiatal hernias, the gastroesophageal junction is usually located in a normal position, and the gastric fundus or other abdominal viscera are displaced through the hiatus and located in the thorax. Some hiatal hernias are a combination of sliding and paraesophageal hernias, with the gastroesophageal junction and gastric fundus both displaced.

DIAGNOSIS

Clinical Presentation

Signalment

Hiatal hernias may occur in a variety of dog and cat breeds; however, males, Shar Peis, and brachycephalic dogs appear to be predisposed. Hiatal hernia was diagnosed in 76% of golden retrievers with muscular dystrophy[14] and in one-third of cats evaluated for dysphagia.[15] Most symptomatic animals have signs relating to congenital hiatal hernia before reaching 1 year of age, although diagnosis may occur later. Patients with acquired hernias may develop signs at any age.

History

Regurgitation is the primary clinical sign in symptomatic individuals, but many patients are asymptomatic. Other signs may include vomiting, hypersalivation, dysphagia, respiratory distress, hematemesis, anorexia, and weight loss. Severe dyspnea or trauma may have occurred.

Physical Examination Findings

Affected patients may be thin on physical examination. Abnormal lung sounds are ausculted with aspiration pneumonia.

Diagnostic Imaging

Hiatal hernias usually appear as a soft tissue or soft tissue/gas–filled mass near the esophageal hiatus in the caudodorsal thoracic region on survey radiographs (Fig. 18.59). However, with sliding hernias, several radiographs may be necessary to identify the herniation, which may be intermittent. Intermittent hernias are particularly common in cats. The presence of gas in the herniated portion aids in identification of the mass as herniated stomach. Varying degrees of megaesophagus and pneumonia may be noted. A positive contrast esophagram should show the gastroesophageal junction, rugal folds, or both, cranial to the hiatus. Occasionally, strictures may be identified. Fluoroscopy may demonstrate hypomotility, delayed clearing of the distal esophagus, or gastroesophageal reflux. Compressing the abdomen during fluoroscopy may help identify hernias. Esophagoscopy can sometimes detect the hernia (i.e., gastric mucosa that has entered the thoracic cavity), as well as esophagitis (inflammation, mucosal erosion), gastric reflux, and strictures. Some hiatal hernias are intermittent (sliding) and require multiple radiographs or fluoroscopy (or both) to diagnose. Do not confuse hiatal hernias with peritoneopericardial (see p. 931) or traumatic (see p. 926) diaphragmatic hernias, despite their sometimes having a similar radiographic appearance. Hiatal hernias are typically located in the plane of the caudal mediastinum.

Laboratory Findings

Hematologic and serum chemistry results are nonspecific in affected animals.

DIFFERENTIAL DIAGNOSIS

Esophageal stricture, neoplasia, extraluminal masses, vascular ring anomalies, esophageal foreign body or perforation, esophagitis, esophageal intussusception, esophageal diverticulum, and megaesophagus are other possible causes of regurgitation.

SURGICAL TREATMENT

Affected patients may benefit from medical treatment for gastroesophageal reflux or esophagitis (see p. 366) because hiatal hernias may be "innocent bystanders" in a patient with symptoms resulting from other problems. Surgery is generally recommended in symptomatic, young animals with congenital disease that does not respond to appropriate medical treatment. A number of surgical techniques have been described for correcting hiatal hernias. Diaphragmatic hiatal reduction and plication, esophagopexy, and left-sided fundic gastropexy are described here. Gastropexy is probably the most important step in the repair. Displacing the terminal esophagus slightly caudally with a left-sided gastropexy increases barrier pressure at the gastroesophageal

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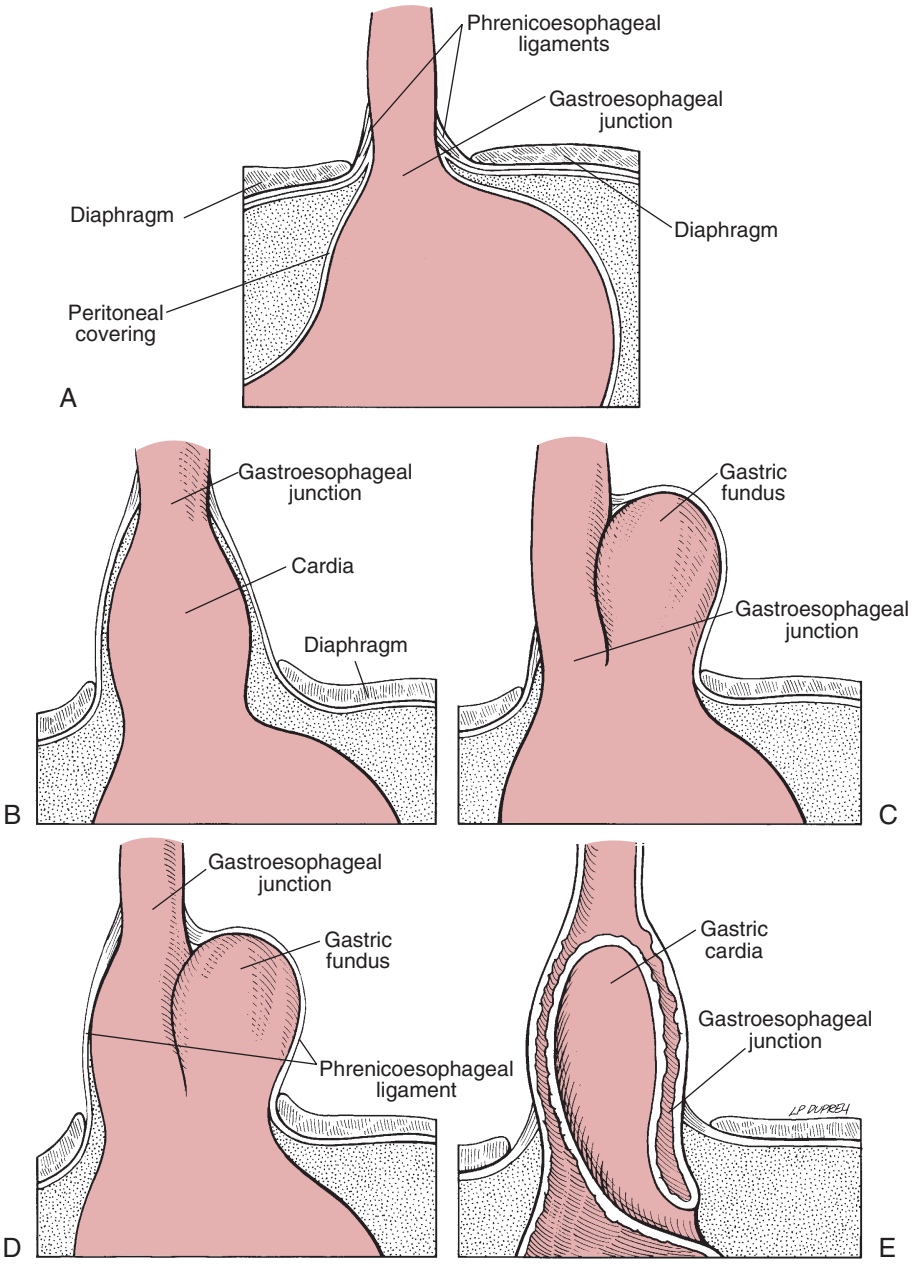


FIG. 18.58 Diagrams of a (A) normal gastroesophageal junction and (B–E) hiatal abnormalities. (B) Sliding or axial hiatal hernia. (C) Paraesophageal or rolling hiatal hernia. (D) Combination sliding and paraesophageal hernia. (E) Gastroesophageal intussusception.

junction, and reflux disease often resolves after the procedure. If esophagitis is severe and oral intake is to be withheld for several days, a gastrostomy tube (see p. 97) allows early alimentation without further esophageal irritation. Some surgeons perform sphincter-enhancing procedures, such as a Nissen fundoplication (antireflux procedure), instead of the aforementioned techniques. However, fundoplication or other antireflux procedures are only indicated in patients with evidence of gastroesophageal reflux. In dogs and cats, primary incompetence of the caudal esophageal sphincter has not been documented in association with hiatal hernia; therefore antireflux procedures are not routinely recommended.

Preoperative Management

Reflux esophagitis and aspiration pneumonia should be treated before induction of anesthesia (see pp. 366–367). Feeding frequent, small meals of high-protein/low-fat foods may be beneficial. If megaesophagus is present, feeding affected animals in a standing, upright position may reduce regurgitation.

Anesthesia

Positive-pressure ventilation may be necessary if pneumothorax is created during hiatal manipulations. Nitrous oxide should not be used in these patients. Negative intrathoracic pressure is reestablished by thoracentesis or tube thoracostomy after hiatal

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manipulations are complete. See p. 367 and Table 18.3 for anesthetic recommendations for patients undergoing esophageal surgery and p. 884, Table 29.1, and Table 30.3 for recommendations for patients undergoing thoracic surgery.

Surgical Anatomy

The esophageal hiatus is one of three openings in the diaphragm. The esophageal hiatus is more centrally located than the caval foramen (located ventrally) or aortic hiatus (located dorsally). The esophagus passes through the esophageal hiatus, along with the vagal nerve trunks and esophageal vessels. The esophageal hiatus is surrounded by the phrenicoesophageal ligament, the thickened collagen fibers of which are weakened, stretched, or in some way defective in hiatal hernias. The terminal 1 to 2 cm of the esophagus is expected to lie within the abdominal cavity caudal to the diaphragm. The esophagogastric junction and gastroesophageal sphincter, which are in the abdomen, regulate movement of ingesta between the esophagus and the stomach.

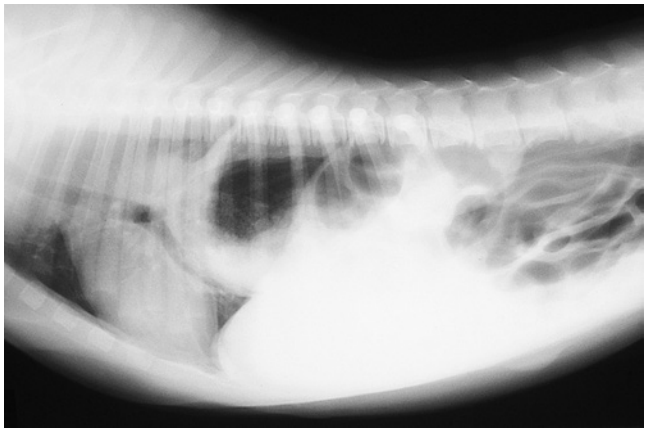


FIG. 18.59 Lateral radiograph of a 12-week-old shar pei with a hiatal hernia. Note the air-filled mass (stomach) in the caudal thorax.

Positioning

Patients are positioned in dorsal recumbency, and the caudal thorax and ventral abdomen are prepared for aseptic surgery.

SURGICAL TECHNIQUE

Make a cranial ventral midline incision extending caudal to the umbilicus to expose the diaphragm and stomach. Retract the left lobes of the liver medially to expose the esophageal hiatus. Pass a stomach tube (28- to 32-Fr) to help identify and manipulate the esophagus. Grasp the stomach and reduce the hernia with gentle traction. Examine the hiatus. Dissect the phrenicoesophageal membrane, freeing the esophagus from the diaphragm ventrally. Preserve the vagal trunks and esophageal vessels during dissection. Place an umbilical tape sling around the abdominal esophagus to displace it caudally and facilitate manipulations. Perform a diaphragmatic hiatal plication-reduction, esophagopexy, and left-sided fundic gastropexy. Accomplish diaphragmatic hiatal plication-reduction by excoriating or debriding the margins of the hiatus and then place three to five sutures (2-0 polydioxanone or polypropylene) to appose the edges and narrow the hiatus (Fig. 18.60).

Perform plication around a large stomach tube (28- to 32-Fr) (see Fig. 18.60). Reduce the hiatus to 1 or 2 cm, a size that allows passage of one finger. Accomplish esophagopexy by placing sutures (3-0 or 2-0 polydioxanone or polypropylene) from the remaining margin of the hiatus through the adventitia and muscular layers of the abdominal esophagus. Complete the repair with either a left-sided tube gastropexy or incisional gastropexy (see pp. 406–408). Fix the fundus with slight to moderate caudal traction to prevent cranial movement of the gastroesophageal junction into the thorax. Evacuate air from the chest by thoracentesis or tube thoracostomy and lavage and close the abdomen.

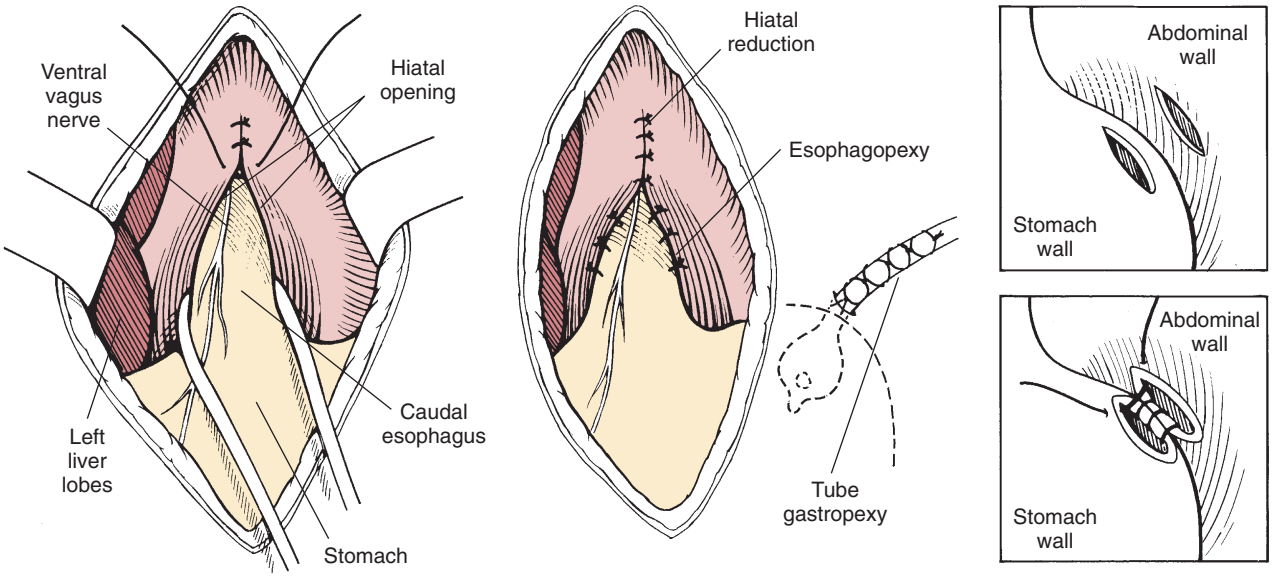


FIG. 18.60 Reduce hiatal hernias and reduce the size of the esophageal hiatus with plicating sutures. Suture the esophagus to the diaphragm (esophagopexy) and perform a tube or incisional gastropexy *(insets)* at the fundus.

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POSTOPERATIVE CARE AND ASSESSMENT

Patients should be monitored after surgery for dyspnea caused by pneumothorax, and air should be evacuated from the thorax as necessary. Nasal oxygen may benefit dyspneic animals. Analgesics should be provided as necessary to control pain (see Table 13.2). Affected animals may continue to regurgitate after surgery because of persistent esophagitis or reflux or if the repair caused obstruction at the lower esophageal high-pressure zone. Treatment of esophagitis and aspiration pneumonia should be continued postoperatively (see pp. 366–367). Feed small portions of low-fat, high-protein, softened or liquefied food three to five times a day. Feeding from an elevated platform may be beneficial in animals with concurrent esophageal weakness. Postoperative esophagoscopy may benefit patients with persistent clinical signs by identifying persistent herniation, obstruction, or ulceration.

COMPLICATIONS

Dysphagia is common for several days; however, if it continues beyond that time, the hiatus may have been overreduced, requiring reoperation. Infection may occur if the esophageal or gastric lumen is penetrated with sutures or tubes. Possible problems after antireflux procedures include gastric dilatation, necrotic gastritis, and acute death.

PROGNOSIS

The prognosis without surgery is good in asymptomatic patients and those that respond to medical therapy; however, symptomatic patients that do not respond to medical therapy and are not surgically repaired may develop severe esophagitis and stricture. The prognosis is good; however, aspiration pneumonia must be controlled for a favorable outcome. Patients with gastroesophageal sphincter incompetence may benefit from additional antireflux procedures.

GASTROESOPHAGEAL INTUSSUSCEPTION

DEFINITION

Gastroesophageal intussusception (*esophageal intussusception, gastroesophageal invagination, esophageal invagination*) is the invagination of the gastric cardia into the distal esophagus with or without the spleen, duodenum, pancreas, and omentum.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Gastroesophageal intussusception is a rare condition that can be confused with esophageal hiatal hernia (see Fig. 18.58E). Most reports in the literature are of solitary cases. The gastroesophageal junction does not move cranially into the thorax as with a sliding hiatal hernia, and the cardia is within the esophageal lumen rather than external to the esophagus as with a paraesophageal hiatal hernia. Gastroesophageal intussusception usually occurs in immature animals with megaesophagus. The etiology of gastroesophageal intussusception is unknown. Idiopathic megaesophagus or incompetency of the gastroesophageal sphincter mechanism or abnormal esophageal motility and subsequent regurgitation may predispose an animal to this disorder. Vomiting and retching cause the esophagus to dilate. Experimental work in puppies has shown that vomiting can cause invagination of the gastric cardia into the esophagus. A large or lax esophageal hiatus may be necessary to allow the gastric cardia to move cranially into the esophagus. Entrapment or strangulation of the invaginated stomach occurs, causing esophageal obstruction, continued regurgitation, and rapid fluid loss. Discomfort is caused by stretching of gastric mesenteric attachments and esophagitis. Severe respiratory distress is caused by the greatly enlarged esophagus compressing the pulmonary parenchyma or by aspiration pneumonia (or both). Cardiovascular collapse occurs secondary to obstruction of venous return and eventually of the arterial blood supply. The resulting congestion, inflammation, and necrosis contribute to the animal's deterioration. The condition is even rarer in cats, but has been seen in conjunction with megaesophagus.

DIAGNOSIS

Clinical Presentation

Signalment

Although several breeds have been reported with gastroesophageal intussusception, German shepherds and other large-breed dogs seem to be at increased risk. More cases have been reported in males than in females. The condition is most common in young dogs, usually under 3 months of age. Gastroesophageal intussusception has been reported in cats.

History

In most cases there is acute onset of clinical signs (e.g., regurgitation, vomiting, dyspnea, hematemesis, abdominal discomfort, rapid deterioration, death) with rapid deterioration and death within 1 to 3 days if the condition is not treated immediately. Signs may mimic those of aspiration pneumonia, making the diagnosis difficult. Approximately half of affected animals have a history of esophageal disease, and a few have been reported with chronic, intermittent clinical signs including recurrent vomiting and regurgitation and lethargy. These chronic cases may be secondary to a sliding intussusception.

Physical Examination Findings

Animals may be thin and show pain on abdominal palpation. Signs of shock may be noted (i.e., poor capillary refill, pale mucous membranes, labored breathing, tachycardia, and thready pulse).

Diagnostic Imaging

Radiographs show a dilated distal esophagus with a luminal soft tissue mass (Fig. 18.61). Rugal folds may be associated with the soft tissue mass. The trachea may be deviated ventrally, and signs of aspiration pneumonia may be apparent. The normal gastric gas bubble usually seen in the cranial abdomen may be absent or diminished in size. The intussusception can be outlined by either positive or negative contrast studies; however, this may be difficult to perform, as these patients are often severely dyspneic. Esophagoscopy reveals a dilated esophagus with a large soft tissue mass having gastric rugal folds in the distal esophageal lumen. Esophagitis may be apparent. It may not be possible to advance the endoscope into the distal esophagus or stomach.

Laboratory Findings

Laboratory findings are nonspecific and related to dehydration and shock.

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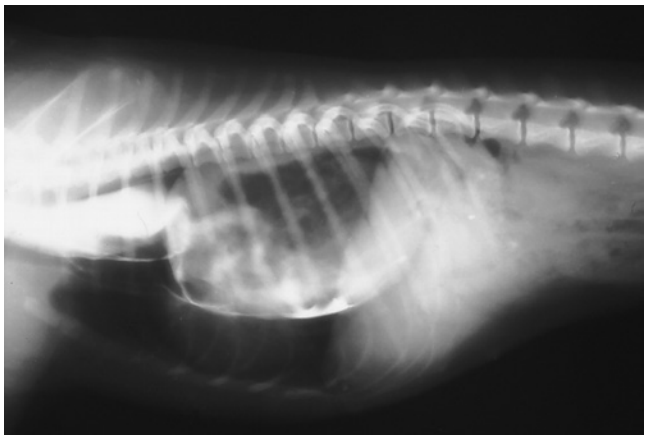


FIG. 18.61 Radiograph of a dog with a gastroesophageal intussusception. Note the luminal soft tissue mass in the dilated distal esophagus.

DIFFERENTIAL DIAGNOSIS

Esophageal hiatal hernia, neoplasia, extraluminal masses, esophageal foreign body, and esophageal diverticulum are differential diagnoses.

SURGICAL TREATMENT

Surgical intervention should be performed as soon as possible after diagnosis. Stabilization of the patient's condition and treatment for shock should be performed before induction of anesthesia; however, surgical treatment should not be delayed in acute cases. A single report of endoscopic reduction and treatment with a percutaneous endoscopic gastrostomy tube has been reported in one dog.[16]

Preoperative Management

Before induction of anesthesia, shock treatment should be initiated (i.e., aggressive fluid therapy and broad-spectrum antibiotics), and electrolyte and acid-base abnormalities should be identified and corrected if possible. An ECG also should be performed before the induction of anesthesia.

Anesthesia

Anesthetic recommendations for animals undergoing esophageal surgery are provided on p. 367 and in Table 18.3.

Surgical Anatomy

The surgical anatomy of the esophageal hiatus is discussed on p. 369.

Positioning

The animal is positioned in dorsal recumbency, and the caudal thorax and ventral abdomen are prepared for aseptic surgery.

SURGICAL TECHNIQUE

Make a ventral midline abdominal incision from the xiphoid process to several centimeters caudal to the umbilicus. Explore the abdomen and locate the duodenum and stomach. Apply gentle traction on the duodenum and stomach to reduce the intussusception. If necessary, digitally dilate or enlarge the esophageal hiatus to allow complete reduction of the intussusception. Examine the distal esophagus, the stomach, and any other involved viscera for evidence of vascular thrombosis, avulsion, ischemia, or necrosis. Resect devitalized tissue. Reduce the size of the esophageal hiatus to 1 to 2 cm if it is too large or lax (see p. 389). Perform an incisional gastropexy (see p. 408) at the gastric fundus to prevent recurrence. In addition to the fundic gastropexy, an incisional right or pyloric antrum gastropexy is sometimes performed. *Lavage and close the abdomen. Place a feeding tube if esophageal disease is severe.*

POSTOPERATIVE CARE AND ASSESSMENT

Fluids should be continued after surgery and acid-base and electrolyte imbalances corrected. Nasal oxygen may benefit dyspneic animals. Postoperative analgesics should be provided (see Table 13.2), and oral intake withheld initially to encourage resolution of esophagitis and gastritis (see p. 366). After 24 to 48 hours, water can be offered. If vomiting or regurgitation does not occur, small amounts of low-fat, high-protein gruel can be offered several times a day. If megaesophagus is present, the animal should be fed in an upright position or through a gastrostomy tube. However, esophageal weakness may not resolve, especially in young animals. Treat esophagitis as needed (see Box 18.13). A gastrostomy tube feeding may be helpful. Deterioration and death may occur if the condition is not recognized and treated promptly. Devitalization of a portion of the esophagus or stomach may occur as a result of preoperative vascular compromise. Dysphagia is common for several days after surgery; however, persistent dysphagia may occur if the hiatus is overly narrowed by surgery. Such patients require reoperation.

PROGNOSIS

Antemortem diagnosis is rare in acute cases (mortality approaches 95%); therefore few cases of gastroesophageal intussusception have been diagnosed and treated successfully. Recurrence is not expected if incisional gastropexy is performed and esophagitis is controlled. Prognosis in chronic intermittent cases is fair if reduction is maintained and esophageal disease manageable.

CRICOPHARYNGEAL ACHALASIA

DEFINITION

Cricopharyngeal achalasia (*congenital cricopharyngeal achalasia, cricopharyngeal dysphagia, cricopharyngeal dysfunction, cricopharyngeal asynchrony*) is one type of pharyngeal dysphagia. The condition is characterized by the interruption of passage of a bolus from the oropharynx through the cranial esophageal sphincter into the cervical esophagus because of failure of the sphincter to open or failure of the opening to coordinate with orophayrngeal swallowing.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Cricopharyngeal achalasia is a rare cause of dysphagia; however, it is important to differentiate it from other forms of oropharyngeal dysphagia because the former is treatable with surgery. There are a number of swallowing disorders and their treatment varies. The cause of cricopharyngeal achalasia is unknown but appears to be a congenital derangement in the coordination of

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the swallowing reflex. The cause is probably neurologic, because transecting the pharyngeal branch of the tenth cranial nerve can reproduce the disorder. Cricopharyngeal achalasia is characterized by inadequate relaxation of the cricopharyngeal muscle and/or lack of coordination with pharyngeal muscle contractions during swallowing. This disrupts the cricopharyngeal phase of swallowing, causing food to remain in the pharynx and produce gagging, regurgitation, aspiration, and coughing. Repeated attempts to swallow are made until retained pharyngeal contents are swallowed, regurgitated, or aspirated. Food remaining in the pharynx may be aspirated into the trachea during inspiration, eliciting a cough and causing pneumonia. The small amount of food that succeeds in passing from the oropharynx into the proximal esophagus passes normally into the stomach.

In normal swallowing, food is grasped by the teeth and formed into a bolus by rapid tongue movements. The bolus is pushed up and back by the base of the tongue into the oropharynx. The hypopharyngeal, pterygopharyngeal, and palatopharyngeal muscles contract and force the bolus through the relaxed upper esophageal sphincter (cricopharyngeal, thyropharyngeal muscles) into the cervical portion of the esophagus. The cricopharyngeal muscle contracts after the bolus passes. Cricopharyngeal muscle tone is linked to deglutition and the respiratory cycle. During swallowing, the airways are protected by the soft palate (which closes the nasopharynx) and the epiglottis (which flips back to close the glottis).

DIAGNOSIS

Clinical Presentation

Signalment

The condition is rare. It seems to be more common in springer and cocker spaniels and miniature poodles but has been seen in a variety of breeds. Signs of dysphagia begin at weaning, although in rare cases animals may not be diagnosed until they are older.

History

Most dogs appear normal until they begin eating solid food. At that time, repeated unsuccessful attempts to swallow are noted, with gagging, retching, and expulsion of saliva-covered food. Regurgitation occurs immediately after swallowing. Other signs may include aspiration, coughing, excess salivation, and nasal reflux during eating. Most patients have a voracious appetite, but some fail to thrive, become anorexic, and lose weight.

Physical Examination Findings

Affected animals may appear normal or stunted at the time of examination. Some patients are emaciated. The animal should be observed eating and drinking to confirm dysphagia and to characterize it as oral or pharyngeal. Patients with oral dysphagia have difficulty with prehension and bolus formation. Those with pharyngeal dysphagia have difficulty transporting the bolus into the esophagus. Patients with cricopharyngeal dysphagia usually have more difficulty with solid food, whereas those with other types of pharyngeal dysphagia may have more difficulty (i.e., may aspirate more readily) when swallowing liquids. Swallowing attempts may trigger the animal to cough, gag, and choke. A neurologic examination should be performed to rule out cranial nerve deficits and concurrent neuromuscular abnormalities. Signs of concurrent aspiration pneumonia, rhinitis, laryngeal paralysis, or megaesophagus may be present.

Diagnostic Imaging

Survey thoracic radiographs should be evaluated for aspiration pneumonia and esophageal size. Definitive diagnosis requires fluoroscopic or cinefluoroscopic evaluation of swallowing barium sulfate. Use liquid barium or paste barium sulfate mixed with food unless aspiration is anticipated. An experienced radiologist may be needed to differentiate cricopharyngeal achalasia from pharyngeal dysphagia. Affected animals will have several ineffectual attempts by the tongue to force the bolus from the pharynx and inadequate relaxation of the cricopharyngeal muscle, prohibiting bolus entry into the proximal esophagus. In normal dogs, the time between onset of swallowing and opening of the upper esophageal sphincter following a bolus of liquid barium is approximately 0.10 seconds; opening in those with cricopharyngeal achalasia is delayed (approximately 0.17–0.45 seconds). A thin stream of contrast may be seen passing through the sphincter. Some contrast may be seen refluxing into the nasopharynx or being aspirated into the airway. Patients with cricopharyngeal achalasia have adequate pharyngeal strength to push the food bolus into the esophagus, but the cricopharyngeal sphincter stays shut or opens at the wrong time during the swallowing reflex. Patients with pharyngeal dysphagia do not have adequate oropharyngeal strength or coordination of the pharyngeal muscles to properly push the food bolus into the esophagus. Positive contrast in the trachea or outlining airways indicates aspiration. Esophageal motility should be evaluated during fluoroscopic studies to identify concurrent problems because many animals with pharyngeal dysphagia have concurrent esophageal dysfunction, and for these patients, cricopharyngeal surgery is absolutely contraindicated. Endoscopic examination of the pharynx and esophagus is normal.

Laboratory Findings

Laboratory findings are normal unless the patient is severely debilitated or has aspiration pneumonia. Then they may have a neutrophilia or hypoproteinemia.

DIFFERENTIAL DIAGNOSIS

Pharyngeal dysphagia caused by inadequate pharyngeal contraction can be challenging to differentiate from cricopharyngeal achalasia. Pharyngeal dysphagia tends to occur in older dogs, whereas cricopharyngeal dysfunction tends to occur in younger dogs, but age is an inadequate criterion to distinguish these two conditions. Sometimes more than one abnormality contributes to the dysphagia. Dental disease, oral masses, foreign bodies, trauma, stomatitis, cleft palate, hypoplastic palate, and other congenital and skeletal abnormalities should also be considered. Esophageal hypomotility and megaesophagus may also be associated with pharyngeal dysphagias. Other considerations include functional abnormalities resulting from rabies, central nervous system disease, peripheral neuropathies, neuromuscular disease, myopathies, and myositis.

MEDICAL TREATMENT

Medical management may be attempted with intralesional injection of botulinum toxin. This therapy has only recently been performed on dogs; its efficacy is uncertain at this time.

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SURGICAL TREATMENT

Cricopharyngeal myectomy is often curative for congenital cricopharyngeal achalasia. Surgery is not necessarily recommended for acquired cricopharyngeal achalasia. Some surgeons also resect a portion of the thyropharyngeus muscle. Cricopharyngeal myectomy for patients with other pharyngeal dysphagias can be disastrous because it allows food retained in the proximal esophagus to more easily reenter the pharynx and be aspirated.

Preoperative Management

Supportive care is important; patients should be well hydrated and nourished before surgery. Preoperative nutritional support should be provided with a gastrostomy tube if necessary (see p. 97). Oral feeding should include feeding food of a consistency (dry vs. canned vs. gruel) most successfully retained by the animal. Aspiration pneumonia should be treated with fluids, appropriate antibiotics, and expectorants. Perioperative antibiotics are recommended for debilitated patients.

Anesthesia

Anesthetic recommendations for esophageal surgery are provided on p. 367 and Table 18.3. Hypothermia and hypoglycemia are serious problems in young patients and should be prevented during anesthesia and surgery.

Surgical Anatomy

The cranial esophagus is located dorsal to the larynx and slightly to the left of midline. The cricopharyngeal muscle lies on the larynx and pharynx immediately caudal to the thyropharyngeal muscle (Fig. 18.62). It arises from the lateral surface of the cricoid cartilage and passes dorsally to insert on the median dorsal raphe. The cricopharyngeal muscle can be identified as a bundle of transverse muscle fibers converging on the dorsal midline and blending into the longitudinal muscle fibers of the cranial esophagus. The thyropharyngeal muscle can be difficult to differentiate from the cricopharyngeal muscle because their muscle fibers are both oriented transversely. The thyropharyngeal muscle lies cranial to the cricopharyngeal muscle and is separated from it by a thin septum of connective tissue. The separation is found dorsal to the attachment of the sternothyroid muscle. The cricopharyngeal muscle controls most of the upper esophageal sphincter function. Branches of the glossopharyngeal and vagus nerves innervate it. Blood supply to the cricopharyngeal muscle is primarily from branches of the cranial thyroid arteries.

Positioning

The animal is positioned in dorsal recumbency with the legs positioned lateral to the thorax. The ventral neck (from the angle of the mandible to the manubrium) should be prepared for aseptic surgery. Alternatively, the animal is positioned in lateral recumbency and a lateral incision is made.

SURGICAL TECHNIQUE

Make a ventral midline cervical incision beginning cranial to the larynx and extending caudally to the midcervical area. Separate and retract the sternohyoid muscles laterally to expose the trachea. Rotate the larynx and trachea laterally by using traction on the sternothyroid muscle to expose the cricopharyngeal musculature (Fig. 18.63A). Place a suture through the lamina of the thyroid cartilage to maintain laryngeal rotation and exposure of the cricopharyngeal muscle and dorsal esophagus. Pass a gastric tube into the esophagus to aid identification of the esophageal wall. Identify

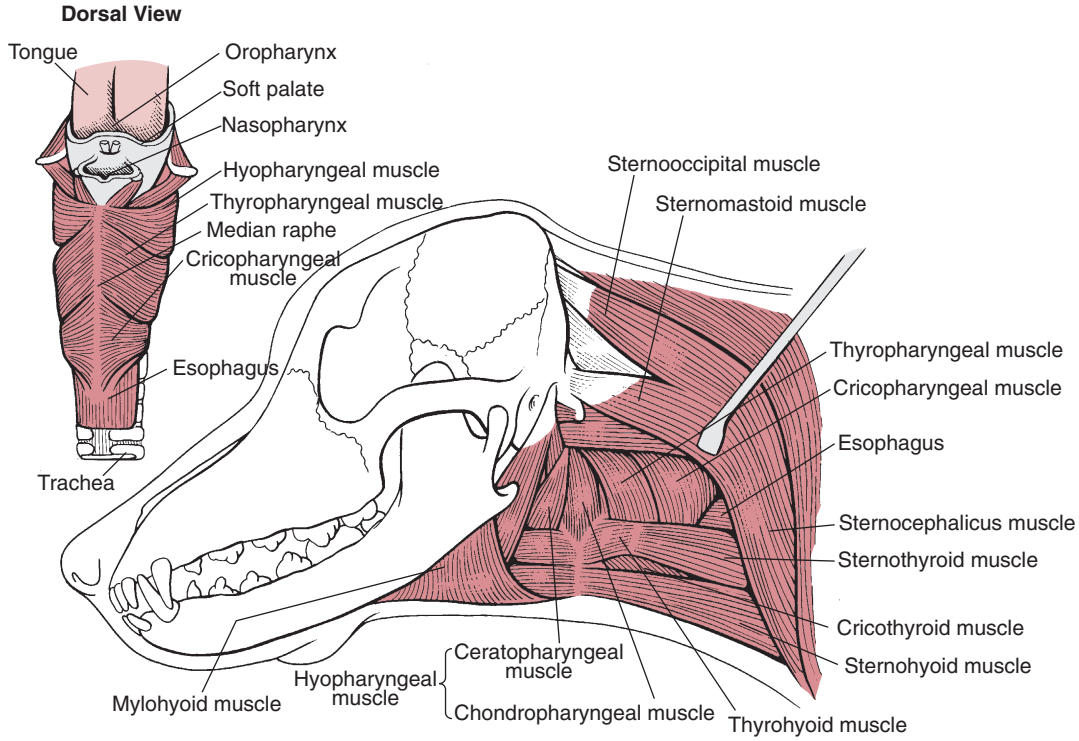


FIG. 18.62 Surgical anatomy of the cranial esophagus and neck.

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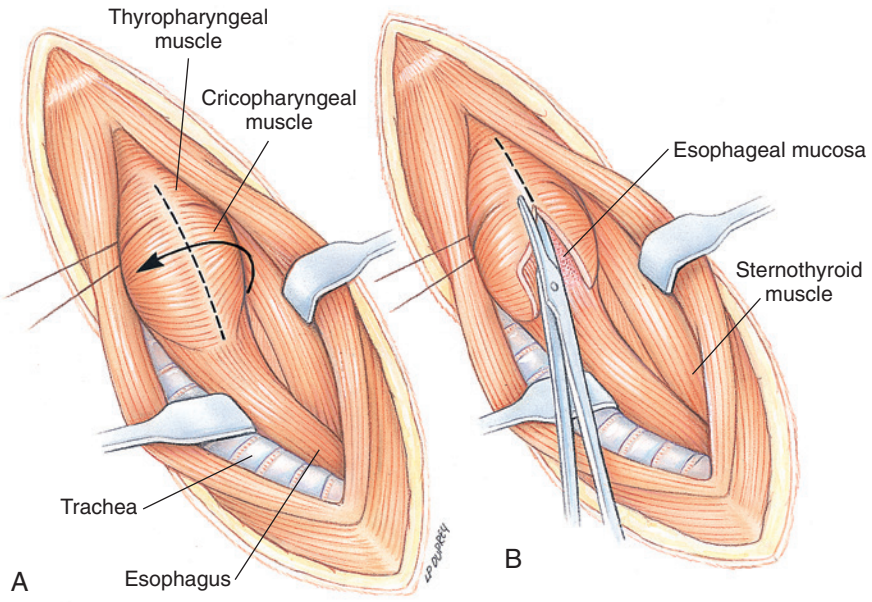


FIG. 18.63 Cricopharyngeal myectomy. (A) Expose the cranial esophagus through a ventral midline cervical incision. Place a stay suture through the thyroid lamina and rotate the cricopharyngeal muscle into view. (B) Resect the lateral portion of each cricopharyngeal muscle.

the cricopharyngeal muscles. Incise the cricopharyngeus (and, if needed, the thyropharyngeus) muscle on its midline (see Fig. 18.63B). Elevate the muscle fibers from the underlying esophageal submucosa with care to avoid perforating the esophageal wall. Resect the lateral portion of each cricopharyngeus (and the thyropharyngeus) muscle. Inspect the esophageal wall for damage and lavage the area. Allow the larynx and trachea to return to their normal position. Appose the sternohyoid muscles with a continuous suture pattern. Close subcutaneous tissues and skin routinely.

POSTOPERATIVE CARE AND ASSESSMENT

Provide analgesia and treat aspiration pneumonia as needed. Immediately after surgery, improvement in swallowing function is expected. When fully recovered from anesthesia, evaluate swallowing function by giving a small amount of soft food. A gruel or canned food should be fed for the first 1 to 2 days after surgery, then the food should gradually be returned to normal consistency over the next 3 to 4 days. If necessary, IV fluids should be continued to maintain hydration. Continue feeding through a gastrostomy tube if necessary to improve body condition. Antibiotics should be continued if aspiration pneumonia is present. Inadequate or unilateral myectomy may not relieve signs of cricopharyngeal achalasia.

COMPLICATIONS

Recurrence of dysphagia because of fibrosis and constriction at the myectomy site can be prevented by adequate removal of muscle. If pharyngeal dysphagias or proximal esophageal dysfunction are present, then cricopharyngeal myectomy will worsen the patient's dysphagia and pneumonia. Aspiration pneumonia may continue to be a problem if esophageal hypomotility exists. Other potential complications include laryngeal paralysis, esophageal perforation, and pharyngocutaneous fistula.

PROGNOSIS

The prognosis is good if the only abnormality present is cricopharyngeal achalasia and guarded if other dysphagias or related conditions are present. Although complete resolution of signs is expected, some have only partial resolution and others have recurrence of clinical signs within 2 to 36 weeks. Persistent regurgitation may result in aspiration pneumonia. The prognosis without surgery is guarded because it is difficult to effectively nourish the patient and control pneumonia.

VASCULAR RING ANOMALIES

DEFINITION

Vascular ring anomalies are congenital malformations of the great vessels and their branches that cause constriction of the esophagus and signs of esophageal obstruction. PRAA, the most common type of vascular ring anomaly, is also known as *persistent fourth right aortic arch.*

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The most common type of vascular ring anomalies are a persistent fourth right aortic arch, right dorsal aortic root, and rudimentary left ligamentum arteriosum (left sixth arch). The left pulmonary artery and the descending aorta are connected by the ligamentum arteriosum. The esophagus is encircled by the ligamentum arteriosum (or patent ductus arteriosus) on the left, the base of the heart and pulmonary artery ventrally, and the aortic arch on the right. The esophagus is constricted by this vascular "ring" and begins to dilate cranially as food accumulates. Food that does not pass beyond the constriction is intermittently regurgitated. Chronic regurgitation predisposes the animal to aspiration

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BOX 18.20 Vascular Ring Anomalies: Key Points

- Regurgitation classically begins at weaning with introduction of solid food
- Some patients are years old before they are diagnosed
- Aspiration pneumonia is a concurrent risk
- 95% are persistent right aortic arch ± other aberrant vessels
- Mostly diagnosed in German shepherds
- Radiographic signs: esophageal dilatation cranial to heart base and tracheal deviation to the left
- Advanced imaging (contrast computed tomography) is recommended to be certain of the diagnosis
- Perform surgery early to prevent irreversible changes
- Treat pneumonia and improve body condition before surgery
- Identify, isolate, occlude, and divide offending vessels
- Dissect constricting fibrous bands from around esophagus
- Feed gruel while standing erect on hind legs; gradually increase food consistency
- Most regurgitate less following surgery; some are clinically normal

pneumonia. Approximately 95% of animals diagnosed with vascular ring anomalies have a PRAA (Box 18.20).

Abnormal location of the great vessels mechanically interferes with functioning of the esophagus and sometimes the trachea and other adjacent structures. The severity of clinical signs and the degree of esophageal stricture depend on the vascular structures involved. Approximately 44% of animals with PRAA have coexisting compressive arterial anomalies. Other types of vascular ring anomalies include (1) PRAA with persistent left subclavian artery, (2) PRAA with persistent left ligamentum arteriosum and left subclavian artery, (3) double aortic arch, (4) normal left aortic arch with persistent right ligamentum arteriosum, (5) normal left aortic arch with persistent right subclavian artery, and (6) normal left aortic arch with persistent right ligamentum arteriosum and right subclavian artery. A patent ductus arteriosus rather than the ligamentum arteriosum persists in approximately 15% of those with vascular ring anomalies. Additionally, persistent left vena cava occurs in conjunction with PRAA in 12% to 40% and a left hemiazygos vein in approximately 6% of the cases.

Six pairs of aortic arches surround the esophagus and trachea during early fetal life. Normal maturation and selective regression of these arches form the adult vasculature. All vascular ring anomalies have resulted from abnormal development of arches three, four, and six. The mechanism of inheritance is complex and polygenic, probably involving multiple recessive genes. In the embryo the first and second aortic arches disappear, and the fifth arches are incomplete and inconsistent. The third arch joins the dorsal aortic arch and continues anteriorly as the right and left internal carotid arteries. The third arch also forms the brachiocephalic trunk. The dorsal aortas disappear between the third and fourth arches. Normally the left fourth aortic arch and the dorsal aortic root persist to form the permanent aortic arch. The left sixth arch becomes the ductus arteriosus, and the right fourth arch contributes to the right subclavian artery.

DIAGNOSIS

Clinical Presentation

Signalment

Vascular ring anomalies occur in both dogs and cats but are more common in dogs. German shepherds, Irish setters, and Boston terriers are the most commonly affected breeds. PRAA may be a hereditary condition in greyhounds. Siamese and Persian cats have been diagnosed more often than other cat breeds. Males and females are equally affected. The condition may affect several animals in a litter. Vascular ring anomalies are present at birth. Clinical signs are usually evident at the time of weaning, most being diagnosed between 2 and 6 months of age; however, the condition may not be recognized until later in life if obstruction is partial and signs are mild or intermittent. Early diagnosis and treatment of PRAA may improve the prognosis.

History

The classic history is acute onset of regurgitation when solid or semisolid food is first fed. Regurgitation of undigested food occurs soon after eating early in the disease; later it may occur at variable times (minutes to hours). Affected animals may grow more slowly than litter mates and appear malnourished. They often have a voracious appetite; some immediately re-eat the regurgitated food. Coughing with respiratory distress may be a result of aspiration pneumonia or tracheal stenosis that occurs secondary to a double aortic arch.

Physical Examination Findings

Affected animals are often thin and small. An enlarged esophagus may sometimes be palpated at the thoracic inlet and neck. The thoracic inlet and caudal neck area may bulge when the chest is compressed. Murmurs are rare; an occasional animal may have a continuous murmur associated with concurrent patent ductus arteriosus. Fever and pulmonary crackles suggest pneumonia.

Diagnostic Imaging

Thoracic radiographs may reveal a dilated esophagus cranial to the heart containing air, water, or food. Focal leftward deviation of the trachea near the cranial border of the heart on dorsoventral or ventrodorsal radiographs strongly suggests PRAA in symptomatic young dogs. The trachea may be displaced ventrally and the esophagus may overlap it. Signs of pneumonia may be identified. Incidental axial skeletal abnormalities of the vertebrae, ribs, or sternebrae have been reported in cats.[17] Positive contrast radiography using a barium sulfate liquid mixed with food demonstrates esophageal constriction at the base of the heart with varying degrees of esophageal dilatation extending cranially (Fig. 18.64). However, other esophageal conditions can sometimes mimic vascular ring anomalies; therefore the contrast study needs to be critically evaluated. The ratio of the area of maximal esophageal dilatation after liquid barium sulfate (8 mL/kg body weight) to the narrowest height of the fifth thoracic vertebrae in normal dogs and cats is ≤1. The animal is considered to have mild dilatation if the ratio is ≤2.5, moderate with a ratio ≤4, and severe with a ratio >4. The caudal esophagus is usually normal in size, although it is sometimes dilated. Fluoroscopy is beneficial in evaluating esophageal motility. The dilated esophagus does not usually demonstrate normal peristaltic contractions. Contrast CT evaluation is more helpful in identifying abnormal vasculature than angiography and is recommended. Echocardiography may also be beneficial. Endoscopic examination of the esophagus helps rule out other causes of esophageal stricture or obstruction and may reveal esophageal ulceration. Sometimes, a dilated esophagus proximal to the heart is due to focal esophageal weakness, but radiographically mimics PRAA. Esophagoscopy can determine if there is a stricture caused by a vascular ring or

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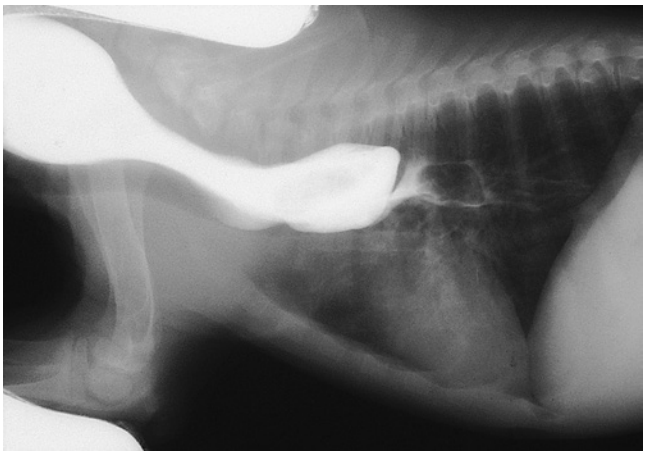


FIG. 18.64 Contrast esophagram of a dog with a persistent right aortic arch. Note the esophageal narrowing at the base of the heart. The cranial esophagus is dilated.

if there is no stricture (and therefore the dilated area is due to esophageal weakness). Tracheoscopy is not routinely performed but may document narrowing of the tracheal lumen caused by external compression.

Laboratory Findings

Laboratory findings are expected to be normal (for the animal's age) unless debilitation is severe or pneumonia is present. In animals with pneumonia, neutrophilia may be present and debilitated animals may be hypoproteinemic.

DIFFERENTIAL DIAGNOSIS

The primary differential diagnoses are generalized megaesophagus and obstruction caused by a foreign body, stricture, mass, or hiatal hernia. It can be difficult to radiographically differentiate between esophageal dilatation caused by a vascular ring anomaly and a large outpouching cranial to the heart associated with megaesophagus.

MEDICAL MANAGEMENT

Patients with vascular ring anomalies are treated both medically and surgically. Surgery should be performed as soon after the onset of clinical signs as possible to hopefully prevent progressive damage to the esophageal muscles and nerves. Clients should be informed that medical treatment without surgery is palliative at best and is not recommended. Medical management includes treating aspiration pneumonia (see p. 367) and improving the animal's nutritional status. An affected animal should be fed gruel while vertical (e.g., using a Bailey chair). The animal should be maintained in an upright position (standing on hind legs) for 5 to 10 minutes after eating to allow gravity to assist in emptying the dilated esophagus. Placement of a gastrostomy feeding tube may be beneficial for severely debilitated patients, if aspiration is present, or in cases of longstanding megaesophagus with delayed presentation for surgery.

SURGICAL TREATMENT

The surgical treatment of PRAA is described later. Other types of vascular ring anomalies can be managed in a similar fashion. A persistent left vena cava often covers the left ventral area of the vascular ring. A persistent right ligamentum arteriosum and some aberrant right subclavians should be approached from the right side. Angiograms are helpful in patients with double aortic arches to determine which arch is dominant and if adequate circulation can be maintained after transection of the other arch. It may not be possible to relieve constrictions caused by a double aortic arch. If the animal is severely debilitated, a gastric feeding tube should be placed for several days before surgery.

Some surgeons attempt to reduce the size of the esophageal lumen if the esophagus is severely dilated and not expected to return to normal size. Placing a series of nonpenetrating "plication" or "gathering" sutures in the accessible lateral esophageal wall accomplishes this goal. As an alternative, a portion of the esophagus may be resected. These techniques are not recommended routinely because they increase the risk of complications.

Preoperative Management

Hydration, electrolyte, and acid-base abnormalities should be corrected before surgery if possible. If pneumonia is present or if the animal is severely debilitated, nutrition should be provided via a gastrostomy feeding tube (see p. 97) for several days to a week. Antibiotics are indicated for debilitated animals and those with pneumonia (see p. 367).

Anesthesia

Anesthetic recommendations for animals undergoing thoracotomies are presented on p. 884, Table 29.1, and Table 30.3. Anesthetic recommendations for animals with esophageal disease are found on p. 367 and Table 18.3. In addition, refer to the general anesthesia section in Chapter 26 on p. 722 for anesthetic concerns with the pediatric patient. Animals younger than 6 months of age should be anesthetized with care. Because hepatic glycogen stores are rapidly depleted during fasting in puppies and kittens, fasting longer than 4 to 6 hours is not generally recommended. If you are unable to monitor the blood glucose level, IV fluids containing balanced electrolytes in a 2.5% dextrose solution should be given for surgeries lasting longer than 1 hour or if anesthetic recovery is delayed. Hypothermia commonly develops in puppies and kittens during surgery because they have a higher surface area to body weight ratio, which causes greater heat loss by radiation and evaporation. Hypothermia may cause bradycardia, low cardiac output, and hypotension, which may prolong drug elimination and anesthetic recovery. Drugs that normally redistribute to muscle or fat have prolonged effects in puppies and kittens after repeated administration. Reduced hepatorenal function may also prolong drug effects. Phenothiazine tranquilizers should be used with care in animals younger than 3 months of age, because the drugs may cause prolonged central nervous system depression. If used, phenothiazines should be given at one-fourth to one-half the adult dose. Opioids can be used in young animals but should be started by using half the adult dose and titrated to effect. Chapter 26 contains additional information on handling and caring for pediatric patients.

Surgical Anatomy

The anatomy of the esophagus is discussed on p. 369 and the anatomy of the heart on p. 794. See also the discussion under General Considerations and Clinically Relevant Pathophysiology on p. 394.

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Positioning

Most patients with vascular ring anomalies should be positioned in right lateral recumbency for a left lateral thoracotomy (see p. 889); however, those with persistent right ligamentum arteriosum are positioned in left lateral recumbency. Positioning of animals with double aortic arches varies, depending on the dominant arch.

SURGICAL TECHNIQUE

Surgical transection of the constricting structure or structures is recommended before esophageal dilatation becomes severe. Transection is feasible with most vascular ring anomalies, with the exception of some double aortic arches. Staples may be used rather than suture to occlude vascular structures. Thoracoscopic-assisted division of PRAA is an alternative approach.

Perform a lateral thoracotomy at the left fourth (fifth) intercostal space for patients with PRAA. Pack the cranial lung caudally to expose the mediastinum dorsal to the heart. Identify the aorta, pulmonary artery, ligamentum arteriosum, and vagal and phrenic nerves (see Figs. 27.3 and 18.43). Identify the anomalous structure or structures. If a persistent left cranial cava is present, dissect and retract the vena cava to improve visualization. If a prominent hemiazygos vein is also present, dissect, ligate, and divide it. If a constricting subclavian artery is identified, isolate, ligate, and transect it. If an atretic left aortic arch is present isolate, ligate and transect it. Incise the mediastinum and dissect and elevate the ligamentum arteriosum. Double ligate the ligamentum arteriosum and then transect it. If a large-diameter patent ductus arteriosus is present rather than a ligamentum arteriosum, oversew both ends after transection. Pass a balloon catheter or large orogastric tube through the constricted esophagus to aid identification of constricting fibrous bands and to dilate the site. Dissect and transect these fibrous bands from the esophageal wall. Lavage the area, reposition the lung lobes, place a thoracostomy tube, and close the thorax routinely.

Thoracoscopic Ligation and Division of the Ligamentum Arteriosum

Division of the ligamentum arteriosum via thoracoscopy has been described for the treatment of PRAA in dogs. *Establish an endoscopic port in the left seventh intercostal space at the costochondral junction. Place three other ports at the same level in the third and fifth intercostal spaces for operating equipment. Place a fourth port in the dorsal aspect of the fifth intercostal space and pass a lung retractor to manipulate the left cranial lung caudally for exposure of the ligamentum. Use a palpation probe to identify the ligamentum if it is not visible at the junction of the dilated and more normal esophagus (Fig. 18.65A). Pass an endoscope or balloon catheter to assist identification if necessary. Bluntly elevate the ligamentum and place hemoclips dorsally and ventrally prior to transection with Metzenbaum scissors (see Fig. 18.65B). Dissect away remaining connective tissue from the surface of the esophagus taking care to avoid its muscular layer; dilation with a balloon may be done if necessary.*

An alternative, caudal approach to the ligamentum can be done with an endoscope port placed in the middle of the 10th intercostal space with instrument ports in the dorsal and ventral aspects of the ninth intercostal space and a port in the ventral aspect of the sixth intercostal space for lung retraction. Visualization and dissection proceed from caudal to cranial, similar to the preceding lateral approach.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative analgesics should be provided as described on p. 886. The patient should be closely monitored for dyspnea and the chest tapped if necessary. Nasal oxygen may benefit dyspneic patients. If a thoracostomy tube has been placed, the thorax should be aspirated at regular intervals (initially every 15–30 minutes) and the volume of air and fluid collected at each interval noted. Thoracostomy tubes can generally be removed the day of surgery or by the next morning in these patients. Antibiotics should be continued in debilitated patients if thoracic contamination occurred or if pneumonia exists.

Pediatric patients should be closely monitored for hypoglycemia in the postoperative period. Oral intake can be resumed within 12 to 24 hours of surgery. Initially, a canned food gruel should be fed with the animal in an upright posture. This stance should be maintained for 5 to 10 minutes after eating to help prevent distention of the dilated esophagus and help reestablish esophageal

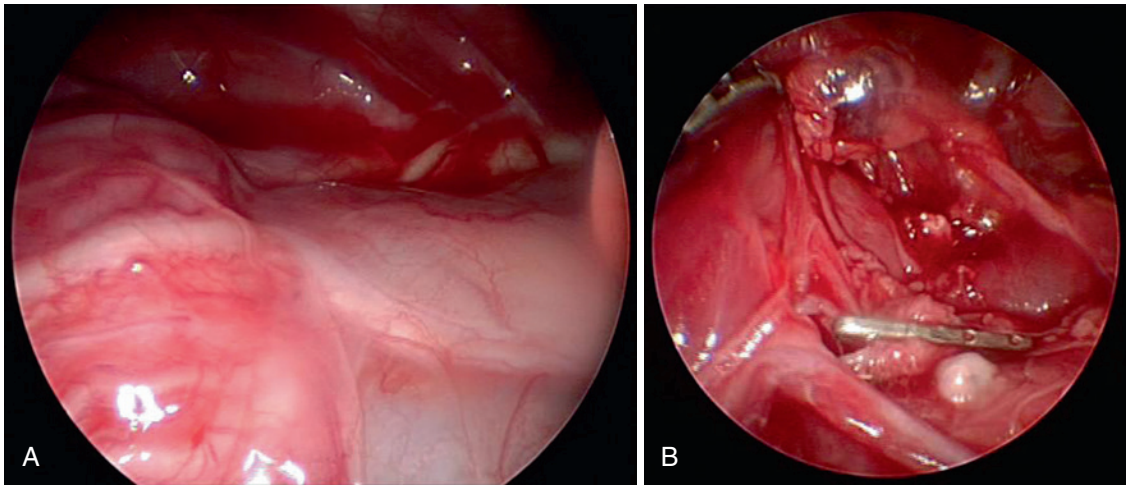


FIG. 18.65 (A) Thoracoscopic view of a dog with a persistent right aortic arch. The dilated esophagus cranial to the ligamentum arteriosum is to the left in the image. The vagus nerve is visible on the ventrolateral surface of the esophagus. (B) Dissection of the ligamentum arteriosum after vascular clip application.

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muscle tone and size. Owners may gradually reduce the amount of water in the food 2 to 4 weeks after surgery if minimal regurgitation has occurred with gruel feeding. Hopefully, the addition of water can ultimately be eliminated without increased regurgitation. Animals that can eat solid food without regurgitation should be allowed to eat with the bowl on the floor while standing normally. This feeding practice is continued unless regurgitation recurs. Some animals can eventually be fed any type of food from a normal stance, whereas others must continue eating gruel from an elevated stand.

If the patient is still having trouble, the esophagus should be reevaluated with an esophagram 1 to 2 months after surgery to assess persistent dilatation and motility. Sometimes the esophagus returns to a normal size and function. Other times the esophagus remains severely dilated because of poor motility. If esophageal stricture occurs at the surgery site, balloon dilatation (see p. 382) may be beneficial, but care must be taken to avoid rupturing the esophagus. Owners should be advised against breeding affected animals because the condition is genetic.

COMPLICATIONS

Surgical complications are common because of the initial malnourished and debilitated condition of affected animals and concurrent aspiration pneumonia. Persistent regurgitation is the most common postoperative problem. Preoperative client education must emphasize the high incidence of continued regurgitation and the need for prolonged dietary management. Aspiration pneumonia and death may occur if regurgitation persists. Esophageal resection or imbrication increases the risk of contamination and infection secondary to esophageal leakage or dehiscence.

PROGNOSIS

Most animals that survive surgery improve; however, perioperative mortality can be as high as 8%.[18] Animals with severe esophageal dilatation (esophageal to fifth thoracic vertebrae ratio >4) prior to surgery may have a worse long-term outcome than those with mild or moderate dilatation. Persistence of esophageal dilation, however, may not be associated with either continued regurgitation or an adverse long-term prognosis. They may not have completely normal esophageal function, but they generally regurgitate less and their body condition improves. If surgery is performed soon after signs appear, normal esophageal tone and function might return. The longer the delay before surgical correction, the more cautious the prognosis; some patients show little or no improvement. Megaesophagus often persists with mild to moderate improvement in motility, although most patients no longer have regurgitation. The prognosis is poor with esophageal dilatation caudal to the constriction because this area is often hypomotile and frequently does not regain normal size. Without surgery, regurgitation usually continues and worsens as the esophagus continues to dilate. Aspiration pneumonia is a continuous threat, and the animal may have difficulty maintaining adequate body condition. Short-term causes of death include cardiopulmonary arrest, aspiration pneumonia, and euthanasia due to failure to improve. Good to excellent outcome can be expected in 87%. Poor outcomes relate to the need for specialized or elevated feeding and recurrent regurgitation.[18]

Surgery of the Stomach

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Gastrotomy is an incision through the stomach wall into the lumen. **Partial gastrectomy** is a resection of a portion of the stomach, and **gastrostomy** is the creation of an artificial opening into the gastric lumen. **Gastropexy** permanently adheres the stomach to the body wall. Removal of the pylorus (**pylorectomy**) and attachment of the stomach to the duodenum (**gastroduodenostomy**) is a **Billroth I** procedure. Attachment of the jejunum to the stomach (**gastrojejunostomy**) after a partial gastrectomy (including pylorectomy) is a **Billroth II** procedure. In a **pyloromyotomy,** an incision is made through the serosa and muscularis layers of the pylorus only. For a **pyloroplasty,** a full-thickness incision and tissue reorientation are performed to increase the diameter of the gastric outflow tract.

PREOPERATIVE CONCERNS

Gastric surgery is commonly performed to remove foreign bodies (see p. 413) and to correct gastric dilatation-volvulus (GDV; see p. 419). Gastric ulceration or erosion (see p. 427), neoplasia (see p. 430), and benign gastric outflow obstruction (see p. 425) are less common indications. Gastric disease may cause vomiting (intermittent or profuse and continuous) or just anorexia. Dehydration and hypokalemia are common in vomiting animals and should be corrected before induction of anesthesia. Alkalosis may occur secondary to gastric fluid loss; however, metabolic acidosis may also be seen. Hematemesis may indicate gastric erosion or ulceration or coagulation abnormalities. Peritonitis arising from perforation of the stomach caused by necrosis or ulceration is often lethal if not treated promptly and aggressively (see p. 527). Aspiration pneumonia or esophagitis may also occur in vomiting animals. If possible, severe aspiration pneumonia (see p. 367) should be treated before induction of anesthesia for gastric surgery.

Mild esophagitis generally can be treated by withholding food for 24 to 48 hours (see p. 367) and treating with proton pump inhibitors. However, severe esophagitis may necessitate a gastrostomy tube (see p. 97). If continued vomiting is likely, an enteral feeding tube is preferred (see p. 101). Treatment with proton pump inhibitors (i.e., omeprazole, esomeprazole, pantoprazole; see Box 18.13) is important. Orally administered sucralfate slurries may help alleviate pain, but they should be given 1 hour after other medications (see p. 429). Administration of prokinetics (e.g., cisapride, erythromycin, metoclopramide, ranitidine) will improve gastric emptying and help minimize reflux.

When possible, food should be withheld for at least 8 to 12 hours before surgery to ensure that the stomach is empty. If gastroscopy will be performed, it is best to fast the patient for at least 18 and preferably 24 hours before the procedure. However, fasting for only 4 to 6 hours may help prevent hypoglycemia in pediatric patients (see the discussion on anesthetic and surgical management of pediatric patients, which follows). Surgery for gastric obstruction, distention, malpositioning, or ulceration should be performed as soon as possible after the animal's condition has been stabilized.

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ANESTHETIC CONSIDERATIONS

Numerous anesthetic protocols have been used in stable animals undergoing abdominal surgery (see Table 19.1). See Table 18.4 for anesthetic recommendations for the acute abdomen.

ANTIBIOTICS

Perioperative antibiotics may be used if the gastric lumen will be entered; however, animals with normal immune function undergoing simple gastrotomy (i.e., proper aseptic technique and no spillage of gastric contents) rarely require them. If antibiotics are used (e.g., cefazolin; 22 mg/kg given intravenously at induction; repeat once or twice at 90-minute to 2-hour intervals), they should be given intravenously before induction of anesthesia and continued for up to 12 hours postoperatively. Except for *Helicobacter* organisms, bacteria are scarce in the stomach compared with other parts of the GI tract because of the low gastric pH.

SURGICAL ANATOMY

The stomach can be divided into the cardia, fundus, body, pyloric antrum, pyloric canal, and pyloric ostium. The esophagus enters

TABLE 18.4 Anesthetic Considerations for the Patient With an Acute Abdomen

Preoperative Considerations

Associated conditions	• Dehydration • Electrolyte abnormalities • Hypotension • Anemia • Arrhythmias
Bloodwork	• HCT • Electrolytes • BUN • Cr • TP • Lactate • Urinalysis • Blood gas if available
Physical exam	• Often a younger patient that was previously healthy • May be dehydrated, tachycardic or bradycardic, hypotensive, retching, vomiting, or hypothermic • Painful or distended abdomen may be present
Other diagnostics	• Blood pressure • ECG • Radiograph (abdominal)

Premedications	• Rehydrate over 4–6 h if possible; if emergent, may have to give more rapid boluses to expedite time to surgery.
	• Correct electrolyte and metabolic abnormalities if time permits
	• Avoid sedatives in depressed or cardiovascularly compromised patients.
	• Avoid alpha-2 agonists and acepromazine.
	• If patient is stable and anxious, give:
	  • Midazolam (0.2 mg/kg IV, IM) *or*
	  • Diazepam (0.2 mg/kg IV)
	• If patient is not depressed, then give:
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM in dogs; 0.05–0.1 mg/kg IV, IM in cats), *or*
	  • Morphine[b] (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM)

Intraoperative Considerations

Induction	• If dehydrated or unstable, give the following:
	  • Etomidate (0.5–1.5 mg/kg IV), *or*
	  • Ketamine (5.5 mg/kg IV) and diazepam (0.28 mg/kg IV)
	• If hydrated and stable, give the following:
	  • Propofol (2–6 mg/kg IV), *or*
	  • Alfaxalone (2–3 mg/kg IV)

Maintenance	• Isoflurane or sevoflurane, *plus*
	  • Fentanyl (2–10 µg/kg IV PRN in dogs; 1–4 µg/kg IV PRN in cats) for short-term pain relief, *plus*
	  • Fentanyl CRI (1–5 µg/kg IV loading dose, then 2–30 µg/kg/h IV), *or*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Oxymorphone (0.05–0.2 mg/kg IV PRN), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV PRN), *plus*
	  • Ketamine (low dose) (0.5–1 mg/kg IV)
	• For hypotension (to keep MAP 60–80 mm Hg) give phenylephrine, ephedrine, norepinephrine, or dopamine as needed

Fluid needs	• Two large IV cephalic or jugular catheters • 10–20 mL/kg/h if open abdomen with higher evaporative losses, plus 3 × EBL; higher rates of fluids are necessary if dehydration not corrected preoperatively and animal is hypotensive • Consider colloids if persistent hypotension

Continued

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TABLE 18.4 Anesthetic Considerations for the Patient With an Acute Abdomen—cont'd

Monitoring	• Blood pressure • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature • U/O
Blocks	Epidural: • Morphine (0.1 mg/kg preservative free) *or* • Buprenorphine (0.003–0.005 mg/kg diluted in saline) • Avoid local anesthetics for spinal or epidural Incisional: • Lidocaine (<5 mg/kg in dogs; 2–4 mg/kg in cats), *or* • Bupivacaine (<2 mg/kg)
Postoperative Considerations	
Analgesia	• Fentanyl CRI (1–10 µg/kg IV loading dose, then 2–20 µg/kg/h IV), *or* • Morphine[b] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.05–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h in cats) if no hypotension, *or* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats), *or* • Hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs), *or* • Oxymorphone (0.05–0.2 mg/kg IV PRN), *or* • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM q4–8h or 0.01–0.02 mg/kg OTM q6–12h in cats), *plus* • ± Ketamine CRI (2 µg/kg/min IV; if no previous loading dose, give 0.5 mg/kg IV prior to CRI) • Avoid NSAIDs in patients with hypotension
Monitoring	• SpO_2 • Blood pressure • HR • Respiratory rate • Temperature • U/O • ECG
Bloodwork	• HCT if significant blood loss • Repeat abnormal preoperative bloodwork • Serial blood glucose checks if necessary • Blood gas if available
Estimated pain score	• Moderate to moderately severe

[a]Monitor for hyperthermia in cats.
[b]Give slowly to prevent histamine release.
[c]Buprenorphine is a better analgesic than morphine in cats.
BUN, Blood urea nitrogen; *Cr,* creatinine; *CRI,* constant-rate infusion; *EBL,* estimated blood loss; ECG, electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *MAP,* mean arterial pressure; *NSAID,* nonsteroidal antiinflammatory drug; *PRN,* as needed; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein; *U/O,* urine output.

the stomach at the cardiac ostium. The fundus is dorsal to the cardiac ostium, and although relatively small in carnivores, it is easy to identify on radiographs because it is typically filled with gas. The body of the stomach (the middle one-third) lies against the left lobes of the liver. The pyloric antrum is funnel shaped and opens into the pyloric canal. The pyloric ostium is the end of the pyloric canal that empties into the duodenum.

NOTE The gastric mucosa accounts for half of the stomach's weight. You can easily separate the mucosa from the submucosa and serosa when raising flaps or making partial-thickness incisions during a gastropexy or pyloromyotomy.

The gastric (lesser curvature) and gastroepiploic (greater curvature) arteries supply the stomach and are derived from the celiac artery. The short gastric arteries arise from the splenic artery and supply the greater curvature. The portion of the lesser omentum that passes from the stomach to the liver is the hepatogastric ligament. The stomach of the beagle holds more than 500 mL of fluid when distended (a mature cat's stomach may hold 300–350 mL). When the stomach is highly distended, it can be palpated beyond the costal arch.

NOTE The short gastric vessels are often avulsed in animals with GDV, which typically accounts for the intraabdominal hemorrhage seen in these cases (see also the discussion of GDV on p. 423).

SURGICAL TECHNIQUES

Gastric surgery is often performed in small animals. Generally, performing a gastrotomy is safer than performing an esophagotomy or enterotomy. Peritonitis is uncommon after gastrotomy if proper techniques are used. Stricture or obstruction is also rare. Billroth procedures are more difficult and may be associated with severe complications.

Gastroscopy

Endoscopic removal of foreign bodies is preferred over surgical removal but requires appropriate endoscopic snares. Likewise,

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endoscopy is more sensitive than gastrotomy when looking for erosions, ulcers, *Physaloptera,* and other small lesions. However, it is imperative that the entire gastric mucosa be systematically examined, including the fundus and lower esophageal mucosa. Similarly, endoscopy is the preferred method for gastric mucosal biopsy because it allows one to obtain more tissue samples than surgery, which is important because gastric mucosal lesions can be very spotty. Scirrhous carcinomas, pythiosis, and submucosal lesions are important gastric lesions but cannot always be diagnosed with endoscopic biopsy. Rarely, intraoperative gastroscopy can be performed to help the surgeon find a mucosal lesion (e.g., ulcer) that is not obvious from the serosal surface. Endoscopic polypectomy using electrosurgery can performed when polyps are causing outflow obstruction or are inflamed.

Gastrotomy

The most common indication for gastrotomy in dogs and cats is removal of a foreign body (see p. 416). *Make a ventral midline abdominal incision from the xiphoid to the pubis. Use Balfour retractors to retract the abdominal wall and provide adequate exposure of the GI tract. Inspect the entire abdominal contents before incising the stomach. To reduce contamination, isolate the stomach from remaining abdominal contents with moistened laparotomy sponges. Place stay sutures to assist in manipulation of the stomach and help prevent spillage of gastric contents. Make the gastric incision in a hypovascular area of the ventral aspect of the stomach, between the greater and lesser curvatures (Fig. 18.66). Make sure the incision is not near the pylorus, or closure of the incision may cause excessive tissue to be enfolded into the gastric lumen, resulting in outflow obstruction. Make a stab incision into the gastric lumen with a scalpel (Fig. 18.67A) and enlarge the incision with Metzenbaum scissors (see Fig. 18.67B). Use suction to aspirate gastric contents and reduce spillage. Close the stomach with 2-0 or 3-0 absorbable suture material (e.g., polydioxanone, polyglyconate) in a two-layer inverting seromuscular pattern (see Fig. 18.67C). Include serosa, muscularis, and submucosa in the first layer, using a Cushing or simple continuous pattern, then follow it with a Lembert or Cushing pattern that incorporates the serosal and muscularis layers (see Fig. 18.67D). As an alternative, close the mucosa in a simple continuous suture pattern as a separate layer to reduce postoperative bleeding. Before closing the abdominal incision, substitute sterile instruments and gloves for those contaminated by gastric contents. Whenever you remove a gastric foreign body, be sure to check the entire intestinal tract for additional material that could cause an intestinal obstruction.*

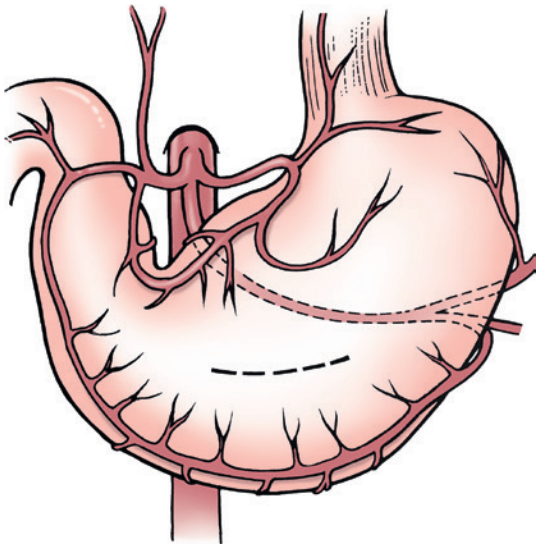


FIG. 18.66 Preferred location of gastrotomy incisions.

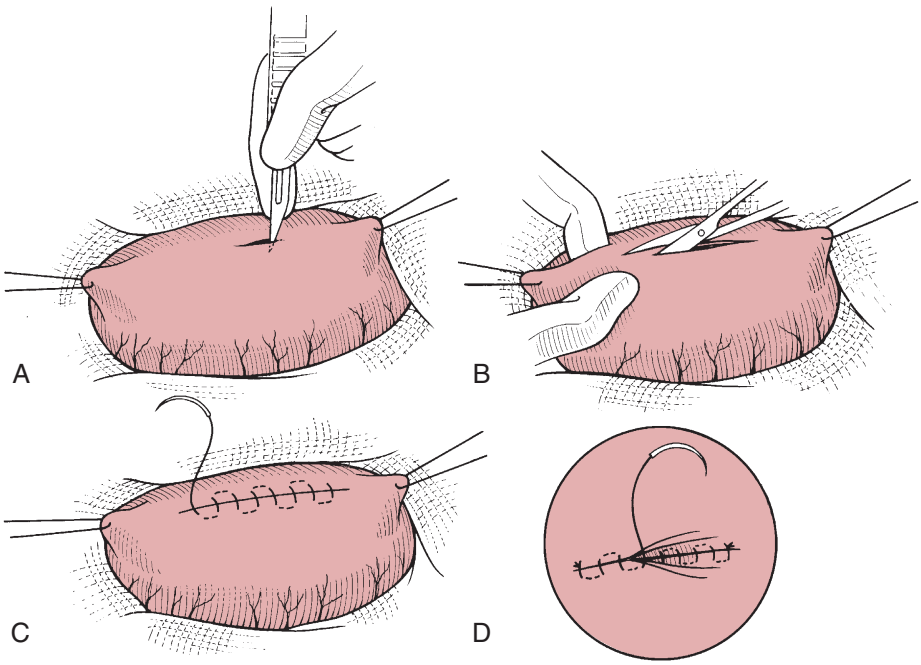


FIG. 18.67 Gastrotomy. (A) Make a stab incision into the gastric lumen with a scalpel. (B) Enlarge the incision with Metzenbaum scissors. (C–D) Close the stomach with a two-layer inverting seromuscular suture pattern.

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Partial Gastrectomy and Invagination of Gastric Tissue

Partial gastrectomy is indicated when necrosis, ulceration, or neoplasia involves the greater curvature, or middle portion, of the stomach. Necrosis of the greater curvature is primarily associated with GDV and may be treated by resection or invagination. Invagination does not require opening of the gastric lumen (Fig. 18.68); however, obstruction from excessive intraluminal tissue and excessive bleeding is possible. Assess the extent of necrosis by observing the serosal color, gastric wall texture, vascular patency, and bleeding on incision; however, in many cases it is difficult to determine tissue viability with these techniques (see p. 436 for a discussion of methods for determining tissue viability). Necrotic tissue may range in color from gray-green to black and often feels thin. A full-thickness incision can be made into the suspected necrotic tissue to assess arterial bleeding. IV fluorescein dye has not proved to be an accurate method of determining gastric viability in dogs with GDV. Generally, if you question the viability of the gastric tissue, remove it or invaginate it. Failure to remove or invaginate necrotic tissue may result in perforation, peritonitis, and death. Melena is commonly observed for a few days after gastric invagination.

NOTE Do not use mucosal color to predict gastric viability; the mucosa is commonly black in dogs with GDV because of vascular obstruction. Damage to the mucosa may predispose these animals to gastric ulceration.

To remove the greater curvature of the stomach, ligate branches of the left gastroepiploic vessels or short gastric vessels (or both) along the section of the stomach to be removed (Fig. 18.69). Excise the necrotic tissue, leaving a margin of normal, actively bleeding tissue to suture. Close the stomach in a two-layer inverting pattern using 2-0 or 3-0 absorbable suture (e.g., polydioxanone, polyglyconate). Incorporate the submucosa, muscularis, and serosal layers in a Cushing or simple continuous pattern in the first layer. Then use a Cushing or Lembert pattern to invert the serosa and muscularis over the first layer. As an alternative, you may use a TA stapling device to close the incision. To invaginate necrotic

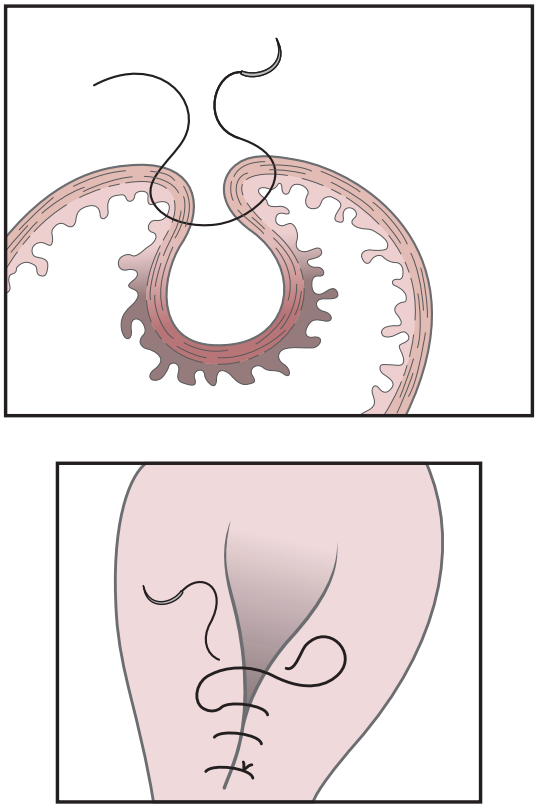


FIG. 18.68 Invagination of necrotic gastric tissue.

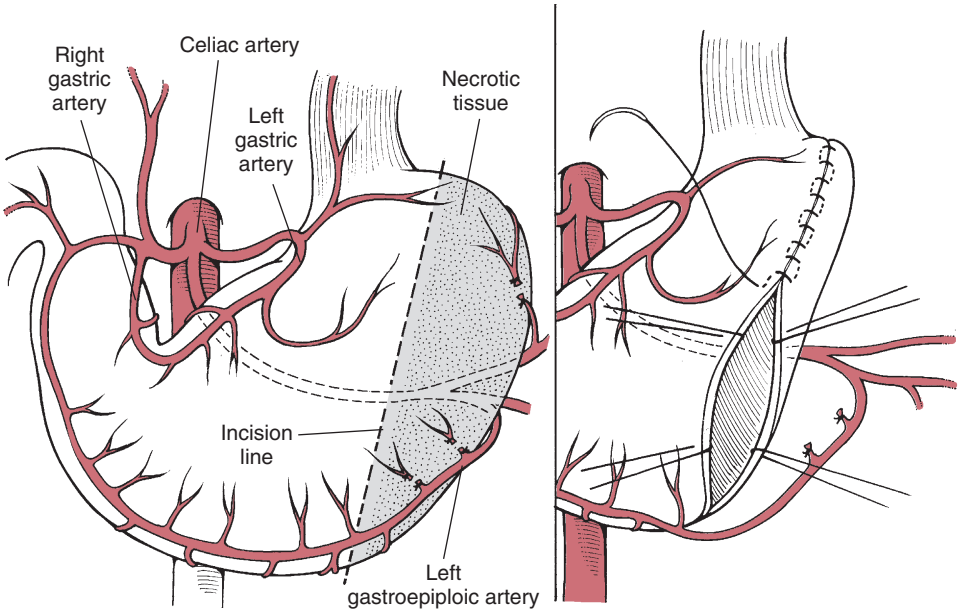


FIG. 18.69 To remove the greater curvature of the stomach, ligate branches of the left gastroepiploic vessels or short gastric vessels (or both) and excise the necrotic tissue. Close the stomach with a two-layer inverting suture pattern.

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tissue, use a simple continuous suture pattern followed by an inverting pattern. Place sutures in healthy gastric tissue on both sides of the tissue that is to be invaginated, bringing the healthy tissue over the top of the necrotic tissue. Make sure the sutures are placed in healthy tissues to prevent dehiscence.

Removal of neoplasia (see p. 430) or ulceration (see p. 427) of the greater or lesser curvature is similar to that described for necrotic tissue. Most neoplasms in the gastric body, except for leiomyomas and leiomyosarcomas, have metastasized by the time they are diagnosed. If the abnormal tissue involves the dorsal or ventral aspect of the stomach, an elliptic incision encompassing the lesion and some adjacent normal tissue is used. Closure is as for a simple gastrotomy. Occasionally the extent of the lesion requires resection of both the dorsal and ventral walls of the stomach. *In such cases, ligate branches of the right and left gastric artery and vein (lesser curvature) and left gastroepiploic artery and vein (greater curvature) and remove the omental attachments. After removal of the suspect tissues, perform a two-layer end-to-end anastomosis of the stomach. If the luminal circumferences are of disparate size, the larger circumference can be partly closed using a two-layer suture pattern (Fig. 18.70B). Close the mucosa and submucosa of the dorsal surface of the stomach in a simple continuous pattern using 2-0 or 3-0 absorbable suture (e.g., polydioxanone, polyglyconate), then with the same suture close the ventral aspect. Suture the serosa and muscularis layers in an inverting pattern (e.g., Cushing or Lembert).*

Temporary Gastrostomy

Temporary gastrostomy is used to decompress the stomach and is occasionally indicated in dogs with GDV until more definitive surgery can be performed but is rarely done.

Pylorectomy With Gastroduodenostomy (Billroth I)

AP Removal of the pylorus and gastroduodenostomy is indicated for neoplasia (see p. 430), outflow obstruction caused by pyloric muscular hypertrophy (see p. 425), or ulceration of the gastric outflow tract (see p. 427). If neoplasia is present, at least 1- to 2-cm margins of normal tissue should be removed with the abnormal tissue. The margins of the resected tissue should be evaluated histologically for evidence of neoplasia. If the common bile duct has been damaged, a cholecystoduodenostomy or cholecystojejunostomy may be required (see p. 574). If the pancreatic ducts are inadvertently ligated, supplementation with pancreatic enzymes may be necessary postoperatively.

NOTE Use extreme care when incising in the pyloric area to avoid damaging the common bile duct where it traverses the lesser omentum.

Identify the common bile duct and pancreatic ducts, then place stay sutures in the proximal duodenum and pyloric antrum. If increased caudoventral retraction of the pylorus is desired, identify and transect a portion of the hepatogastric ligament. Ligate branches of the right gastric and right gastroepiploic artery and vein to the affected tissues, and remove the omental and mesenteric attachments (see Fig. 18.70A). Alternatively, use a vessel sealer divider to transect branches along the segment to be excised. Use noncrushing (Doyen) forceps or fingers to occlude the stomach and duodenum proximal and distal to the area to be resected. With Metzenbaum scissors or a scalpel blade, excise the area of pylorus to be removed and inspect the remaining edges to ensure that all abnormal tissue has been excised. If the gastric and duodenal lumens are markedly different in size, incise the duodenum at an angle or partly close the antrum (see Fig. 18.70B). Perform a one- or two-layer end-to-end anastomosis of the pyloric antrum to the duodenum using 2-0 or 3-0 absorbable suture material (polydioxanone, polyglyconate, or poliglecaprone 25) in a simple continuous, crushing, or simple interrupted pattern. There does not appear to be a difference in the prevalence of postoperative leakage and incisional dehiscence between one- and two-layer closures. *Close the far (dorsal) aspect of the incision first (see Fig. 18.70C), followed by the near (ventral) aspect (see Fig. 18.70D). Avoid inverting excessive tissue, which might reduce the diameter of the gastric outflow tract.*

Partial Gastrectomy With Gastrojejunostomy (Billroth II)

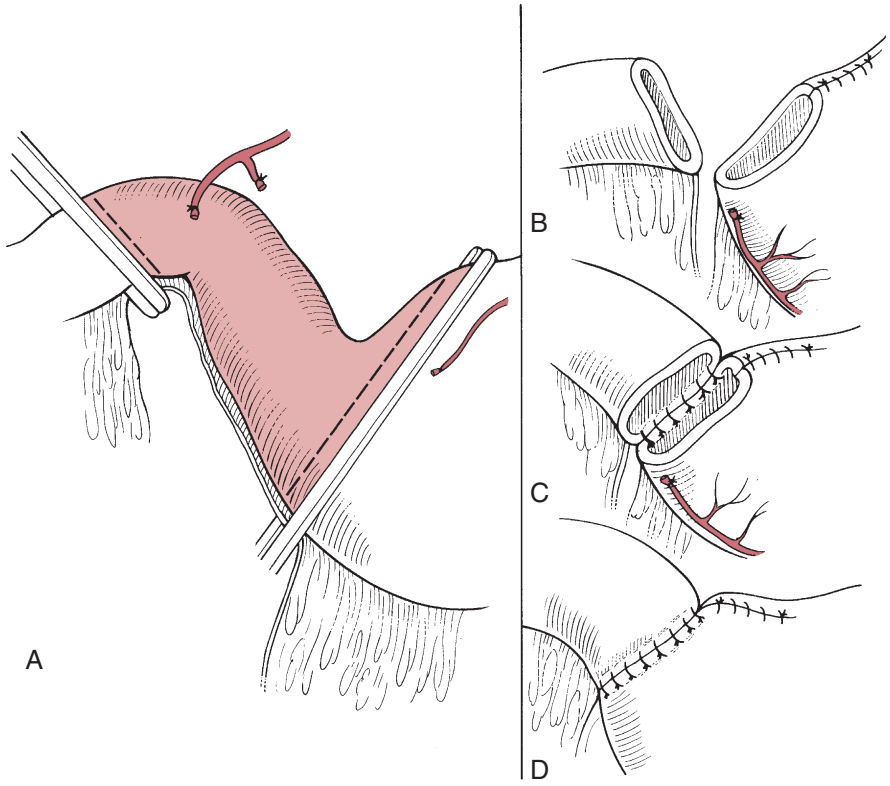
AP If the extent of the lesion precludes end-to-end anastomosis of the pyloric antrum to the duodenum, consider a Billroth II procedure. If only mucosal hypertrophy is present, a Y-U pyloroplasty (discussed later) is easier to perform and is effective. Before undertaking this procedure, make sure that there is no gross evidence of metastasis (see p. 430 for information regarding gastric neoplasia). In most cases a cholecystojejunostomy or cholecystoduodenostomy is required in addition to the gastrojejunostomy (see p. 574). Exocrine insufficiency may occur if the pancreatic ducts are damaged. Exocrine plus endocrine pancreatic insufficiency (i.e., diabetes mellitus) may occur as a result of pancreatic resection or severe damage to the pancreatic blood supply.

The procedure is similar to a Billroth I except that the distal stomach and proximal duodenum are closed after pylorectomy, and the jejunum is attached with a side-to-side anastomosis to the diaphragmatic surface of the stomach. *Resect the pylorus, antrum, and proximal duodenum as described earlier, ligating appropriate branches of the right and left gastric and gastroepiploic vessels (Fig. 18.71A). Close the duodenal and pyloric antral stumps in a two-layer pattern. For the first layer, incorporate the mucosa and submucosa in a simple interrupted or simple continuous pattern using 2-0 or 3-0 absorbable suture (see Fig. 18.71B). Then use an inverting suture pattern (e.g., Lembert) in the seromuscular layer. Identify an avascular area between the gastric incision and greater curvature. Bring a loop of proximal jejunum to the selected site and attach it to the stomach with stay sutures. Suture the seromuscular layers of the stomach and intestine together using a simple continuous pattern with 2-0 or 3-0 absorbable suture (see Fig. 18.71C). Make full-thickness, longitudinal incisions into the stomach and intestinal lumens near the suture line (see Fig. 18.71D). Suture the mucosa and submucosa of the stomach to the intestine in a continuous pattern using 3-0 or 4-0 absorbable suture (see Fig. 18.71E). Next, place a continuous suture pattern in the serosa and muscularis.* Use of stapling devices has also been described for stump closure and creation of the gastrojejunostomy.

Pyloromyotomy and Pyloroplasty

Pyloromyotomy and pyloroplasty increase the diameter of the pylorus and are used to correct gastric outflow obstruction (i.e., chronic antral mucosal hypertrophy or pyloric stenosis). These procedures must be done with care as errors can be difficult or impossible to revise. They should not be performed routinely in dogs without evidence of pyloric dysfunction (e.g., most dogs with GDV) because they can slow gastric emptying. Gastric outflow procedures that increase the diameter of the pyloric

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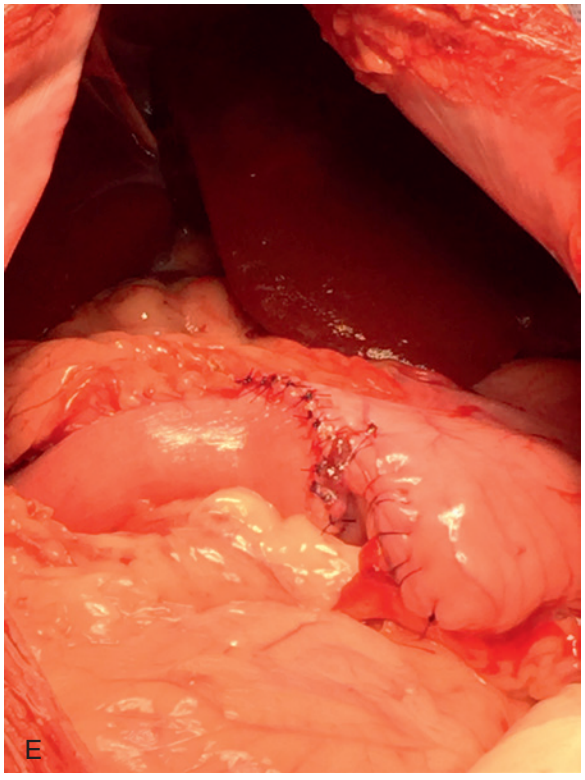


FIG. 18.70 Billroth I procedure. (A) Ligate the vasculature, remove the omental and mesenteric attachments, and excise the area of the pylorus to be removed. (B) If the gastric and duodenal lumens are markedly different in size, incise the duodenum at an angle or partly close the antrum. (C) Close the far (dorsal) aspect of the incision first. (D) Close the near (ventral) aspect. (E) Appearance of the stomach following a Billroth I procedure.

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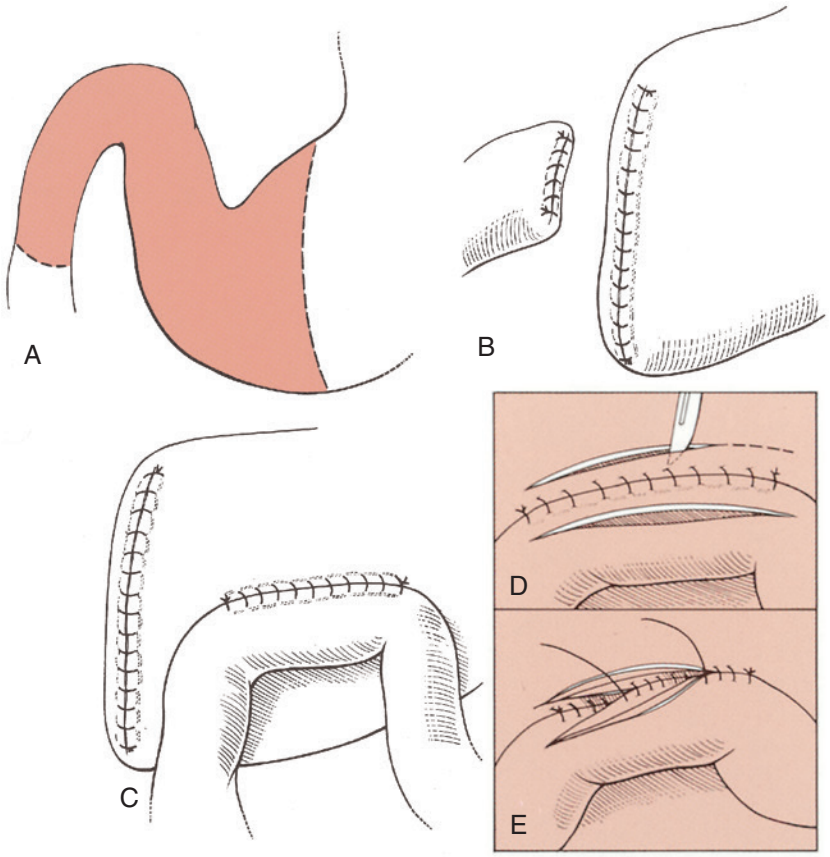


FIG. 18.71 Billroth II procedure. (A) Resect the pylorus, antrum, and proximal duodenum. (B) Close the duodenal and pyloric antral stumps with a two-layer suture pattern. (C) Bring a loop of proximal jejunum to the stomach and suture the seromuscular layers of the stomach and intestine together. (D) Make full-thickness, longitudinal incisions into the stomach and intestinal lumens. (E) Suture the mucosa and submucosa of the stomach to the intestine with a continuous suture pattern.

BOX 18.21 Prokinetic Drugs

Cisapride (Pharmacy Compounded)
Dogs: 0.1–0.5 mg/kg PO q8–24h
Cats: 2.5–5 mg/cat PO q8–12h

Ranitidine
Dogs: 2 mg/kg IV, PO q8–12h
Cats: 2.5 mg/kg IV q12h or 3.5 mg/kg PO q12h

Erythromycin
0.5–1 mg/kg PO or IV q8–12h

Metoclopramide (Reglan)
0.25–0.5 mg/kg PO, IV or SC q6–24h

IV, Intravenous; *PO,* orally; *SC,* subcutaneous.

lumen seem to favor early passage of viscous, nonhomogeneous, and hyperosmolar gastric contents into the duodenum. This early passage may overstimulate the enterogastric reflex, prematurely inhibiting antral motor activity and delaying gastric emptying. In addition, gastroduodenal reflux may occur if pyloric function is altered by surgery. Metoclopramide, cisapride, ranitidine, or low-dose erythromycin (Box 18.21) may stimulate gastric emptying.

Fredet-Ramstedt Pyloromyotomy

Fredet-Ramstedt pyloromyotomy is the simplest and easiest of these procedures. It does not allow inspection or biopsy of the pyloric mucosa and probably provides only temporary benefit because healing may reduce the size of the lumen.

Hold the pylorus between the index finger and thumb in the nondominant hand. Select a hypovascular area of the ventral pylorus and make a longitudinal incision through the serosa and muscularis but not through the mucosa (Fig. 18.72). Make sure the muscularis layer is completely incised to allow the mucosa to bulge into the incision site. If the mucosa is inadvertently penetrated, close it with interrupted sutures of 2-0 or 3-0 absorbable suture material.

Heineke-Mikulicz Pyloroplasty

Heineke-Mikulicz pyloroplasty allows limited exposure of the pyloric mucosa for biopsy and is easy to perform. *Make a full-thickness, longitudinal incision in the ventral surface of the pylorus (Fig. 18.73). Place traction sutures at the center of the incision, and orient the incision transversely. Suture the transverse incision in a one-layer pattern (simple interrupted or crushing) using 2-0 or 3-0 absorbable suture. Place the sutures carefully so that the incision edges are properly aligned and tissue inversion is avoided.*

Y-U Pyloroplasty

The Y-U pyloroplasty allows greater accessibility for resection of the pyloric mucosa in dogs with mucosal hypertrophy while

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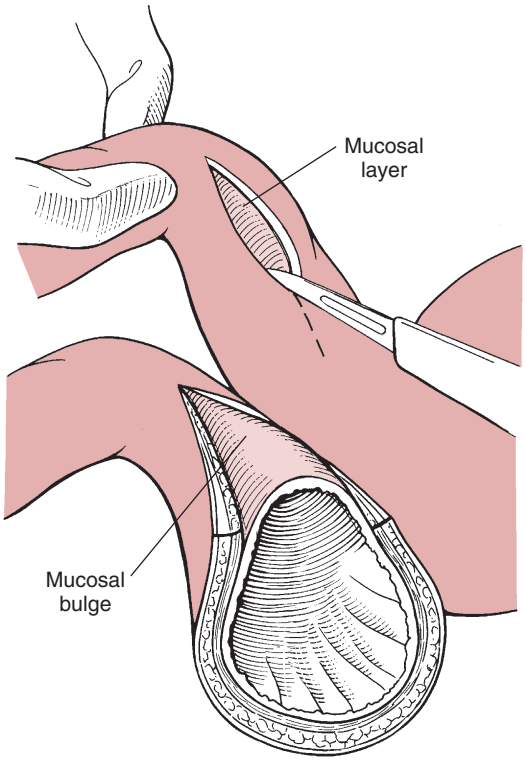


FIG. 18.72 Fredet-Ramstedt pyloromyotomy.

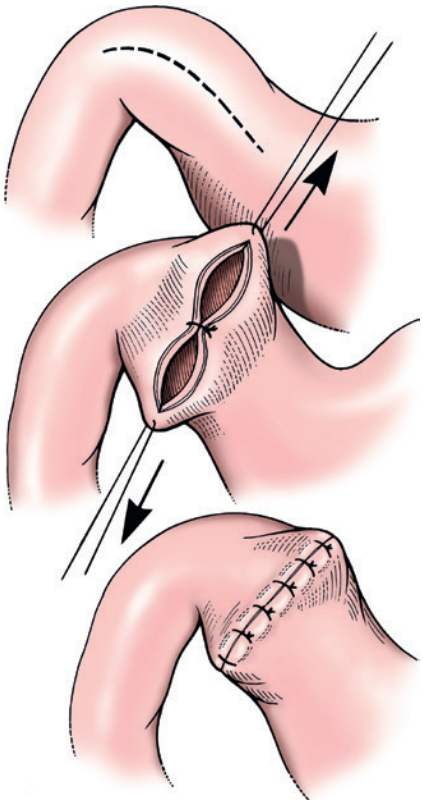


FIG. 18.73 Heineke-Mikulicz pyloroplasty.

simultaneously increasing the luminal diameter of the outflow tract. *Make a longitudinal incision (limb) in the serosa overlying the ventral pylorus and extend it into the stomach by making two incisions (arms) that run parallel to the lesser and greater curvature of the stomach (creating a Y-shaped incision) (Fig. 18.74A). Make sure the angle of the Y is not overly narrow, or necrosis may result. The limbs and arms of the Y-shaped incision should be approximately the same length. Make a full-thickness incision. Inspect the mucosa and resect it if necessary. If the mucosa is resected, appose the remaining mucosal edges in a continuous pattern using absorbable 3-0 to 4-0 suture before closing the Y incision. Suture the base of the antral flap to the distal end of the duodenal incision in a simple interrupted pattern using absorbable 2-0 or 3-0 suture, creating a U-shaped closure (see Fig. 18.74B). Close the remainder of the incision (the limbs) with simple interrupted sutures (see Fig. 18.74C). Make sure that tissue approximation is adequate to prevent leakage and that minimal tissue has been enfolded into the pyloric lumen.*

NOTE To reduce necrosis of the pointed tip of the gastric tissue flap, you may wish to excise the point of the Y before suturing it.

Gastropexy

Gastropexy techniques are designed to permanently adhere the stomach to the body wall. The most common indications are GDV (pyloric antrum to right body wall) and hiatal herniation (fundus to left body wall). Numerous gastropexy techniques have been described. Although the strength and extent of adhesions created by these techniques differ, all of them (when properly performed) prevent movement of the stomach. Careful attention to the normal anatomy and orientation of the stomach and descending duodenum is important to avoid postoperative gastric outflow abnormalities due to gastric malpositioning.[19]

NOTE To create a permanent adhesion, the gastric muscle must be in contact with the muscle of the body wall; intact gastric serosa does not form permanent adhesions to an intact peritoneal surface.

A technique for gastropexy has been described in which the stomach is incorporated into the abdominal incision during closure (ventral midline gastropexy). The recurrence rate after this procedure seems to be similar to other gastropexy techniques and the concern that the adherence of the stomach to the midline abdominal incision would interfere with subsequent celiotomy has not been borne out.

Tube Gastropexy

Tube gastropexy (gastrostomy) is quick and relatively simple (Fig. 18.75). In addition, it allows postoperative gastric decompression and placement of medications directly into the stomach in inappetent animals. The tube should be left in place for 7 to 10 days to form a permanent adhesion. Although this may lengthen the postoperative hospitalization period compared with other techniques, the tube can be capped and secured against the trunk and the patient sent home on oral feeding. The risk of leakage is minimal if proper technique is used; however, improper placement may result in peritonitis.

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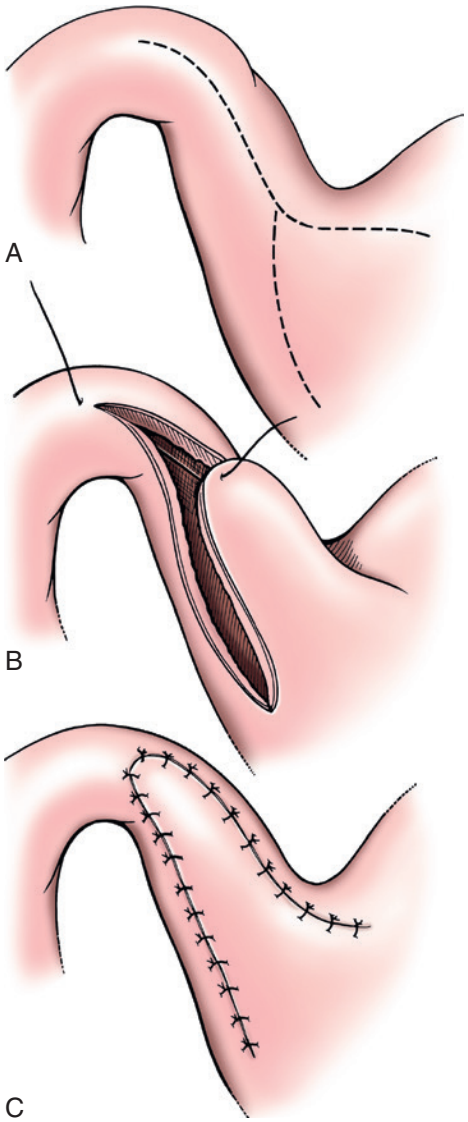


FIG. 18.74 Y-U pyloroplasty. (A) Make a Y-shaped incision in the pylorus and pyloric antrum. (B) Begin by suturing the base of the antral flap to the distal point of the duodenal incision. (C) Close the remainder of the incision in a U-shape.

Make a stab incision into the right abdominal wall caudal to the last rib and 4 to 10 cm lateral to the midline. Place a Foley catheter (18–30 Fr) or a mushroom-tipped silicone catheter through the stab incision. Select a site in a hypovascular region of the seromuscular layer of the ventral surface of the pyloric antrum where the balloon of the catheter will not obstruct gastric outflow. Place a purse-string suture of 2-0 absorbable suture (e.g., polydioxanone, polyglyconate) at this site. Make a stab incision through the purse-string suture and insert the catheter tip into the gastric lumen (Fig. 18.76A). (Note: the catheter can be placed through the omentum before entry into the stomach so that the omentum is secured between the stomach and body wall, or the omentum can be wrapped around the site after the stomach has been secured to the body wall.) *If using a Foley catheter, inflate the bulb with saline (not air), and secure the purse-string suture around the tube. Preplace three or four absorbable sutures between the pyloric antrum and the body wall where the tube exits (see Fig. 18.76B). Avoid penetrating the catheter or balloon when placing the sutures. Draw the stomach to the body wall by placing traction on the catheter, and tie the preplaced sutures (see Fig. 18.76C). Secure the tube to the skin with a Roman sandal suture pattern (see Fig. 30.10), but avoid penetrating it with a suture. Place a bandage around the dog's abdomen and over the tube to prevent premature removal (use an Elizabethan collar if necessary). Leave the tube in place for 7 to 10 days, then deflate the balloon and remove it. Leave the skin incision open to facilitate drainage. Place a light bandage over the open wound if desired.*

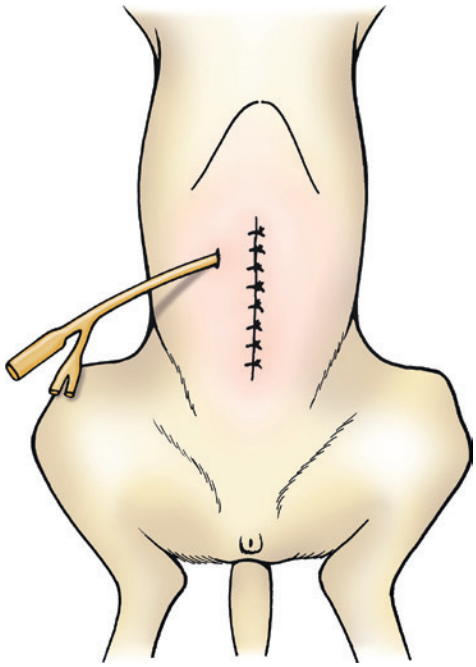


FIG. 18.75 Tube gastropexy using a Foley catheter.

The Roman sandal suture pattern is preferred over friction sutures tied to the skin along the course of the tube, as the Roman sandal pattern requires more force to failure and it is the suture that tends to fail, rather than tube slippage. Silicone tubing has a higher force to failure than latex for gastrostomy tubes.

NOTE Cut the tip off the end of the Foley catheter if food or viscous fluids are to be injected through it. Inflate the balloon with saline, not air.

Circumcostal Gastropexy

Circumcostal gastropexy forms a stronger adhesion than most other techniques but is technically more challenging (Box 18.22). Because the lumen of the stomach is not entered, the risk of gastric leakage and abdominal contamination is diminished compared with tube gastropexy. Possible complications associated with circumcostal gastropexy include pneumothorax and rib fracture.

Make either a one- or two-layer hinged flap (approximately 5–6 cm long in large dogs) by incising through the seromuscular layer of the pyloric antrum (Fig. 18.77A). Do not incise the gastric mucosa or enter the lumen (if this occurs, suture the mucosa with

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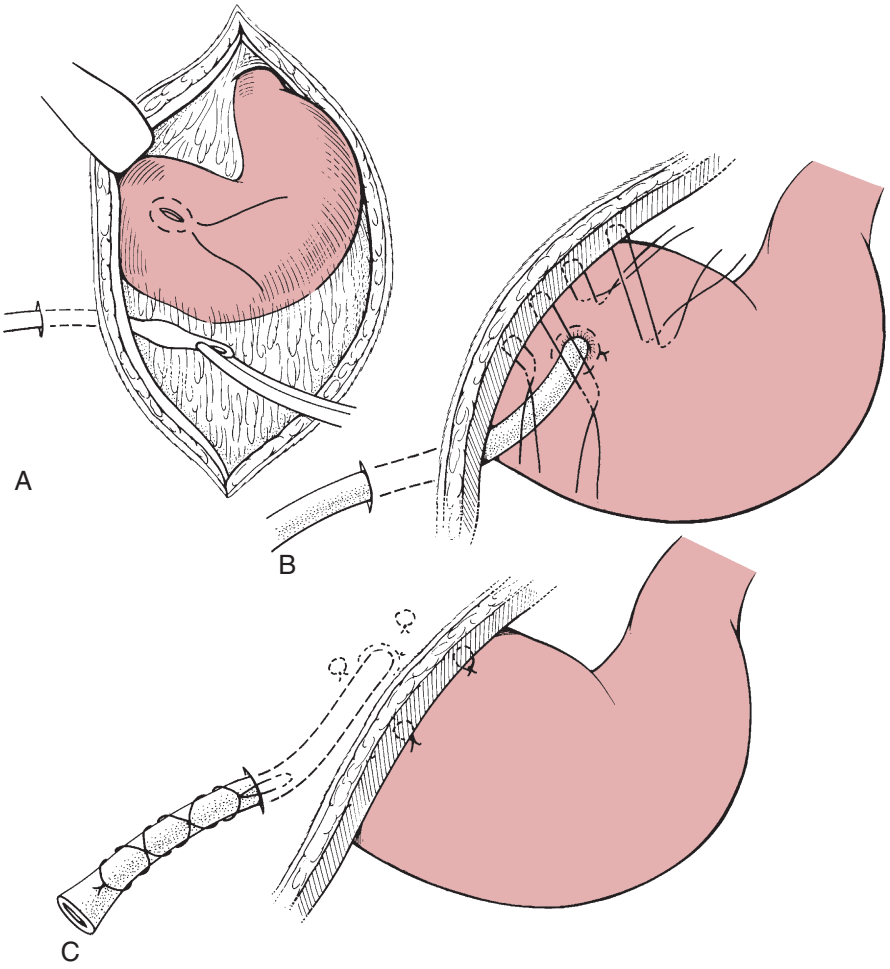


FIG. 18.76 Tube gastropexy using a Foley catheter. (A) Insert the tip of the catheter into the gastric lumen through a purse-string suture. (B) Preplace three or four absorbable sutures between the pyloric antrum and the body wall where the tube exits. (C) Draw the stomach to the body wall and tie the preplaced sutures. Secure the tube to the skin with a Roman sandal suture pattern.

BOX 18.22 Advantages and Disadvantages of Circumcostal Gastropexy

Advantages

- Strong adhesion to body wall
- Gastric lumen not opened

Disadvantages

- More difficult to perform
- Risk of pneumothorax
- Possibility of rib fractures
- Does not provide direct access to gastric lumen if postoperative decompression is necessary

3-0 absorbable suture). Elevate the flap by dissecting under the muscularis. If a one-hinged flap is made, place the hinge toward the lesser curvature. Make a 5- to 6-cm incision over the 11th or 12th rib at the level of the costochondral junction (see Fig. 18.77B). Make sure the incision does not penetrate the diaphragmatic attachments to the body wall, causing pneumothorax. Form a tunnel under the rib using a Carmalt clamp or hemostat (see Fig. 18.77C). Place stay sutures on the flap (if using a two-flap technique, place the sutures on the flap nearest the lesser curvature). Pass the gastric antral flap craniodorsal under the rib (see Fig. 18.77D), and suture it with 2-0 absorbable suture to the original gastric margin (one-flap technique; Fig. 18.78A) or the other flap (two-flap technique; see Fig. 18.78B).

Muscular Flap (Incisional) Gastropexy

Muscular flap (incisional) gastropexy is easier than circumcostal gastropexy and avoids the possible complications associated with tube gastropexy (Box 18.23). *Make an incision in the seromuscular layer of the gastric antrum. Then make an incision in the right ventrolateral abdominal wall by incising the peritoneum and internal fascia of the rectus abdominis or transverse abdominis muscles (Fig. 18.79A). Suture the edges of the incisions in a simple continuous pattern using 2-0 absorbable or nonabsorbable suture (see Fig. 18.79B). Make sure the muscularis layer of the stomach is in contact with the abdominal wall muscle (see Fig. 18.79C). Suture the cranial margin first, then the caudal margin.* As an alternative, you may raise flaps in the stomach and body wall to increase the extent of muscle contact between these tissues.

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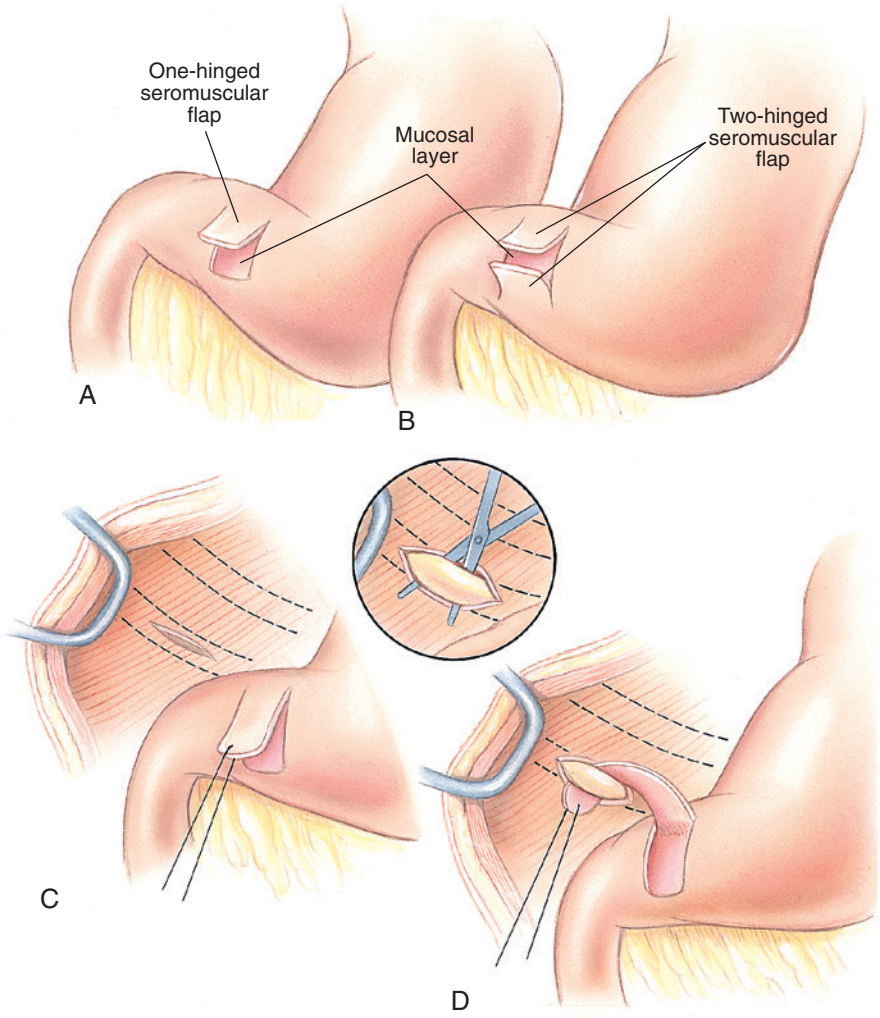


FIG. 18.77 Circumcostal gastropexy. (A) Make a single- or double-layer hinged seromuscular flap in the pyloric antrum. (B) Make an incision over the 11th or 12th rib at the level of the costochondral junction. (C) Form a tunnel under the rib using a Carmalt clamp or hemostat. (D) Pass the gastric antral flap craniodorsal under the rib and suture it to the original gastric margin or to the other flap (see Fig. 18.78).

BOX 18.23 Advantages and Disadvantages of Muscular Flap and Belt-Loop Gastropexy

Advantages

- Easy and quick to perform
- Gastric lumen not opened

Disadvantages

- Less strong than circumcostal gastropexy
- Does not provide direct access to gastric lumen if postoperative decompression is necessary

Alternatively, a horizontal incision may be made in the pyloric antrum (Fig. 18.80A), sutured to an incision crossing the fibers of the transversus abdominus (see Fig. 18.80B). The transverse orientation of the incisions may lessen the difficulty of the procedure for a surgeon without an assistant. Suture the dorsal, or aborad, aspect of the antral incision to the transversus muscle first (see Fig. 18.80C) followed by the ventral, or orad, aspect of the incision (see Fig. 18.80D).

Two studies have shown that incisional gastropexy is adequate for the prevention of recurrent GDV or the prevention of GDV in normal dogs; however, it will not prevent gastric dilatation.[20,21]

Belt-Loop Gastropexy

A belt-loop gastropexy is similar to a muscle flap gastropexy except that a single flap is elevated and passed beneath a tunnel created in the abdominal wall. It is technically simple and seems to result in adequate adhesions. *Elevate a seromuscular flap in the gastric antrum. Make two transverse incisions in the ventrolateral abdominal wall by incising the peritoneum and abdominal musculature (Fig. 18.81A). The incisions should be 2.5 to 4 cm apart and 3 to 5 cm long. Create a tunnel under the abdominal musculature with forceps. Place stay sutures in the edge of the antral flap and use them to pass the flap from cranial to caudal under the*

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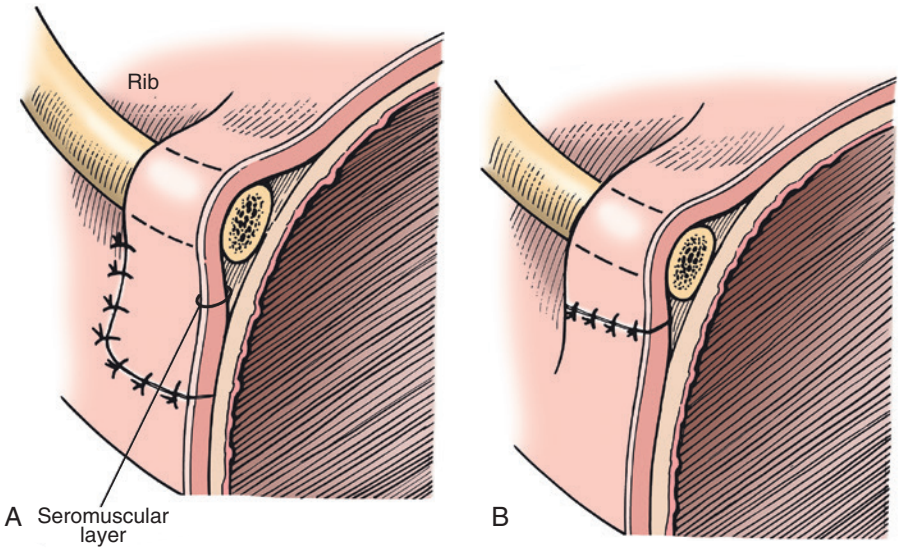


FIG. 18.78 (A) For a single-flap technique, suture the circumcostal flap to the original gastric margin. (B) For a double-flap technique, pass the seromuscular flap under the rib and suture it to the second flap.

muscular flap (see Fig. 18.81B). Suture the flap to its original gastric margin in a simple continuous pattern using 2-0 absorbable or nonabsorbable suture (see Fig. 18.81C) or skin staples. You may wish to place additional sutures between the body wall and the stomach to reduce tension on the gastropexy.

Gastrocolopexy

Gastrocolopexy has also been described as a method of preventing recurrence of GDV. In this technique, the greater curvature of the stomach is sutured to the transverse colon. This technique may result in a recurrence rate as high as 20%.

Laparoscopic-Assisted Prophylactic Gastropexy

This minimally invasive technique allows a rapid gastropexy. *With the dog in dorsal recumbency, place the first cannula just caudal to the umbilicus and place the second 10-mm cannula just right of midline, approximately 2 to 4 cm behind the last rib (make this incision parallel to the rib (Fig. 18.82A). Use Babcock forceps to grasp the antrum, midway between the greater and lesser curvatures and 6 to 8 cm back from the pylorus. Pull this portion of the stomach up to the end of the cannula, and then pull the cannula, Babcock forceps, and stomach out of the abdomen as one unit until the stomach wall can be seen protruding from the peritoneal cavity (see Fig. 18.82B). Enlarge the incision through which the cannula was placed to 4 to 5 cm. Grasp the gastric wall with Allis tissue forceps or Babcock forceps, and place stay sutures between it and the body wall (see Fig. 18.82C). Release the endoscopic Babcock forceps. Perform the gastropexy as described previously under Muscular Flap Gastropexy. Take care to not twist the antrum as it is brought up to and out of the incision.* It is best to reexamine the stomach laparoscopically before final closure to ensure that malpositioning did not take place.

Endoscopically Assisted Gastropexy

Endoscopically assisted gastropexy may be performed in dogs. *Place the dog in a 30-degree, left oblique recumbency. With the surgeon standing toward the dog's spine and the endoscopist positioned at the dog's ventrum (Fig. 18.83), pass the flexible endoscope into the stomach, and insufflate until rugal folds are minimized.* An assistant may be required to manually occlude the cervical esophagus during insufflation. *Palpate and indent the body wall caudal to the 13th rib and stomach at the proposed gastropexy site at the pyloric antrum. Pass polypropylene suture (0 or 2-0; see Fig. 18.83) percutaneously in a mattress suture fashion through the skin, body wall, pyloric antrum, and back through the body wall and skin. Temporarily tighten the suture with hemostatic forceps and place another suture in the same manner 4 to 5 cm aborad to the first suture.* These stay sutures guide the paracostal approach for the gastropexy, which is approached as for the laparoscopic-assisted gastropexy. *Incise the seromuscular layer of the pyloric antrum and suture it to the transversus abdominis muscle in two simple continuous patterns, avoiding the stay sutures. Remove the stay sutures and close the external abdominal oblique muscle in a simple continuous pattern using 2-0 PDS and routinely close the skin and subcutis. Decompress the stomach and remove the endoscope.*

Laparoscopic Sutured Gastropexy

Position the dog in dorsal recumbency, and establish an endoscope portal in the umbilical position and two operating ports 6 to 7 cm cranial to the umbilical port to the left and right of midline. Introduce and grasp the pyloric antrum with a 5-mm bowl grasper and retract the stomach to the right ventral abdominal wall. Use a coagulation hook probe to create four coagulated spots (0.5 seconds, 8 W power) on the peritoneal surface oriented cranial to caudal, separating each by 0.5 to 1 cm. Incise the skin approximately 4 cm over the coagulated region of the body wall under endoscopic guidance. Pass four metric PDS (1 USP) sutures on a 36-mm, half-circle needle through the body wall and seromuscular layer of the stomach in a mattress pattern, exiting the body wall through the skin incision. Coagulate the serosal surface of the stomach incorporated by the horizontal mattress suture. Place three more horizontal mattress sutures through the body wall and coagulated region of the stomach, separating each by 2 to 2.5 cm. Reduce abdominal insufflation and secure the sutures to form a gastropexy. Close the portal sites routinely.

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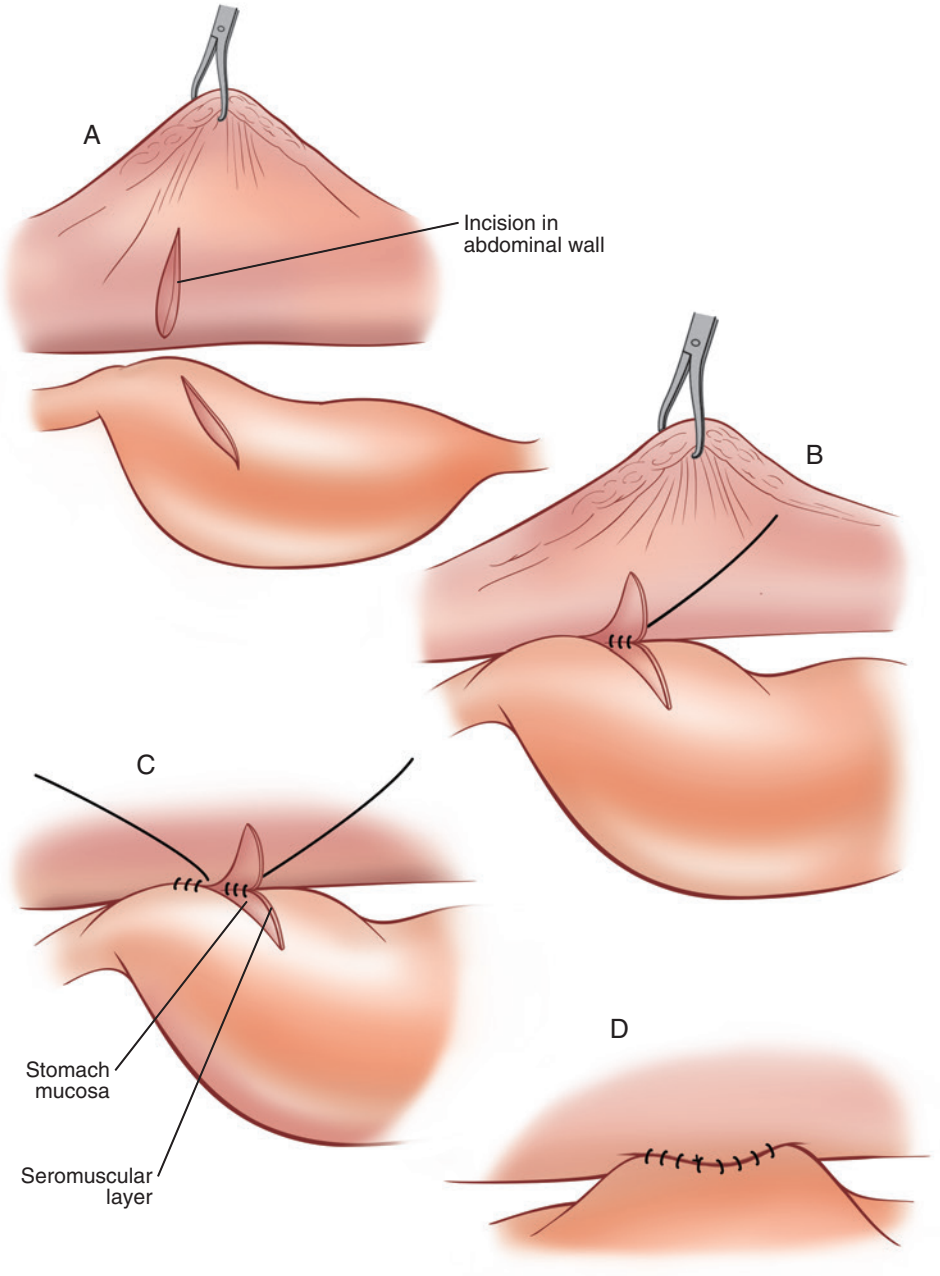


FIG. 18.79 Incisional gastropexy. (A) Incise the seromuscular layer of the pyloric antrum midway between the greater and lesser curvatures longitudinally. Incise the transversus muscle parallel to its fibers. (B) Suture the dorsal half of the cranial transversus and seromuscular layers in a simple continuous pattern. (C) Suture the dorsal half of the caudal transversus and seromuscular layers in a simple continuous pattern. (D) Appearance of the gastropexy following closure of the cranial then caudal ventral portions of the gastropexy.

Intracorporeally Sutured Laparoscopic Gastropexy

Intracorporeally sutured laparoscopic gastropexy may be performed. *Place an initial endoscope port in the infraumbilical site. Establish two instrument ports on the ventral midline with one 3 to 4 cm caudal to the xiphoid process and the second midway between the established ports on midline. Move the endoscope to the middle port. Place a mattress suture (2-0 nonabsorbable monofilament) through the body wall 2 to 3 cm caudal to the last rib and 5 to 8 cm lateral to the linea alba on the right side of the abdomen. Take a full-thickness bite of the pyloric antrum and exit through the body wall adjacent to the entry site. Incise the transversus abdominis and seromuscular layer of the stomach at the gastropexy site. Orient the incision parallel to the linea alba to facilitate visualization during suturing of the aborad then orad sides of the gastropexy.* The gastropexy may be done by hand with intracorporeal knot tying or using an automatic suture assist device (e.g., Endostitch).

Development of barbed suture has led to the knotless endoscopic gastropexy.[22,23] The technique is similar to that described previously; however, the suture is passed through a loop in the

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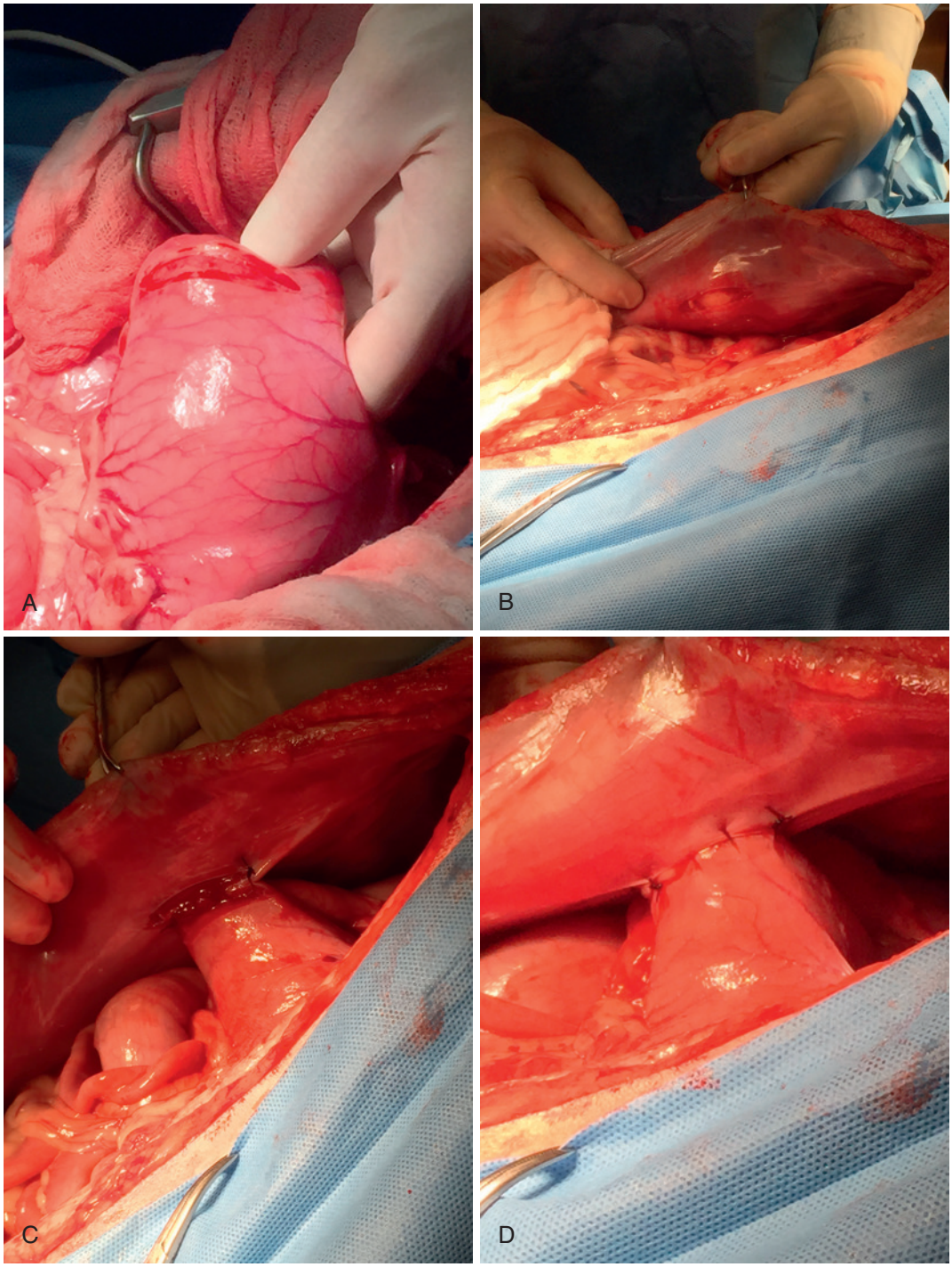


FIG. 18.80 Transverse incisional gastropexy. (A) Make a transverse incision through the seromuscular layer of the pyloric antrum. (B) Make a transverse incision in the adjacent transversus abdominus muscle. (C) Suture the dorsal aspect of the transversus muscle and gastric incisions with a simple continuous pattern. (D) Complete the gastropexy by suturing the ventral aspect of the incisions.

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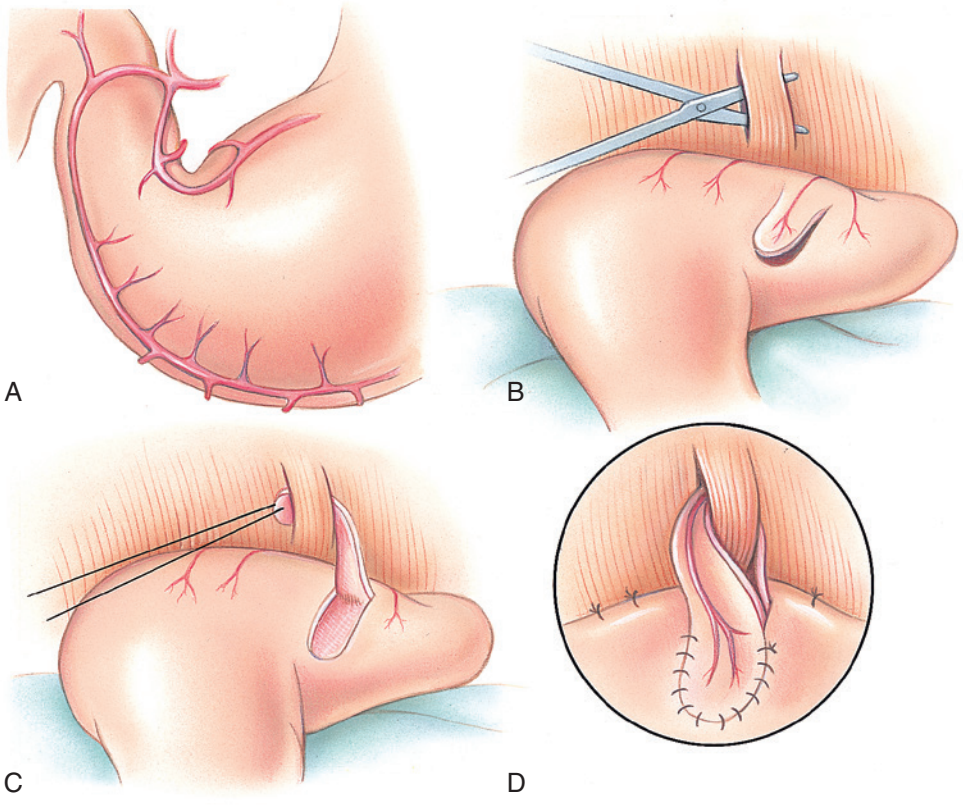


FIG. 18.81 Belt-loop gastropexy. (A) Elevate a seromuscular flap in the gastric antrum. (B) Make two transverse incisions in the ventrolateral abdominal wall and create a tunnel under the abdominal musculature with forceps. Pass the flap from cranial to caudal under the muscular flap. (C–D) Suture the flap to its original gastric margin.

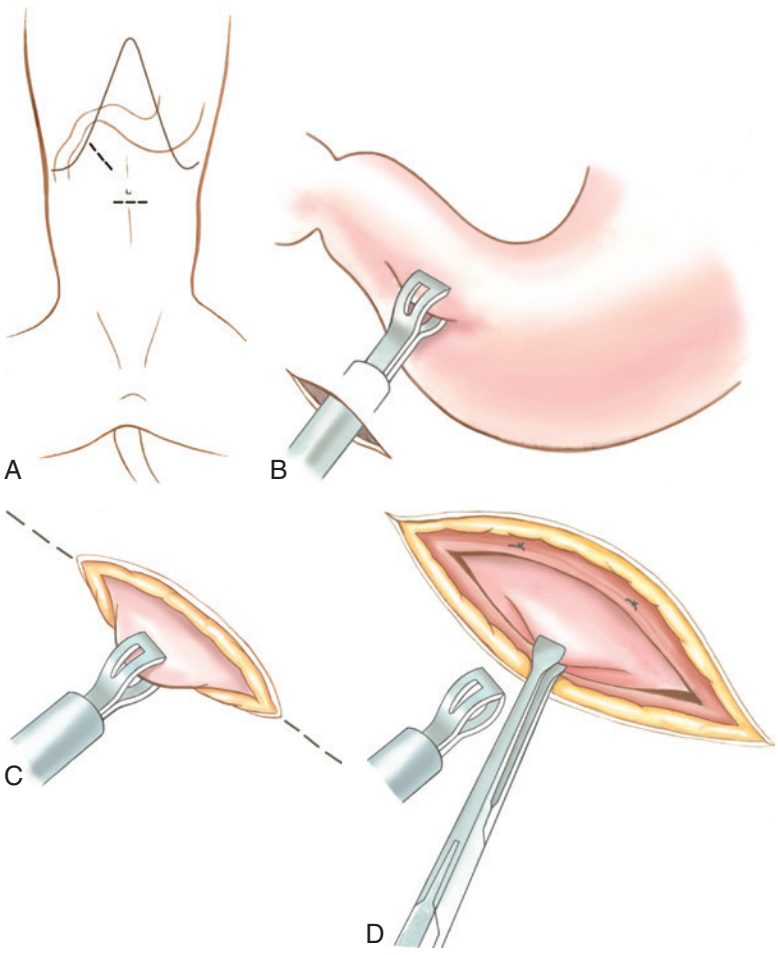


FIG. 18.82 For a laparoscopic assisted gastropexy, (A) place the first cannula just caudal to the umbilicus and place the second cannula just right of midline, approximately 2 to 4 cm behind the last rib (make this incision parallel to the rib). (B) Pull the stomach out through the incision. (C) Place stay sutures between it and the body wall. (D) Perform a gastropexy as described in Fig. 18.79.

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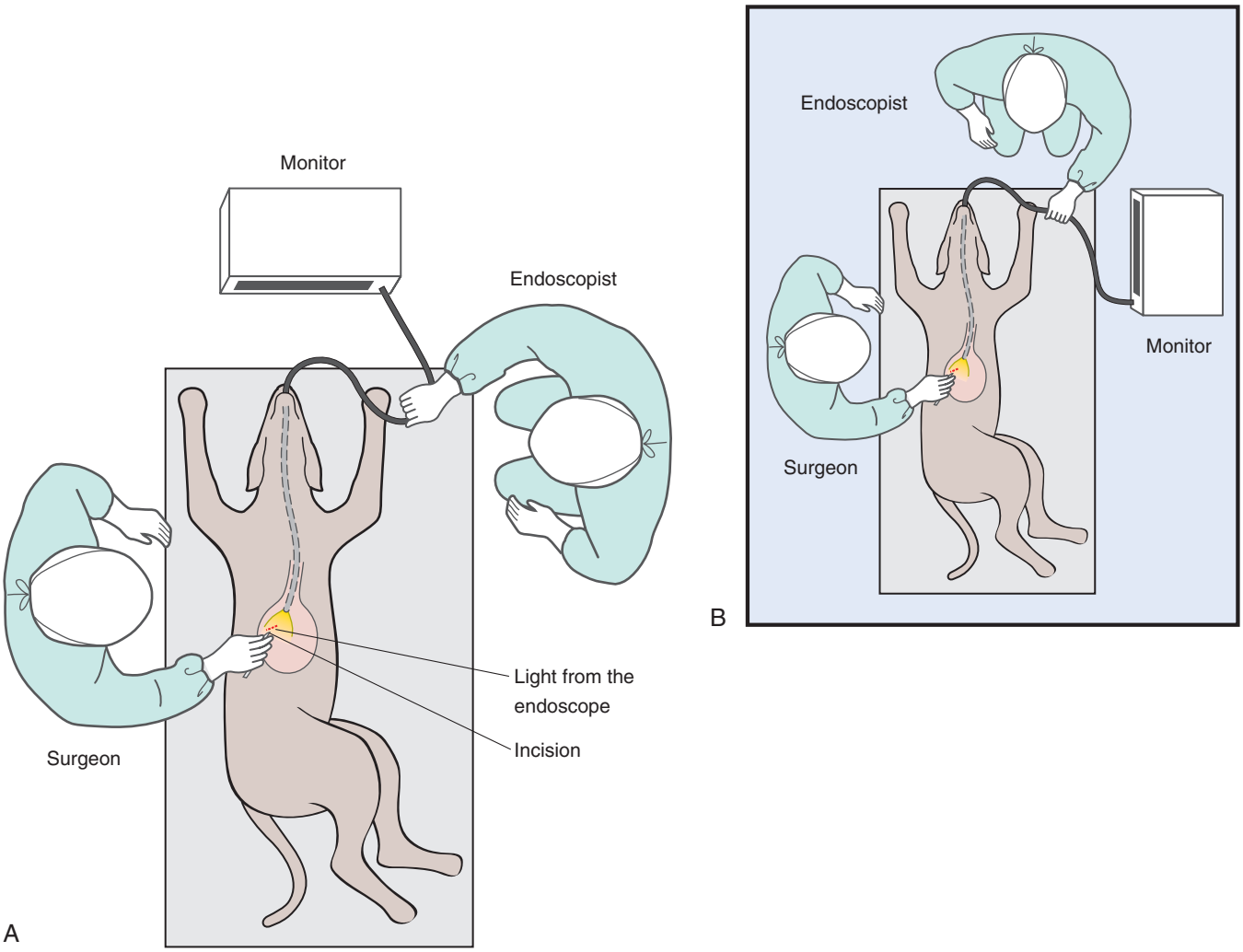


FIG. 18.83 (A) Positioning for endoscopically assisted gastropexy as described in the literature with the endoscopist positioned to the patient's left and the surgeon at the patient's dorsum. (B) Alternatively, the endoscopist may be at the patient's head and the monitor to the patient's left. Strict adherence to asepsis must be maintained with the monitor positioned adjacent to the surgery site.

tail or backstitched to finish the line of closure without knotting. *Pass the barbed suture into the abdomen as described prior to apposing the antrum to transversus muscle.* Two types of barbed suture are the Quill suture, which has bidirectional barbs and two needles, and the V-Loc suture, which has a loop wielded at one end. *For the Quill suture, start the suture centrally and suture in two directions; for the V-Loc suture, start at one end and pass it through the loop prior to suturing the antrum to transversus abdominus.*

Single-Port Laparoscopic Gastropexy

A specialized endoscopic port that allows insertion of the endoscope and multiple laparoscopic instruments may also be used for incisional gastropexy in dogs.[24–26] The multitrochar port is placed in the same position as the endoscopic port for laparoscopic-assisted gastropexy. Elective intraabdominal procedures, usually ovariectomy, are generally done with straight or reticulating instruments, while the port is used to grasp the pyloric antrum and exteriorize it for laparoscopic-assisted gastropexy. *To use the SILS Port (Covidien, Inc.) make a 2 to 3 cm incision in the body wall and place a multiple-trocar port through it while maintaining external traction using stay sutures at either end. Lubricate, then grasp the port with two Rochester Carmalt forceps, staggered on the port for insertion through the incision. To use the ENDOCONE (Karl Storz), which is a reusable metal multiple-trochar port, place the port in the location described for laparoscopic-assisted gastropexy. Make a 30-mm incision extending through the oblique muscles. Retract the oblique muscles with a self-retaining retractor and incise the transversus muscle. Use stay sutures for external traction prior to incising the transversus muscle. Digitally evaluate for adhesions prior to insertion of the flange on the port and clockwise rotating the port. Use the port to identify and grasp the pyloric antrum for performing laparoscopic-assisted gastropexy (see previous description).*

Mini-Laparotomy Prophylactic Gastropexy

This procedure may be considered for prophylactic gastropexy. *Place the dog in left lateral recumbency. Make a 6-cm vertical skin incision*

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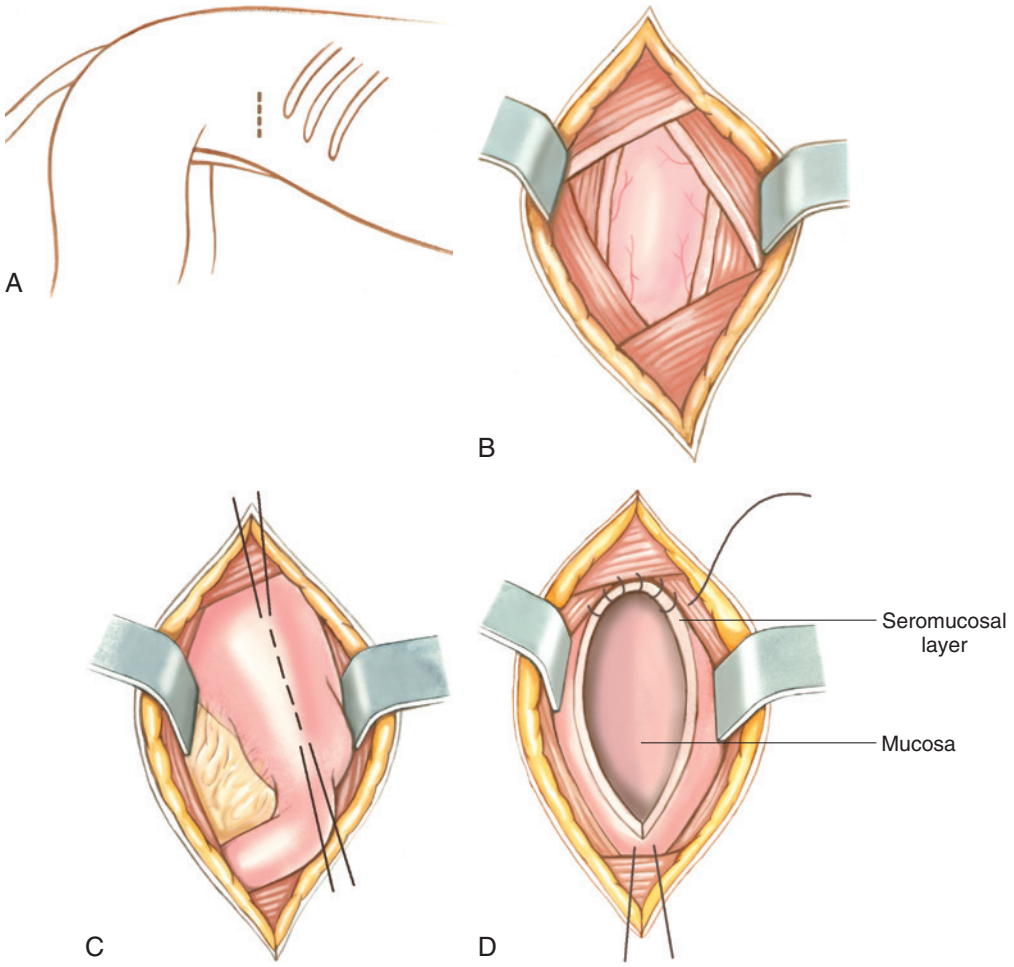


FIG. 18.84 For a mini-laparotomy, (A) make a 6-cm vertical skin incision just caudal and ventral to the right 13th rib. Bluntly dissect through the muscles. (B) Enter the abdomen and visualize the stomach. Grasp the stomach with Babcock intestinal forceps and retract the gastric antrum into the surgical field. (C) Place stay sutures at each end of the intended gastric incision to hold the stomach in the surgical field. Allow the gastric antrum to fall back into the abdomen. Palpate the pylorus to ensure proper placement of the gastric incision. (C–D) Make a 3-cm longitudinal incision through the serosa and muscularis layers of the gastric antrum, approximately 5 cm orad to the pylorus. Beginning at the dorsal edge, suture the edges of the incision to the abdominal muscles using a simple continuous suture pattern.

just caudal and ventral to the 13th rib (Fig. 18.84A). Bluntly dissect through the external and internal abdominal oblique and transversus abdominis muscles. Enter the abdomen via blunt dissection and visualize the stomach (see Fig. 18.84B). If the stomach cannot be visualized, palpate the duodenum and pylorus with two fingers and locate the antrum of the stomach. Grasp the stomach with Babcock intestinal forceps and retract the gastric antrum into the surgical field. Identify the stomach by palpating the pylorus and visualizing the omental attachments, right gastric vessels, and right gastroepiploic vessels. Place stay sutures at each end of the intended gastric incision to hold the stomach in the surgical field. Allow the gastric antrum to fall back into the abdomen (see Fig. 18.84C). Palpate the pylorus to ensure proper placement of the gastric incision. Make a 3-cm longitudinal incision through the serosa and muscularis layers of the gastric antrum approximately 5 cm orad to the pylorus (see Fig. 18.84C). Beginning at the dorsal edge, suture the edges of the incision to the abdominal muscles using a simple continuous suture pattern (see Fig. 18.84D). Close the muscles, subcutaneous tissues, and skin routinely.

HEALING OF THE STOMACH

The extraordinarily rich blood supply, reduced bacterial numbers (caused by gastric acidity), rapidly regenerating epithelium, and defense mechanisms provided by the omentum allow gastric incisions to heal quickly. Because the stomach has a thicker wall than the bowel, it can be difficult to find and stop bleeding vessels. Gentle pressure applied to the bleeding tissues usually is effective; crushing clamps or forceps and electrocautery should be avoided.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Monofilament absorbable suture material (i.e., polydioxanone [PDS], polyglyconate [Maxon], or poliglecaprone 25 [Monocryl]) is preferred for GI surgery. These sutures are strong, have minimal tissue drag, and maintain tensile strength for longer than 10 days. Chromic gut suture is rapidly removed by digestion and phagocytosis and should be avoided. Nonabsorbable suture

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material that penetrates the lumen may cause gastric ulcers to form along the suture line if a continuous suture pattern is used; therefore nonabsorbable suture material should be avoided in the stomach. Small-diameter, swaged-on, taper point needles are generally preferred for GI surgery. However, cutting needles are sometimes used because they more readily penetrate submucosa and require less stabilization of tissue, which might result in less crushing.

Noncrushing forceps (e.g., Doyen forceps or some vascular clamps) are useful for occluding the gastric and duodenal stumps during gastroduodenostomy or gastrojejunostomy procedures (see p. 403). Pediatric Doyen forceps are the appropriate size for occluding the duodenal lumen. If noncrushing forceps are not available, the straight portion of an Allis tissue forceps can be wrapped with a moistened sponge. Crushing forceps, such as Carmalt or Allen forceps, are useful for occluding the portion of the GI tract to be removed but should not be placed on tissues that are not to be excised.

Endoscopic gastropexy requires specialized barbed suture for knotless surgery, and multitrochar laparoscopic ports for single incisional laparoscopic surgery may be more efficiently done with reticulating instrumentation.

POSTOPERATIVE CARE AND ASSESSMENT

Electrolytes, especially potassium, should be monitored postoperatively. Analgesics should be provided as needed. IV fluids are continued until the patient is drinking adequate amounts to maintain hydration. If prolonged vomiting or anorexia is anticipated, enteral hyperalimentation should be provided by means of a gastrostomy or enterostomy tube (see Chapter 10). With planning, feeding tubes can be placed during the initial surgery so as to avoid a second procedure. Food can be offered 12 hours postoperatively if there is no vomiting. ECGs should be monitored if arrhythmias were present before surgery or are anticipated postoperatively.

COMPLICATIONS

Complications associated with gastric surgery may include vomiting, anorexia, peritonitis secondary to intraoperative or postoperative leakage, ulceration at anastomotic sites, gastric outlet obstruction, and pancreatitis. Malpositioning of the stomach following gastropexy can result in gastric outflow obstruction and chronic vomiting or gastric dilation. Potential complications of endoscopically-assisted gastropexy include needle breakage and loss, and organ entrapment or damage during passage of the stay sutures. Use of No. 2 polypropylene on a 76-mm-long cutting needle is recommended for stay suture placement. Postoperatively, some dogs may develop short-term (<7 days) intermittent vomiting or diarrhea. With intracorporeally sutured gastropexy, technical difficulties associated with the automated suture assist device (among other complications) may result in the need for supplemental suture placement in some dogs. Although laparoscopic-assisted gastropexy may be faster, activity levels are often greater in the postoperative period with the intracorporeally sutured gastropexy.

SPECIAL AGE CONSIDERATIONS

Young animals and those that are severely emaciated and have little or no body reserve should not have food withheld for longer than 4 to 6 hours before surgery and should be fed as soon as they are fully recovered from the anesthesia. If they cannot be fed, blood glucose concentrations should be monitored and, if necessary, maintained by adding glucose to the IV fluids (see the discussion of anesthesia on p. 399). Polydioxanone suture may cause calcinosis circumscripta in young dogs. Because the skin of puppies and kittens is delicate and elastic, skin sutures should be placed loosely. Geriatric animals may have delayed wound healing and may be immunosuppressed as a result of concurrent disease (e.g., diabetes mellitus, Cushing's disease). Strong absorbable or nonabsorbable suture material should be used in these patients and prophylactic antibiotic therapy initiated at induction of anesthesia.

PROGNOSIS

Evaluation of 24 dogs undergoing Billroth I showed that 88% survived to discharge; death or euthanasia at less than 3 months after surgery is generally related to progression of neoplastic disease. Postoperatively, hypoalbuminemia and anemia are common, and approximately one-third may require blood products postoperatively. Preoperative weight loss and malignant neoplasia may be associated with a decrease in median survival time. Complications included hypotension, aspiration pneumonia, pancreatitis, and septic peritonitis resulting from anastomotic leakage.

SPECIFIC DISEASES

GASTRIC FOREIGN BODIES

DEFINITIONS

A gastric foreign body is anything ingested by an animal that cannot be digested (i.e., rocks, plastic) or that is slowly digested (bones). **Linear foreign bodies** are usually pieces of string, yarn, thread, cloth, or dental floss.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Gastric foreign bodies usually cause vomiting because of outflow obstruction, gastric distention, and/or mucosal irritation (Box 18.24). Occasionally, however, gastric foreign bodies are asymptomatic,

BOX 18.24 Important Considerations for Gastric Foreign Bodies

- Initial clinical signs may not alert the owner to seriousness of condition.
- Linear foreign objects must be removed as soon as possible to avoid intestinal perforation and peritonitis.
- Not all animals with gastric foreign objects vomit.
- Finding a foreign object in the stomach does not guarantee that it is the cause of vomiting.
- Linear foreign bodies are more common in cats; always check under the tongue (this often requires sedation).
- Most gastric foreign objects can be removed endoscopically.
- Complete exploration of the entire intestinal tract is mandatory in surgical patients.
- Always repeat the radiographs immediately before surgery to make sure that the object has not moved.

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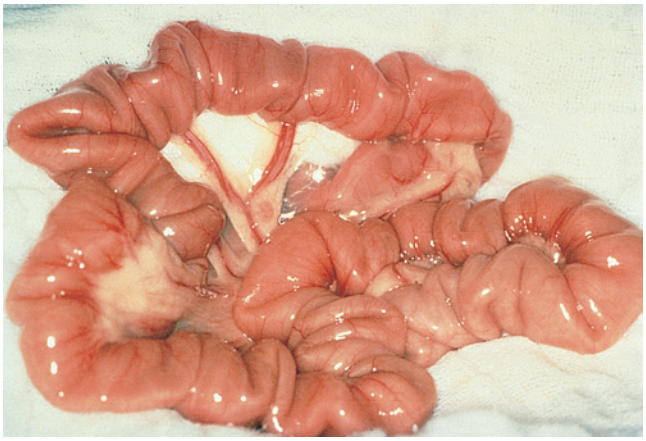


FIG. 18.85 Plication in the intestines of a cat caused by a string foreign body. (Courtesy Dr. J. Hauptman, Michigan State University.)

incidental findings on abdominal radiographs. Dogs are indiscriminate eaters and often ingest rocks, plastic toys, cooking bags, and other objects. Cats more commonly ingest linear material (e.g., yarn or sewing thread attached to a needle). In cats, linear foreign bodies are frequently anchored under the tongue or at the pylorus and often cause intestinal plication (Fig. 18.85). Noxious stimuli or distention of the duodenum or pyloric antrum stimulates vomiting, whereas similar stimulation of the gastric body often does not. Therefore vomiting is often intermittent, occurring when the object is forced into the pyloric antrum.

NOTE Foreign bodies may occur in the stomach and small intestine concurrently; therefore the entire intestinal tract should be explored thoroughly whenever surgery is done to remove a gastric foreign body.

If endoscopic removal is performed, examine the small intestine as far as possible and consider radiographing the patient before waking it up; the gas that has been insufflated will act as a negative contrast. Small, blunt foreign bodies may pass through the digestive system without causing harm; however, most should be removed when they are diagnosed because of the risk of distal obstruction and perforation. Repeat radiographs are indicated immediately before surgery, even if only a few hours have elapsed since the initial radiographs; the object may exit the stomach and be located in either the small intestine or the colon. Foreign objects in the colon are usually eliminated without difficulty unless they have sharp edges that catch in the anus. Opening the colon is seldom justified; if a colonic foreign body must be removed, endoscopy is the preferred technique.

DIAGNOSIS

Clinical Presentation

Signalment

Young animals more commonly ingest foreign bodies than older animals, and gastric or intestinal foreign bodies should be suspected in any puppy or kitten presented for treatment of acute or persistent vomiting.

History

Most animals with gastric foreign bodies have vomiting, anorexia, and/or depression. One study showed 89% of patients with sewing needle foreign bodies exhibited clinical signs and the remainder had no clinical signs associated with extragastrointestinally located needles.[27] Vomiting may be intermittent, and some animals may continue to eat and remain active. Vomiting is often absent if the foreign body is in the gastric fundus and does not obstruct the pylorus. Occasionally abdominal pain is noted. Be sure to consider parvoviral enteritis in young animals that acutely vomit and feel bad.

Physical Examination Findings

Physical examination often is unremarkable. The patient may be dehydrated; however, many animals with gastric foreign bodies continue to drink water. The object usually cannot be palpated because of the stomach's proximal location in the abdomen. Plicated intestines may be felt if a linear foreign object is present, and pain may be evident if gastric perforation has caused peritonitis or if the intestines are bunched together. A thorough examination of the mouth, especially the area ventral to the tongue, is mandatory in all cats suspected of having a linear foreign object. General anesthesia or sedation may be necessary to properly evaluate the base of the tongue.

NOTE Don't forget to check under the tongue in cats that may have a linear foreign object.

Diagnostic Imaging

Radio-opaque foreign bodies may be diagnosed with survey radiographs, but many foreign bodies are radiolucent. Some radiolucent foreign bodies can be identified ultrasonographically if the stomach is fluid filled and an appropriate acoustic window is obtained. A positive contrast or double-contrast gastrogram can be utilized to delineate radiolucent foreign bodies, although these studies have largely been replaced with endoscopy. If contrast studies are performed, remember that barium sulfate should not be utilized if GI perforation is suspected (i.e., pneumoperitoneum, abdominal effusion); an iodinated, water-soluble contrast agent should be used in such cases. Any effusion should be cytologically examined; evidence of sepsis is adequate to recommend surgery, and contrast radiographs are unnecessary. Likewise, spontaneous pneumoperitoneum is automatically assumed to indicate perforation and justifies exploratory surgery. Double-contrast studies using both air and a positive contrast agent (i.e., barium sulfate) are more sensitive than positive contrast procedures but are more difficult to perform. Contrast procedures are especially useful if the foreign object absorbs or is coated by the contrast material. Radiographic findings in cats with linear foreign bodies include plication of the small bowel, increased intraluminal gas bubbles, and peritonitis secondary to bowel perforation (see p. 528).

Currently, radiographic contrast procedures are seldom performed to look for foreign objects. Gastroduodenoscopy is more sensitive than radiographs in finding foreign objects (unless the stomach if filled with food) and may afford the chance to remove it at the same time. In addition, one can find other gastric lesions and biopsy them (see Chapter 14).

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NOTE Do not do an upper gastrointestinal series using barium sulfate if you will perform endoscopy within 24 hours, and don't use barium sulfate if perforation is likely!

Gastroduodenoscopy

If a foreign body is located, the endoscopist should first examine and study it to determine the best retrieval device to use and whether any special equipment (e.g., an overtube) is needed. If there are sharp edges or points, then consideration must be given to which end of the foreign body will be grasped. Endoscopic removal of linear foreign bodies should probably only be attempted when the linear foreign body has been present for a relatively short time (e.g., <3–4 days) and the orad-most aspect is lodged at the pylorus. One may try to gently pull the foreign object out of the pylorus, but if it does not readily come out, this tactic must be quickly abandoned. A better technique is as follows: *Insert the endoscope between the foreign object and the pylorus, and then advance the tip of the endoscope to as close to the end of the foreign object as possible.* This can be tedious, so be patient! *Then grasp the linear foreign object as close to the distal end as possible and retrieve the foreign object by pulling the distal end out first. Take a plain abdominal radiograph afterward to check for spontaneous pneumoperitoneum, a sign of perforation.*

In rare cases, when the orad end of the linear foreign object is lodged at the pylorus, one may use the endoscope to push the orad end into the duodenum, thus relieving the point of fixation. This approach is similar to cutting a linear foreign body off the base of the tongue and seeing if it will then pass without incident. This may allow the entire foreign object to pass in the feces. In these cases, if the patient does not clearly feel better within 6 hours, surgery is indicated to remove the foreign object.

Laboratory Findings

The laboratory findings depend on the severity and duration of the obstruction; they cannot be predicted. Laboratory parameters may be normal or may show only changes caused by dehydration (hemoconcentrations, increased serum albumin, prerenal azotemia). If vomiting causes loss of gastric secretions, a hypochloremic, hypokalemic metabolic alkalosis with paradoxical aciduria may occur. Sometimes a metabolic acidosis occurs because of dehydration and subsequent lactic acidosis.

DIFFERENTIAL DIAGNOSIS

Gastric neoplasms sometimes cause filling defects in the gastric lumen that could be confused with a foreign object. However, such lesions should remain in the same location when the animal is positioned for different radiographic views. Radiographs and endoscopy distinguish animals with gastric foreign objects from those with other causes of pyloric obstruction (e.g., chronic antral mucosal hypertrophy or pyloric stenosis; see p. 425) or gastric ulceration (see p. 427).

MEDICAL MANAGEMENT

If the object is small with rounded edges or is cloth, one can attempt to expel it by inducing vomiting with apomorphine (dogs) or xylazine (cats) (Box 18.25). However, this should be attempted only when the clinician is relatively certain the object will be expelled without causing harm. Factors that must be considered include whether the esophagus is apt to be lacerated, the likelihood of the object lodging in the esophagus, and whether the object or gastric contents might be aspirated. Esophageal surgery carries more risk than gastric surgery because the esophagus does not heal as readily as the stomach (see p. 374). Indiscriminate use of antibiotics may mask clinical signs of peritonitis or pyothorax and delay treatment.

BOX 18.25 Induction of Vomition

Dogs
Apomorphine
0.02–0.04 mg/kg IV or IM; 0.1 mg/kg SC

Cats
Xylazine (Rompun)
0.4–0.5 mg/kg IV

IM, Intramuscular; *IV,* intravenous; *SC,* subcutaneous.

SURGICAL TREATMENT

Preoperative Management

If possible, metabolic and acid-base abnormalities should be identified and corrected, and food should be withheld for 12 hours. Radiographs should be taken immediately before surgery (especially before endoscopy) to verify the position of the object in the digestive tract. Perioperative antibiotics may be given at induction of anesthesia and continued for up to 12 hours postoperatively (see p. 399).

Anesthesia

See p. 399 for suggested anesthetic protocols in dogs with gastric disorders.

Surgical Anatomy

See p. 399 for the surgical anatomy of the stomach.

Positioning

The animal is placed in dorsal recumbency and the abdomen is prepared for a ventral midline incision. The prepped area should extend from midthorax to the pubis to allow the entire digestive system to be explored for foreign objects.

SURGICAL TECHNIQUE

Most gastric foreign bodies can be easily removed via a gastrotomy incision (see p. 401). *Inspect the entire digestive system for material that could cause obstruction or perforation. If a linear foreign body is found in the pylorus and extends into the intestinal tract, do not try to pull it into the stomach unless it moves easily. Instead, make several incisions into the stomach and intestines to avoid causing further damage to the intestinal tract. Inspect the stomach for perforation or necrosis and, depending on the location, remove or patch abnormal tissue (see p. 447 for a description of a serosal patch). Close the gastric incision as described on p. 401.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Absorbable suture material (2-0 or 3-0) should be used to close the gastrotomy incision (see p. 415). Instruments helpful in performing gastric surgery are listed on p. 415.

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BOX 18.26 Treatment of Vomiting[a]

Maropitant (Cerenia)
1 mg/kg IV or 2 mg/kg PO q24h for 5 days if <7 months old; otherwise, can use as long as desired

Ondansetron (Zofran)
0.1–0.2 mg/kg IV or SC, q6–8h; 0.5–1.0 mg/kg given 30 min before administration of chemotherapeutic drugs

[a]Metoclopramide (see Box 18.21), although a poor antiemetic, may be useful in conjunction with these drugs if single-drug therapy fails.
IM, Intramuscular; *PO,* orally; *SC,* subcutaneous.

POSTOPERATIVE CARE AND ASSESSMENT

The patient's fluid status should be monitored and hydration maintained with IV fluids postoperatively until the animal is drinking. Electrolyte abnormalities should be corrected. Hypokalemia is likely if the animal has been vomiting, especially if it was also anorexic. A bland diet should be fed starting 12 to 24 hours after surgery if the patient is not vomiting. If vomiting continues, centrally acting antiemetics such as maropitant or ondansetron (Box 18.26) may be administered, and oral food and water should be withheld. See p. 429 for treatment of ulcers that may occur secondary to foreign bodies.

PROGNOSIS

The prognosis is good if the stomach has not been perforated and the foreign body is removed. If perforation has occurred, the prognosis is guarded (see the discussion of peritonitis on pp. 449 and 527).

GASTRIC DILATATION-VOLVULUS

DEFINITIONS

Enlargement of the stomach associated with rotation on its mesenteric axis is referred to as **gastric dilatation-volvulus (GDV)**. The term **simple dilatation** refers to a stomach that is engorged with air or froth but not malpositioned. **Dilatation** refers to a condition in which an organ or structure is stretched beyond its normal dimensions; **dilation** is the act of stretching a cavity or orifice. GDV is also called *gastric torsion* or *bloat.*

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Classically, GDV syndrome is an acute condition with a mortality rate of 20% to 45% in treated animals. The gastric enlargement is thought to be associated with a functional or mechanical gastric outflow obstruction. The initiating cause of the outflow obstruction is unknown; however, once the stomach dilates, normal physiologic means of removing air (i.e., eructation, vomiting, and pyloric emptying) are hindered because the esophageal and pyloric portals are obstructed.

The stomach becomes enlarged as gas, fluid, or both accumulate in the lumen. The gas probably comes from aerophagia, although bacterial fermentation of carbohydrates, diffusion from the bloodstream, and metabolic reactions may contribute. Normal gastric secretion and transudation of fluids into the gastric lumen secondary to venous congestion contribute to fluid accumulation.

BOX 18.27 Recommendations for Dogs at High Risk for Gastric Dilatation Volvulus

- Feed several small meals a day rather than one large meal.
- Avoid stress during feeding (if necessary, separate dogs in multiple-dog households during feeding).
- Restrict exercise before and after meals (of questionable benefit).
- Do not use an elevated feed bowl.
- Do not breed dogs with a first-degree relative that has a history of gastric dilatation-volvulus.
- For high-risk dogs, consider prophylactic gastropexy.
- Seek veterinary care as soon as signs of bloat are noted.

The cause of GDV is unknown, but exercise after ingestion of large meals of highly processed food or water has been suggested to contribute to it (this is largely unsupported by clinical data). Epidemiologic studies have also not supported a causal relationship between feeding soy-based or cereal-based dry dog food and GDV. However, Irish setters fed a single feed type appear to have an increased risk of GDV compared with those fed a mixture of feed types. Likewise, adding table food or canned food to the diet of large- and giant-breed dogs is associated with a decreased incidence of GDV. Dogs fed a larger volume of food per meal may be at increased risk of GDV, regardless of the number of meals fed daily; however, the risk of GDV is highest in dogs fed a larger volume of food once daily. Feeding dry dog foods in which one of the first four ingredients is an oil or a fat may also increase the risk of GDV. Other contributing causes include an anatomic predisposition, ileus, trauma, primary gastric motility disorders, vomiting, and stress. Recommendations for clients of animals at high risk are provided in Box 18.27. Male sex, increasing age, being underweight, being fed a large volume of food per meal, eating one meal (especially a large volume meal) per day, eating rapidly, having a raised feeding bowl, and having a fearful temperament are predisposing factors that may increase a dog's risk of GDV. Having a deeper and narrower thorax may change the anatomic relationship between the stomach and esophagus such that the dog's ability to eruct is impaired. Feeding dogs from a raised feed bowl may increase the risk of GDV because it may promote aerophagia. Some authors have recommended gastropexy to prevent GDV following splenectomy because of the possible loss of gastric stabilization by the gastrosplenic ligament, but this has not been proved necessary. Finally, military working dogs appear to be more likely to develop a GDV in November, December, and January, but the reasons for this are uncertain. Atmospheric data have been related to the occurrence of GDV; minimum and maximum atmospheric pressure and the maximum atmospheric pressure the day prior were positively associated with GDV.

NOTE Having a first-degree relative with a history of gastric dilatation-volvulus (GDV) is significantly associated with an increased risk of GDV. Thus it may be wise to suggest that dogs with a first-degree relative that has had GDV not be used for breeding.

Generally, with GDV the stomach rotates in a clockwise direction when viewed from the surgeon's perspective (with the dog on its back and the clinician standing at the dog's side, facing cranially; Fig. 18.86). The rotation may be 90 to 360 degrees but is usually 220 to 270 degrees. The duodenum and pylorus move

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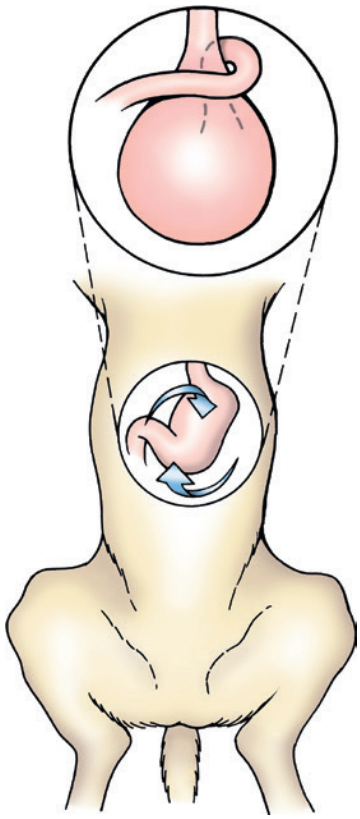


FIG. 18.86 Direction of gastric rotation in most dogs with gastric dilatation-volvulus.

ventrally and to the left of the midline and become displaced between the esophagus and stomach. The spleen is usually displaced to the right ventral side of the abdomen.

Caudal vena cava and portal vein compression by the distended stomach reduces venous return and cardiac output, causing myocardial ischemia. Central venous pressure, stroke volume, mean arterial pressures, and cardiac output are reduced. Obstructive shock and inadequate tissue perfusion affect multiple organs, including the kidneys, heart, pancreas, stomach, and small intestine. Cardiac arrhythmias occur in many dogs with GDV, particularly those with gastric necrosis. Arrhythmias may contribute to mortality and require appropriate monitoring and treatment (see discussion under Postoperative Care and Assessment on p. 423). Myocardial depressant factor has also been recognized in affected dogs, and cardiac damage is common, as seen by increased serum concentrations of troponin. Reperfusion injury has been implicated as causing much of the tissue damage that ultimately results in death after correction of GDV. Early treatment with IV lidocaine (2 mg/kg IV bolus) before decompression followed by CRI of lidocaine (0.05 mg/kg per min) for 24 hours appears to decrease the occurrence of cardiac arrhythmias, acute kidney injury, and hospitalization in dogs with GDV, possibly by mitigating reperfusion injury.

Partial or chronic GDV may occur in dogs, can last weeks to months, and is usually a progressive but non–life-threatening syndrome that may be associated with vomiting, anorexia, and/or weight loss. These dogs may have chronic, intermittent signs and appear normal between episodes. Gastric malpositioning may be intermittent or chronic but without dilatation. Plain or contrast radiographs are diagnostic, but repeat radiographs may be necessary if the stomach is intermittently malpositioned. Endoscopic evaluation has been reported as helpful in the diagnosis of chronic GDV, but the intermittent nature of the condition makes diagnosis somewhat difficult.

DIAGNOSIS

Clinical Presentation

Signalment

GDV primarily occurs in large, deep-chested breeds (e.g., Great Dane, Weimaraner, Saint Bernard, German shepherd, Irish and Gordon setters, Doberman pinscher) but has been reported in cats and small-breed dogs. Shar peis may have an increased incidence compared with other medium-sized breeds and basset hounds may have a higher risk of GDV, despite their not being a large-breed dog. GDV may occur in dogs of any age but is most common in middle-aged or older animals. The thoracic depth to width ratio appears to be highly correlated with the risk of bloat. In one study, intact female dogs had the highest risk of GDV.[28]

History

A dog with GDV may have a history of a progressively distending and tympanic abdomen, or the owner may simply find the animal recumbent and depressed with a distended abdomen. The dog may appear to be in pain and may have an arched back. Nonproductive retching, hypersalivation, and restlessness are common.

Dogs that are fed dry kibble, are anxious, are a family pet, and those that spend 5 hours or more with the owner appear to be at an increased risk of GDV. A decreased risk may be associated with playing with other dogs, being fed egg supplements, and spending equal time indoors and outdoors. Splenectomy has been purported as a risk factor for GDV; however, whether this is true is uncertain. In one study, approximately 8% of dogs undergoing splenectomy and 6.4% of controls undergoing enterotomy developed GDV after initial surgery.[29] In another study of 453 cases the odds of GDV in dogs with a history of previous splenectomy was 5.3 times that of dogs without such a history.[29a] Interestingly, in a retrospective study, large- or giant-breed dogs with gastric foreign body were five times as likely to develop GDV compared with those with no foreign body; however, a causal relationship could not be established.[30]

Physical Examination Findings

Abdominal palpation often reveals various degrees of abdominal tympany or enlargement; however, it may be difficult to feel gastric distention in heavily muscled large-breed or very obese dogs. Splenomegaly is occasionally palpated. Clinical signs associated with shock may be present, including weak peripheral pulses, tachycardia, prolonged capillary refill time, pale mucous membranes, and/or dyspnea.

Diagnostic Imaging

Radiographs are necessary to differentiate simple dilatation from dilatation plus volvulus. Affected animals should be decompressed before radiographs are taken. Right lateral and dorsoventral radiographic views are preferred to facilitate filling the abnormally displaced pylorus with air so that it can be easily identified. The pylorus is normally located ventral to the fundus on the lateral view and on the right side of the abdomen on the dorsoventral

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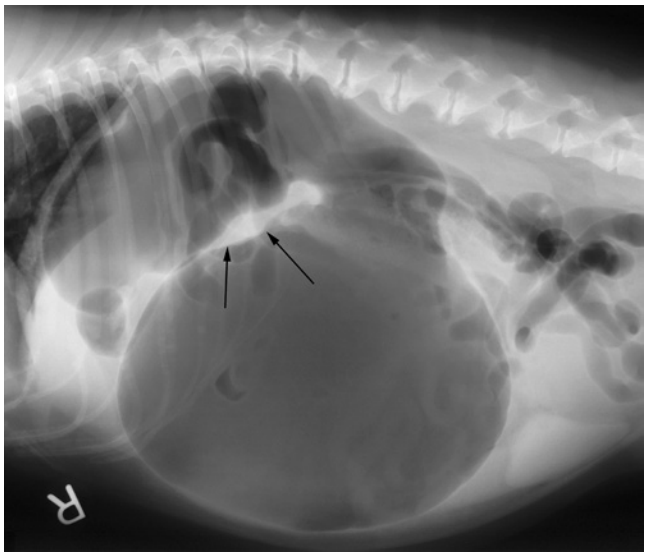


FIG. 18.87 Right lateral abdominal radiograph of a dog with gastric dilatation-volvulus showing a distended, gas-filled stomach. Note the reverse C sign or "double bubble" caused by the shelf of soft tissue *(arrows)*. The pylorus is located dorsal to the shelf of tissue.

view. On a right lateral view of a dog with GDV, the pylorus lies cranial to the body of the stomach and is separated from the rest of the stomach by soft tissue (reverse C sign or "double bubble") (Fig. 18.87). On the dorsoventral view, the pylorus appears as a gas-filled structure to the left of midline (Fig. 18.88). Free abdominal air suggests gastric rupture and air within the wall of the stomach indicates necrosis, both of which warrant immediate surgery.

NOTE Caution! Positioning these animals for a ventrodorsal view may lead to aspiration. Remember that the right lateral and dorsoventral views are preferred when attempting to diagnose GDV.

Laboratory Findings

The CBC is seldom informative unless disseminated intravascular coagulation (DIC) causes thrombocytopenia. Potassium concentrations may be normal or elevated, but hypokalemia is more common. Vascular stasis may cause increased lactic acid and metabolic acidosis. However, metabolic alkalosis caused by sequestration of hydrogen ions in the gastric lumen can offset the metabolic acidosis, causing the blood pH to be normal (i.e., a mixed acid-base disorder). Respiratory acidosis may be caused by hypoventilation secondary to gastric impingement on the diaphragm and diminished ventilatory compliance. Therefore routine administration of sodium bicarbonate is inappropriate. Plasma lactate concentrations are prognostic; higher values (7.4 mmol/L) have been associated with gastric necrosis and a poor prognosis, and were 88% accurate for prediction of necrosis. Its sensitivity, however, was 50%.[31]

DIFFERENTIAL DIAGNOSIS

Simple gastric dilatation occurs commonly in young puppies from overeating and seldom requires specific treatment. The stomach, although greatly enlarged with ingesta and gas, is not malpositioned. Small intestinal volvulus is a differential diagnosis

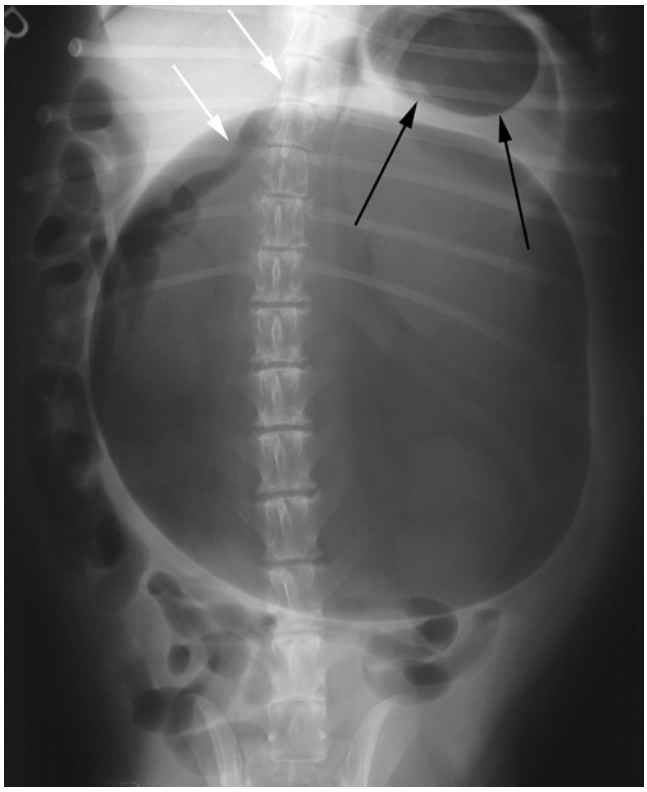


FIG. 18.88 Dorsoventral radiograph of a dog with gastric dilatation-volvulus. The pylorus appears as a gas-filled structure to the left of the midline *(black arrows)*. Notice the duodenum coursing from the pylorus toward the right abdomen *(white arrows)*.

BOX 18.28 Medical Management of Gastric Dilatation-Volvulus

- Fluids (see text)
- Antibiotics such as cefazolin (22 mg/kg IV) or a combination of enrofloxacin (Baytril), 7–15 mg/kg IV (give diluted and slowly, over 30 minutes) plus ampicillin at 22 mg/kg IV

IV, Intravenous.

because it results in a tympanic and enlarged abdomen (see p. 464); however, dilatation of the intestinal tract is apparent on radiographs. Primary splenic torsion (see p. 643) often causes acute abdominal pain; however, abdominal distention is absent to mild. Diaphragmatic herniation may produce clinical signs similar to GDV, particularly if the stomach is herniated and outflow is obstructed (see p. 926). Ascites can cause abdominal distention, but a fluid wave should be felt during ballottement, which distinguishes it from the tympanic abdomen found in GDV.

NOTE You cannot differentiate gastric dilatation-volvulus from gastric dilatation without volvulus simply because you are able to pass a stomach tube. Stomach tubes can frequently be passed in dogs with twisted stomachs.

MEDICAL MANAGEMENT

Stabilizing the patient's condition is the initial objective (Box 18.28). One or more large-bore IV catheters should be

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placed in either a jugular or both cephalic veins. Either isotonic fluids (given at 90+ mL/kg per hour to effect), hypertonic 7% saline (4–5 mL/kg over 5–15 minutes), hetastarch (5–10 mL/kg over 10–15 minutes), or a mixture of 7.5% saline and hetastarch (dilute 23.4% saline with 6% hetastarch for a 7.5% solution; administer at 4 mL/kg over 5 minutes) is administered. If hypertonic saline or hetastarch is given, the rate of subsequent crystalloid administration must be adjusted. Blood should be drawn for blood gas analyses, a CBC, and a biochemical panel. Broad-spectrum antibiotics (e.g., cefazolin, ampicillin plus enrofloxacin) should be administered. If the animal is dyspneic, oxygen therapy may be given by nasal insufflation (see p. 31) or mask.

Gastric decompression should be performed while shock therapy is initiated. The stomach may be decompressed percutaneously with several large-bore IV catheters or a small trocar, or (preferably) a stomach tube may be passed. The stomach tube should be measured from the point of the nose to the xiphoid process and a piece of tape applied to the tube to mark the correct length. A roll of tape can be placed between the incisors and the tube passed through the center hole. Attempts should be made to pass the tube to the measured point. Placing the animal in different positions (i.e., sitting, reclining on a tilt-table) may help if it is difficult to advance the tube into the stomach. Do not perforate the esophagus with overly vigorous attempts to pass the tube. If these attempts fail, percutaneous decompression of the stomach should be attempted (Fig. 18.89). This may relieve pressure on the cardia and allow the tube to enter the stomach. Once the air has been removed, the stomach should be flushed with warm water. Failure to lavage the stomach usually results in rapid redilatation after the tube is withdrawn. If blood is seen in the fluid from the stomach, prompt surgical intervention is warranted because this may indicate gastric necrosis. If the stomach tube still cannot be passed and immediate surgical correction is not possible, temporary decompression may be achieved by performing a temporary gastrostomy, but this technique is not recommended. A Foley catheter should not be placed in the stomach percutaneously unless the stomach is simultaneously tacked to the body wall (see p. 406) because of the high risk of peritonitis if the stomach pulls away from the tube. The disadvantages of a temporary gastrostomy are that the stomach must be closed when the permanent gastropexy is performed and there is a high risk of peritoneal contamination. Keeping the stomach from redilating is important; however, this may not reduce gastric necrosis since the latter appears to be associated with malpositioning of the stomach rather than dilatation.

SURGICAL TREATMENT

Surgery should be performed as soon as the animal's condition has been stabilized, even if the stomach has been decompressed. Rotation of an undistended stomach interferes with gastric blood flow and may potentiate gastric necrosis.

Preoperative Management

The animal should be given IV fluids and antibiotics before surgery (see the discussion of Medical Management). Significant electrolyte and acid-base abnormalities should be corrected. A greatly enlarged stomach may hinder respiration and make it difficult for the animal to ventilate during induction of anesthesia. An ECG should be monitored to detect cardiac arrhythmias, which should be treated with lidocaine before surgery if they are significant (i.e., long runs of ventricular tachycardia, which can decrease cardiac output).

Anesthesia

Numerous anesthetic protocols have been described for dogs with GDV. If the animal has been decompressed and its condition is stable without significant cardiac arrhythmias, then hydromorphone and diazepam may be given intravenously and the patient induced with etomidate, thiobarbiturates, propofol, or alfaxalone (see Table 18.4). Refer to the surgery of the small intestine preoperative concerns (p. 433) and anesthesia (p. 435) sections for a more complete discussion of management of the patient with an acute abdomen. If the animal is depressed, premedication is probably not needed. Preoxygenation followed by a rapid induction with either ketamine plus a benzodiazepine (e.g., midazolam, diazepam) or etomidate should be done. Etomidate is a good choice for induction if the animal's condition has not been well stabilized because it maintains cardiac output and is not arrhythmogenic. Lidocaine and thiobarbiturate (e.g., thiopental) may be used if arrhythmias are present; 9 mg/kg of each is drawn up, and half is given initially intravenously. Additional drug is given to effect to allow the dog to be intubated. Generally, no more than 6 mg/kg of lidocaine is given intravenously to prevent toxicity. If bradycardia occurs, anticholinergics (e.g., atropine or glycopyrrolate) may

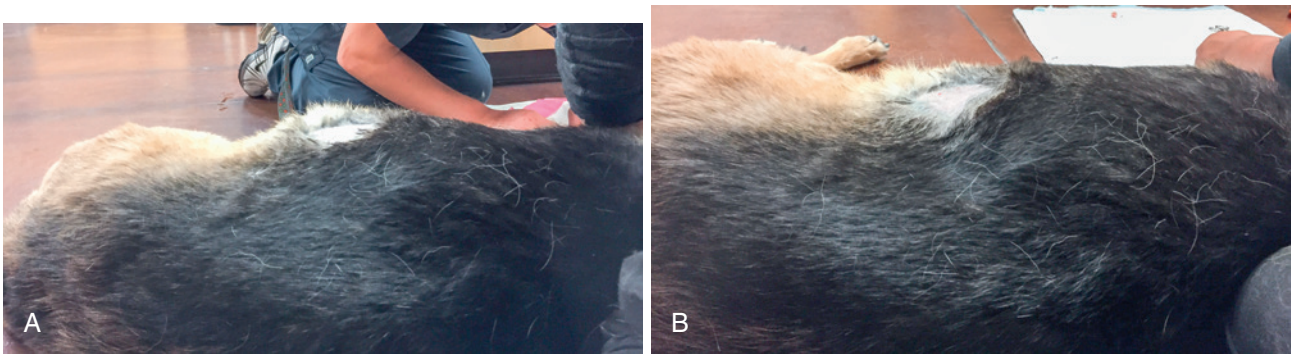


FIG. 18.89 Abdominal contour in a dog with gastric dilatation-volvulus. (A) Significant abdominal distension due to gastric dilatation-volvulus. (B) Appearance of the abdomen following trocharization.

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be given. Nitrous oxide should not be used in dogs with GDV (see p. 435). Isoflurane or sevoflurane are the inhalation agents of choice.

Surgical Anatomy

Normally, when viewed from the surgeon's perspective (i.e., with the animal in dorsal recumbency), the pylorus is located on the dog's right side, and the greater omentum arises from the greater curvature of the stomach and covers the intestines. The gastric (lesser curvature) and gastroepiploic (greater curvature) arteries supply the stomach and are derived from the celiac artery. The short gastric arteries arise from the splenic artery and supply the greater curvature. Rupture of the short gastric arteries in dogs with GDV is common and may contribute to blood loss and gastric infarction or necrosis. Eighty percent of the arterial flow is to the mucosa, and the remainder is to the muscularis and serosa; therefore observation of mucosal color is not a reliable indicator of gastric wall viability. The mucosa often appears darkened because of vascular compromise, even when full-thickness necrosis is not present.

Positioning

The dog is placed in dorsal recumbency, and the abdomen is prepared for a midline abdominal incision. The prepped area should extend from midthorax to the pubis. If a tube gastropexy is to be performed, the prepped area should be extended cranially and dorsally to allow the tube to be exteriorized behind the caudal right rib.

SURGICAL TECHNIQUE

The goals of surgical treatment are threefold: (1) to inspect the stomach and spleen so as to identify and remove damaged or necrotic tissues, (2) to decompress the stomach and correct any malpositioning, and (3) to adhere the stomach to the body wall to prevent subsequent malpositioning. Upon entering the abdominal cavity of a dog with GDV, the first structure noted is the greater omentum, which usually covers the dilated stomach.

Decompress the stomach before repositioning by using a large-bore needle (i.e., 14 or 16 gauge) attached to suction. If the needle becomes occluded with ingesta, have an assistant pass an orogastric stomach tube and perform gastric lavage. Intraoperative manipulation of the cardia usually allows the tube to be passed into the stomach without difficulty. If adequate decompression is still not achieved or an assistant is not available, a small gastrotomy incision can be performed to remove the gastric contents. A right-sided gastrotomy can be incorporated into an incisional gastropexy.

For a clockwise rotation, once the stomach has been decompressed, rotate it counterclockwise by grasping the pylorus (usually found below the esophagus) with the right hand and the greater curvature with the left. Push the greater curvature, or fundus, of the stomach toward the table while simultaneously elevating the pylorus toward the incision. Check to make sure the spleen is normally positioned in the left abdominal quadrant. If there is splenic necrosis or significant infarction, perform a partial or complete splenectomy (see pp. 641–642). Remove or invaginate (see Fig. 18.68) necrotic gastric tissues (Fig. 18.90). If you are uncertain whether gastric tissue will remain viable, invaginate or resect the abnormal tissue (see p. 402). Verify that the gastrosplenic ligament is not torsed, and before closure palpate the intraabdominal esophagus to ensure that the stomach is derotated.

Perform a permanent gastropexy (see pp. 406–414). Gastropexy is usually curative for dogs with partial or chronic GDV.

NOTE To prevent recurrence of GDV, the stomach must be permanently adhered to the body wall. However, gastropexy does not guarantee that dilatation or volvulus will not recur; it simply makes it less likely. Gastropexy should always be performed in conjunction with abdominal exploration and derotation of the stomach.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Absorbable (polydioxanone or polyglyconate) or nonabsorbable (polypropylene) suture material may be used for the gastropexy (0 or 2-0). A Foley catheter or a mushroom-tipped silicone catheter is needed for a tube gastropexy. Balfour retractors, handheld retractors (i.e., Army-Navy retractors or malleable retractors), and extra towel clamps (for placement on the rib when doing a circumcostal gastropexy) are helpful.

POSTOPERATIVE CARE AND ASSESSMENT

Electrolyte, fluid, and acid-base status should be monitored closely postoperatively. Many dogs with GDV are hypokalemic postoperatively and require potassium supplementation (see Table 4.6). Small amounts of water and soft, low-fat food should be offered 12 to 24 hours after surgery and the patient observed for vomiting. Gastritis that occurs secondary to mucosal ischemia is common and may be associated with gastric hemorrhage or vomiting. If vomiting is severe or continuous, a centrally acting antiemetic may be given (see p. 419). Secondary gastric ulcers may occur and require treatment (see p. 429). Proton pump inhibitors (omeprazole, pantoprazole; see Box 18.13) are the recommended drugs to reduce gastric acidity if ulceration is present. IV fluid therapy should be continued until the patient's oral fluid intake is adequate to maintain hydration. Patients should be monitored for hypoalbuminemia and anemia in the early postoperative period.

NOTE Lidocaine toxicity may be enhanced in patients given cimetidine concurrently.

Ventricular arrhythmias are common in dogs with GDV and usually begin 12 to 36 hours after surgery. Their cause is unknown, but myocardial depressant factor, reduced cardiac output, and myocardial ischemia may contribute. Treatment of cardiac arrhythmias includes maintenance of normal hydration and correction of electrolyte imbalances. Sometimes the arrhythmia can be corrected simply by correcting hypokalemia. Some antiarrhythmic drugs (i.e., lidocaine) are ineffective when the animal is hypokalemic (see Table 4.6). If the arrhythmias interfere with cardiac output (as noted by poor peripheral pulses), are multiform, have subsequent premature beats inscribed on the wave of the previous complex (R on T), or have a sustained ventricular rate above 160 beats per minute, the animal should be treated with IV drugs. A test bolus of lidocaine given intravenously (2 mg/kg bolus, up to 8 mg/kg total dose) can be used to determine responsiveness to this drug. If the arrhythmias diminish or stop, lidocaine should be given by a continuous IV infusion of 50 to 75 µg/kg per minute (Box 18.29). Low doses should be used initially and increased only if necessary. Signs of lidocaine toxicity include

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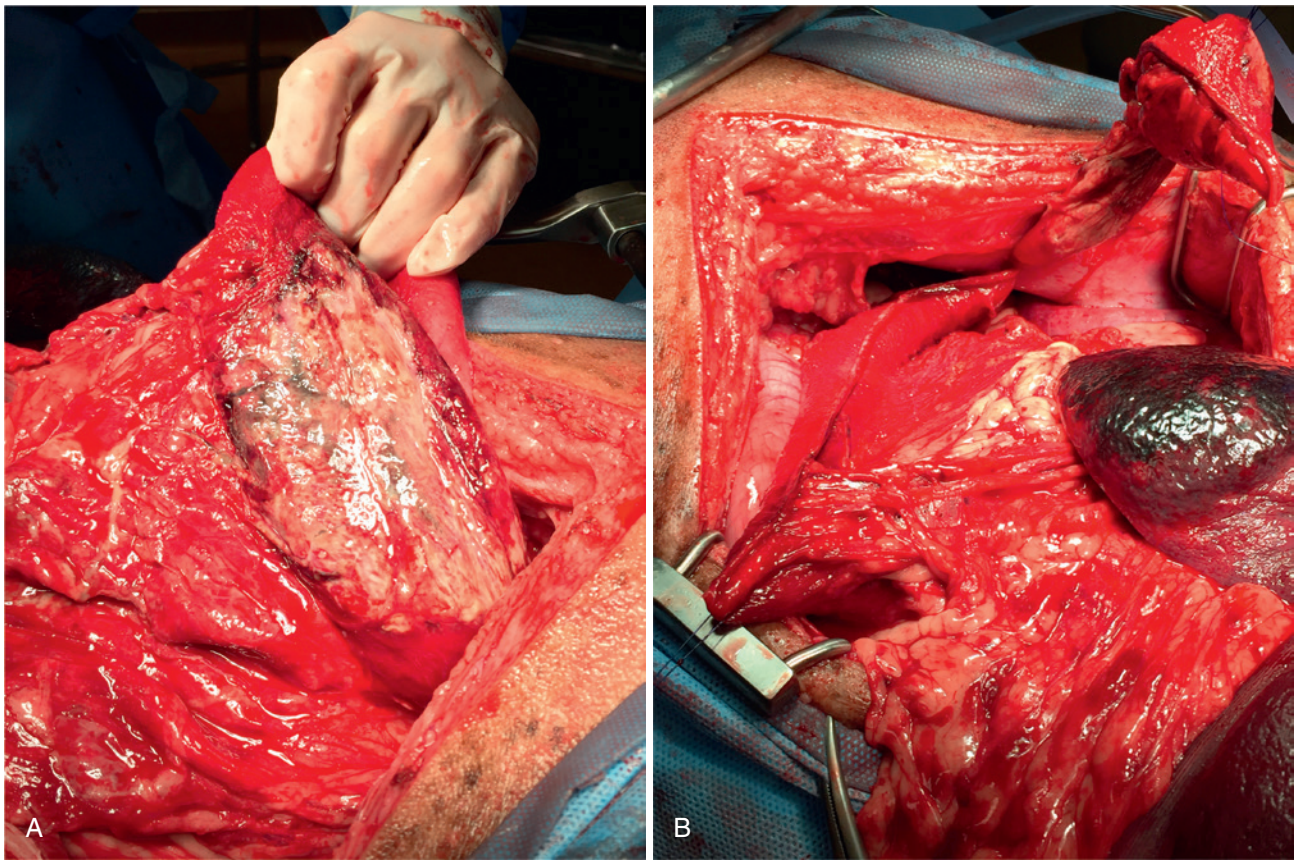


FIG. 18.90 Intraoperative appearance of the stomach of a dog with gastric dilatation-volvulus. (A) Gastric compromise due to gastric dilatation-volvulus. (B) Appearance of the stomach following resection of the compromised portion.

muscle tremors, vomiting, and seizures; lidocaine therapy should be discontinued if these signs occur. Other possibly effective antiarrhythmic drugs are procainamide and sotalol. Procainamide may be given as an IV bolus, by continuous infusion, intramuscularly, or orally (see Box 18.29). Sotalol may be effective in animals that have not responded to lidocaine or procainamide.

COMPLICATIONS

Sepsis and peritonitis may be caused by gastric necrosis or perforation if devitalized tissue is not adequately removed. Diagnostic peritoneal lavage (see p. 534) may help diagnose peritonitis. Peritonitis requires immediate surgical intervention. DIC occurs in approximately one-fifth of dogs with GDV. These dogs are more likely to die than those that do not have DIC; therefore assessment of clotting parameters and appropriate treatment with plasma, fluids, and heparin (see Box 22.7) is important to improve survival. Cardiac arrhythmias are common in dogs with GDV (~45.5% are affected), but arrhythmias do not appear to be associated with an increased risk of death in these dogs.

PROGNOSIS

Mortality rates of 45% and higher have been reported; however, mortality rates as low as 10% are more common with timely, aggressive, rapid therapy. Gastric dilatation without volvulus has a better prognosis than GDV; however, the degree of rotation is not associated with survival in dogs with GDV. Breeds at greatest risk for mortality may be the bloodhound, German longhaired pointer, and Neapolitan mastiff. The prognosis is poor if gastric necrosis or perforation occurs or if surgery is delayed. Preoperative measurement of plasma lactate may be a good predictor of gastric necrosis and outcome for dogs with GDV. Plasma lactate concentrations under 7.4 mmol/L suggest that gastric necrosis is not present, and thus a fair prognosis is warranted. Dogs with gastric necrosis are approximately 10 times more likely to die than those that do not have gastric necrosis. Recurrence rates for GDV differ, depending on techniques used, but most have reported rates of less than 10%. Tube gastropexy has the highest reported recurrence rate, varying from 5% to 29%.

Some dogs with GDV respond to tube decompression and medical stabilization alone. Occasionally the stomach becomes normally positioned after the air is removed, or if it was only partly rotated (less than 180 degrees), or merely dilated. However, these dogs still have a high likelihood of recurrence, and gastropexy should be recommended even when conservative management successfully alleviates the gastric malpositioning. The reported recurrence rates in dogs operated on for GDV in which the stomach was repositioned but gastropexy was not performed approaches 80%.

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BOX 18.29 Antiarrhythmic Therapy in Dogs

Intravenous Antiarrhythmic Drugs—Ventricular

Lidocaine

IV bolus (2 mg/kg increments up to total dose of 8 mg/kg) then IV drip at 50 µg/kg/min (500 mg in 500 mL of fluid, administered at maintenance rate [66 mL/kg per day]). CRI can be increased to 75–100 µg/kg/min if indicated clinically.

NOTE: If seizures occur, stop the drug and consider using another antiarrhythmic agent.

Procainamide (Pronestyl)

5–25 mg/kg slow IV bolus then 25–50 µg/kg/min as CRI

Amiodarone[a]

1 mg/kg IV repeated up to a total cumulative dose of 3 mg/kg; alternatively 150 µg/kg/min as a CRI.

Intravenous Antiarrhythmic Drugs—Supraventricular

Diltiazem (Cardizem)

1–8 µg/kg/min CRI for acute SVT

Oral Antiarrhythmic Drugs—Ventricular

Amiodarone[a]

10–20 mg/kg PO q24h for 5–7 days, then 5–10 mg/kg PO q24h (may be able to reduce to q48h)

Mexiletine (Mexitil)

4–8 mg/kg PO q8h

Sotalol (Betapace)

1–3 mg/kg PO q12h

Oral Antiarrhythmic Drugs—Supraventricular

Extended-Release Diltiazem (Dilacor XR; Cardizem CD)

2–4 mg/kg PO q12h

Sotalol (Betapace)

1–3 mg/kg PO q12h

Amiodarone[a]

10–20 mg/kg PO q24h for 5–7 days, then 5–10 mg/kg PO q24h (may be able to reduce to q48h)

Digoxin (Lanoxin)

0.005–0.01 mg/kg PO q12h

Atenolol (Tenormin)

0.1–2.0 mg/kg PO q12–24h (gradually uptitrate the dose)

[a]Solvents used in the commercial intravenous formulations can result in acute anaphylactic reactions.

CRI, Constant-rate infusion; *IV,* intravenous; *PO,* orally; *SVT,* supraventricular tachycardia.

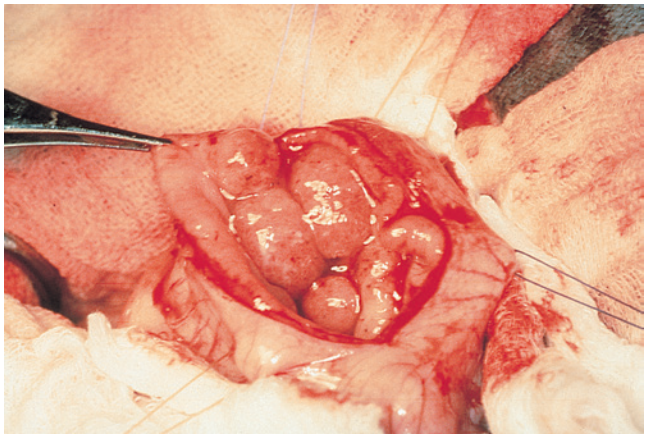


FIG. 18.91 Intraoperative image showing mucosal hypertrophy in a dog with chronic hypertrophic pyloric gastropathy.

BOX 18.30 Important Considerations for Gastric Outlet Obstruction

- Not all older patients with proliferative masses causing outlet obstruction have malignancies.
- Not all obstructed patients have hypokalemic, hypochloremic, metabolic alkalosis.
- Not all animals with hypokalemic, hypochloremic, metabolic alkalosis have gastric outlet obstruction.
- Benign gastric outflow obstruction usually has a good prognosis with appropriate therapy.
- Gastroduodenoscopy is usually more appropriate than contrast radiographs; with endoscopy, you can biopsy and often determine if malignancy is present, as well as remove foreign objects.

BENIGN GASTRIC OUTFLOW OBSTRUCTION

DEFINITIONS

Pyloric stenosis refers to benign muscular hypertrophy of the pylorus. **Chronic antral mucosal hypertrophy** refers to benign hypertrophy of the pyloric mucosa causing outflow obstruction (Fig. 18.91). **Chronic hypertrophic pyloric gastropathy (CHPG)** is a term that denotes pyloric hypertrophy without specifying whether the mucosa or the muscularis is involved. CHPG has been used specifically to refer to acquired mucosal hypertrophy by some authors and to either muscular (type I) or mucosal (types II and III) hypertrophy by others. **Polyps** are benign, adenomatous proliferations that can be single or multiple. Pyloric stenosis is also known as *benign antral muscular hypertrophy, congenital hypertrophic stenosis*, and *congenital pyloric muscle hypertrophy*. Chronic antral mucosal hypertrophy is also called *pyloric* or *gastric mucosal hypertrophy, chronic hypertrophic gastritis, multiple polyps of the gastric mucosa,* and *acquired hypertrophy.*

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Gastric outlet obstruction may be caused by pyloric abnormalities, disorders of gastric motility, or extrinsic lesions compressing the outflow tract (e.g., pancreatic, duodenal, or hepatic neoplasia; Box 18.30). Hypertrophy of the pyloric mucosa or muscle may be isolated or may occur in conjunction with other abnormalities. A syndrome of polycystic kidneys, hepatic disease, polyneuropathy, and hypertrophic gastropathy has been described in the Drentse Patrijshond breed. Hyperplastic pyloric polyps have been reported in French bulldogs with a history of chronic vomiting since weaning.

The cause of pyloric stenosis is unknown, but excessive production of gastrin has been suggested. Gastrin is the major regulator

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of gastric acid secretion and is trophic for gastric smooth muscle and mucosa. Congenital pyloric stenosis has been produced in puppies by administering gastrin to pregnant bitches. Neurogenic dysfunction may also play a role. Acute stress, inflammatory disease, or trauma might stimulate the sympathetic nervous system, reducing gastric motility and causing retention. Prolonged gastric distention may then lead to increased gastrin secretion and subsequent hypertrophy.

DIAGNOSIS

Clinical Presentation

Signalment

In dogs, pyloric stenosis is most commonly seen in brachycephalic breeds (i.e., boxers, bulldogs, and Boston terriers). Siamese cats have also been reported with this condition. Affected cats may have both vomiting (caused by gastric outlet obstruction) and regurgitation (caused by secondary esophagitis and esophageal dysfunction). Pyloric stenosis is more common in young animals, although animals of any age may be affected. Chronic antral mucosal hypertrophy occurs most commonly in small-breed dogs (less than 10 kg), particularly Lhasa apso, shih tzu, and Maltese breeds. Some dogs reported with chronic antral mucosal hypertrophy have been considered particularly excitable or vicious. Males may be more commonly affected than females. Chronic antral mucosal hypertrophy is more common in middle-aged or older dogs and may mimic neoplasia.

History

The clinical signs are caused by obstruction of gastric outflow. Vomiting is the most common sign, and it may be intermittent or delayed hours after feeding or both. Cats commonly have regurgitation and vomiting. Liquids often pass through the pylorus; therefore severe dehydration is uncommon and vomiting may occur for months to years before diagnosis. Animals with congenital pyloric stenosis often begin vomiting when they start eating solid food. The frequency of vomiting varies from several times daily to once or twice a week.

Physical Examination Findings

The physical examination findings are generally nonspecific. They may include weight loss, anorexia, depression, and/or dehydration. Abdominal pain is rare. Aspiration pneumonia or reflux esophagitis (or both) may occur secondary to chronic vomiting.

Diagnostic Imaging

Survey abdominal radiographs and ultrasound may reveal gastric distention (usually filled with fluid) and are helpful for eliminating extrinsic causes of pyloric obstruction. Ultrasonography usually reveals pyloric wall thickening and may detect extrinsic lesions (e.g., abscesses or neoplasms), obstructing gastric outflow as well as neoplastic metastases. Contrast radiographs may show delayed emptying, pyloric wall thickening, and/or a filling defect in the pylorus. However, normal emptying of liquid barium does not rule out gastric outflow obstruction, and it can be difficult to interpret studies when barium is mixed with food. Contrast radiographs are now infrequently used to detect gastric outflow obstruction because endoscopy is probably at least as sensitive and also allows biopsy of any lesions present. Radiographs and ultrasound cannot accurately distinguish inflammation, hypertrophy, or neoplasia, whereas endoscopy usually can, thereby hopefully avoiding unnecessary surgery.

NOTE Neoplastic pyloric disease and benign disease causing hypertrophy are often difficult to distinguish visually; biopsy is imperative!

Laboratory Findings

The hematologic and biochemical changes in animals with benign gastric outlet obstruction are usually nonspecific. If vomiting has caused loss of gastric secretions, a hypochloremic, hypokalemic metabolic alkalosis may be present (but it is not invariable). Prerenal azotemia may occur, and mild hypoalbuminemia is sometimes seen in young dogs that are severely emaciated.

DIFFERENTIAL DIAGNOSIS

Any condition that causes vomiting is a differential diagnosis. GI foreign bodies, pythiosis, inflammation, neoplasia, and ulceration may cause gastric outlet obstruction. Other causes of vomiting that should be eliminated before surgery are uremia, hypoadrenocorticism, hypercalcemia, diabetic ketoacidosis, hepatic insufficiency, peritonitis, pancreatitis, feline hyperthyroidism, early right-sided heart failure in cats, gastritis, and inflammatory bowel disease (IBD).

MEDICAL MANAGEMENT

Dehydration, electrolyte, and acid-base abnormalities should be corrected before surgery or endoscopy (see p. 398). Proton pump inhibitors may be used to treat esophagitis caused by frequent exposure of the esophagus to gastric acid (Box 18.31). Antibiotics may be indicated for aspiration (see p. 367). Gastric prokinetics (e.g., metoclopramide, cisapride) should not be used if outflow obstruction is suspected.

SURGICAL TREATMENT

Surgery is recommended for benign pyloric obstruction. The goal is to remove the obstruction and reestablish normal gastric emptying. A full-thickness biopsy should be submitted to ensure that the thickening is benign (Fig. 18.92). Benign polyps should be removed if they are obstructing gastric outflow or if they are bleeding.

Preoperative Management

Food should be withheld for 24 hours before surgery. Presurgical endoscopy can define the extent of the lesion and usually confirm its benign or malignant nature by histologic or cytologic examination. IV prophylactic antibiotics (e.g., cefazolin; 22 mg/kg given IV; repeat once or twice at 2- to 4-hour intervals) may be given

BOX 18.31 Proton Pump Inhibitors

Omeprazole (Prilosec)[a]
1–2 mg/kg PO q12h

Pantoprazole (Protonix)
1 mg/kg IV q12–24h

[a]Inhibits hepatic P450 enzymes and decreases hepatic blood flow.
IV, Intravenous; *PO,* orally.

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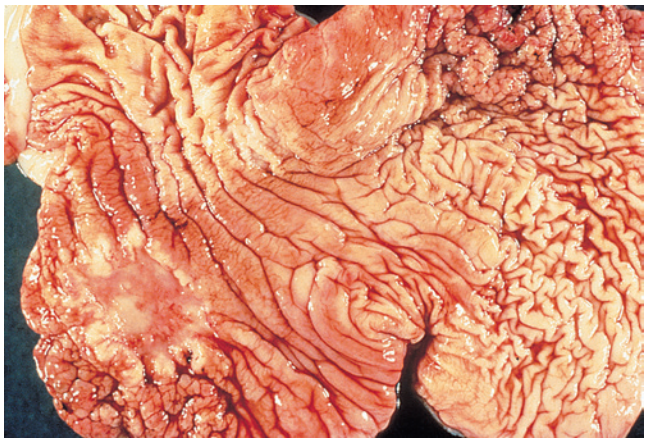


FIG. 18.92 Gastric ulceration associated with a gastric carcinoma.

at induction of anesthesia if antibiotic therapy has not already been initiated, but this is not essential.

Anesthesia

See p. 399 for suggested anesthetics to use in animals with gastric disorders. See Table 18.3 for management of patients at risk for aspiration.

Surgical Anatomy

See p. 399 for the surgical anatomy of the stomach.

Positioning

The animal is placed in dorsal recumbency and the abdomen is prepared for a ventral midline incision. The prepped area should extend from midthorax to near the pubis.

ENDOSCOPIC REMOVAL OF POLYPS

Polyps with a narrow stalk can be removed by using an electrosurgical snare. The area should be free of fluids and debris. *Carefully place the snare around the base of the polyp and then pull tight. Do not touch the metallic part of the snare to other aspects of the stomach, and hold the polyp up such that it is not bent over and touching gastric mucosa. Keep the tip of the snare several centimeters away from the tip of the endoscope lest the current go back up the endoscope and damage the video processor. Apply a mixed coagulation/cutting setting while gradually tightening the snare around the base of the polyp. Start at lower settings and gradually increase the current as needed.* If excessive coagulation current is applied, a severe electrical burn may cause mucosal inflammation with subsequent anorexia and vomiting, or in more severe cases, perforation. *Do the process slowly to be sure that the vascular supply to the polyp is coagulated and will not cause hemorrhage.* This procedure should generally only be done by individuals trained in using endoscopic electrosurgery. After the mass is cut off, it can be retrieved for histopathology.

SURGICAL TECHNIQUE

Surgical procedures to correct outlet obstruction caused by mucosal and/or muscular hypertrophy include pyloroplasty or Billroth I procedures. Pyloromyotomy (see p. 403) is often ineffective and is not recommended. If muscular hypertrophy without significant mucosal hypertrophy is present, a Heineke-Mikulicz pyloroplasty (see p. 405) is easy to perform. However, mucosal exposure is limited, and it does not allow adequate resection of hypertrophied mucosa. When mucosal hypertrophy is present, a Y-U pyloroplasty (see p. 405) or Billroth I procedure (see p. 403) is preferred. A Y-U pyloroplasty allows hypertrophied mucosa to be removed and the outflow tract widened (see Fig. 18.74). A Billroth I procedure is more difficult and carries the additional risk of dehiscence or leakage but has been successfully performed in numerous dogs with benign outflow obstruction. A Billroth I procedure, rather than a pyloroplasty, should be considered when the mucosa or muscular layers are so thickened as to be inflexible.

For mucosal hypertrophy, perform a Y-U pyloroplasty as described on p. 405. Be sure to perform a full-thickness biopsy and to resect the hypertrophied mucosa. Approximate incised edges of the mucosa in a continuous pattern with 3-0 absorbable suture before closing the pyloroplasty.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

If the animal is severely hypoproteinemic or debilitated, wound healing may be delayed. Strong absorbable suture (2-0 or 3-0) should be used and chromic gut suture avoided.

POSTOPERATIVE CARE AND ASSESSMENT

Small amounts of water should be given the day after surgery and the patient observed for vomiting. If vomiting does not occur, small amounts of moist food can be given 24 hours postoperatively. Fluid therapy should be continued until the animal is eating and drinking normally. Electrolyte abnormalities should be monitored postoperatively and corrected as necessary.

PROGNOSIS

The prognosis with surgical correction of these conditions is good. Most dogs at long-term evaluation will have good or excellent outcomes. A poor outcome is generally the result of technical failures (i.e., dehiscence or leakage) or of an inappropriate surgical technique for the lesion.

GASTRIC ULCERATION AND EROSION

DEFINITIONS

An **ulcer** is a mucosal defect extending through the muscularis mucosae into the submucosa or deeper layers of the stomach, whereas an **erosion** does not penetrate the muscularis mucosae. **Gastrinomas** are gastrin-secreting tumors of the alimentary tract. **Zollinger-Ellison syndrome** is a condition in which duodenal ulceration occurs as a result of hypersecretion of gastrin from a gastrinoma of the pancreas. The terms *gastrinoma* and *Zollinger-Ellison syndrome* are often used interchangeably; however, gastrinomas can be located anywhere in the alimentary tract, whereas Zollinger-Ellison syndrome refers specifically to a gastrin-secreting tumor in the pancreas.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Gastric ulceration/erosion (GUE) in small animals is often iatrogenic (i.e., NSAIDs, dexamethasone) or occurs secondary

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BOX 18.32 Important Considerations for Gastroduodenal Ulcers and Erosions

- Initial clinical signs (e.g., anorexia, depression) may not alert owner to seriousness of disease.
- Not all animals with ulcers vomit, and those that vomit may not vomit blood.
- Nonsteroidal antiinflammatory drugs are a very common cause, yet without careful questioning the history may not reveal their use.
- Look for underlying causes; do not just treat symptomatically.
- Anytime a patient with severe hepatic disease suddenly worsens, consider gastroduodenal ulceration even if there is no vomiting.
- Perforation may occur unexpectedly and cause potentially fatal peritonitis (see pp. 449 and 527).
- Any patient with a spontaneous pneumoabdomen or septic peritonitis should be presumed to have a perforated ulcer and should be treated accordingly, regardless of lack of historical findings suggestive of ulceration.
- Surgical resection should be considered for ulcers resistant to medical therapy and those causing the patient to hemorrhage vigorously.
- Intraoperative endoscopy may be helpful in locating ulcers.

to an underlying disease process (e.g., shock, gastric tumor, hepatic failure) (Box 18.32).

NSAIDs (e.g., aspirin, phenylbutazone, naproxen, flunixin meglumine, piroxicam, deracoxib, ibuprofen) are common causes of GUE in dogs. Newer NSAIDs primarily inhibiting cyclooxygenase (COX)-2 (e.g., carprofen, etodolac, deracoxib, meloxicam) are regarded as safer than the more traditional NSAIDs that inhibit COX-1. However, although these newer NSAIDs are safer and have fewer gastric complications, they can still cause GUE, especially if used improperly. Administration of doses higher than approved or concurrent administration of another NSAID or glucocorticoid increases the risk of GI perforation in dogs receiving COX-2 inhibitors. The mechanism of ulcer formation secondary to NSAIDs is probably multifactorial, but inhibition of prostaglandin synthesis is important. Prostaglandins exert a protective effect on the mucosal barrier by stimulating mucus and bicarbonate production. Prostaglandin agonists (e.g., misoprostol) may help prevent NSAID-induced lesions. In fact, inhibition of COX-2 delays healing of gastric ulcers.

Glucocorticoids (particularly dexamethasone) may be ulcerogenic in dogs, especially when used at high dosages. Prednisolone, when administered at appropriate dosages (1 mg/kg per day or less), infrequently causes GUE unless the dog is hypoxic (e.g., immune-mediated hemolytic anemia) or hypoperfused. Chronic steroid administration may reduce gastric mucus production, diminish the ability of mucosal cells to replicate, and increase exfoliation of mucosal cells into the gastric lumen. Concurrent use of steroids and NSAIDs (except for ultra-low dose aspirin used to prevent thrombosis) clearly increases the likelihood of GUE.

Gastric ulceration may be caused by neoplasia (see Fig. 18.92), either as a direct or paraneoplastic effect. Gastric adenocarcinoma and lymphoma are probably the most common gastric tumors, but leiomyomas in the body of the stomach are especially prone to ulcerate and bleed. Paraneoplastic ulceration caused by mast cell tumors (common) or gastrinomas (rare) primarily occurs in the duodenum, just past the pylorus. Gastroduodenal ulceration is a common complication of mast cell disease because histamine is a potent stimulator of gastric acid secretion. The cytoplasmic granules of mast cell tumors contain vasoactive amines (e.g., histamine and serotonin) and heparin. Histamine also causes vasodilation of gastric vessels and alters endothelial permeability, which promotes intravascular thrombosis and gastric necrosis.

Gastrin, which is normally secreted by the antral G cells in response to vagal stimulation and gastric distention, is a potent stimulator of gastric acid secretion. Zollinger-Ellison syndrome is a condition in which hypersecretion of gastrin is associated with neoplasia of the non-β pancreatic islet cells. Severe duodenal ulceration is seen with this disease, and removal of the pancreatic mass may be necessary to alleviate clinical signs (see p. 613). Because of the aggressive biological behavior of this malignant neoplasm, the prognosis for long-term cure is poor; however, aggressive medical management with omeprazole (1–2 mg/kg given orally twice a day) may palliate the disease for a year or more.

Acute and chronic hepatic disease may be associated with GUE with or without bleeding. Chronic hepatic disease causes gastric mucosal injury through a variety of mechanisms, which are poorly understood. Thrombosis associated with DIC may reduce gastric blood flow and enhance ulcer formation.

Circulatory shock causing poor gastric perfusion is one cause of "stress" GUE. Ulcers and erosions may also form secondary to SIRS (also known as *septic shock*), extreme exertion, and intervertebral disc disease. Administration of high doses of glucocorticoid to dogs with severe neurologic disease probably contributes to the high prevalence of GUE in these patients. Other contributing factors in dogs with intervertebral disc disease include alterations in mucosal blood flow, sympathetic and parasympathetic stimulation of the bowel, and the physiologic stress of major surgery. Colonic perforation of dogs with neurologic disease is associated with high mortality. Other conditions associated with GUE in small animals include gastroduodenal reflux of bile, major surgery, and pythiosis.

NOTE Use steroids with caution in patients with neurologic disease.

The stomach has an enormous ability to increase local blood flow, which helps remove caustic substances from the gastric lumen. In addition, the rapid rate of cell turnover in the gastric mucosa helps heal minor erosions in 1 to 2 days, providing the cause is removed. Other normal defenses that help prevent the formation of ulcers include those properties that interfere with absorption of hydrogen ions (e.g., phospholipid membranes, tight junctional complexes), neutralization of acid by bicarbonate (secreted by the oxyntic, pyloric, and duodenal mucosae), and a thick, alkaline mucus coating that traps and neutralizes hydrogen ions. For mucosal damage to occur, the gastric pH must usually be lower than 3 to 5 (there are other factors besides acid which promote gastric ulceration). Deep ulcers do not heal rapidly, and they heal by the formation of scar tissue rather than by reepithelialization.

DIAGNOSIS

Clinical Presentation

Signalment

GUE occurs more commonly in dogs than cats. Most noniatrogenic gastric ulcers in dogs occur in middle-aged or older dogs. The disorder shows no breed predisposition.

History

Although vomiting is a common clinical sign of GUE, some dogs are presented for anorexia or anemia (or both) without

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vomiting, and sometimes GUE is an unexpected finding in dogs without clinical signs of disease. Vomitus may or may not contain digested blood, fresh blood, or blood clots. Digested blood looks like coffee grounds. Owners only rarely report that the stools of dogs with ulcers are black (melena) and that the dog has a poor appetite.

Physical Examination Findings

Most patients have no abnormalities on physical examination unless they are anemic from blood loss. Some have abdominal pain when palpated. Rectal examination sometimes reveals melena. However, most of the time what people describe as "dark feces" is normal feces that is dark brown or dark green. Melena is pitch-black feces that, when put on white, absorbent paper, will be seen to have red color diffuse out from it. Melena only occurs when there has been substantial hemorrhage over a short period of time; most dogs with gastric bleeding do not have melena.

Diagnostic Imaging

Radiography and ultrasonography will not detect erosions. Positive contrast radiographs or ultrasonography may detect ulceration, but the sensitivity of these tests is relatively low. Gastroduodenoscopy is the most sensitive test for finding GUE. However, the entire mucosal surface must be examined. The patient should not receive Carafate or barium for at least 24 hours prior to endoscopy lest the lesions be obscured. If there has been GI bleeding, one must be prepared to flush and aspirate repeatedly until the debris has been removed and the mucosa can be examined. Ulcers just inside the pylorus are particularly hard to visualize.

Laboratory Findings

A hemogram and serum biochemical profile should be performed in animals in which GUE is suspected, to assess the severity of blood and protein loss and to identify underlying potential causes (e.g., hepatic failure). Animals with GUE may be anemic, hypoproteinemic, or both. Platelet counts and clotting profiles should be performed if the patient is bleeding and a coagulopathy is possible. Electrolyte and acid-base abnormalities may occur if vomiting has been severe (e.g., hypochloremic, hypokalemic metabolic alkalosis, or a metabolic acidosis). Gastrinomas (see p. 612) are uncommon, but if no other underlying cause is identified, serum gastrin levels can be measured.

DIFFERENTIAL DIAGNOSIS

Coagulopathies may mimic GUE. Gastric neoplasia and gastritis are best distinguished from other causes of GUE by endoscopic biopsy. Exploratory gastrotomy is required if full-thickness biopsies are necessary because the lesion is submucosal or cirrhotic and the flexible endoscope is unable to tear off a diagnostic piece. Coagulopathies from ingestion of toxins, DIC, or inherent clotting abnormalities occasionally cause gastric bleeding. Coagulation profiles should be performed if bleeding is noted.

MEDICAL MANAGEMENT

Therapy depends on the severity of the bleeding, the depth of the ulcer, the likelihood of perforation, the animal's status, and whether an underlying cause is found or suspected. Medical treatment is typically recommended to control bleeding if perforation seems unlikely and if the cause of the bleeding is known or strongly suspected. Symptomatic therapy (i.e., fluids, blood, antiemetics) is usually administered when trying to identify and remove the initiating cause (e.g., discontinue NSAIDs, reestablish mucosal perfusion, remove mast cell tumors or gastrinomas, or treat hepatic disease). Gastric foreign bodies will inhibit GUE healing even if they are not causing it. Therefore they should be removed promptly to facilitate healing.

BOX 18.33 Medical Therapy of Animals with Gastric Ulceration

Omeprazole (Prilosec)
1–2 mg/kg PO q12h

Sucralfate (Carafate; Only Use Suspension, Not Tablets)
0.5–1.0 g PO q6–8h

Misoprostol (Cytotec)
1.0–5.0 µg/kg PO q8–12h

Pantoprazole (Protonix)[a]
1 mg/kg IV q12–24h

[a]Dose is anecdotal.
IV, Intravenous; *PO,* orally.

Drugs used to treat GUE include those that lessen gastric acidity and those that protect the gastric mucosa from damage (Box 18.33). Omeprazole, esomeprazole, pantoprazole, and other proton pump inhibitors are more potent inhibitors of gastric acid secretion than the H_2-receptor antagonists. They require 2 to 5 days to achieve their maximal effect, but their initial effect is usually better than that of the H_2-receptor antagonists. Combining H_2-receptor antagonists with proton pump inhibitors is not more effective than proton pump inhibitors alone and may lessen the efficacy of the proton pump inhibitors.[32] Antacids (e.g., magnesium hydroxide) stimulate endogenous prostaglandin release, neutralize acids, and bind bile salts. Because they are most effective if administered frequently (i.e., up to six times a day), they are less useful in dogs and cats than in human beings. Misoprostol is a prostaglandin analog that helps prevent ulceration in dogs receiving NSAIDs, and it can be used to cure gastric ulcers. However, proton pump inhibitors have fewer side effects and are probably as effective.

Sucralfate forms a protective coating over the ulcer or erosion. However, its major drug actions that contribute to ulcer healing are related to stimulation of mucosal defense and reparative mechanisms and antipeptic effects, which are induced by both prostaglandin-dependent and prostaglandin-independent pathways. The suspension is probably more effective than the tablet. Sucralfate should be given 1 hour after administration of other oral medications because it may interfere with their absorption. Drugs that interact with sucralfate include fluoroquinolones, tetracycline, theophylline, aminophylline, and digoxin. The major disadvantage of sucralfate is that it must be administered orally, which can be problematic in vomiting animals.

ENDOSCOPIC TREATMENT

Endoscopic electrocautery may be used to stop hemorrhage in emergency situations and allow surgery to occur at a more opportune time. *Once the bleeding lesion has been identified, advance an electrocautery probe with suction capability until the*

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tip of the probe is in contact with the bleeding lesion. Keep the tip of the electrocautery probe several centimeters away from the tip of the endoscope. If there is fluid, blood, or debris, suction it away to allow for as dry a field as possible, and then apply a low coagulation current for 2 to 5 seconds. If that is not successful, aspirate the field again and use more current with a coagulation setting. However, do not apply a coagulation current for long periods of time because this increases the risk of perforation. "Hot" biopsy forceps should not be used as their use is associated with an increased risk of complications. Endoscopic electrosurgery should only be done by individuals trained in its use, or damage to the scope and patient may occur. Alternatively, one may use sclerotherapy needles to inject alcohol or other such agents into bleeding lesions, but there are no reports of this being done in clinical veterinary medicine.

SURGICAL TREATMENT

Surgical resection of ulcers is indicated if medical therapy does not substantially alleviate clinical signs within 6 to 7 days, if bleeding is profuse and life-threatening, or if perforation is believed imminent.

NOTE Gastric ulcers, even very deep ones, cannot be reliably detected from the serosal surface of the stomach. Preoperative (or intraoperative) endoscopy will allow accurate localization of all important gastric ulcers.

Preoperative Management

If possible, the animal's condition should be stabilized before surgery. Whole blood should be given if the animal is severely anemic (i.e., has a packed cell volume [PCV] under 20%). If the animal has DIC, one may administer plasma with or without heparin therapy. Electrolyte and acid-base abnormalities should be corrected and fluid therapy initiated.

Anesthesia

See p. 399 for anesthetic recommendations for animals undergoing gastric surgery. See Table 19.1 for anesthetic recommendations for stable animals undergoing abdominal surgery and Table 19.2 for the septic patient needing abdominal surgery.

Surgical Anatomy

See p. 399 for the surgical anatomy of the stomach.

Positioning

The dog is placed in dorsal recumbency, and the abdomen is prepared for a ventral midline incision. The prepped area should extend from midthorax to the pubis.

SURGICAL TECHNIQUE

If possible, remove the ulcer with a full-thickness gastric resection and submit tissue for histopathologic examination. Assess the regional lymph nodes and liver for evidence of metastatic neoplasia or pythiosis, and biopsy them if they appear abnormal. Check both limbs of the pancreas for masses. Occasionally, location of the ulcer near the pylorus makes full-thickness resection difficult. *If the ulcer is located at the pylorus and perforation is present or imminent, perform a serosal patch (see p. 447) over the site to help prevent leakage and promote healing of the ulcer.* A serosal patch is simpler to perform than a pylorectomy and gastroduodenostomy (Billroth I; see p. 403). Occasionally an abscess will be noted where an ulcer has perforated and the omentum or other abdominal structures have walled-off the site. *If this is the case, carefully drain the abscess and resect or patch the ulcer. Consider preoperative or intraoperative endoscopy to help locate ulcers that are difficult to discern from the serosal surface. If there is extensive disease secondary to a condition that may not resolve quickly (e.g., IBD or hepatic failure), place an enterostomy feeding tube (see p. 101).*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

If the animal is severely hypoproteinemic or anemic, wound healing may be delayed. Polydioxanone, polyglyconate, polyglycolic acid, or polyglactin 910 suture (2-0 or 3-0) is preferred to close the gastrotomy incision (see p. 415). Gut suture should be avoided for gastric surgery.

POSTOPERATIVE CARE AND ASSESSMENT

Small amounts of water should be given the day after surgery and the patient observed for vomiting. If vomiting does not occur, small amounts of food can be given 24 hours postoperatively. To aid gastric emptying, the diet should be low fat and contain moderate amounts of protein and carbohydrates. Moist diets are usually preferable to dry diets. Fluid therapy should be continued until the animal can maintain hydration with oral fluids.

PROGNOSIS

The prognosis depends on identification and treatment of underlying diseases and whether peritonitis is present. The prognosis is good if the ulcer is the result of treatable disease and perforation has not occurred; however, euthanasia or death is common in dogs that develop perforation of the GI tract while receiving COX-2 inhibitors. If peritonitis is present, the prognosis is guarded (see the discussion of peritonitis on pp. 449 and 527).

GASTRIC NEOPLASIA AND INFILTRATIVE DISEASE

DEFINITIONS

Adenocarcinomas arise from glandular tissue or are composed of tumor cells that form glandular structures. The term **lymphoma** denotes a malignant neoplasm arising from the lymphoid system. **Leiomyosarcomas** are malignant tumors and **leiomyomas** are benign tumors that arise from smooth muscle. **Pythiosis** is a fungal infection caused by *Pythium insidiosum* that may cause a severe inflammatory and infiltrative lesion in the stomach. **Phycomycosis** is a more general term for mycoses caused by fungi of the group Phycomycetes. The terms *lymphoma* and *lymphosarcoma* are used synonymously to denote a malignant neoplasm of the lymphoid system.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Benign gastric tumors are more commonly found in dogs than in cats; however, most gastric neoplasms are malignant

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BOX 18.34 Important Considerations for Gastric Neoplasia

- Most gastric tumors are malignant.
- Anorexia, not vomiting, is the most common sign.
- Some patients are anemic.
- Most patients do not vomit until the neoplasm is well advanced or is causing gastric outflow obstruction.
- Neoplasia is a possible cause of ulceration in the dog and cat, but many neoplasms do not cause ulceration.
- Not all obstructed patients have hypokalemic, hypochloremic, metabolic alkalosis.
- Gastroduodenoscopy is usually more appropriate than contrast radiographs; with endoscopy, you can biopsy and often (not always) diagnose malignancy.

(Box 18.34). Adenocarcinoma is the most common canine gastric tumor, accounting for approximately 60% to 70% of cases. Adenocarcinomas tend to metastasize to the regional lymph nodes, the liver, the lungs, or all three, and they may appear diffusely infiltrative or nodular. They usually occur in the pyloric antrum or lesser curvature. Infection with *Helicobacter pylori* has been linked with gastric carcinoma and gastric mucosa–associated lymphoma in human beings, but dogs and cats have other species of *Helicobacter* in their stomachs. Other reported malignant gastric tumors in dogs include leiomyosarcoma, lymphosarcoma, and fibrosarcoma.

Lymphoma is the most common gastric tumor in cats; adenocarcinomas are rare. Most affected cats test negative for feline leukemia virus (FeLV). Lymphoma may be solitary or diffuse in the stomach and may or may not simultaneously affect the intestine.

Leiomyomas are the most common benign canine gastric tumor. They tend to be slow growing, submucosal, and expansile. Clinical signs may not be apparent until the tumors are large. Leiomyomas usually occur at the cardia, and complete surgical excision may be possible. Adenomatous polyps are occasionally found in dogs. They may be multiple and rarely cause clinical signs, but vomiting or anorexia (or both) may be seen if the polyps occur at the pylorus and cause obstruction. Other benign tumors rarely found in dogs include adenomas, lipomas, and fibromas.

Pythiosis is a fungal infection caused by *P. insidiosum* that can affect any part of the alimentary tract, as well as the skin. It is primarily found in the southeastern United States, particularly near the Gulf Coast, but has been found from California to Ohio to the east coast of the United States. The fungus causes intensive submucosal infiltration of fibrous connective tissue and a profound inflammatory reaction in the mucosa (often with eosinophils) and deeper layers of the gastric wall (Fig. 18.93). The organism is often difficult to find histologically, and large tissue samples that include substantial submucosa should be submitted.

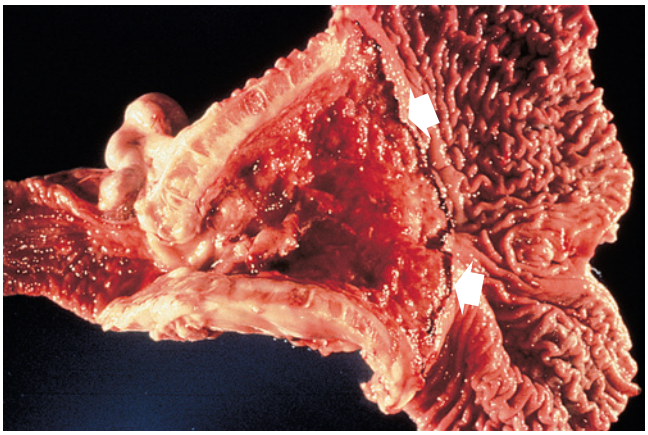


FIG. 18.93 Pythiosis affecting the pylorus and antrum of a dog's stomach. Note the sharp line of demarcation between normal and abnormal tissue *(arrows)*.

DIAGNOSIS

Clinical Presentation

Signalment

Belgian shepherds and chow chows may have a higher than normal incidence of gastric carcinoma, whereas beagles appear to have a higher incidence of leiomyoma. Males appear to be more commonly affected than females. Adenocarcinoma is most common in dogs 7 to 10 years of age. Gastric adenocarcinoma is extremely rare in cats. Lymphoma affects primarily middle-aged and older dogs (average age, 6 years) and cats but can occur in dogs and cats of any age. Pythiosis may affect dogs of any age.

History

Animals with gastric neoplasia or other infiltrative disease usually have a history of anorexia. Vomiting, hematemesis, melena, lethargy, weight loss, and/or edema may also occur. Some animals are relatively asymptomatic until the tumor becomes large enough to affect gastric emptying or starts bleeding. Clinical signs with pythiosis are generally the result of gastric outflow obstruction and gastric stasis.

Physical Examination Findings

The physical examination findings in animals with gastric neoplasia or pythiosis are often nonspecific (e.g., weight loss, anemia, and/or edema). Weight loss may be caused by anorexia, chronic vomiting, or cancer cachexia. Occasionally a large mass may be palpated in the stomach; however, detailed palpation of the stomach is usually difficult. Abdominal pain may be present with ulceration or pancreatitis.

Diagnostic Imaging

Noncontrast (survey) radiographs are generally nondiagnostic. Survey thoracic radiographs should be taken to rule out pulmonary metastasis (rare). Contrast radiographs may reveal filling defects, delayed gastric emptying, ulceration, loss of normal rugal folds, mucosal thickening, or loss of gastric wall compliance; however, contrast radiographs are being done less frequently because of the advantages that ultrasound and endoscopy have over such procedures.

Ultrasonographically, gastric neoplasia is associated with mural thickening with loss of normal wall and diminished to absent local motility. Ultrasonography is not sensitive for gastric neoplasia because of difficulties inherent in examining a motile hollow viscus that may contain air or food. An ultrasound-guided fine needle aspirate or core needle biopsy can sometimes provide a preoperative diagnosis. Ultrasonography may also detect metastasis to the liver or regional lymph nodes and help define the gastric lesion.

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Endoscopy

Endoscopy is the preferred diagnostic procedure because it finds almost all gastric tumors, as well as other mucosal diseases, plus it allows one to biopsy the lesion. However, tumors may be difficult to biopsy if they are completely submucosal or if they are scirrhous. Gross appearance can be very suggestive and help the client decide if surgery is appropriate. Finding a deep ulcer with a hard, dense, necrotic base surrounded by raised or distorted mucosa is suggestive of scirrhous carcinoma. Scirrhous tumors are very dense, and it may be impossible to endoscopically tear a diagnostic piece of tissue from the lesion. Leiomyomas and leiomyosarcomas are usually submucosal masses with or without ulcers. They are also hard to biopsy with flexible endoscopic forceps, but a diagnostic sample can usually be obtained if the endoscopist is persistent. Pythiosis causes severe mucosal inflammation and necrosis; however, the organism is best found in the dense, hard fibrotic reaction under the diseased mucosa. This makes it difficult to obtain a diagnostic tissue sample endoscopically.

Laboratory Findings

Clinical pathologic changes in animals with gastric neoplasia are usually nonspecific. A microcytic, hypochromic or a normocytic, normochromic anemia may occur. The anemia may be the result of blood loss or chronic disease. If biliary drainage is obstructed, icterus ensues. If vomiting causes a loss of gastric secretions, a hypochloremic, hypokalemic metabolic alkalosis with or without paradoxical aciduria may occur. Pythiosis may often be diagnosed serologically.

NOTE If the animal is icteric, the lesion is probably near the pylorus. Be prepared to perform a cholecystoenterostomy (see p. 574).

DIFFERENTIAL DIAGNOSIS

Anorexia may be associated with many systemic diseases (e.g., uremia, hypoadrenocorticism, hepatic insufficiency, hypercalcemia, inflammatory disease). Gastric outlet obstruction caused by nonneoplastic disease (i.e., mucosal hypertrophy, foreign object) may produce similar clinical signs (see p. 425). Cytologic or histologic examination of tissues (or both) is necessary to differentiate these conditions. Gastric foreign bodies can be differentiated from neoplasia with imaging or endoscopy. Pythiosis and neoplasia may be differentiated by cytology or histopathology (see Fig. 18.125).

MEDICAL MANAGEMENT

Medical management depends on the severity of the clinical signs. If possible, electrolyte, acid-base, hydration, and coagulation abnormalities should be corrected before surgery.

SURGICAL TREATMENT

Preoperative Management

Food should be withheld for 12 hours before surgery. Perioperative antibiotics may be given at induction of anesthesia and continued for up to 12 hours postoperatively.

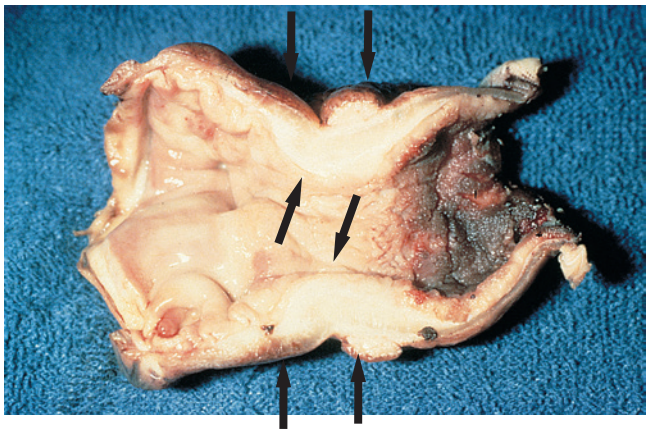


FIG. 18.94 A resected pylorus from a dog that had gastric outflow obstruction caused by a submucosal adenocarcinoma *(arrows)* that did not disrupt the mucosa.

Anesthesia

See p. 399 for suggested anesthetics to use in dogs with gastric disorders. See Table 19.1 for anesthetic recommendations for stable animals undergoing abdominal surgery and Table 19.2 for the septic patient.

Surgical Anatomy

See p. 399 for the surgical anatomy of the stomach.

Positioning

The animal is placed in dorsal recumbency, and the abdomen is prepared for a ventral midline incision. The prepped area should extend from midthorax to the pubis.

SURGICAL TECHNIQUE

With the exception of lymphoma, surgery is the only potentially curative treatment for gastric neoplasia (Fig. 18.94). Unfortunately, most carcinomas are not diagnosed until they are so far advanced as to be unresectable. *Palpate the regional lymph nodes for evidence of metastasis. Inspect the liver and other abdominal structures for metastasis or thickening, and biopsy suspicious lesions. If the lesion appears localized to the stomach, consider gastric resection, because it might be curative.*

If wide excision and bypass procedures, such as gastrojejunostomy and cholecystojejunostomy, are necessary (see p. 574), then surgery is of dubious value because of the likelihood of tumor recurrence, plus the likelihood of unacceptable morbidity from surgery. Solitary gastric lymphoma is rarely cured by surgery alone, and chemotherapy is only palliative for diffuse lymphoma. Likewise, wide surgical excision is currently the only potentially curative therapy available for pythiosis; however, obtaining wide surgical margins is difficult because of the extensive nature of the disease at diagnosis. Medical therapy of pythiosis is infrequently beneficial and almost never curative if the lesion cannot be resected.

If the lesion appears small enough that it can be resected without causing unacceptable morbidity, then wide surgical margins (including some normal tissue) should be obtained for both gastric malignancies and pythiosis. Cytologic examination of tissues submitted during the surgical procedure or frozen

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sections are helpful for determining the adequacy of the tissue margins. Surgical techniques for gastric resection are described on pp. 402 to 403.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Absorbable suture materials, such as polydioxanone or polyglyconate (2-0 or 3-0), should be used. Chromic gut suture should be avoided.

POSTOPERATIVE CARE AND ASSESSMENT

The patient's electrolyte and fluid status should be monitored postoperatively and deficiencies corrected. The animal can be fed a low-fat, bland diet beginning 24 hours after surgery if vomiting does not occur. If vomiting continues, centrally acting antiemetics such as maropitant and ondansetron (see Box 18.26) may be beneficial. An enterostomy feeding tube should be considered to help provide nutrition in the postoperative period (see p. 101).

PROGNOSIS

The prognosis is guarded for most malignant gastric neoplasms because they are not diagnosed until they are advanced (making resection impossible) and have metastasized. Despite aggressive surgery, most dogs with gastric adenocarcinoma or leiomyosarcoma will have recurrence of clinical signs and will be euthanized in less than a year. For animals with benign lesions, surgery may be curative. Pythiosis may be difficult to treat surgically because of its rapid growth rate and extensive nature, but surgical cures have been achieved if the infection is confined to the stomach.

Surgery of the Small Intestine

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Enterotomy is an incision into the intestine, and **enterostomy** is removal of a segment of intestine. **Intestinal resection and anastomosis** is an enterostomy with reestablishment of continuity between the divided ends. **Enteroenteropexy**, or **intestinal plication**, is surgical fixation of one intestinal segment to another; **enteropexy** is fixation of an intestinal segment to the body wall or another loop of intestine.

PREOPERATIVE CONCERNS

Surgery of the small intestine is most often indicated for GI obstruction (e.g., foreign bodies, masses). Other indications include trauma (e.g., perforation, ischemia), malpositioning, infection, and diagnostic or supportive procedures (e.g., biopsy, culture, cytology, feeding tubes).

Diagnosis of small intestinal disease is based on the history, clinical signs, physical examination, radiographs, ultrasound scans, laboratory data, endoscopy, and/or biopsy. Diet, medications, stressful events, and response to prior therapy should be ascertained from owners. Clinical signs of small intestinal disease vary and are nonspecific, although weight loss, diarrhea, vomiting, anorexia, and/or depression are the most common (Table 18.5). Pain and shock may result from trauma, vascular occlusion, or complete intestinal obstruction. Severe vomiting, shock, or an acute abdomen suggests intestinal malposition, ischemia, perforation, or upper intestinal obstruction. Visual examination provides information about the animal's mental state, temperament, nutritional state, and comfort. Abdominal palpation may identify pain, thickened intestine, abdominal masses, or malpositioned organs.

Hematologic and biochemical profiles should be performed on animals suspected of having small intestinal abnormalities to help identify concurrent systemic disease (e.g., renal disease, hepatic disease, hypoadrenocorticism, hypercalcemia, diabetes mellitus, pancreatitis) and to direct preoperative therapy (Box 18.35). Dehydration, acid-base abnormalities, and electrolyte imbalances are common sequelae to vomiting, diarrhea, and fluid sequestration. These abnormalities should be corrected before induction of anesthesia if possible. Alleviating hypotension is important because it is associated with intense portal vasoconstriction that causes the breakdown of the intestinal mucosal barrier, allowing increased endotoxin absorption. Profuse vomiting typically results in dehydration and may cause hypochloremia, hypokalemia, and/or hyponatremia. Duodenal vomitus may cause greater sodium, potassium, and water losses than gastric vomiting. Alkalosis generally occurs with loss of gastric fluid; however, metabolic acidosis may occur due to fluid depletion from vomiting, insensible water losses, lack of intake, and/or catabolism of body stores. Cross-matched whole blood or red cells should be administered when the PCV drops below 20% or if the dog is weak or clinically hypoxic. Chronically ill, anemic patients should be given whole blood if hypovolemic and packed red blood cells if normovolemic. Clotting factor deficiencies should be corrected with whole fresh blood or fresh or fresh-frozen plasma. Platelet-rich plasma or platelet transfusions should be used if the animal is severely thrombocytopenic. Administration of plasma (5–20 mL/kg), whole blood transfusions, or hetastarch several hours before surgery should be considered if serum albumin concentrations are below 1.5 g/dL. If the patient has a severe protein-losing enteropathy, administration of plasma is seldom effective in raising the serum albumin concentration because most of the albumin is quickly lost into the GI tract. Therefore hetastarch is usually preferred. There is some evidence that blood transfusions may impair intestinal healing and increase susceptibility to intraabdominal sepsis.

Plain radiographs may demonstrate abnormal gas-fluid patterns, masses, foreign bodies, abdominal fluid, or displaced viscera

BOX 18.35 Preoperative Management of Patients Undergoing Intestinal Surgery

- Obtain minimum database: complete blood cell count, chemistry profile, urinalysis, coagulation profile (if possible), with or without an electrocardiogram.
- Localize the lesion with abdominal palpation, radiographs, ultrasonography, and/or endoscopy.
- Correct hydration, electrolyte, and acid-base abnormalities.
- Transfuse if the packed cell volume is less than 20% or if the animal is clinically weak or debilitated (see Box 4.1).
- Withhold food from mature animals for 12–18 hours and from pediatric patients 4–8 hours before induction.
- Administer prophylactic antibiotics if indicated.

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TABLE 18.5 Clinical Signs of Chronic Intestinal Disease

Clinical Sign	Small Intestine	Large Intestine
Weight loss	Consistent	Only patients with severe infiltrative colonic disease
Appetite	Variable	Usually normal; variable
Vomiting	Occasional	Rare
Belching	Occasional	Rare
Flatulence and borborygmus	Occasional	Occasional
Distended abdomen	Variable	Rare
Defecation quantity	Normal to large	Small to normal
Defecation frequency	Normal to slightly increased	Normal to very frequent
Blood in feces	If present, usually dark, black (melena)	If present, usually red to red-brown (hematochezia)
Mucus in feces	Absent	Present or absent
Steatorrhea	Occasional	Absent
Fecalith	Absent	Sometimes
Urgency or tenesmus	Absent	Sometimes present
Dyschezia	Absent	Present with rectal disease
Rectal examination	Normal	May be normal or abnormal (blood, mucus, pain, mass)
Abdominal pain	Variable	Variable
Poor hair coat	Variable	Uncommon
Depression	Variable	Uncommon

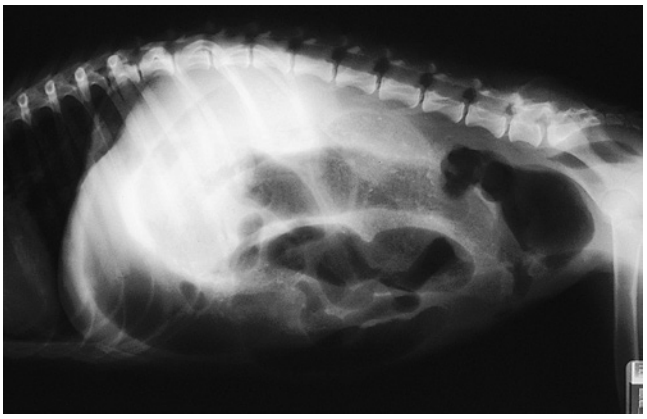


FIG. 18.95 Lateral radiograph of a depressed, anorectic, constipated, 2-year-old boxer with a "doughy" abdomen. Note the massive intestinal distention with gas and ingesta. A corncob was removed from the distal jejunum.

(Fig. 18.95). Both lateral recumbent views and a ventrodorsal projection should be taken. A ratio of the maximum diameter of the small intestine to the height of the cranial end plate of the second lumbar vertebrae of greater than 3 is strongly associated with intestinal obstruction in cats, whereas a ratio of greater than 4 is nearly always associated with obstruction. Contrast studies can demonstrate foreign bodies, obstructions, abnormal displacements, abnormal bowel wall thickness, irregular mucosal patterns, and distortion of the bowel wall. The positive contrast agent usually used for GI radiology is micropulverized barium sulfate suspension; however, iodinated contrast or iohexol should be used when intestinal perforation is suspected but septic peritonitis cannot be demonstrated with abdominocentesis or diagnostic peritoneal lavage (see p. 534). However, abdominal ultrasound is typically done before contrast studies because it often provides the diagnosis and allows the contrast study to be circumvented. Even when ultrasound is not definitive, many clinicians next move on to endoscopy instead of upper GI contrast studies.

NOTE Do not use barium sulfate for a gastrointestinal radiographic study if intestinal perforation is suspected. Instead, document peritonitis by abdominocentesis, diagnostic peritoneal lavage, or exploratory surgery.

Ultrasonography can often detect and define intestinal and other abdominal masses plus evaluate intestinal wall thickness (a normal small intestinal wall is 2–3 mm thick), the appearance and symmetry of the various wall layers, the number of peristaltic contractions, the pattern of intestinal contents (gas: hyperechoic, mucus: echogenic without acoustic shadowing, fluid: anechoic), the location of the lesion, and the extent of disease. Five layers are normally visible ultrasonographically in the intestinal wall: the hyperechoic mucosal surface, hypoechoic mucosa, hyperechoic submucosa, hypoechoic muscularis, and hyperechoic serosa. Ultrasound is very useful in helping the clinician decide whether to perform endoscopic or surgical biopsy of the intestines. If the intestine generally looks the same (even if it all looks normal) throughout all or most of its length, then endoscopic biopsy is usually appropriate (remember that ultrasound is relatively insensitive for GI disease). However, if ultrasound reveals that the only sections of bowel that appear involved are out of reach of the endoscope, then surgical biopsy is often preferred.

Gastroduodenoscopy allows visualization and biopsy of the duodenum (and sometimes the upper jejunum), whereas colonoileoscopy allows visualization and biopsy of the ileum.

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Visualization of intestinal mucosa may detect ulcers, erosions, infiltrated mucosa, and/or lymphangiectasia not detected with radiographs or ultrasound. Endoscopy also allows multiple biopsies of the small intestine and in particular allows one to direct the biopsy to obvious mucosal lesions.

The benefits of stabilizing the animal's condition before surgery must be weighed against the risk of ischemic necrosis. Explore the animal without delay if one or more of the following is true: (1) the animal has received a penetrating abdominal injury; (2) large numbers of neutrophils (>25,000/μL) or very toxic neutrophils are identified on cytology of the effusion; (3) bacteria are found in abdominal effusion; (4) the blood-to-peritoneal fluid glucose difference is greater than 20 mg/dL; (5) the peritoneal fluid lactate concentration is greater than 2.5 to 5.5 mmol/L; (6) extraluminal gas bubbles (i.e., spontaneous pneumoperitoneum) or volvulus are identified during imaging; (7) esophageal or gastric intussusception is identified during imaging; or (8) bacterial culture of the fluid is positive for pathogenic bacteria. Consider exploring the animal without delay if the previous factors are not identified but an emergency is suspected and one or more of the following is true: (1) the peritoneal fluid has greater than 13,000 nucleated cells/μL; (2) the peritoneal fluid pH is less than 7.2; (3) the peritoneal fluid carbon dioxide partial pressure is greater than 55 mm Hg; (4) the peritoneal fluid oxygen partial pressure is less than 50 mm Hg; (5) the peritoneal fluid glucose concentration is less than 50 mg/dL; (6) a negative blood-to-fluid lactate difference exists; or (7) the animal deteriorates clinically despite aggressive medical management and complete obstruction, perforation, strangulation, necrosis, or sepsis is suspected (Box 18.36).

ANESTHETIC CONSIDERATIONS

Special anesthetic considerations are needed for patients with bowel obstruction, ischemia, perforation, torsion, or volvulus (see Table 18.4). These patients may be dehydrated, hypotensive, and tachycardic, with marked electrolyte and acid-base abnormalities. Cardiac arrhythmias are not uncommon. Enlarged viscera may compress the vena cava, causing circulatory and vascular compromise. Furthermore, the diaphragm may be displaced cranially, compressing the thorax and decreasing lung volumes. Placing two large cephalic catheters will facilitate rehydration; shock doses of fluids at 90 mL/kg or greater may be needed in affected dogs (e.g., those with GDV or intestinal torsion). Often the patient is depressed and premedication is unnecessary. Complications at induction may arise because these animals may present emergently and volume replacement may be incomplete. These patients should be monitored for arrhythmias or tachycardia. An ECG, a pulse oximeter, and direct and indirect blood pressure measurements should be monitored throughout surgery. Preoxygenate these patients for 3 to 5 minutes prior to induction. Because of cardiovascular compromise, induction with either ketamine and a benzodiazepine (e.g., midazolam or diazepam) or etomidate may be warranted. Propofol should be avoided because of the marked hypotension it causes in hypovolemic patients. Be prepared for a drop in blood pressure at induction, even in volume-resuscitated patients (see Box 19.6). For maintenance of anesthesia, sevoflurane or isoflurane may have to be used at reduced levels. Nitrous oxide increases the volume of air trapped in body viscera and therefore should be avoided in patients with intestinal obstruction, volvulus, or torsion. Visceral manipulation may induce bradycardia; however, atropine or glycopyrrolate can resolve this if necessary. Anticipate large fluid shifts. Large volumes of saline or lactated Ringer's solution may have to be given initially to correct hypovolemia and continued during surgery because of ongoing evaporative and third space losses. Hetastarch may be given preoperatively, intraoperatively, and/or postoperatively for a total dose of 20 mL/kg per day. When giving hetastarch, monitor the patient closely. Sometimes the hetastarch enters the extravascular compartment and draws fluid into the interstitium or a third space. If possible, fluid warmers should be utilized in the preoperative period as well as in the operating room. Extra care needs to be taken to avoid hypothermia, which has numerous deleterious consequences (coagulopathy, decreased drug metabolism, impaired renal function, poor wound healing, cardiac arrhythmias). Care should be taken during surgery to try to maintain the patient's body temperature above 35°C (95°F).

Selected anesthetic protocols for animals with peritonitis are provided in Table 19.2. Selected anesthetic protocols for animals in a stable condition undergoing abdominal surgery are provided in Table 19.1.

BOX 18.36 Emergency Surgery Indications

Explore Without Delay If:

Penetrating abdominal injury
Effusion with intracellular bacteria
Blood-to-peritoneal fluid glucose difference >20 mg/dL
Peritoneal fluid lactate >2.5–5.5 mmol/L
Imaging reveals spontaneous extraluminal gas bubbles or volvulus
Esophageal or gastric intussusception is identified
Bacteria isolated from peritoneal fluid

Consider Exploring Without Delay If:

Peritoneal fluid has ≥13,000 nucleated cells/μL
Peritoneal fluid pH <7.2
Peritoneal fluid pCO_2 >55 mm Hg
Peritoneal fluid pO_2 <50 mm Hg
Peritoneal fluid glucose <50 mg/dL
Blood-to-peritoneal fluid lactate is negative
Animal deteriorates despite aggressive medical management and complete obstruction, perforation, strangulation, necrosis, or sepsis is suspected

pCO_2, Carbon dioxide partial pressure; *pO_2,* oxygen partial pressure.

ANTIBIOTICS

Bacteria populate the GI tract. Normally, bacterial numbers are smaller in the duodenum and jejunum than in the ileum, colon, and rectum. The colon has the greatest number of bacteria, both aerobic and anaerobic. Normally, fewer potentially pathogenic bacteria reside proximal to the ileocecal valve unless peristalsis is interrupted by ileus or obstruction. However, some patients have surprising large numbers of bacteria (i.e., $>10^8$/mL of intestinal fluid) in the upper small intestine, including potential pathogens such as *Bacteroides, Clostridium, Enterococcus, Staphylococcus,* and *Escherichia coli*. Resident bacteria typically proliferate in diseased bowel because stagnant luminal contents and devitalized wall are excellent growth media. Six hours of abnormal conditions may allow bacterial numbers to increase from 10^4/mL of ingesta to 10^{11}/mL. Holding animals off food reduces bacterial numbers in the small intestine and stomach. Antibiotic therapy alters the normal intestinal flora and promotes resistant strains of bacteria. Nonetheless, antibiotics can be indicated in

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BOX 18.37 Prophylactic Antibiotics for Animals Undergoing Intestinal Surgery

Cefazolin (Ancef, Kefzol)
22 mg/kg given IV

Cefmetazole (Zefazone)
15 mg/kg IV[a]

Cefoxitin (Mefoxin)
30 mg/kg IV

[a]Dose is anecdotal.
IV, Intravenous.

animals with severe mucosal damage or acute GI disease associated with fever, leukocytosis, leukopenia, and/or shock.

Surgical techniques that involve entering the intestinal lumen are classified as clean-contaminated or contaminated procedures, depending on the amount of spillage (see p. 84). The risk of infection in contaminated wounds increases with patient stress, the organism's pathogenicity, tissue susceptibility, and time. Pathogens that often cause peritonitis after intestinal surgery are *E. coli*, *Enterococcus* spp., and coagulase-positive *Staphylococcus aureus*. Although less frequently isolated, anaerobes are also common and may cause peritonitis (see pp. 449 and 527). Prophylactic antibiotics are indicated in animals with intestinal obstruction because there is an increased risk of contamination associated with bacterial overgrowth. They are also indicated when devascularized and traumatized tissue is present and when surgery is expected to last longer than 2 to 3 hours. First-generation cephalosporins (e.g., cefazolin; Box 18.37) should be administered before surgery on the upper and middle small intestine, whereas second-generation cephalosporins (e.g., cefoxitin; see Box 18.37) or a penicillin plus a beta-lactamase inhibitor (e.g., ampicillin plus sulbactam; piperacillin plus tazobactam; see Box 18.3) should be considered for procedures involving the distal small intestine and large intestine. Antibiotics should be redosed 2 hours after the initial dose.

SURGICAL ANATOMY

The intestines in dogs are approximately five times the body (crown to rump) length, with 80% being small intestine. Duodenum, jejunum, and ileum make up the small intestine. The duodenum is the most fixed portion, beginning at the pylorus to the right of midline and extending approximately 25 cm. It courses dorsocranially for a short distance, turns caudally at the cranial duodenal flexure, and continues on the right as the descending duodenum. The duodenum turns cranially at the caudal duodenal flexure where the duodenocolic ligament attaches. The ascending duodenum lies to the left of the mesenteric root. The common bile duct and pancreatic duct open in the first few centimeters of the duodenum at the major duodenal papilla in dogs. The accessory pancreatic duct enters caudal to this at the minor duodenal papilla.

The jejunum forms most of the small intestinal coils lying in the ventrocaudal abdomen. It is the longest and most mobile segment of the small intestine. It begins to the left of the mesenteric root where the ascending duodenum turns to the right at the duodenojejunal flexure. The ileum has an antimesenteric vessel and is approximately 15 cm long. It passes from the left to the right side in a transverse plane through the midlumbar region caudal to the root of the mesentery and joins the ascending colon on the right of the midline at the ileocolic orifice. The root of the mesentery attaches the jejunum and ileum to the dorsal body wall. Branches of the celiac and cranial mesenteric arteries supply the small intestine. Mesenteric lymph nodes lie along vessels in the mesentery.

The layers of the intestinal wall are the mucosa, submucosa, muscularis, and serosa. Mucosa is an important barrier that separates the luminal environment from that of the abdominal cavity. Mucosal health and the intestinal blood supply are important for normal intestinal secretion and absorption. The submucosal layer provides blood vessels, lymphatics, and nerves. It is the layer of greatest tensile strength. The muscularis is needed for normal motility. The serosa is important for forming a quick seal at a site of injury or incision.

BOX 18.38 Principles of Intestinal Surgery

- Early diagnosis and good surgical technique prevent most complications.
- Perform surgery as soon as anesthesia is possible in patients with perforation, strangulation, or complete obstruction.
- Optimal healing requires a good blood supply, accurate mucosal apposition, and minimal surgical trauma.
- Systemic factors may delay healing and increase the risk of dehiscence, hypovolemia, shock, hypoproteinemia, debilitation, and infection.
- Use approximating suture patterns: simple interrupted, Gambee, crushing, or simple continuous; stapling techniques are feasible.
- Engage submucosa in all sutures or staples.
- Select a monofilament, synthetic absorbable suture such as polydioxanone, polyglyconate, poliglecaprone 25, or glycomer 631.
- Cover surgical sites with a serosal patch if dehiscence is a concern.
- Replace contaminated instruments and gloves before closing the abdomen.

NOTE Because the submucosa is the intestinal layer that provides mechanical strength, it must be engaged when suturing intestine to provide a secure closure.

SURGICAL TECHNIQUES

Surgical correction of mechanical obstructions is preferably performed within 12 hours of diagnosis, allowing time for partial to complete correction of fluid, acid-base, and electrolyte abnormalities (Box 18.38). The benefits of stabilizing the patient's condition must be weighed against the risk of ischemic necrosis caused by vascular disruption, which increases with time. Perforation, loss of mucosal integrity, and systemic exposure to intestinal bacteria and toxins are life-threatening developments. Surgery for penetrating abdominal wounds, intestinal perforation, volvulus, or peritonitis should be performed as soon as the diagnosis is made.

Ischemic necrosis of the bowel wall may occur with obstruction (complete or partial), strangulation, and thrombosis. Routine criteria for assessing bowel viability include observation of intestinal color (pink to red rather than blue to black), wall texture, peristalsis, pulsation of arteries, and bleeding when incised. Because these factors are subjective, assessment of viability is often difficult. Bathing the involved segment in warm saline for a few minutes may improve color and peristalsis. However, a normal appearance does not guarantee that the bowel will heal after surgery; therefore bowel of questionable viability should be resected. A number of techniques have been proposed for

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increasing the accuracy of standard clinical criteria for viability assessment, and their most common error is that viable bowel would be resected. Viability assessment techniques include the use of electromyography, radioactive microspheres, microtemperature probes, and pH measurements. These techniques are technically cumbersome, expensive, and not generally suited for clinical use. Doppler ultrasonic flow probes have been used to detect pulsatile mural blood flow with an accuracy of 80%. Pulse oximetry measures oxygen saturation via pulse probes and may be superior to Doppler ultrasound in determining intestinal viability. Pulse oximetry of the intestinal wall as compared with peripheral oxygen saturation has shown that normal intestine remains within 1 cm of a normal pulse oximetry reading. Pulse oximetry is a reliable, reproducible means of assessing arterial perfusion of ischemic intestine, exceeding the overall accuracy of either standard clinical criteria or Doppler ultrasound when compared with fluorescein dye. Pulse oximetry is not as sensitive as fluorescein dye in detecting viability in segments with combined arterial and venous occlusion.

IV injection of various agents (primarily fluorescein dye) is practical but of limited accuracy (~95% accurate in detecting nonviable bowel, less than 60% accurate in detecting viable bowel). Fluorescein dye is injected intravenously (15–25 mg/kg), allowed to equilibrate for 2 to 3 minutes, and then the intestine is viewed with a Wood's lamp in a darkened operating room. Viable intestine has fluorescing areas of a smooth, uniform, green-gold color or a finely mottled pattern with no areas of nonfluorescence larger than 3 mm in diameter. Fluorescein can be used only once in a 24-hour period. Dyes such as fluorescein assess only perfusion and not mucosal integrity, which is essential for maintaining the mucosal barrier. Although this technique is a test of vascularity but not viability, it still can be a valuable adjunct in predicting viability.

Biopsy Techniques

Intestinal biopsy is indicated to diagnose intestinal diseases that have not been defined by other tests. The small intestine may be biopsied during endoscopy, ultrasonography, laparoscopy, or laparotomy. All biopsy techniques require general anesthesia or sedation.

Ultrasonographic Biopsy

The main advantages of ultrasound-guided biopsy of the small intestine are (1) these procedures are applicable to the entire intestine, (2) they are generally safe and quick and can be done on an outpatient basis, (3) they are likely to be specific if a diagnosis of neoplasia or fungal infection is made, and (4) one may also biopsy abdominal masses, if they are present. The main disadvantages are that (1) ultrasound is insensitive in detecting mucosal lesions, (2) it is very hard to examine the entire intestinal tract with ultrasound, meaning that it is easy for ultrasound to miss focal lesions of the intestines, and (3) there are no data on how diagnostic these samples are for inflammatory lesions of the intestines. Ultrasound-guided biopsy would appear to be the biopsy method of choice for intestinal masses, with surgery being indicated if the ultrasound-guided technique fails to make a diagnosis. Fine needle aspiration biopsy, microcore biopsy, or automated microcore biopsy may be performed during ultrasonographic examination of intestinal lesions; however, they may be difficult to perform if mild or moderate infiltration is present. For fine needle aspiration of lesions smaller than 2 cm in diameter, a 23- to 25-gauge, half-inch spinal needle or a Westcott biopsy needle may be used. If aspirated samples are nondiagnostic and the lesion is larger than 2 cm in diameter, an automated microcore biopsy may be performed (Bard Biopty-Cut biopsy needle). As an alternative, a microcore biopsy may be performed using a 20- or 22-gauge needle, which is passed repeatedly through the lesion. A syringe with a small amount of air is then attached to the needle, and the cells are blown out onto a slide. Possible complications include peritonitis, hematoma, tumor seeding, and trauma to adjacent organs.

Localize the lesion with the transducer and aseptically prepare the skin over this site. Tense the skin and puncture with the needle. Using ultrasound guidance, direct the needle into the lesion but not through the mucosa. For aspiration biopsies, remove the stylet and apply suction with a 6-mL syringe three to six times. After releasing suction, remove the needle and syringe. Collect two to four samples and evaluate the cytologic preparations. For a microcore biopsy, select a site as far from the intestinal lumen as possible. Using the Tru-Cut biopsy instrument with ultrasound guidance, collect one or two biopsies. Transfer the samples to biopsy traps and place in 10% formalin. After sampling, observe the biopsy site with ultrasound for fluid collection, suggestive of leakage or hemorrhage. Abdominal radiography may be used to look for pneumoperitoneum. *Monitor the mucous membrane color, capillary refill time, pulse, and respiratory rate during recovery.*

Flexible Endoscopic Biopsy

The main advantages of flexible endoscopic biopsy are (1) it is the least invasive technique available and it can be done as an outpatient procedure, (2) it allows visualization of the mucosa and directed biopsy of mucosal lesions not visible with imaging or laparotomy, and (3) it allows multiple biopsies of each organ, which is important because lesions can be very spotty, even in very ill animals. The main disadvantages of flexible endoscopy are (1) the jejunum is generally out of reach, except in small patients, (2) only a limited amount of submucosa and no muscular tunic is included in these biopsies, and, most importantly, (3) it is easy to obtain poor quality, nondiagnostic tissue samples unless the operator is well trained. Unless careful attention is paid to technique, endoscopic biopsy of duodenal mucosa often results in artifact-ridden, twisted, nonoriented biopsies that are difficult to impossible to meaningfully evaluate. See Chapter 14 for one technique for obtaining flexible endoscopic biopsies of the duodenum.

Laparoscopic Biopsy of the Small Intestine

The main advantages of laparoscopic biopsy of the small intestine are (1) it is relatively noninvasive and can be performed as an outpatient procedure, (2) it can be performed at the same time as hepatic and pancreatic biopsies, (3) one may biopsy the jejunum, which is usually out of reach of flexible endoscopes, and (4) one obtains a full-thickness biopsy. The main disadvantages of laparoscopic biopsy of the small intestine are (1) one cannot readily access the duodenum or ileum, (2) one is limited in the number of biopsies that may be taken, (3) the abdomen cannot be examined as well as it can be with laparotomy, and (4) one cannot see mucosal lesions and direct the biopsy to affected spots. *Perform a routine, exploratory laparoscopy using two to three ports. At the end of the procedure, use Babcock forceps to gently grasp a section of jejunum and bring it up to the cannula. Then pull the cannula, Babcock forceps, and intestine from the abdomen as a single unit.* It helps if the cannula used is a 10-mm cannula (if necessary, a 5-mm adapter may be placed over the top to accommodate smaller diameter instruments). *If necessary, enlarge the skin incision to make it easier to exteriorize that portion of the small intestine. Place stay sutures in the intestine and remove*

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the cannula and Babcock forceps. Next, perform an incisional biopsy, as described later under "Enterotomy." There are variations on this technique. *Alternatively, use a wound retractor to enlarge one port site to "run the bowel" by exteriorizing it and evaluate it visually and via palpation.* Each segment must be returned to the abdomen, as exteriorization of the entire small intestine is not possible without compromising the blood supply. The enlarged wound is too small for maintenance of blood flow through the root of the mesentery.

Enterotomy

The main advantages of laparotomy and enterotomy are that (1) it allows access to the entire GI tract, (2) it provides full-thickness biopsies, which are important in submucosal masses, and (3) one can examine and sample the rest of the abdomen at the same time. The main disadvantages of laparotomy are that (1) it is the most expensive and most invasive technique (i.e., it is not an outpatient procedure), (2) it does not allow one to detect mucosal lesions, (3) it does not allow one to obtain as many mucosal samples as flexible endoscopy, and (4) it is possible to take nondiagnostic tissue samples if proper technique is not followed. Laparotomy should be performed if other techniques are not possible or if other techniques have been or are likely to be nondiagnostic. Consideration should be made for taking samples of multiple segments of the GI tract to include stomach, duodenum, jejunum, and ileum, because disease processes may not be present in every segment. Samples of the liver and mesenteric lymph node (and sometimes pancreas in the cat) should be taken to identify concurrent hepatopathy or to diagnose conditions such as mast cell tumor that may only be present in lymph nodes.

Longitudinal or transverse enterotomy incisions can be made to collect biopsy samples. Multiple biopsies should be performed, and the samples should be reasonably large (4–5 mm in diameter) and should contain adequate amounts of mucosa. The entire abdomen should be explored thoroughly before biopsies are performed. Samples should be collected from the lymph nodes, liver, or other tissues before gastric or intestinal procedures to prevent cross-contamination. Other indications for enterotomy include removal of foreign bodies and luminal examination.

Exteriorize and isolate the diseased or desired intestine from the abdomen by packing with towels or laparotomy sponges. Gently milk chyme (intestinal contents) from the lumen of the identified intestinal segment. To minimize spillage of chyme, occlude the lumen at both ends of the isolated segment by having an assistant use a scissor-like grip with the index and middle fingers 4 to 6 cm on each side of the proposed enterotomy site (Fig. 18.96A). If an assistant is not available, use noncrushing intestinal forceps (Doyen) or a Penrose drain tourniquet to occlude the intestinal lumen. Make a full-thickness stab incision into the intestinal lumen on the antimesenteric border with a No. 11 scalpel blade. Obtain full-thickness biopsy samples 2 to 3 mm wide either by making a second longitudinal incision parallel to the first with the scalpel blade or by removing an ellipse of intestinal wall at one margin of the first incision with Metzenbaum scissors (see Fig. 18.96B–C).

Transverse enterotomy incisions can be made or a skin biopsy punch used to obtain biopsies. *Place the biopsy serosal side down on a heavy piece of sterile paper to help prevent curling of the*

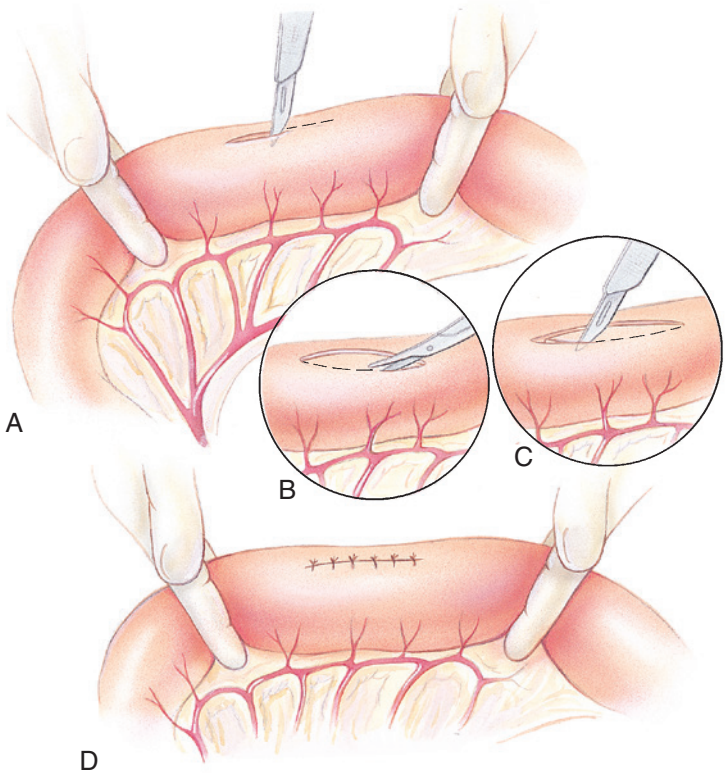


FIG. 18.96 Intestinal biopsy. (A) Occlude the lumen, then make a stab incision into the lumen with a No. 11 blade. (B) Remove a 2- to 3-mm ellipse of tissue with Metzenbaum scissors, or (C) make a second incision approximately parallel to the first with a scalpel. (D) Close the incision with simple interrupted sutures.

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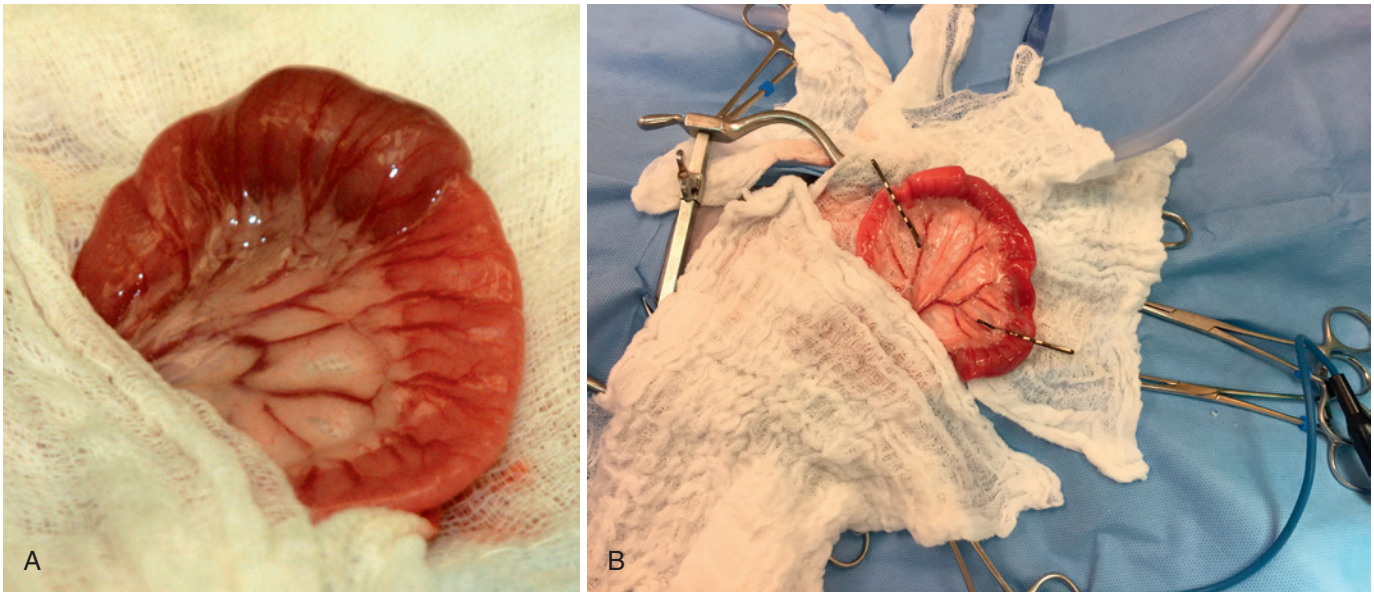


FIG. 18.97 Intestinal foreign body in a cat. (A) The intestinal segment has been exteriorized and packed off from the rest of the abdomen with moist laparotomy sponges to maintain asepsis. (B) The intestine has been occluded proximal and distal to the foreign body using sterile hair (bobby) pins.



FIG. 18.98 Intestinal segment with a foreign body that was removed via an enterotomy. Note the dilated proximal intestine with some ischemic areas and that the incision was made in healthier distal intestine.

specimen. Close the incision as described later with simple interrupted sutures (see Fig. 18.96D). Simple continuous or crushing sutures may also be used to close the enterotomy. Successful use of skin staples has also been described for intestinal closure.

If a foreign body is present (Fig. 18.97), make the incision in healthy-appearing tissue distal to the foreign body (Fig. 18.98). Lengthen the incision along the intestine's long axis with Metzenbaum scissors or scalpel as necessary to allow you to remove the foreign body without tearing the intestine.

After biopsy or removal of the foreign body, prepare the incision for closure by trimming everted mucosa so that its edge is even with the serosal edge (if necessary) or use a modified Gambee suture (see p. 69). Suction the isolated lumen. Close the incision with gentle appositional force in a longitudinal or transverse direction using simple interrupted sutures (Fig. 18.99). A transverse

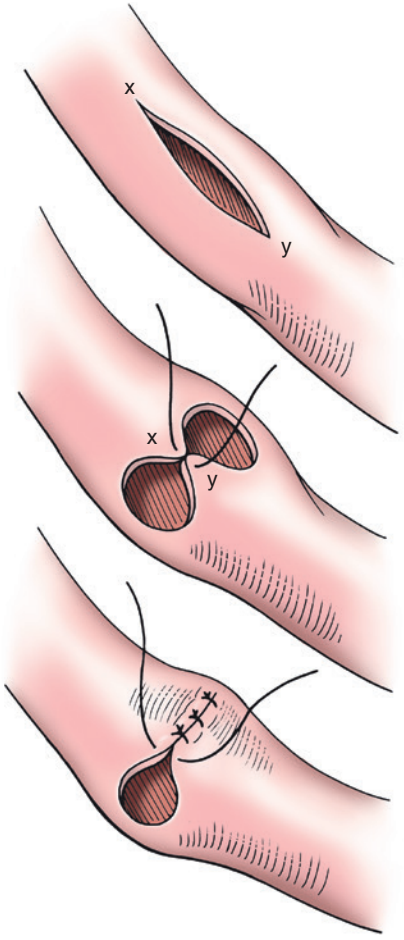


FIG. 18.99 Enterotomy incisions may be closed transversely if the intestinal lumen is small. Join the extremes (x and y) of the longitudinal incision with a simple interrupted suture to transpose the incision to a transverse orientation. Place remaining sutures 2 to 3 mm apart.

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enterotomy can be done for sampling of the intestine when no foreign material is present (Fig. 18.100). *Place sutures through all layers of the intestinal wall 2 mm from the edge and 2 to 3 mm apart with extraluminal knots. Angle the needle so that the serosa is engaged slightly farther from the edge than the mucosa (Fig. 18.101) to help reposition everting mucosa within the lumen. Tie each suture carefully, without cutting through layers of the intestinal wall, to gently appose all intestinal layers without crushing the tissue. Use a monofilament, absorbable suture material (4-0 or 3-0 polydioxanone, polyglyconate, or poliglecaprone 25) with a swaged-on taper or tapercut point needle. Consider a slowly absorbable monofilament suture (see p. 62) or even a monofilament, nonabsorbable suture (4-0 or 3-0 polypropylene, nylon, or polybutester) if the patient has an albumin level of 2 g/dL or lower. While maintaining luminal occlusion near the enterotomy site, moderately distend the lumen with sterile saline, apply gentle digital pressure, and observe for leakage between sutures or through needle holes (Fig. 18.102). Place additional sutures if leakage occurs between sutures. Lavage the isolated intestine and the entire abdomen if contamination has occurred. Place omentum over the suture line before closing the abdomen. Use a serosal patch (see p. 447) rather than omentum if intestinal integrity is questionable or if leakage occurs from needle holes. Replace contaminated instruments and gloves before closing the abdomen.*

Intestinal Resection and Anastomosis

Intestinal resection and anastomosis are recommended for removing ischemic, necrotic, neoplastic, or fungal-infected segments of intestine. Irreducible intussusceptions are also managed by resection and anastomosis. End-to-end anastomoses are recommended.

Sutured Anastomoses

Make an abdominal incision long enough to allow exploration of the abdomen. Thoroughly explore the abdomen and collect any nonintestinal specimens; then exteriorize and isolate the diseased intestine from the abdomen by packing with towels or laparotomy sponges. Assess intestinal viability and determine the amount of intestine needing resection. Occlude (double ligate, staple, or heat seal) and transect the arcadial mesenteric vessels from the cranial mesenteric artery that supplies this segment of intestine (Fig. 18.103). Occlude (double ligate, staple, or heat seal) the terminal arcade vessels and vasa recta vessels within the mesenteric fat at the points of proposed intestinal transection. Gently milk chyme (intestinal

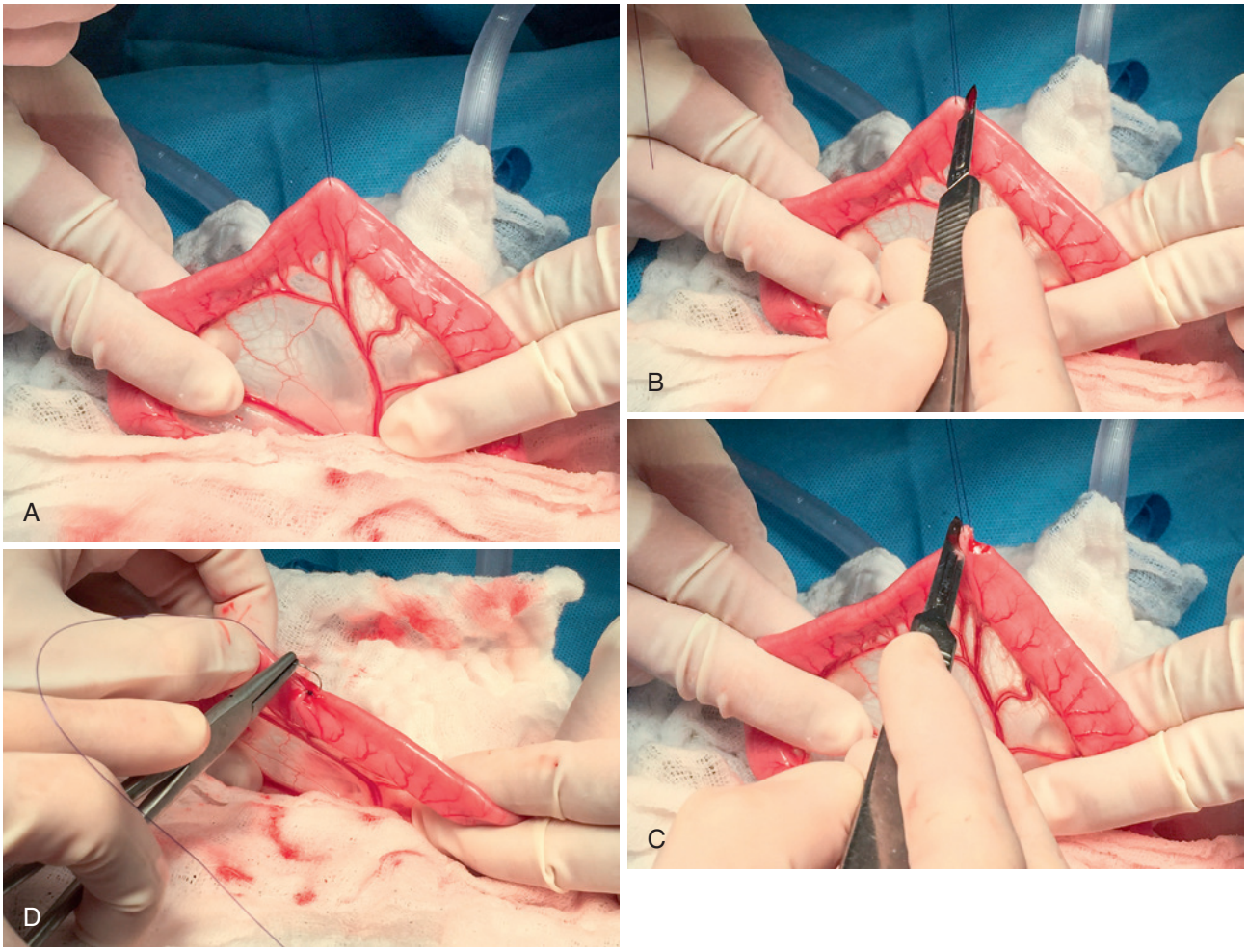


FIG. 18.100 Transverse wedge biopsy of the small intestine. (A) Place a stay suture in the antimesenteric surface. (B) Incise the intestine at an angle orad and (C) aborad to the suture to remove a sample. (D) Close the defect transversely in a simple interrupted pattern.

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contents) from the lumen of the identified intestinal segment. Use fingers or intestinal forceps to occlude the lumen at both ends of the segment to minimize spillage of chyme (discussed earlier). Place forceps across each end of the diseased bowel segment (these forceps may be either crushing or noncrushing because this segment of the intestine will be excised). Transect the intestine with either a scalpel blade or Metzenbaum scissors along the outside of the forceps. Make the incision either perpendicular or oblique to the long axis.

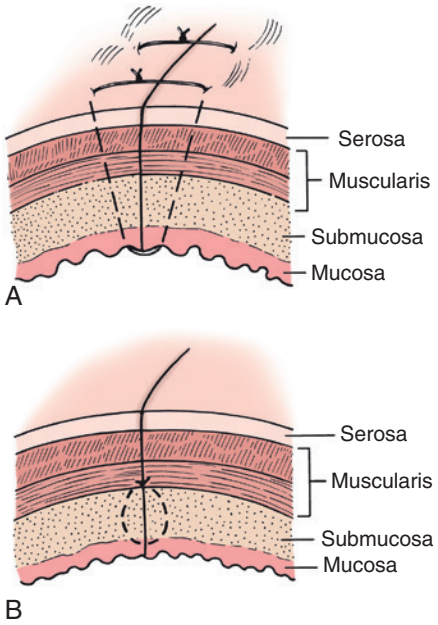


FIG. 18.101 (A) For an approximating suture closure of the intestine, place simple interrupted sutures 2 mm from the edge and 2 to 3 mm apart. Engage slightly more serosa than mucosa to force everted mucosa back into the lumen. (B) Place crushing sutures similarly, but pull them tight to cut through all layers except the submucosa when tying. This suture pattern is no longer generally recommended.

Use a perpendicular incision (75- to 90-degree angle) at each end if the luminal diameters are the same. When the luminal sizes of the intestinal ends are expected to be unequal, use a perpendicular incision across the intestine with the larger luminal diameter and an oblique incision (45- to 60-degree angle) across the intestine with the smaller luminal diameter to help correct size disparity (Fig. 18.104). Make the oblique incision such that the antimesenteric border is shorter than the mesenteric border. If further correction for size disparity is needed, space sutures around the larger lumen slightly farther apart than around the smaller lumen or remove a wedge from the antimesenteric border of the smaller segment (Fig. 18.105). Suction the intestinal ends and remove any debris clinging to the cut edges with a moistened gauze sponge. Trim everting mucosa with Metzenbaum scissors just before beginning the end-to-end anastomosis.

Use 3-0 or 4-0 monofilament, absorbable suture (polydioxanone, polyglyconate, or poliglecaprone 25) with a swaged-on taper or tapercut point needle. In animals with peritonitis, consider using a more slowly absorbable monofilament suture (see p. 62). Place simple interrupted sutures through all layers of the intestinal wall. Angle the needle so that the serosa is engaged slightly farther from the edge than the mucosa (see Fig. 18.101A). This helps reposition everting mucosa within the lumen. *Tie each suture carefully to gently appose the edges of the intestine with the knots positioned extraluminally.*

Tying sutures roughly or with too much tension causes the suture to cut through the serosa, muscularis, and mucosa, creating a crushing suture (see Fig. 18.101B). Some surgeons prefer this suture, but most prefer a simple interrupted or simple continuous pattern. Pulling continuous sutures too tight has a purse-string effect, and significant stenosis may occur. A continuous pattern around the intestine may limit dilation at the anastomotic site and cause a partial obstruction. Therefore a divided, modified simple continuous suture pattern is used to avoid these effects (Fig. 18.106). Two stay sutures are placed at the mesenteric and antimesenteric borders, then one simple continuous suture is placed between the sutures on each side. Experimentally, skin staplers may be used successfully in lieu of interrupted sutures. The bursting pressure of intestinal anastomoses using glycomer



FIG. 18.102 Leak testing of the enterotomy site of the enterotomy closure of the intestine.

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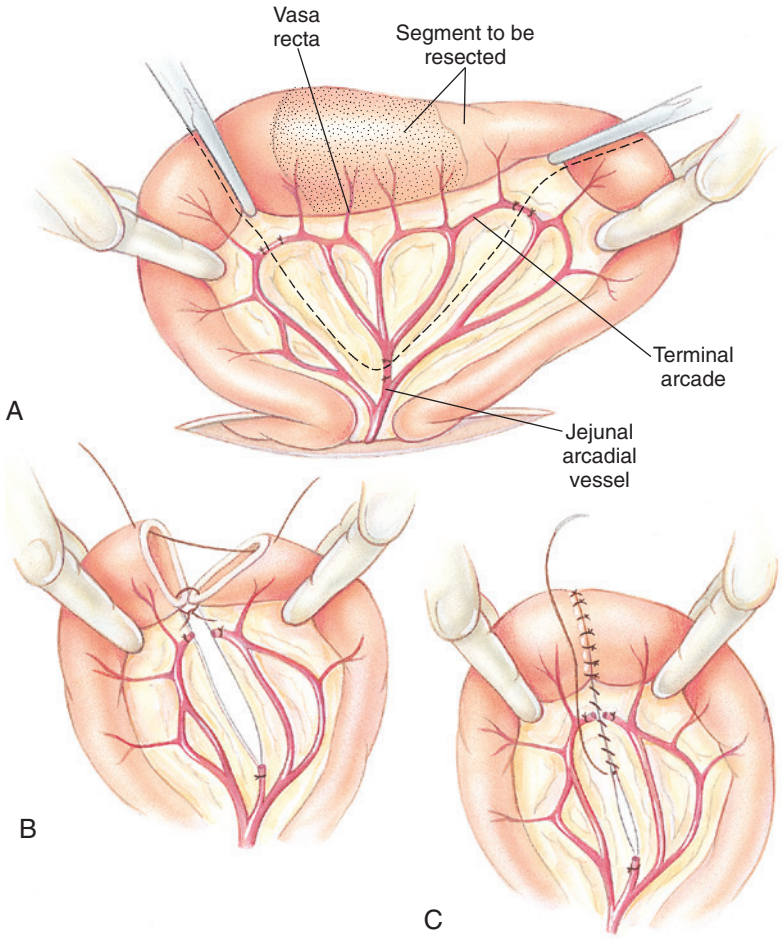


FIG. 18.103 For small intestinal resection and anastomosis, place forceps transversely across the dilated proximal intestine and obliquely across the distal intestine. Ligate vessels as indicated. (A) Occlude the lumen of the normal intestine, then transect the intestine and mesentery where the *dashed lines* indicate. (B) Place the first suture at the mesenteric border and the second at the antimesenteric border. (C) Place additional simple interrupted sutures to complete the anastomosis. Appose the mesentery in a simple continuous pattern.



FIG. 18.104 To perform an end-to-end anastomosis when the intestinal segments are of disparate size, transect the dilated intestine at a right angle and the smaller segment at an oblique angle (45–60 degrees).

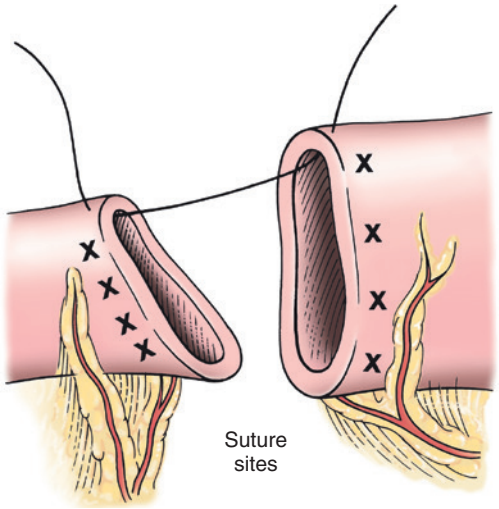


FIG. 18.105 In addition to angling the incisions (see Fig. 18.104), further correct for size disparity by spacing sutures around the larger lumen slightly farther apart than around the smaller lumen.

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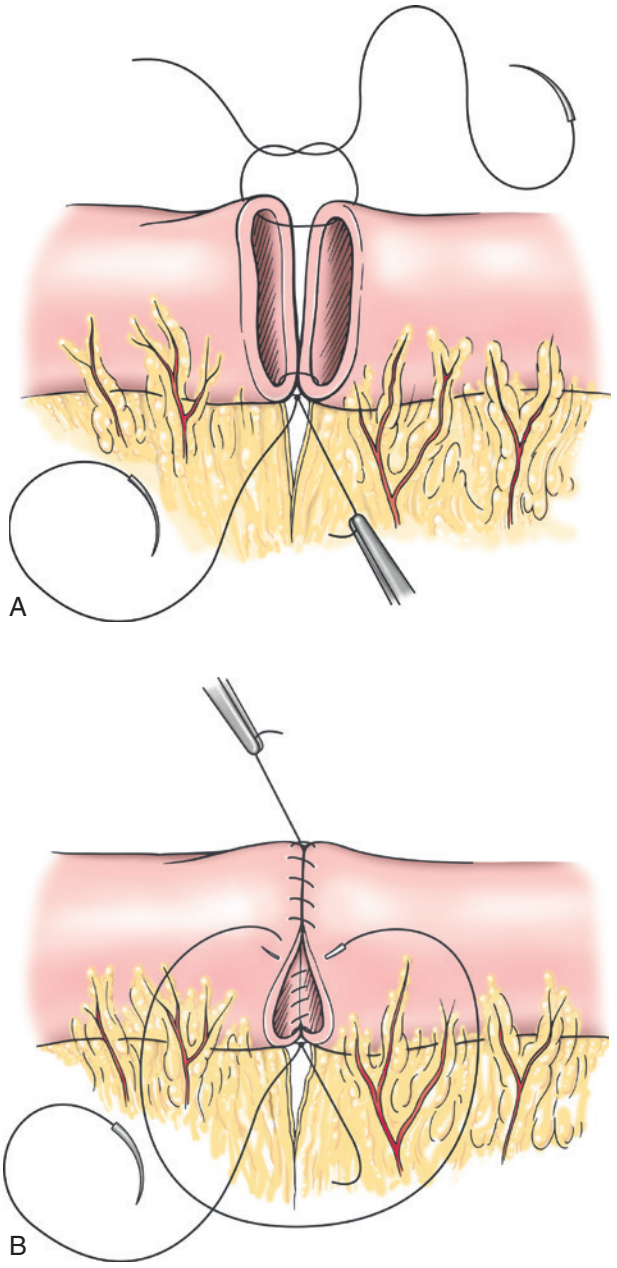


FIG. 18.106 End-to-end anastomosis using a modified simple continuous pattern. (A) Place and tie appositional sutures at the mesenteric and antimesenteric borders, leaving the needles attached. (B) Using the suture tags as stay sutures to maintain tension, place a continuous suture pattern between the antimesenteric and mesenteric sutures. Reposition the intestine and begin a second continuous suture line on the opposite side.

631 barbed suture versus 3-0 to 4-0 glycomer 631 suture was reported in an experimental study in dogs.[33] In this study, the barbed suture leaked at a higher pressure than the nonbarbed suture, leading the investigators to conclude that barbed suture was an acceptable alternative to nonbarbed suture for the closure of enterectomies in dogs.

Appose the intestinal ends by first placing a simple interrupted suture at the mesenteric border (see Fig. 18.103) and then placing a second suture at the antimesenteric border approximately 180 degrees from the first (this divides the suture line into equal halves

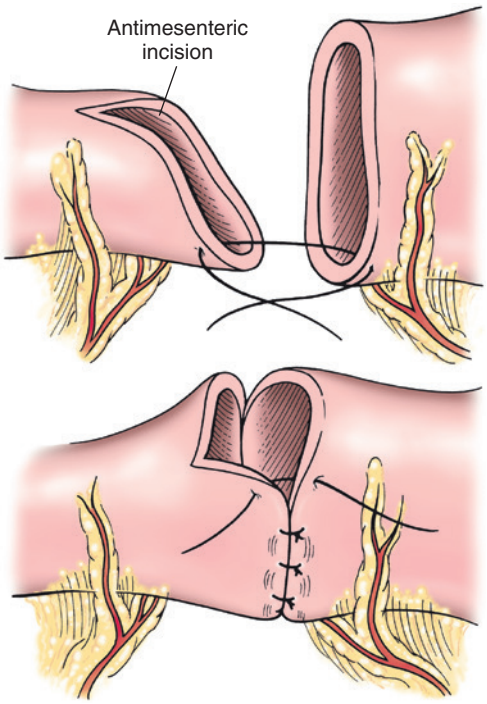


FIG. 18.107 If incision angling and suture spacing do not totally accommodate the luminal size differences, remove a wedge from the antimesenteric border of the distal intestine.

and allows determination of whether the ends are of approximately equal diameter). The mesenteric suture is the most difficult suture to place in the anastomosis because of mesenteric fat. It is also the most common site of leakage. *If the ends are of equal diameter, space additional sutures between the first two sutures approximately 2 mm from the edge and 2 to 3 mm apart (see Fig. 18.101A). If minor disparity still exists between lumen sizes, space the sutures around the larger lumen slightly farther apart than the sutures in the intestine with the smaller lumen (see Fig. 18.105). To correct luminal disparity that cannot be accommodated by the angle of the incisions or by suture spacing, resect a small wedge (1–2 cm long and 1–3 mm wide) from the antimesenteric border of the intestine with the smaller lumen (Fig. 18.107). This enlarges the perimeter of the stoma, giving it an oval shape. Do not suture together the edges of the intestine with the larger lumen in an attempt to reduce luminal size to that of the smaller intestine.* Narrowing the larger lumen is not recommended because there is greater tendency for stricture at the anastomotic site when the dilated intestine contracts to a normal size.

After suture placement, inspect the anastomosis and check for leakage. While maintaining luminal occlusion adjacent to the anastomotic site, moderately distend the lumen with sterile saline, apply gentle digital pressure, and observe for leakage between sutures or through needle holes (see Fig. 18.102). This is a subjective test, because all anastomoses can be made to leak if enough pressure is applied.

Place additional sutures if leakage occurs between sutures. Close the mesenteric defect in a simple continuous or interrupted pattern using 4-0 monofilament absorbable suture, taking care not to penetrate or traumatize arcadial vessels near the defect. Lavage the isolated intestine and the entire abdomen if abdominal contamination has occurred. Wrap the anastomotic site with omentum before

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closing the abdomen, or use a serosal patch (see p. 447) if intestinal integrity is questionable and leakage is likely.

Stapled Anastomoses

Resection may also be accomplished with staples. Three stapled anastomosis techniques are available: (1) triangulating end-to-end, (2) inverting end-to-end, and (3) side-to-side or functional end-to-end anastomoses. The small size of the intestine (less than 20 mm) often precludes the use of everting triangulating and inverting stapling techniques. A functional end-to-end anastomosis creates a larger stoma than the original intestinal lumen and is the preferred technique because the other two stapling techniques may reduce the size of the lumen. Stapled anastomoses have a higher tensile strength than sutured anastomoses after 7 days. They heal by primary intention with minimal inflammation. Thick, inflamed, and edematous tissues may prevent proper firing of the stapler by preventing complete penetration and formation of the staples into a B-shaped configuration. Dilation causes thinning of the visceral walls and may make tissues too thin for the staples to be effective. Stapled anastomoses do not have a lower rate of dehiscence compared to hand-sutured anastomoses; however, the surgical time is approximately 30 minutes longer for hand-sutured anastomoses.[34] The use of disposable skin staplers has been reported in dogs.[35] Careful evaluation for hemorrhage during and after the procedure is important, and staples should be avoided in the colon to avoid colonic stricture following surgery.

NOTE Staple cartridges are expensive; weigh the cost against the value of your time and the patient's condition when determining which technique to use.

A triangulating end-to-end anastomosis is performed with a transverse (TA) stapling instrument or a disposable skin stapler. This technique is expensive, as it requires three staple cartridges. The skin stapler is more economical and allows rapid application of individual 4.8 × 3.4 mm rectangular staples. *Remove the diseased intestine, then place three stay sutures to divide the stoma into three equal segments and appose the divided intestinal ends. Apply the TA stapler across each segment, partly overlapping the previous staple line (Fig. 18.108). Trim protruding tissue before removing the instrument.* Each application of the stapler applies a double staggered row of staples. This technique everts the edges. *Inspect the anastomosis for leakage and lavage. Appose the mesentery in a continuous suture pattern.*

Using the disposable skin stapler, apply tension between two of the triangulation sutures to appose the serosa, and compress the mucosa into the lumen with a moistened sponge. Then position the center of the stapler over the junction of the two edges, apply firm pressure, and fire the instrument (Fig. 18.109). Space staples 2 to 3 mm apart between the triangulation sutures. Edges of the intestinal wall will be slightly everted.

An end-to-end anastomosis is performed using a circular, inverting, end-to-end anastomosis stapler (EEA, Premium CEEA, or ILP staplers). These instruments are composed of a staple cartridge with a circular blade attached to a dome-shaped anvil and rod (Fig. 18.110). They are available in several sizes that create anastomotic stomas approximately 10 mm smaller than their cartridge size (EEA: 31, 28, 25, and 21 mm; ILP: 33, 29, 25, and 21 mm). The intestinal lumen should be 0.6 mm larger in diameter than the stapler. Activation applies a circular double row of staples and simultaneously resects a doughnut of intestinal wall at the anastomotic site. End-to-end staplers are used less often in the small intestine than in other areas of the GI tract because of the small lumen size of the small intestine. End-to-end staplers are commonly used during Billroth I, esophageal, and large bowel anastomotic procedures. Do not use the stapler if the tissue is too thick (i.e., will not compress to 2 mm) or too thin (i.e., compresses to <2 mm) and only if sufficient tissue is available to allow proper inversion of tissue edges.

Ligate and divide vessels to the diseased intestine as usual. Dissect the mesentery away from each intestinal segment (31-mm cartridge, 1.5 cm; 28-mm cartridge, 1 cm; 25- or 21-mm cartridge, 0.5 cm) because these tissues or ligatures may interfere with closing of the instrument. Place the purse-string instrument around the proximal intestine at the point of desired transection. Place the purse-string suture and transect the intestine using the purse-string instrument as the cutting guide. Place a purse-string suture and make the distal transection using the same technique. Insert a lubricated ovoid sizer through an enterotomy to determine the appropriate staple cartridge size and to dilate the intestine. Insert the stapler cartridge into the intestinal lumen through an enterotomy 3 to 4 cm from the transection site. Insert the anvil into the other intestinal end. Facilitate placement by placing three or four stay sutures at the edge of the intestine. Using the stay sutures, first pull the mesenteric border of the intestine over the anvil and then over the antimesenteric border. If it appears that the lumen of the intestine will not easily accommodate the anvil or the sizer of the desired diameter, insert a well-lubricated, 26- to 30-Fr Foley catheter with a 30-mL balloon. Slowly inflate the balloon with sterile water to dilate the intestine adequately. After dilation, insert the stapler components. Tie both purse-string sutures securely around the shaft of the stapler (see Fig. 18.110). Twist the wing nut to compress the intestinal segments between the cartridge and the anvil until the unit is aligned. Examine the anastomotic site for evidence of intestinal slippage. Release the safety and activate the instrument by squeezing the handles. Partly separate the anvil and cartridge by loosening the wing nut and remove the stapling instrument. Facilitate removal of the instrument by placing a traction suture around the staple line and lift the edge of the staple line over the anvil while gently rotating the instrument. Inspect the severed, inverted intestinal segment; to prevent leakage, make sure all tissue layers are present. Inspect the anastomotic site for hemorrhage and integrity. Close the enterotomy with sutures or a transverse stapler. Close the mesenteric defect in a continuous pattern. Lavage the surgical site and place an omental or a serosal patch (see p. 447) before closing the abdomen.

A side-to-side anastomosis or a functional end-to-end anastomosis is created using a linear cutting stapler (GIA stapler) and a transverse anastomotic stapler (TA). This is the preferred technique for small intestinal anastomosis because the resulting stoma is larger than the original, and disparity in luminal size is easily accommodated. The staple lines of the GIA device should be partially offset to avoid their apposition during the TA stapler closure and to provide the highest leakage pressure.[36] *Resect diseased intestine and use the linear cutting stapler to join the bowel segments at their antimesenteric borders, creating an antiperistaltic side-to-side anastomosis. Fully insert (50 mm) the linear cutting stapler into the stomas of each intestinal loop and activate it (Fig. 18.111).* Activation results in the placement of two double staggered staple lines that join the intestinal loops as the knife simultaneously incises between them. *Separate the stapled suture*

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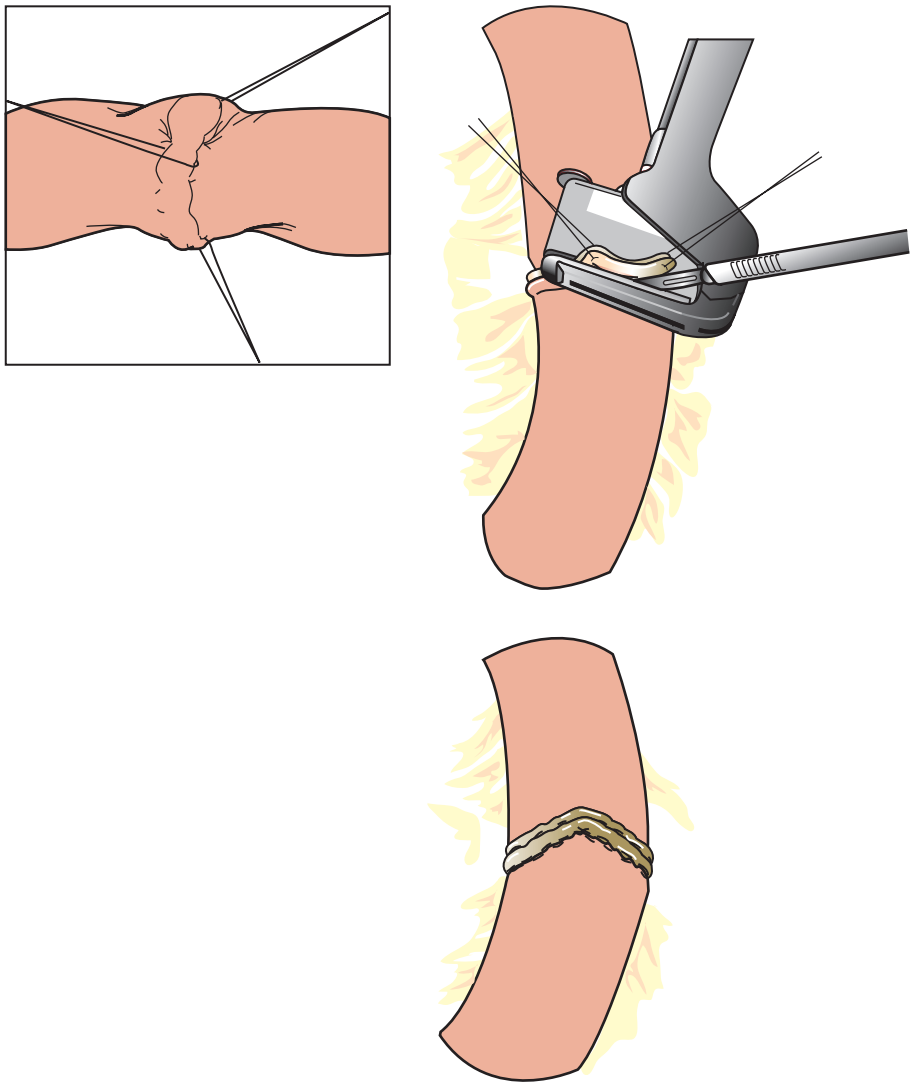


FIG. 18.108 End-to-end anastomosis using a triangulation technique and linear stapler. Place three stay sutures that appose the ends of the intestine, and divide the circumference into three equal parts. Apply tension between two of the sutures, and fire the stapler, leaving a double staggered row of sutures. Apply tension between the next two sutures and position the stapler so it overlies the end of the first row of staples and fire again. These steps are repeated a third time to complete the anastomosis.

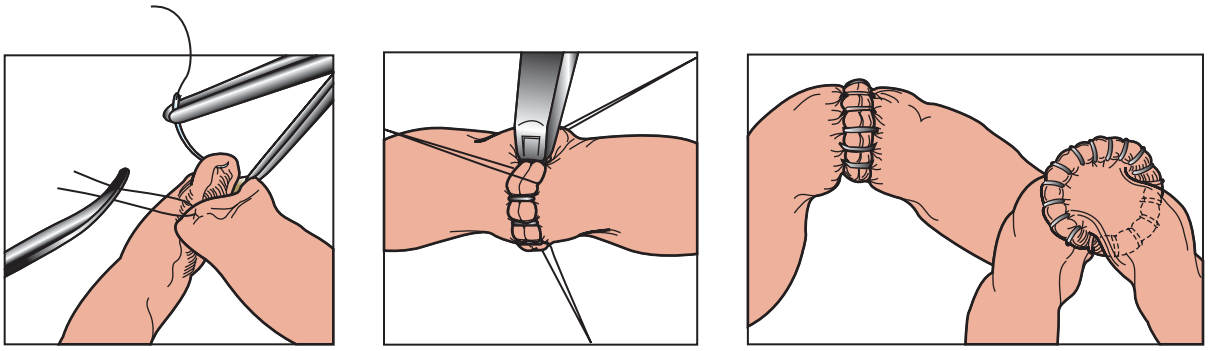


FIG. 18.109 End-to-end anastomosis using a triangulation technique and a skin stapler. Place three stay sutures to appose the ends of the intestine, and divide the circumference into three equal parts. Apply tension between two sutures. Center the skin stapler between the two segments, then apply staples with gentle pressure approximately 2 to 3 mm apart.

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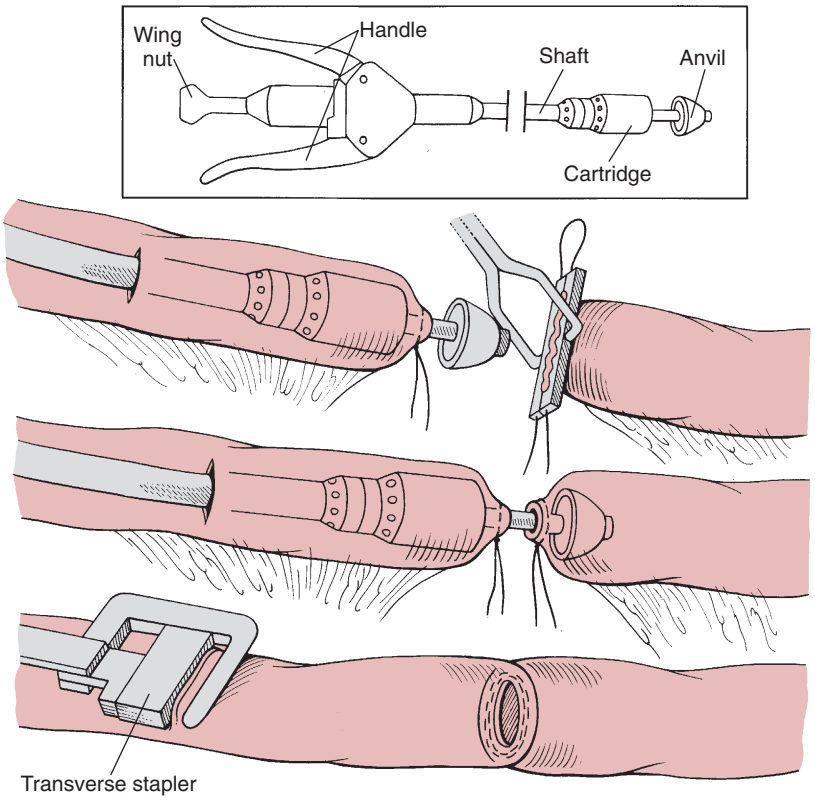


FIG. 18.110 For an inverting end-to-end anastomosis, use an end-to-end anastomosis stapler and a transverse stapler. Insert the stapler cartridge into the intestinal lumen through an enterotomy 3 to 4 cm from the transection site. Insert the anvil into the other intestinal end. Tie purse-string sutures securely around the shaft of the stapler. After completing the anastomosis, close the enterotomy with sutures or a transverse stapler.

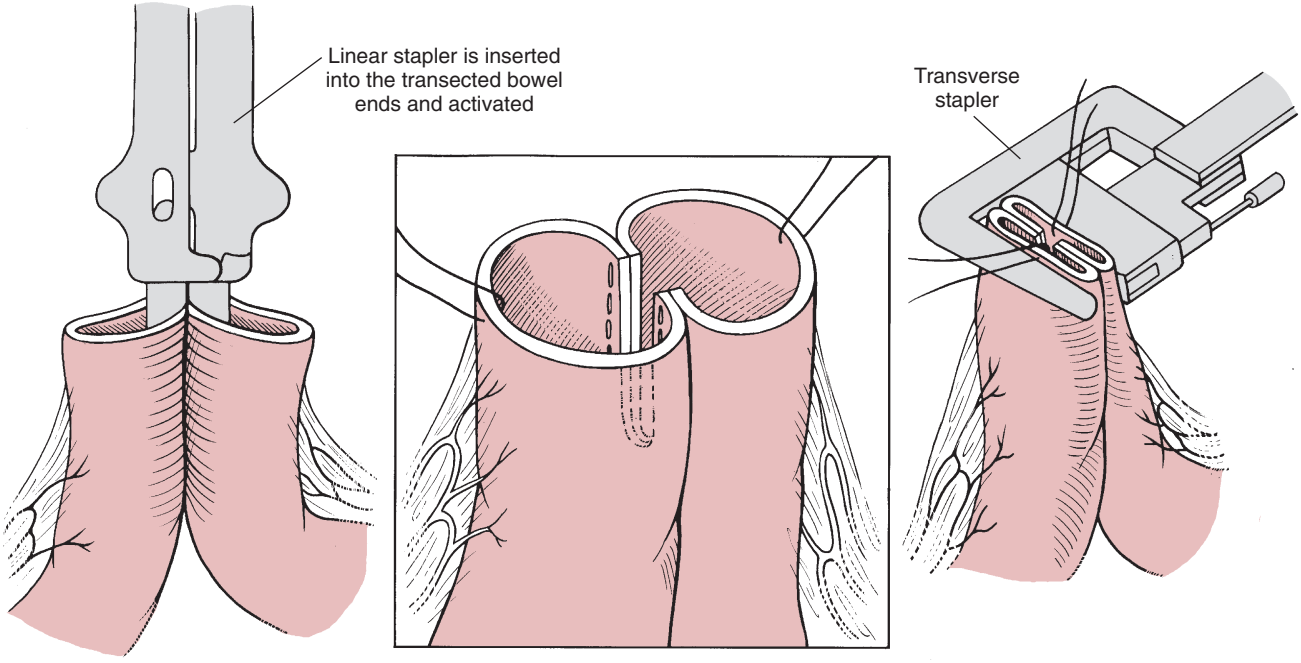


FIG. 18.111 For a functional end-to-end anastomosis, use a linear cutting stapler and a transverse stapler. Fully insert (50 mm) the linear cutting stapler into the stomas of each intestinal loop and activate it. Separate the stapled suture line, and apply the transverse stapling instrument to close the anastomosis.

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line and apply the transverse stapling instrument to close the anastomosis. The transverse stapler places a double staggered row of staples but has no cutting action. *Transect protruding intestinal wall flush with the stapler. Alternatively, place a second cartridge in the GIA stapler and fire it across the intestine, as done with the transverse stapling instrument. Remove the stapler and place an anchoring suture at the base of the staple line, where tension is greatest, to discourage staple pullout. Close the mesenteric defect in a continuous pattern before lavaging, patching, and closing the abdomen.* A similar stapling method, the closed, one-stage functional end-to-end anastomosis, involves creating the side-to-side anastomosis with the linear cutting stapler by inserting it through small antimesenteric stab incisions before resection of the diseased bowel. *Transect the diseased bowel after application of the transverse stapler.*

Serosal Patching

Serosal patching consists of putting the antimesenteric border of a loop of small intestine over a suture line or organ defect and securing it with sutures (Fig. 18.112). Serosal patching provides support, a fibrin seal, increased resistance to leakage, and blood supply to the damaged area plus it may prevent intussusception. Patches are commonly used after intestinal surgery when closure integrity is questioned or when dehiscence is repaired. Patches that span visceral defects are covered with mucosal epithelium within 8 weeks. Most commonly, jejunum adjacent to the defect or area of questionable viability is used for the serosal patch. Other sources could include the stomach, other intestinal segments, abdominal wall, or the urinary bladder. Serosal patching has not been shown to protect dogs from septic peritonitis or prevent mortality in dogs with preexisting septic peritonitis.[37]

Use one or more loops of intestine to form the patch. Use gentle loops to avoid stretching, twisting, or kinking the intestine and mesenteric vessels. If using more than one loop of intestine, suture these loops together before securing the patch to the damaged area (see Fig. 18.112). All sutures used to create or secure the patch engage the submucosa, muscularis, and serosa; theoretically they should not penetrate the intestinal lumen, although doing so is unlikely to lead to complications. *Place interrupted or continuous sutures in healthy tissue to secure the patch and isolate the damaged area. Alternatively, to patch over an anastomosis, use a piece of normal intestine and loop it perpendicular to the area to be patched (Fig. 18.113A). Be sure that the loop is gentle so as not to cause obstruction. Using a simple continuous suture pattern, suture between the looped piece of normal intestine, starting at the*

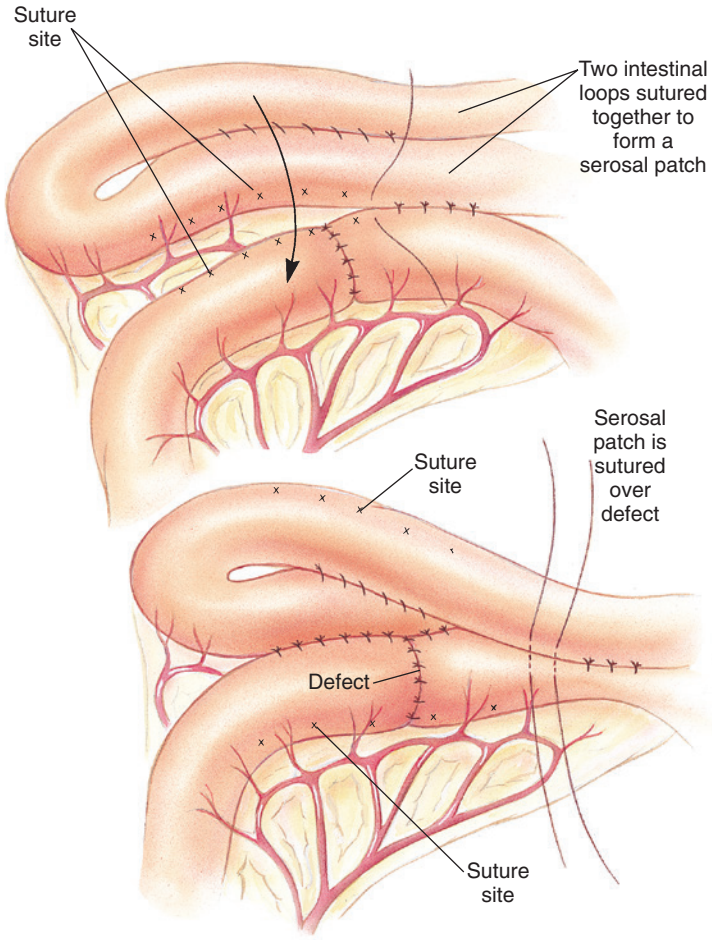


FIG. 18.112 Create a serosal patch by suturing adjacent intestinal loops together with simple continuous or interrupted sutures. Suture the patch over the defect or suture line.

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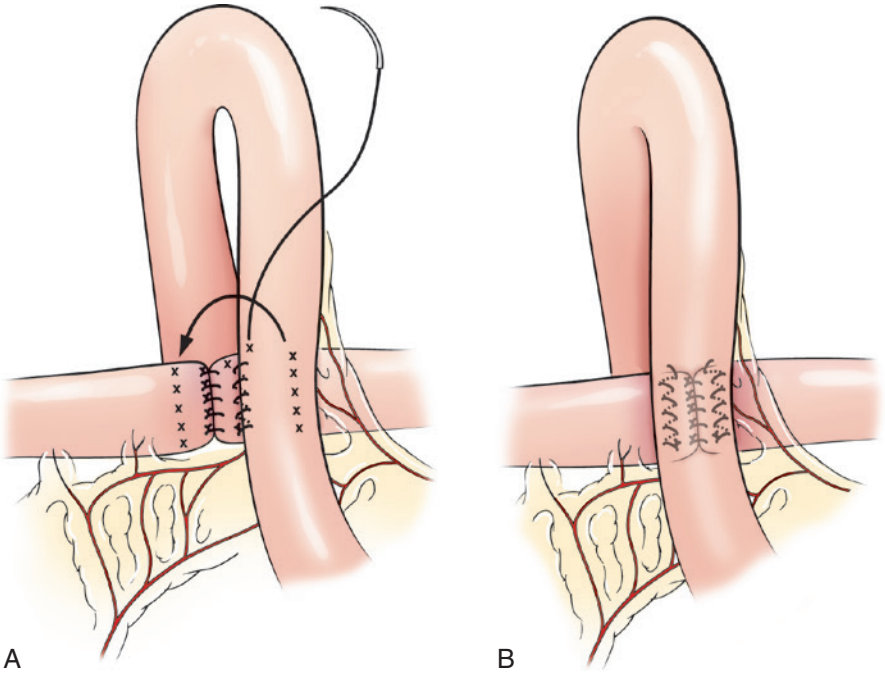


FIG. 18.113 To patch over an anastomosis, (A) use a piece of normal intestine and loop it perpendicular to the area to be patched. Be sure that the loop is gentle so as not to cause obstruction. (B) Using a simple continuous suture pattern, suture between the looped piece of normal intestine starting at the mesenteric border and continuing up to the antimesenteric border. Then suture across the anastomosis and back down to the mesenteric border on the same side. Repeat the process on the opposite side of the anastomosis. Be careful not to compromise the vasculature at the mesenteric border with your sutures.

mesenteric border and continuing up to the antimesenteric border (see Fig. 18.113B). Then suture across the anastomosis and back down to the mesenteric border on the same side. Repeat the process on the opposite side of the anastomosis. Be careful not to compromise the vasculature at the mesenteric border with your sutures.

Externalization of an anastomotic site may be considered in dogs with leakage following intestinal resection and anastomosis. The anastomotic segment can be externalized into the subcutaneous space, allowing for visual inspection via bandaging until the site can be replaced in the abdomen. *For this procedure, make a paramedian incision and pass suture through the body wall and mesentery in an interrupted horizontal mattress pattern to avoid vascular trauma and compression. Tighten the sutures to allow passage of ingesta through the intestinal lumen. Return the segment to the abdomen once the peritonitis has resolved.*

Bowel Plication

Enteroenteropexy, or bowel plication, is performed to prevent recurrence of intussusception. The small intestine from the duodenocolic ligament to the ileocolic junction is sutured to decrease the potential for intestinal strangulation. The bends in the intestine are gentle to prevent obstruction, and plication sutures are placed at intervals that will prevent entrapment and strangulation of other intestinal segments (Fig. 18.114).

NOTE Although one small study suggested that enteroplication was not helpful in preventing recurrence, it is the authors' belief that this technique, when properly performed, is an important tool for preventing reintussusception.

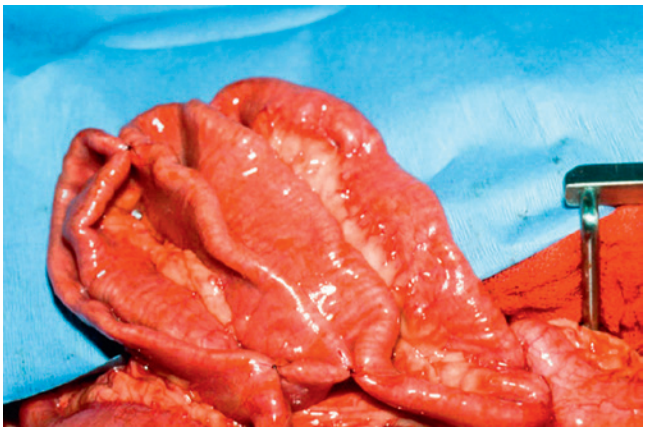


FIG. 18.114 The appearance of the small intestine following enteroplication.

Place small intestinal loops side by side to form a series of gentle loops from the distal duodenum to the distal ileum. Secure the loops by placing sutures that engage the submucosa, muscularis, and serosa 6 to 10 cm apart. Use 3-0 or 4-0 monofilament, absorbable or nonabsorbable sutures with a swaged-on taper point needle. Avoid positioning the intestinal loops at acute angles, or intestinal obstruction may occur. Entering the lumen with pexy sutures may theoretically increase the risk of leakage and abdominal contamination.

HEALING OF THE SMALL INTESTINE

Optimal intestinal healing depends on a good blood supply, accurate mucosal apposition, and minimal surgical trauma.

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Approximating suture patterns facilitate rapid healing. Everting and inverting suture patterns retard intestinal healing and may cause greater stricture formation. Healing is facilitated by adjacent serosal surfaces and omentum, which help seal wounds and contribute to the blood supply. The intestine generally heals rapidly, but healing can be delayed by local and systemic factors. Systemic factors such as hypovolemia, shock, hypoproteinemia, debilitation, and concurrent infections may delay healing and increase the risk of incisional breakdown. Tension on the repair caused by accumulated ingesta, fluid, gas, or poor mobilization of the bowel increases the risk of intestinal suture breakdown. Serosal and peritoneal adhesions occur because of trauma from excess handling or rough technique, foreign material, and drying during surgery. Administration of glucocorticoids or NSAIDs can inhibit the housekeeping prostaglandins and may render the GI tract more susceptible to injury.

The three overlapping phases of healing are the lag phase, the proliferative phase, and the maturation phase. The *lag phase* occurs during days 0 to 4 and is associated with inflammation and edema of the healing intestine. A fibrin seal forms during the first few hours. Although the fibrin clot contributes to wound strength, during this phase most of the wound strength is attributed to sutures. During the lag phase, macrophages are important in wound debridement and the production of growth factors that modulate fibroplasia and angiogenesis (transforming growth factor-β, platelet-derived growth factor, epidermal growth factor, and cytokines). Healing is functionally weakest at the end of the lag phase because of fibrinolysis and collagen deposition; therefore dehiscence most commonly occurs 3 to 5 days after intestinal surgery. Inflammation is more severe and healing time is slower with inverting patterns than with approximating patterns. Everted intestinal anastomoses have reduced tensile and bursting strength during the lag phase and therefore have a greater tendency to leak. The *proliferative phase* occurs between days 3 and 14. Fibrous repair occurs, accompanied by a rapid gain in wound strength. The strength of the repair site approximates that of normal intestine 10 to 17 days after surgery. The *maturation phase* occurs between 10 and 180 days. Collagen is reorganized and remodeled during this phase.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Instruments recommended to facilitate intestinal surgery include self-retaining abdominal retractors, malleable retractors, Doyen noncrushing forceps, Babcock forceps, Metzenbaum scissors, No. 11 scalpel blade, Penrose drains, and suction. Although most absorbable suture materials can be used, 3-0 or 4-0 monofilament polydioxanone, polyglyconate, or poliglecaprone 25 is preferred. Long-lasting monofilament, absorbable suture (polydioxanone, polyglyconate, poliglecaprone 25, glycomer 631) or nonabsorbable suture (nylon, polybutester, or polypropylene) should be selected for patients with low albumin levels. Patching techniques should also be considered in these patients to reinforce and facilitate healing of the surgical site. As an alternative, surgical stapling equipment can be used for some procedures (i.e., transverse stapling instrument, end-to-end anastomosis stapler, linear stapler, ligate-and-divide instrument, and skin stapler). Other instruments useful for diagnosis of digestive disorders are Westcott biopsy needles and Bard Biopty-Cut needles.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative care must be individualized to each patient and its problems. The animal should be monitored closely for vomiting during recovery. Analgesics (e.g., hydromorphone or buprenorphine; see Table 13.2) should be provided as needed. Ischemic intestine produces inflammatory mediators and stimulates their release or production in the distant organs, such as liver and lung, that may result in systemic inflammation. Care should be taken in aggressive monitoring and treatment of cases of intestinal ischemia. Hydration should be maintained with IV fluids, and electrolyte and acid-base abnormalities should be monitored and corrected. Small amounts of water may be offered 8 to 12 hours after surgery. If no vomiting occurs, small amounts of food may be offered 12 to 24 hours after surgery. Early feeding is important as it preserves or increases GI blood flow, prevents ulceration, increases IgA concentrations, stimulates other immune system defenses, and stimulates wound repair. Animals should be fed a bland, low-fat food (e.g., i/d [Hill's Pet Products] or boiled rice, potatoes, and pasta combined with boiled, skinless chicken, yogurt, or low-fat cottage cheese) three or four times daily. The normal diet should be reintroduced gradually, beginning 48 to 72 hours after surgery. Debilitated patients may require feeding tubes or parenteral nutrition. An elemental diet is best for patients undergoing tube feeding following GI surgery. Antibiotics should be discontinued within 2 to 6 hours of surgery unless peritonitis is suspected. Early ambulation and feeding should be encouraged to minimize ileus.

After intestinal surgery, clinical signs (e.g., depression, high fever, excessive abdominal tenderness, vomiting, and/or ileus) and response to abdominal palpation should be monitored for evidence of leakage and subsequent peritonitis or abscess formation. If peritonitis is suspected, an abdominocentesis, chemistry profile, and CBC should be performed. Abdominal fluid should be submitted for culture and sensitivity testing, and antibiotics (e.g., cefazolin, cefoxitin, enrofloxacin plus ampicillin, clindamycin plus enrofloxacin; Box 18.39) and fluid therapy should be initiated. Continuation of antibiotics should be based on the results of culture and susceptibility testing. The abdomen should be explored

BOX 18.39 Antibiotic Therapy for Treatment of Postoperative Peritonitis

Cefazolin (Ancef, Kefzol)
22 mg/kg IV q8h

Cefoxitin (Mefoxin)
30 mg/kg IV q6h

Enrofloxacin (Baytril)
Dogs: 10–20 mg/kg IV q24h; give diluted in IV fluids slowly over 30 min

Ampicillin
22 mg/kg IV, IM, SC q6–8h

Amikacin (Amiglyde-V)
Dogs: 15–22 mg/kg IV q24h
Cats: 10–14 mg/kg IV q24h

Clindamycin (Antirobe)
Dogs: 11–33 mg/kg PO q8–12h, *or* 11 mg/kg IV q8–12h; when giving IV dilute and administer slowly
Cats: 11–33 mg/kg PO or SC q24h

Ampicillin Sodium & Sulbactam Sodium (Unasyn)
15–30 mg/kg IV q6–8h (off-label use)

IM, Intramuscular; *IV,* intravenous; *PO,* orally; *SC,* subcutaneous.

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BOX 18.40 Perioperative Factors Contributing to the Risk of Leakage or Dehiscence

Peritonitis
Intestinal foreign body
Hypoalbuminemia <2.0–2.5 g/dL
Malnutrition
Preoperative weight loss (>4.5 kg in humans)
Sepsis, concurrent infection
Bowel obstruction
Increased age
Systemic disease: diabetes, heart failure, malignancy
High blood urea nitrogen
Neutrophilia
Preoperative glucocorticoids
Blood transfusions
Supplemental nutrition
Poor blood supply or perfusion
Tension
Poor apposition
Sutures placed in nonviable tissue
Traumatic tissue handling
Long operative time
Contaminated or dirty surgery
Intraabdominal drains
Colonic resection
Increased anesthetic risks
Prolonged hospitalization
Emergency surgery
Hypovolemia, hypotension, shock

if toxic neutrophils with engulfed bacteria or intestinal debris are present. Generalized peritonitis should be treated aggressively. Peritoneal drainage may be necessary (see p. 534). The small intestinal dehiscence rate may be as high as 16%, with the majority of those patients dying. There is a high risk for developing leakage if two or more of the following factors are identified: preoperative peritonitis, intestinal foreign body, or serum albumin concentration less than or equal to 2.5 g/dL. In one study evaluating stapled anastomoses, a dehiscence rate of 11% was noted on an average of 4 days after surgery.[38] That study identified hypotension, preexisting IBD, and stapling of the colon as risk factors for dehiscence; the mortality rate associated with dehiscence in that study was 83%. Skin staplers have been reported to have a similar complication rate as other methods of closure for anastomoses in dogs.[39] Other factors may also contribute to dehiscence, especially those indicating general debility and malnourishment (Box 18.40). It is important to note that the effect of abdominal drains on abdominal fluid is not yet determined; however, in one study, abdominal fluid was more likely to be normal in dogs without abdominal drains than in those with abdominal drains.[40]

COMPLICATIONS

Shock, leakage, ileus, dehiscence, perforation, peritonitis, stenosis, short bowel syndrome (SBS), recurrence, and death are possible complications of intestinal surgery (Box 18.41). Hypoalbuminemic dogs have the same postoperative complication rate as dogs with normal plasma albumin levels in some studies, but they have a higher risk in others. Clinically significant strictures are rare unless inverting or everting suture patterns are used or excessive tension exists at the resection site. Recurrent intestinal obstruction

BOX 18.41 Common Errors in Surgical Patients With Small Intestinal Disorders

- Failure to diagnose and treat the condition before ischemia and necrosis occur
- Failure to prevent abdominal contamination
- Failure to prevent intestinal leakage
- Failure to maintain hydration and nutritional homeostasis

BOX 18.42 Selected Drugs Used to Treat Short Bowel Syndrome

Loperamide (Imodium)
Dogs: 0.1 mg/kg PO q8–12h
Cats: 0.08–0.16 mg/kg PO q12h

Omeprazole (Prilosec)
1–2 mg/kg PO q12h

PO, Orally.

may occur after using polypropylene suture in a continuous pattern for closure.

Resection of excessively long sections of bowel puts the patient at risk for SBS. SBS has been defined many different ways. It is not simply resection of a long section of intestine; rather, it is resection of so much intestine that the body cannot compensate without parenteral/enteral nutritional therapy. There will be considerable variation between patients, but it usually does not occur unless more than 70% to 80% of the small intestine is resected. Weight loss, diarrhea, and malnutrition are the predominant clinical signs. Treatment is based on the severity of clinical signs and must be planned individually and modified as needed. The goal of treatment is to provide nutritional support until intestinal adaptation occurs (1–2 months) and diarrhea is controlled. Acute signs should be treated by correcting hydration and electrolyte imbalances, providing adequate nutrition (i.e., enteral and/or total parenteral nutrition), and controlling diarrhea. Proton pump inhibitors (e.g., omeprazole) may be useful in reducing gastric hypersecretion, which contributes to diarrhea and damages duodenal mucosa. Opiate antidiarrheals (e.g., loperamide; Box 18.42) may help decrease intestinal secretion and augment intestinal absorption. Intestinal bacterial populations should be controlled with antibiotics (e.g., tylosin). Growth factors given immediately after resection of large segments of intestine may facilitate intestinal adaptation and minimize clinical signs.

The most important aspect of treating SBS is to provide nutritional support. By definition, SBS requires more than just a highly digestible diet (e.g., i/d [Hill's Pet Products]). Enteral and parenteral nutrition are discussed in Chapter 10. Most patients with SBS will require elemental enteral diets, at least initially. It is critical that enteral nutrition be included, even if the patient requires total parenteral nutrition. The clinician must be prepared to treat for 1 to 2 months to allow the intestines to adapt. If the patient responds and is able to be taken off parenteral and elemental enteral diets, then the patient should be fed several times daily when traditional oral feeding begins. Daily vitamin-mineral supplements should be given. Surgical attempts to control SBS should be made only when medical and dietary therapy fails.

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The prognosis of SBS depends on the extent and site of resection, the degree of intestinal adaptation, the preoperative condition, and the postoperative care, but it is guarded at best. Because of the guarded prognosis and the difficulty and cost involved, it is better to proactively try to avoid SBS. If faced with a patient with extensive disease and a potentially long section of bowel to resect, it may be preferable to resect a short section and then do a second-look procedure 2 to 3 days later in hopes that some bowel can be preserved. Preservation of the ileocolic valve is thought to help prevent colonic bacteria from easily gaining access to the upper small intestine.

Resection of large portions of the small intestine may result in short- and long-term complications less severe than SBS. A 2012 study suggested that glutamine may be of value in maintaining or increasing body weight in animals undergoing 70% bowel resection.[41]

SPECIAL AGE CONSIDERATIONS

Young animals more frequently have GI parasitic infestations or garbage- or foreign body–induced gastroenteritis and intussusceptions. Young animals can quickly become hypothermic and hypoglycemic during surgery and require special care. Healing may be delayed in older animals because of concurrent problems.

SPECIFIC DISEASES

INTESTINAL FOREIGN BODIES

DEFINITION

Intestinal foreign bodies are ingested objects that may cause complete or partial intraluminal obstruction.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The oropharyngeal opening is larger than any other orifice in the GI tract. Foreign bodies that traverse the esophagus and stomach may lodge in the smaller diameter intestine (Box 18.43). Common intestinal foreign bodies include bones, balls, toys, rocks, corncobs, cloth, metal objects (e.g., fishhooks, needles), peach pits, acorns, pecans, hairballs, tampons, and linear objects (i.e., string, thread, fabric, pantyhose, plastic, cassette tape, or ribbon). Some foreign bodies continue to move slowly through the intestine, whereas others become lodged in an intestinal segment, where they cause complete or partial obstruction.

Partial or incomplete obstruction allows limited passage of fluid or gas, whereas complete obstruction does not allow fluid or gas to advance past the obstruction (Fig. 18.115). The clinical course and signs are more severe in animals with complete intraluminal obstruction, particularly a "higher" obstruction, than in those with a partial obstruction. With complete intraluminal obstruction, the intestine oral to the lesion distends with gas and fluid. The gas is a combination of swallowed air, carbon dioxide formed in the lumen by bicarbonate neutralization, and organic gases from fermentation. Fluid accumulation is caused both by retention of ingested fluid in the intestinal lumen and by secretion (i.e., salivary, biliary, gastric, intestinal, and pancreatic glands) (Fig. 18.116). Secretion increases and absorption diminishes (secretions are normally reabsorbed in the lower jejunum and ileum) during obstruction. Absorption is reduced because of lymphatic and venous congestion, increased intraluminal osmolality, and decreased enterocyte turnover rate. After 24 hours of complete obstruction, the distended bowel may lose

BOX 18.43 Intestinal Foreign Bodies: Key Points

- Many different objects can cause obstruction
- Signs vary with location and degree of obstruction
- Dilatation occurs proximal to the obstruction
- Imaging detects most foreign bodies
- Explore for multiple objects and evidence of peritonitis
- Incise through healthy tissue distal to the object for removal
- Assess bowel viability after removal of the object
- Resect devitalized intestine and anastomosis if necessary
- Lavage thoroughly
- Place omentum or create a serosal patch at the site

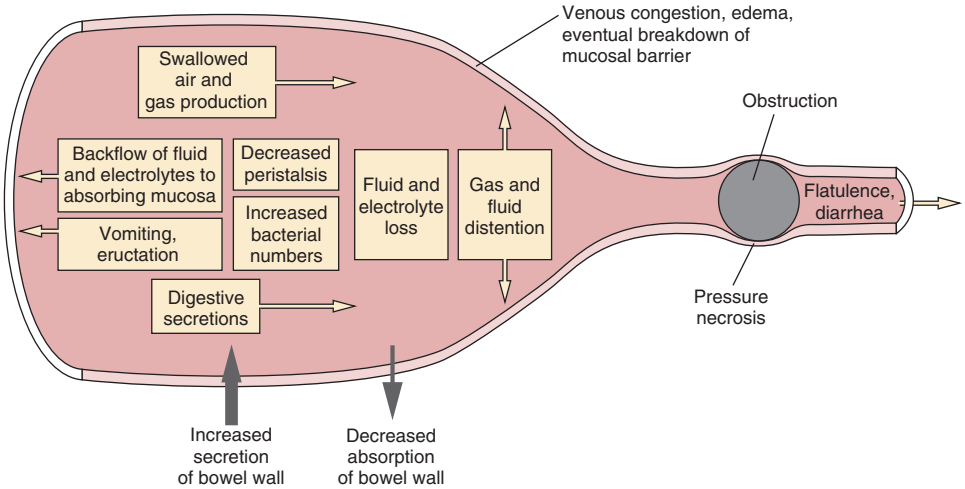


FIG. 18.115 Pathophysiologic events associated with mechanical obstruction of the intestinal lumen.

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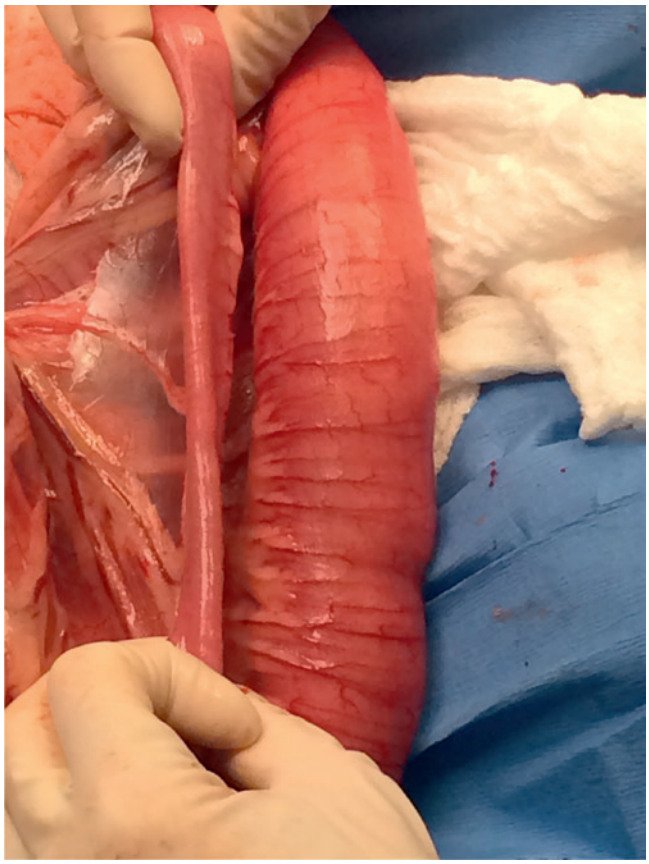


FIG. 18.116 Significant distension of the small intestine in the right of the image, orad to a foreign body.

its ability to absorb fluids, and local hypersecretion occurs. The major mechanisms of hypersecretion and decreased absorption are believed to originate from four sources: (1) hypersecretion mediated by enteric bacterial toxins secreted by noninvasive pathogenic bacteria that bind specific enterocyte receptors and stimulate salt and water production via the messenger camp or cyclic guanosine monophosphate pathways; (2) increased concentrations of bile and fatty acids and products of tissue ischemia at the obstruction site; (3) increased blood flow in the proximal parts of the obstructed intestine that may stimulate secretory activity; and (4) release of serotonin (5-hydroxytryptamine) by enteroendocrine cells that may be stimulated by increased luminal distention, which activates reflex pathways that increase chloride ion secretion. Other sources include chemical mediators of the enteric nervous system (acetylcholine, vasoactive intestinal polypeptide, substance P) that activate chloride ion–rich fluid secretion.

Intraluminal pressure proximal to the obstruction gradually increases because of the accumulation of fluid and gas. Lymphatic and capillary stasis occurs when intraluminal pressures reach 30 mm Hg (normal is 2–4 mm Hg with peristaltic pressures of 15–25 mm Hg); venous drainage is prevented when pressures reach 50 mm Hg. The arterial supply is not affected, and hydrostatic pressure increases at the capillary bed, producing a net shift of fluid into the interstitium and causing intestinal wall edema. Eventually fluid shifts not only into the lumen but also from the serosa into the peritoneal cavity. At a pressure of 44 mm Hg the intestinal segment may be so compromised that blood is shunted away from the intestinal capillaries and into arteriovenous anastomoses. Circulation in the mucosa and submucosa is impaired, oxygen consumption declines, arteriovenous shunting occurs, and the mucosa becomes ischemic. Full-thickness wall necrosis may occur at the obstruction site. Small intestinal stasis leads to luminal bacterial overgrowth. If the normal mucosal barrier is impaired by distention and ischemia, permeability may increase, with bacterial migration and absorption of toxins into the systemic circulation or peritoneal cavity or both.

Intestinal luminal distention causes increased myoelectrical activity proximal to the obstruction and decreased activity distally. The clusters of intense myoelectrical activity that migrate distally are interrupted by periods of absent motor activity as the duration of obstruction increases. The increased myoelectric activity proximal to the obstruction may be cholinergically mediated, whereas the distal inhibition of spike bursts is thought to be due to noncholinergic nonadrenergic pathways.

Large foreign bodies apply pressure to the intestinal wall. This may cause venous stasis and edema, followed by arterial flow compromise, ulceration, necrosis, and perforation.

More proximal and complete obstructions cause more acute and severe signs, with increased likelihood of dehydration, electrolyte imbalance, and shock. Proximal or high obstructions (i.e., duodenum or proximal jejunum) cause persistent vomiting, loss of gastric secretions, electrolyte imbalances, and dehydration. Large volumes of secretions and ingested fluid do not establish contact with jejunal and ileal mucosa for reabsorption. The major cause of mortality from upper small intestinal obstruction is severe hypovolemia with electrolyte disturbances. Untreated dogs with high, complete obstructions usually die within 3 to 4 days. Distal or low obstructions (i.e., distal jejunum, ileum, or ileocecal junction) cause varying degrees of metabolic acidosis. Clinical signs of distal and incomplete obstructions may be insidious, with vague, intermittent anorexia, lethargy, diarrhea, and occasional vomiting spanning several days or weeks. Signs are associated with maldigestion and malabsorption of nutrients. Diarrhea may be attributed to combined osmotic effects of unabsorbed substances in the intestinal lumen and to secretory activity of enterocytes. These animals usually lose weight but may live for more than 3 weeks if water is available. Causes of death associated with complete distal obstruction are fluid loss and toxemia related to bacterial proliferation.

A number of objects can assume a linear configuration, including string, thread, dental floss, nylon stockings, cloth, sacks, ribbon, and plastic. Part of the object lodges, usually at the base of the tongue (cats) or pylorus (dogs and cats), and the remainder advances into the intestine. As peristaltic waves attempt to advance the object, the intestine gathers around it, causing partial or complete obstruction (Fig. 18.117). Linear foreign bodies can cause partial or complete intestinal obstruction, but the clinical presentation may change. Continued peristalsis may cause the object to become taut, cut into the mucosa, and then lacerate the mesenteric border of the intestine, causing peritonitis. Multiple perforations may occur, and this is associated with high mortality. Some animals with linear foreign bodies have concurrent intussusceptions.

NOTE Linear foreign bodies typically perforate at the mesenteric border of the small intestine.

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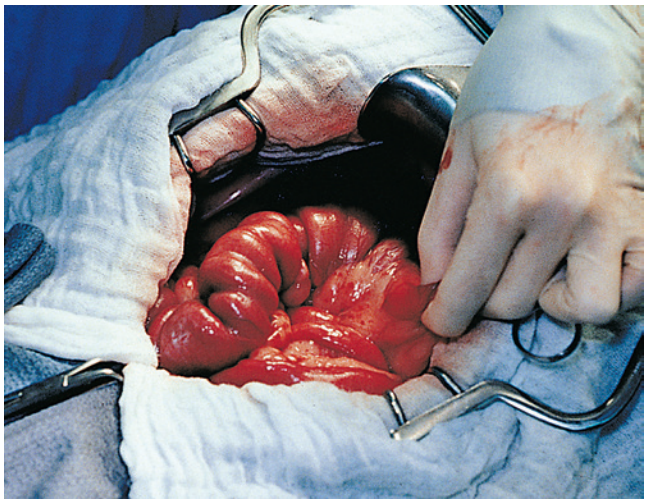


FIG. 18.117 Intraoperative appearance of bunched intestines that resulted secondary to a towel string foreign body.

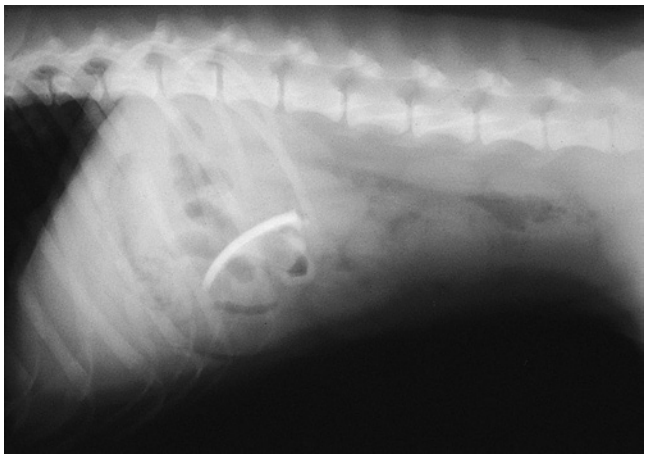


FIG. 18.118 Lateral abdominal radiograph of a 3-year-old female Labrador retriever. Note the radiopaque foreign body in the proximal small intestine. The multiple, well-circumscribed, soft tissue masses visible throughout the caudal abdomen are fetuses in the uterus. One fourth of a tennis ball was removed from the proximal duodenum.

DIAGNOSIS

Clinical Presentation

Signalment

There is no breed or sex predisposition; however, cats more commonly ingest linear foreign bodies than do dogs. Most dogs and cats with linear foreign bodies are under 4 years of age (for dogs, the mean age is 4.5 years and the median age is 2 years; for cats, the mean age is 2.7 years and the median age is 1 year). Other types of foreign bodies are more commonly found in dogs. Playful young animals seem more prone to foreign body ingestion.

History

The presentation and clinical signs depend on the location, completeness, the duration of the obstruction, and the vascular integrity of the involved segment. Acute onset of vomiting and anorexia are the most common presenting complaints. Depression, diarrhea, and abdominal pain are sometimes noted. Occasionally the animal is seen swallowing the object. Profuse vomiting may be seen with complete proximal obstruction; vomiting with partial distal obstructions is usually intermittent. Defecation may be absent or decreased in frequency, and the stool is occasionally bloody. Diarrhea is more common in animals with partial obstruction.

Physical Examination Findings

The physical examination may reveal abdominal distention, diarrhea, abdominal pain, abnormal posture, and/or shock. Classically, animals with high obstructions tend to be severely dehydrated; those with low obstructions tend to be thin. Abdominal palpation may identify a corrugated-feeling loop of bowel, an abnormal mass of bunched intestines, or may elicit pain. Linear foreign bodies may sometimes be visualized around the base of the tongue, but sedation/anesthesia may be required to visualize this area well enough to detect thin strings or thread. Abdominal pain is common if linear foreign bodies have caused bunching of the intestines. Abdominal auscultation may detect noise from peristaltic activity or silence associated with ileus.

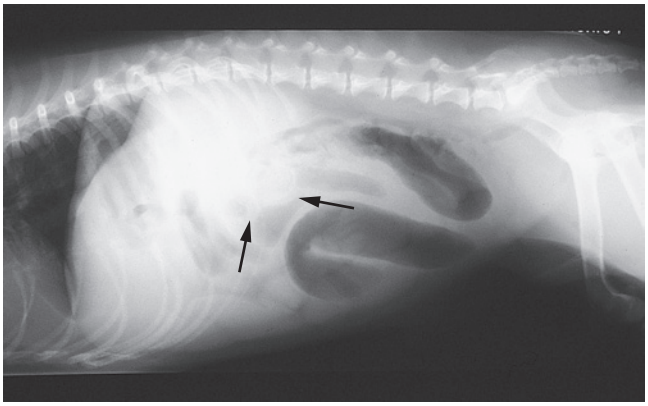


FIG. 18.119 Lateral abdominal radiograph of a dog with segmental ileus. Note the gas and fluid accumulation and the midabdominal, 2.5-cm circular, radio-opaque foreign body *(arrows)*. A hazelnut was removed from the jejunum.

Diagnostic Imaging

Survey radiography often reveals intestinal ileus resulting from a complete or near complete obstruction and may allow identification of the cause, especially if radiopaque foreign bodies are present. Radiolucent foreign objects are sometimes seen if surrounded by gas. Obstructed intestinal loops often become distended with air, fluid, and/or ingesta (Figs. 18.118 and 18.119). "Stacking" of distended intestines and sharp bends or turns in the dilated intestine suggest anatomic ileus as opposed to physiologic ileus. Assessment of intestinal distention can be determined by one of two methods. The maximum intestinal diameter is compared to the height of the body of the fifth lumbar vertebrae at its narrowest point. A ratio higher than 1.6 indicates distention, and those greater than 2 indicate a high probability of obstruction. Another method is to compare the intestinal diameter to the width of a rib; normal is less than 2. Linear foreign bodies may cause the intestines to appear bunched or pleated together, with small gas bubbles in the lumen (comma shaped) and without

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gas-distended intestinal loops (plication). However, some patients (especially cats) with linear foreign bodies have no obvious lesions on plain radiographs.

Contrast studies can often differentiate anatomic from physiologic ileus, although in general, anatomic or mechanical ileus is often focal, whereas physiologic or functional ileus is usually generalized. These two categories may be difficult to differentiate in cases of a distal obstruction. Contrast may delineate the foreign body, reveal luminal-filling defects, or demonstrate delayed transit time or displacement of intestinal loops. Prolonged transit time or complete stasis characterizes simple obstruction. However, severe ileus caused by low jejunal/ileal obstruction may be more difficult to accurately define with contrast studies because the contrast may or may not be able to reach the lesion. In general, radiographic contrast studies are infrequently done for this purpose.

Ultrasonography may identify foreign objects that cannot be seen radiographically, especially those with a hyperechoic margin with or without fluid accumulation. However, if there are large amounts of intestinal gas, a negative ultrasound examination should be viewed with skepticism. Ultrasound also allows assessment of motility, and finding loops of hypermotile bowel filled with fluid is very suggestive of obstruction. However, it is difficult to examine the entire intestinal tract ultrasonographically, and some foreign objects will not be revealed with ultrasound.

Endoscopy

Endoscopy rarely diagnoses intestinal foreign bodies that were not detected radiographically or with ultrasound. This is because the scope can seldom be advanced very far beyond the ascending duodenum. However, endoscopy is useful in diagnosing and removing gastric and some duodenal foreign bodies. In addition, endoscopy can find linear foreign bodies lodged at the pylorus, which can sometimes be removed endoscopically.

NOTE Be sure to repeat radiographs of animals with gastrointestinal foreign bodies immediately before surgery to ensure that the foreign body is still in the stomach or small intestine. Most foreign bodies that enter the large intestine are eliminated in the feces.

Laboratory Findings

Fluid, electrolyte, and acid-base abnormalities are often identified on CBCs and biochemistry profiles. PCV and total protein are sometimes increased in dehydrated patients. However, hypoalbuminemia may occur because of GI losses. Leukocytosis with a left shift or degenerative leukopenia accompanied by septic abdominal effusion indicates intestinal ischemia or perforation with peritonitis. Electrolyte and acid-base changes absolutely cannot be predicted in a given patient. In general, vomiting of gastric contents from any cause may lead to a hypochloremic hypokalemic, metabolic alkalosis, whereas vomiting associated with duodenal and proximal jejunal obstructions may be associated with mild metabolic acidosis and dehydration. Distal obstructions are more commonly associated with hypokalemia and metabolic acidosis. A slight increase in alanine aminotransferase, alkaline phosphatase, blood urea nitrogen, and creatinine may be seen with intestinal obstruction.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis includes all other causes of intestinal obstruction: intussusception, intestinal volvulus or torsion, intestinal incarceration, adhesions, strictures, abscesses, granulomas, hematomas, neoplasia, or congenital malformations. Physiologic ileus may be due to inflammatory diseases (e.g., parvovirus, peritonitis, pancreatitis).

MEDICAL MANAGEMENT

Some foreign bodies pass through the intestine without requiring therapy. Foreign body advancement may be monitored radiographically unless vomiting is severe, debilitation occurs, or evidence of peritonitis is seen (i.e., abdominal pain, fever, neutrophilia; see also p. 528). Radiographs should always be repeated shortly before surgery, even if previous ones were taken several hours before, because the foreign body may have moved into the colon or passed in the feces. Hairballs in cats are treated by administration of semisolid petrolatum-based laxatives for lubrication and a commercial hairball diet. Linear foreign bodies lodged at the base of the tongue in cats that are presented 1 to 3 days after ingestion may be cut and then monitored for passage, depending on how ill the cat appears. If the cat does not obviously feel better within 6 hours of cutting the foreign body off the tongue, it should be taken to surgery.

SURGICAL TREATMENT

In cases of partial obstruction, failure to radiographically demonstrate foreign body movement within the intestine over an 8-hour period or failure to pass the object within approximately 36 hours indicates the need for surgery. Surgery should not be delayed to observe for passage of the object through the intestinal tract if abdominal pain, fever, vomiting, or lethargy is apparent. Most foreign bodies can be removed by enterotomy rather than resection and anastomosis unless intestinal necrosis or perforation is present. If a linear object has been present a long time, it may become embedded in the mucosa, requiring intestinal resection. Multiple enterotomies (two to four) are often necessary to remove linear foreign bodies. Iatrogenic laceration of the mesenteric border may occur if excessive tension causes the object to saw through the wall before or during extraction.

Preoperative Management

Fluid, electrolyte, and acid-base deficits should be corrected before surgery if possible. Prophylactic antibiotics should be administered according to the recommendations on p. 433.

Anesthesia

Anesthetic recommendations for animals undergoing intestinal surgery are provided on p. 435, Table 19.1, and Table 18.4. Nitrous oxide should be avoided in animals with obstructions to prevent further gas accumulation in the intestinal tract.

Surgical Anatomy

The surgical anatomy of the small intestine is described on p. 436.

Positioning

The patient should be positioned in dorsal recumbency for a ventral midline celiotomy. The surgically prepared area should extend from the midthorax to the perineum.

ENDOSCOPIC REMOVAL OF FOREIGN BODIES

The endoscopic technique of removal of duodenal foreign bodies consists of advancing the scope until the foreign body is

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visualized and studying it before attempting to grasp it. *Consider which foreign body retrieval device (e.g., four-wire basket, snare, alligator jaws, W-type coin forceps) should be used. Always be sure that the foreign body can be released if necessary (it is frustrating to grab a foreign object and not be able to remove it or release it).* It can sometimes be hard to pull the foreign body out of the pylorus. Administering ketamine as a preanesthetic might relax the pylorus and lower esophageal high-pressure zone, making it easier to retrieve the object after it has been snared. The endoscopic technique for removing linear foreign bodies is given on p. 418 in the Gastric Foreign Bodies section.

SURGICAL TECHNIQUE

Make an incision through the linea alba that is sufficient to allow complete exploration of the abdomen. Explore the entire abdomen and GI tract to avoid overlooking concurrent abnormalities or multiple foreign bodies present in different parts of the GI tract. Once the foreign body has been located, isolate this loop of intestine from the remainder of the abdominal cavity with laparotomy pads or sterile towels. Complete obstructions may cause the bowel to be severely distended and appear cyanotic; however, reserve determination of intestinal viability until the bowel has been decompressed and the foreign body has been removed by enterotomy. Bathe the intestine in warm saline for a few minutes to help improve its color and peristalsis. Normally the appearance of the intestine improves rapidly after decompression. *If the intestinal segment is determined to be viable, close the enterotomy with simple interrupted or continuous sutures as described on p. 438. Resect nonviable or questionable intestine and reestablish bowel continuity by end-to-end anastomosis (see p. 440). After removing the foreign body, carefully examine the intestine for evidence of perforation that might require resection of the involved segment or segments. Create a serosal patch when the intestinal wall is contused to prevent leakage.*

Removal of linear foreign bodies may require gastrotomy and multiple enterotomies. *Release the foreign body from where it is lodged. Cut the thread that is embedded sublingually or perform a gastrotomy. Attach a hemostat to the distal end of the object when the mass is removed from the pylorus. Pull gently on the hemostat, and identify the next more distal point of attachment. Perform an enterotomy and withdraw the more proximal foreign body. Again, attach a hemostat distally, transect the foreign body, apply gentle tension on the remaining intraluminal foreign body, and identify the next point of attachment. Repeat until the entire linear foreign body is removed. Avoid excess tension on the foreign body as this may lead to full-thickness laceration of the mesenteric border. Close the enterotomies (see p. 438) and inspect the mesenteric border for evidence of perforation.*

A single-enterotomy catheter technique has been described for removing linear foreign bodies. It is most successful when foreign bodies have not penetrated the mucosa. This technique may not be successful if the condition is chronic and the foreign body is embedded in the mesenteric border. *Make an incision into the stomach or intestine at the site where the object is fixed. Suture the linear object to a soft catheter and then completely advance the catheter into the distal intestine. Close the enterotomy site and milk the catheter and foreign body through the intestinal tract and out through the anus.* This technique reduces the number of enterotomies and may thereby reduce the risk of leakage and dehiscence.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Instruments for enterotomy or intestinal resection and anastomosis are discussed on p. 449. Polydioxanone, poliglecaprone 25, polyglyconate, and glycomer 631 (3-0 or 4-0) are the preferred suture materials for these procedures; however, nonabsorbable suture (nylon or polypropylene) is sometimes used in hypoalbuminemic animals.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative treatment includes further correction of fluid, electrolyte, and acid-base deficits. Analgesics should be given as needed to control pain (see Table 13.2). Antibiotics should be continued if peritonitis was diagnosed or if gross abdominal contamination occurred. If no vomiting has occurred, water can be offered 8 to 12 hours after surgery, and food can be given 12 to 24 hours after surgery. These patients should be monitored for signs of leakage and peritonitis (see p. 449).

COMPLICATIONS

Early diagnosis of intestinal foreign bodies and good surgical technique are necessary to prevent complications (e.g., intestinal necrosis, perforation, leakage, dehiscence, peritonitis, endotoxic shock, and stenosis). The risk of anastomotic leakage is greater when associated with a foreign body, debilitation, and hypoalbuminemia. The risk of peritonitis and death is much higher if free gas is present on preoperative radiographs. Resecting large segments of intestine may result in a patient with SBS (see p. 450) and a guarded prognosis.

PROGNOSIS

The prognosis is good if peritonitis and extensive resections are avoided. Mortality is greater in animals with a longer duration of clinical signs, linear foreign body, or those needing more than one GI incision. Chronic obstruction, greater bowel compromise, longer surgery time, increased risk of contamination, and multiple incisions required to remove linear foreign bodies make the association between linear foreign body and poorer outcome logical. The location of the foreign body and degree of obstruction are generally not associated with outcome. The prognosis without surgery is guarded because animals may die from hypovolemic or endotoxic shock, septicemia, peritonitis, or starvation.

INTESTINAL NEOPLASIA

DEFINITION

Intestinal neoplasia is a condition in which tumors arise from one of the layers of the intestinal wall, its glands, or associated cells or lymphatics.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Intestinal tumors most often occur in the canine rectum or colon and the feline small intestine (Box 18.44). Most intestinal tumors are malignant. Intestinal tumors may cause intramural or intraluminal mechanical obstruction. They most commonly invade the muscular layer of the intestinal wall where they

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BOX 18.44 Intestinal Neoplasia: Key Points

- Most are malignant; most common: adenocarcinoma, lymphosarcoma, leiomyosarcoma
- Cause intramural or intraluminal mechanical obstruction
- Resect with 4–8 cm margins of normal appearing intestine
- Prognosis varies with tumor type

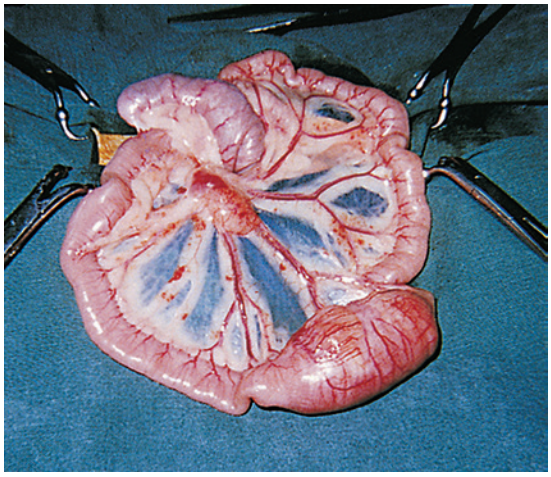


FIG. 18.120 Intraoperative photograph of an adenocarcinoma that was partly obstructing the jejunum of a 15-year-old cat.

compromise the lumen diameter and reduce distensibility. The proximal bowel distends with fluid and gas, and its function is compromised as with foreign body obstruction. The disease is usually advanced at the time of diagnosis, and most malignant tumors have metastasized. Malignant tumors spread by local invasion (e.g., serosa, mesentery, omentum, local lymph nodes) and distant metastasis (i.e., lungs, liver, and spleen). The most common small intestinal malignancies are adenocarcinomas (Fig. 18.120) and lymphosarcomas. Other small intestinal neoplasms are leiomyomas, leiomyosarcomas, fibrosarcomas, mast cell tumors, hemangiosarcomas, anaplastic sarcomas, carcinoids, plasmacytomas, neurolemmomas, adenomas, and adenomatous polyps.

Adenocarcinomas are locally invasive and slow-growing. They most commonly arise in the duodenum and colon of dogs and in the distal jejunum and ileum of cats. These tumors have three main morphologic forms: (1) infiltrative adenocarcinomas cause a thickened stenotic area that obstructs the intestinal lumen; (2) ulcerative adenocarcinomas have a deep indurated mucosal ulcer with raised edges; and (3) proliferative adenocarcinomas are lobulated, expanding intraluminal masses. Mucosal ulceration may cause chronic bleeding, melena, and iron deficiency anemia. The tumors spread to adjacent serosal surfaces, mesentery, omentum, and regional lymph nodes by local invasion and may metastasize distally to the lungs and liver. Mucinous intestinal adenocarcinomas extend transmurally to the mesentery with accumulation of variable amounts of mucin.

Lymphosarcomas (lymphomas) are neoplastic proliferations of lymphocytes. In cats, they may be caused by FeLV or feline immunodeficiency virus (FIV), but most are caused by unknown mechanisms. The etiology in dogs is unknown. Affected animals may have multicentric disease. Lymphomas have two intestinal types: diffuse and nodular. Diffuse infiltration of the lamina propria and submucosa with neoplastic lymphocytes causes malabsorption and occasional deep ulceration. Nodular lymphoma is an expanding intestinal mass that causes obstruction, often but not invariably in the ileocecocolic area. Involvement of regional lymph nodes and other organs is common. Cats in particular may have intestinal lymphoma and no gross or ultrasonographic evidence of intestinal disease. Dogs typically have lymphoblastic lymphoma, whereas there are multiple forms of lymphoma in cats including small cell lymphoma, lymphoblastic lymphoma, epitheliotropic lymphoma, and large granular lymphoma.[42]

Small intestinal smooth muscle tumors are separated into leiomyomas and GI stromal tumors (GIST); however, immunophenotyping is necessary to positively distinguish between these tumor types; many tumors previously classified as leiomyosarcomas have been reclassified as GIST after immunohistochemistry analysis (KIT, DOG1) was performed. Imatinib mesylate–targeted therapy may be effective in some dogs with GIST.

Intestinal leiomyosarcomas are slow-growing, malignant smooth muscle tumors of older dogs that usually occur in the cecum and jejunum. Neoplastic spread is by local invasion, and metastasis is slow. Metastasis occurs to the mesentery, mesenteric lymph nodes, peritoneum, and liver. Median survival of approximately 2 years is expected after resection of a localized lesion. This is also true of animals with metastasis at the time of surgery. Intestinal leiomyomas are slow-growing, benign smooth muscle tumors. Adenomatous polyps are found in the feline duodenum and canine rectum. Recurrence after complete resection is not expected.

DIAGNOSIS

Clinical Presentation

Signalment

Adenocarcinomas are more common in dogs than in cats. In dogs, lymphoma, adenocarcinoma, and leiomyosarcomas are the most common intestinal tumors. Boxers, collies, and German shepherds may be predisposed to intestinal tumors. In cats, lymphosarcomas are most common followed by adenocarcinomas and mast cell tumors. Siamese cats may be predisposed to small intestinal adenocarcinomas. Intestinal tumors generally occur in older animals. Carcinomas are seen at a mean age of 9 years in dogs and 10 years in cats. Leiomyosarcomas occur at a mean age of 11 years in dogs. Lymphosarcomas occur in dogs and cats at a mean age of 10.6 years. The mean age for cats with intestinal mast cell tumors is 13 years.

History

Patients initially have vague clinical signs of depression, anorexia, and lethargy, which may progress to diarrhea, and/or vomiting. Weight loss is progressive. Other clinical signs may include dehydration, melena, hematemesis, anemia, fever, icterus, polyuria, polydipsia, and/or abdominal effusion. Signs of intestinal obstruction, abscessation, and malabsorption may also occur. Lymphatic obstruction may cause steatorrhea from lymphangiectasia. Signs involving other organs may develop secondary to metastasis.

Physical Examination Findings

The animal may be in poor body condition. Abdominal palpation may reveal a firm abdominal mass, thickened intestinal loops, or mesenteric lymphadenomegaly.

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Diagnostic Imaging

Masses, abnormal gas and fluid patterns, visceral displacement, and abdominal fluid may be seen on survey abdominal radiographs. Contrast radiographs can be helpful for delineating regions of mucosal irregularity, luminal narrowing, and intramural infiltration, thickening, or nodularity; however, they are seldom necessary if ultrasound is available. If neoplasia is suspected, thoracic radiographs or CT should be requested.

Abdominal ultrasonography often (not always) delineates intestinal masses or evidence of metastasis, and may facilitate percutaneous fine needle aspiration. Intestinal tumors produce a broad spectrum of ultrasonic patterns. Ultrasonographic findings may include intestinal wall thickening and loss of discrete wall layers, but lymphosarcoma may be present despite a normal-appearing intestine. Smooth muscle tumors may appear as eccentric, poorly echogenic masses with anechoic cavities. Ileus, fluid accumulation, and lymphadenomegaly may be recognized. The overall agreement between ultrasound-guided fine needle aspirates and histopathologic diagnosis is approximately 50%; however, sensitivity and specificity of aspirates varies greatly depending on the tumor type.

Endoscopy

Flexible endoscopic evaluation of intestinal tumors beyond the duodenum may be difficult. Mucosal irregularity, inflammation, ulceration, and a narrowed duodenal or high jejunal lumen may be detected, or the intestine may appear normal. Mucosal biopsies can be diagnostic if the tumor involves the mucosa.

NOTE Ultrasonography is preferable to contrast radiography in affected animals.

Laboratory Findings

Hematologic and biochemical profiles are often normal. Laboratory evaluation may reveal blood loss anemia, iron deficiency anemia, neutrophilic leukocytosis with/without left shift, hypoalbuminemia, hypoglycemia, and/or elevated serum hepatic enzyme concentrations. A definitive diagnosis of intestinal neoplasia can only be made by cytology or histopathology.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis includes all other causes of intestinal obstruction (e.g., foreign bodies, intussusception, intestinal volvulus or torsion, adhesions, strictures, abscesses, granulomas, hematomas, or congenital malformation). Another cause might be physiologic ileus that occurs secondary to severe inflammation (e.g., parvovirus or peritonitis). Eosinophilic masses of the GI tract with no identifiable underlying cause should be considered as a differential diagnosis in dogs, especially in those with multiple mass lesions. These masses may occur anywhere along the GI tract, and surgical treatment alone is associated with a poor prognosis. Anthelmintic and glucocorticoid therapy are required for this condition.

MEDICAL MANAGEMENT

Feline small cell intestinal lymphoma often responds relatively well to chemotherapy. (Refer to an oncology text for additional information and chemotherapeutic protocols.) Other tumor types tend to have modest to poor response to chemotherapy. Radiation therapy is used primarily for tumors in the distal half of the rectum and anal canal.

SURGICAL TREATMENT

Surgical resection is the treatment of choice for focal intestinal tumors; however, many tumors are too advanced to allow complete resection by the time they are diagnosed. If metastasis has occurred, surgical resection may be palliative. However, if resection is attempted, it is important to suture healthy tissue to healthy tissue; suturing tumor-containing tissue to healthy tissue makes dehiscence likely.

Preoperative Management

Fluid, electrolyte, and acid-base deficits should be corrected before surgery if possible. Transfusions should be considered if the PCV is below 20%. Prophylactic antibiotics should be given according to the recommendations on p. 435. Debilitated animals, or those that are likely to remain anorectic or have continued vomiting, should have enteral feeding tubes placed at the time of surgery (see p. 101).

Anesthesia

General anesthetic recommendations for animals undergoing intestinal surgery are provided on p. 435, Table 19.1, and Table 18.4.

Surgical Anatomy

The surgical anatomy of the small intestine is presented on p. 436.

Positioning

Patients should be positioned in dorsal recumbency for a ventral midline celiotomy. The entire abdomen and caudal thorax should be clipped and prepared for aseptic surgery.

SURGICAL TECHNIQUE

Make an incision through the linea alba from the xiphoid process to the pubis to allow complete exploration of the abdomen. Explore the entire abdomen and GI tract to avoid overlooking concurrent abnormalities. Biopsy mesenteric lymph nodes and other organs as needed before incising intestine. Resect the mass with 4- to 8-cm margins of grossly normal tissue and perform an end-to-end anastomosis (see p. 440). Pay special attention to surgical technique because these patients often are debilitated. Submit tissues for histopathologic evaluation and tumor staging. Change gloves and use uncontaminated instruments and suture for abdominal closure.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Instruments for enterotomy or intestinal resection and anastomosis are discussed on p. 449. Polydioxanone, poliglecaprone 25, polyglyconate, and glycomer 631 (3-0 or 4-0) are the preferred suture materials for these procedures; however, nonabsorbable suture (nylon, polypropylene, or polybutester) may be used in hypoalbuminemic animals.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative care should be individualized according to the patient's status and concurrent diseases. Fluid support should

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be maintained until the animal is drinking enough to maintain hydration. Electrolyte and acid-base deficits should be corrected. Analgesics should be provided as necessary (see Table 13.2). If peritonitis, abdominal contamination, or severe debilitation occurs, therapeutic antibiotics should be given. Nutritional support should be provided by enterostomy tube if vomiting or anorexia persists (see p. 101). Adjuvant chemotherapy has been recommended for some malignant intestinal tumors, but its efficacy is unproved (see the section on Medical Management presented earlier) except for lymphoma. However, chemotherapy of intestinal large cell lymphoma is often associated with morbidity, directly or indirectly related to the drugs and destruction of malignant cells in the intestinal wall. Second-look laparotomy or laparoscopy is helpful in evaluating disease progression or response to adjuvant therapy and allows further biopsy or further resection if indicated.

COMPLICATIONS

Leakage, dehiscence, and peritonitis are possible complications and may occur more frequently in debilitated patients. Stenosis after surgical resection or tumor recurrence may cause recurrent signs of obstruction. SBS (see p. 450) is a possible complication if large portions of the bowel are resected.

PROGNOSIS

The prognosis is excellent when benign tumors or polyps are completely excised. The prognosis is good for patients with a localized intestinal adenocarcinoma or leiomyosarcoma if complete resection is possible. The median survival time is 10 months; the 1-year survival rate is 40.5%, and the 2-year survival rate is 33% for dogs with an adenocarcinoma or a leiomyosarcoma. Cats with an intestinal adenocarcinoma may live longer than 2 years after surgery. The prognosis for solitary nodular lymphoma is better than for diffuse lymphoma, which carries a poor prognosis. The prognosis associated with lymphoblastic lymphoma is poor; however, cats with small cell lymphoma can have survival times of years.[42] The prognosis for leiomyosarcoma is good, even with metastasis at the time of resection (discussed earlier). Intestinal sclerosing mast cell tumor has been identified as a type variant of mast cell neoplasia in the intestine of cats that seems to have a high risk of metastasis and guarded prognosis. A guarded to poor prognosis should be given if nonresectable tumors are present because other modes of therapy are ineffective, of questionable value, or not advised because of severe side effects.

INTUSSUSCEPTION

DEFINITIONS

Intussusception is the telescoping or invagination of one intestinal segment **(intussusceptum)** into the lumen of an adjacent segment **(intussuscipiens)** (Fig. 18.121A).

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

GI tract intussusceptions may occur anywhere; however, ileocolic and jejunojejunal intussusceptions are most common (Box 18.45). Intussusceptions are often associated with enteritis (i.e., parasitism, viral or bacterial infection, dietary indiscretion or change, foreign

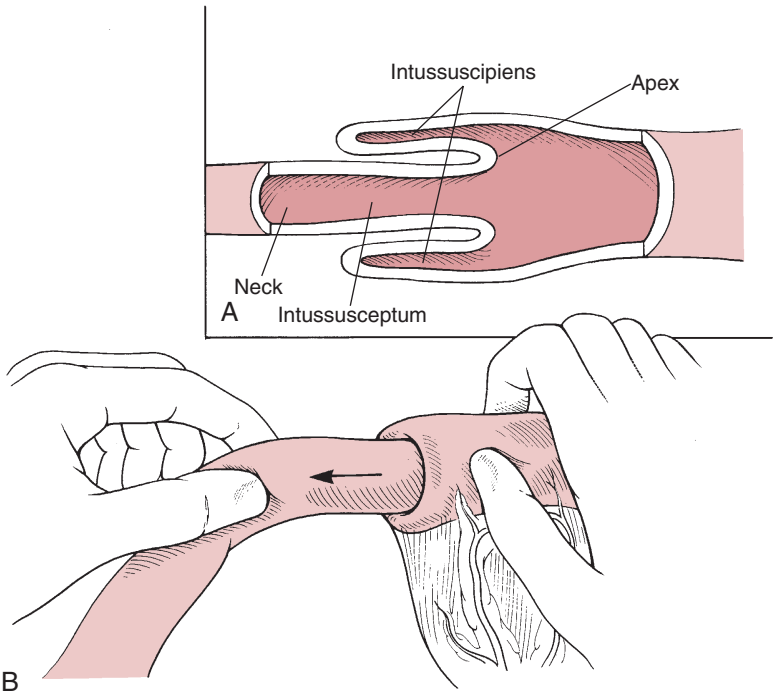


FIG. 18.121 (A) Configuration of an intussusception: neck, intussusceptum, apex, intussuscipiens. (B) To reduce an intussusception, place traction on the neck as you milk the apex out of the intussuscipiens. The arrow denotes the direction of reduction of the intussusceptum.

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BOX 18.45 Intussusception: Key Points

- Young animals are most often affected, especially after parvoviral enteritis.
- This and parasites are major causes of protein-losing enteropathies in young dogs.
- Older animals with intussusceptions often have masses.
- Associated with enteritis or systemic illness.
- Signs progress from partial to complete obstruction.
- Clinical course may span several weeks.
- Invagination is usually normograde but occasionally retrograde.
- Ultrasound reveals target or bull's-eye pattern (concentric intestinal layers).
- Manually reduce or resect and anastomose.
- Enteroenteropexy prevents recurrence.

bodies, masses) or systemic illness; however, the cause of most intussusceptions is unknown. The presence of an intussusception in a cat is more likely associated with neoplasia than in dogs, and affected cats are usually older than affected dogs. They have also been reported after environmental change and surgery. Intussusceptions after surgery may be associated with ileus, adhesions, or anastomotic malfunction. Intestinal irritation that results in hypermotility may cause one intestinal loop to invaginate into another. The direction of the intussusception can be from proximal to distal or vice versa. The intussusceptum is more commonly a proximal intestinal segment, and the intussuscipiens a more distal segment (i.e., the intussusception occurs in the direction of normal peristalsis; direct or normograde intussusception). Intussusceptions can occur at more than one site and are sometimes double (two invaginations at same site). Reverse peristalsis may increase the length of intestine involved in the intussusception. The amount of available mesentery limits the extent of intestinal involvement and the degree of vascular compromise.

The formation of an intussusception is thought to be due to nonhomogeneity of the wall caused by any abnormality within the wall that alters local intestinal motility or pliability. The number and mean ganglion cell density of myenteric plexuses are decreased in resected segments of intussusceptions compared with normal intestine, and the degree of vacuolization of the myenteric plexuses is greater, suggesting abnormal innervation of the bowel. As the intussusception is formed, longitudinal and circular contractions of normal intestine in an adjacent area cause displacement of the intestine and a fold in the intestine is formed. The fold is propagated circumferentially, and longitudinal muscle contraction completes the invagination.

Initially, invagination causes partial intestinal obstruction, which may progress to complete obstruction. Vessels attached to the intussusceptum collapse because of increased intraluminal pressure or kinking, and those vessels may avulse. The wall becomes edematous, ischemic, and turgid. Blood extravasates into the lumen, and the serosa fissures. Fibrin seals the layers of the intestine together and may help localize peritonitis as wall necrosis occurs. Eventually intestinal devitalization occurs, with subsequent contamination of the abdominal cavity. Intussusceptions may occur as agonal events (i.e., are incidental findings and not the cause of death). Agonal intussusceptions are easily reduced and are associated with minimal inflammation; intestinal walls are not edematous, and fibrin does not seal the layers of intestine together.

DIAGNOSIS

Clinical Presentation

Signalment

Intussusceptions occur more commonly in dogs. German shepherds and Siamese cats may be more commonly affected than other breeds. Intussusceptions appear to be more common in immature animals (75% <1 year). Parasitism or enteritis should be suspected as a cause for intussusception in young dogs, and intestinal thickening or masses should be suspected in adults. The occurrence of intussusception in cats may be bimodal, with young cats (<1 year) having idiopathic lesions and cats 6 years or older having concurrent IBD or lymphosarcoma.

History

Most animals have been ill, have changed environments, or have had recent surgery before signs of intussusception begin. The severity and type of clinical signs depend on the location, completeness, vascular integrity, and duration of the intestinal obstruction. Scant, bloody diarrhea, vomiting, abdominal pain, and a palpable mass may occur with intussusceptions. Acute intussusceptions must be considered in puppies with parvoviral enteritis that suddenly become worse or that persist much longer than expected. In particular, a diagnosis of chronic parvoviral enteritis is very suggestive of intussusception. Anecdotally, intussusceptions have been associated with leptospirosis. Chronic cases can have less marked clinical signs. Patients with chronic intussusception often have intractable, intermittent diarrhea, and hypoalbuminemia; intussusceptions and parasites are the two major causes of chronic protein-losing enteropathy in dogs less than 8 to 12 months of age. Other clinical signs include depression, anorexia, and emaciation.

NOTE Consider chronic intussusception as a possible reason why a puppy with an apparently acute episode of enteritis (e.g., parvovirus) has persistent diarrhea.

Physical Examination Findings

A presumptive diagnosis of intussusception can be made when an elongated, thickened intestinal loop (sausage-shaped mass) is palpated. Jejunojejunal intussusceptions are easier to palpate than ileocolic intussusceptions because they are usually more caudal and ventral in the abdomen. Some intussusceptions slide in and out of the colon and can be missed during palpation. Others may protrude from the rectum and can be mistaken for a rectal prolapse. To distinguish rectal prolapse from protruding intussusception, the area around the protruding tissue should be palpated; the existence of a fornix indicates rectal prolapse rather than intussusception.

Diagnostic Imaging

Survey radiographs of patients with intussusception occasionally reveal obstruction; however, most of the time the radiographs do not reveal pathology. Jejunojejunal intussusception is more likely to produce an obstructive pattern than ileocolic intussusception (Figs. 18.122 and 18.123). A tubular soft tissue mass may be identified. If sufficient gas accumulates in the distal intestinal segment, the apex of the intussusception may be outlined. A barium enema or upper GI tract study can localize the obstruction. A ribbon of contrast material may be seen in

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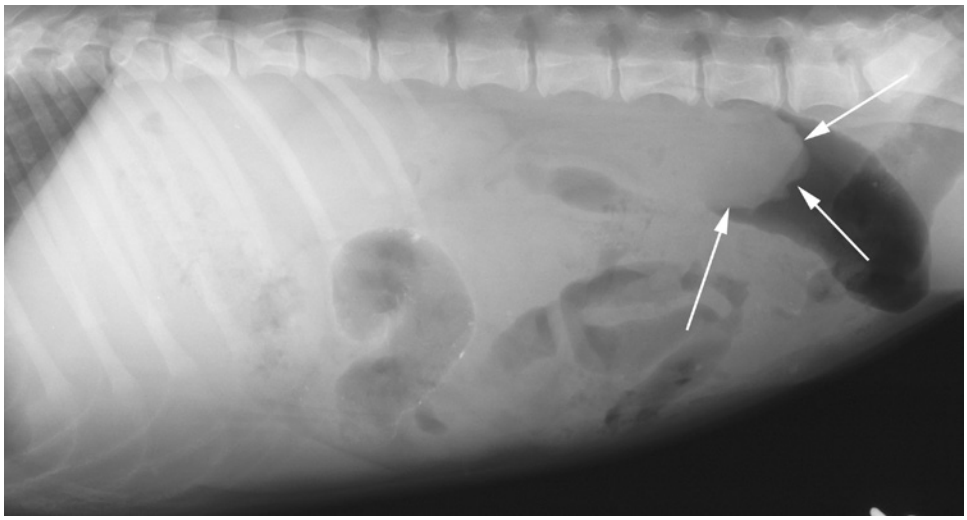


FIG. 18.122 Lateral radiograph of a dog with an ileocolic intussusception. Notice the soft tissue mass within the air-filled colon *(arrows)*.

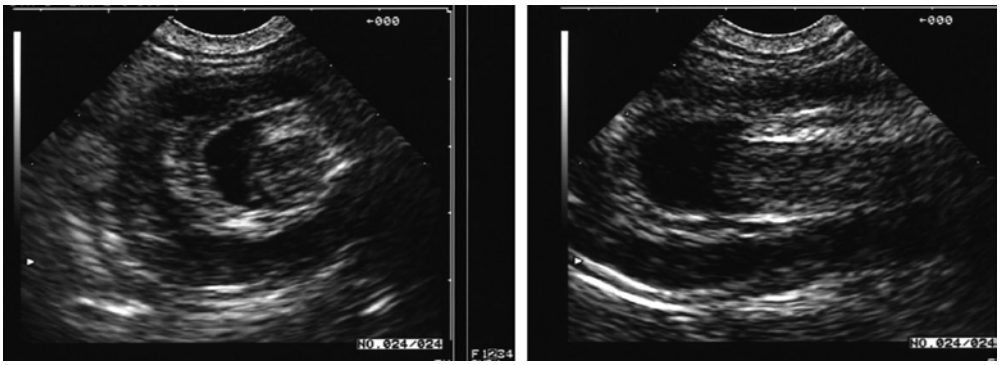


FIG. 18.123 Transverse *(left)* and sagittal *(right)* images of an intussusception. Notice the target-like appearance on the transverse view.

the intussusceptum aboral to a dilated intestinal segment. A large "coiled-spring"–appearing filling defect may be present when barium within the lumen surrounds the intussusceptum. Occasionally contrast material accumulates in the lumen between the intussusceptum and the intussuscipiens.

Ultrasonography is the most useful method for detecting intussusceptions. The ultrasonographic appearance of an intussusception in the transverse plane is that of a multilayered, target-like lesion (concentric hyperechoic and hypoechoic rings with an overall width greater than 8–9 mm) with associated proximal fluid accumulation and diminished intestinal motility. The juxtaposition of the wall layers of the intussusceptum and intussuscipiens create more than the normal 10 hyperechoic and hypoechoic concentric rings, although these layers may be indistinct. Longitudinal scans demonstrate a layered appearance with alternating parallel hyperechoic and hypoechoic lines. Identification of distinct layers on ultrasound examination is an indication that the intussusception may be reducible. Multiplane imaging; the presence of an eccentric, semilunar, or G-shaped hyperechoic center (mesenteric fat); and visualization of the inner intussusceptum are helpful in differentiating an intussusception from similar conditions where target-like lesions may also be identified. Ultrasonography may identify concurrent abdominal abnormalities such as lymphadenopathy or infiltrative intestinal lesions.

Colonoscopy

Colonoscopy sometimes identifies invaginated intestine protruding into the colon in patients with ileocolic or cecocolic intussusception. In such cases, colonoscopy can usually be performed with little more than manual restraint, if the endoscopist is gentle and careful.

Laboratory Findings

Abnormal laboratory findings may include dehydration, stress leukograms, anemia, and electrolyte and acid-base abnormalities. Chronic intussusception may cause hypoalbuminemia because of protein loss from congested mucosa. Fecal examination sometimes reveals parasitic infestation.

DIFFERENTIAL DIAGNOSIS

The differential diagnoses include all other causes of intestinal obstruction (e.g., foreign bodies, intestinal volvulus or torsion, intestinal incarceration, adhesions, strictures, abscesses, granulomas,

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hematomas, tumors, or congenital malformations). Another cause might be physiologic ileus that occurs secondary to inflammation (e.g., parvovirus or peritonitis).

MEDICAL MANAGEMENT

Occasionally percutaneous manual reduction of the intussusception is successful and the intussusception does not recur. In rare cases, intussusceptions self-correct by forming adhesions and sloughing the intussusceptum. However, most intussusceptions require surgical reduction and ancillary procedures to prevent recurrence. Medical therapy should be aimed at correcting fluid and electrolyte imbalances and determining the underlying cause of the intussusception (e.g., enteritis, parasitism).

SURGICAL TREATMENT

Because recurrence is common, intussusceptions should be treated surgically even if they can be manually reduced. Biopsying the intestine at the time of surgical correction may help identify the cause of the intussusception. The tip of the intussusceptum should be evaluated for masses.

Preoperative Management

Hydration, electrolyte, and acid-base deficits should be corrected before surgery if possible. Pediatric patients should not be fasted for longer than 4 to 8 hours to reduce the chances of hypoglycemia. Prophylactic antibiotics should be given according to the recommendations on p. 435.

Anesthesia

General anesthetic recommendations for compromised patients undergoing GI surgery are provided on p. 435 and Table 18.4. See Table 19.1 for anesthetic recommendation for stable patients needing abdominal surgery.

Surgical Anatomy

The surgical anatomy of the small intestine is presented on p. 436.

Positioning

Animals should be positioned in dorsal recumbency for a ventral midline celiotomy. The entire abdomen and caudal thorax should be clipped and prepared for aseptic surgery.

SURGICAL TECHNIQUE

Explore the abdomen, collect specimens, and isolate the involved intestine with laparotomy pads (Fig. 18.124). Reduce intussusceptions manually if possible by gently applying traction on the neck of the intussusceptum while milking its apex (leading edge) out of the intussuscipiens (see Fig. 18.121B). Avoid excessive traction because this may tear the compromised intestine. Push on the intussuscipiens more than pull on the intussusceptum. Manual reduction is successful only if fibrin has not formed firm serosal adhesions. *Evaluate the reduced intestine for viability and perforation. Carefully palpate the leading edge of the intussusceptum to detect mass lesions. Perform a resection and anastomosis if a mass is detected, manual reduction is impossible, tissue is devitalized, or mesenteric vessels have been avulsed from a portion of the involved intestine. Submit biopsies of the involved intestine to help identify the cause of the intussusception.*

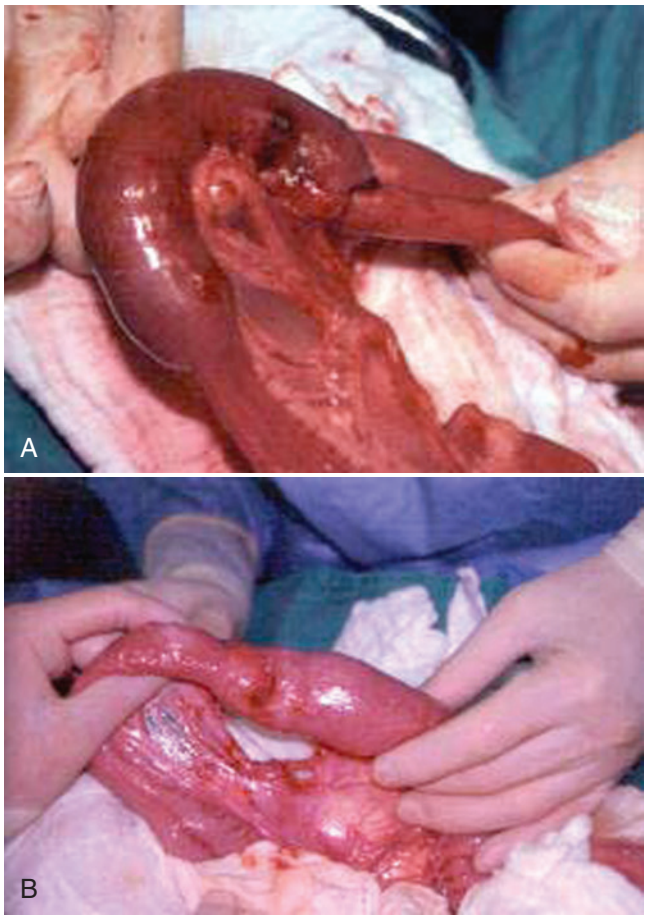


FIG. 18.124 (A) Intraoperative appearance of an intussusception. (B) Following partial manual reduction of this intussusception, serosal and mesenteric tearing are noted.

NOTE Consider performing an enteroenteropexy (see p. 448) on these patients to help prevent recurrence (see Fig. 18.114).

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Instruments for enterotomy or intestinal resection and anastomosis are discussed on p. 449. Polydioxanone, poliglecaprone 25, polyglyconate, and glycomer 631 (3-0 or 4-0) are the preferred suture materials for these procedures; however, nonabsorbable suture (nylon, polybutester, or polypropylene) may be used in hypoalbuminemic animals. Chromic gut suture should be avoided in young animals and those that are debilitated because the suture may be rapidly catabolized and weakened.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative management should be individualized according to the patient's status and concurrent diseases. Hydration, electrolyte, and acid-base abnormalities should continue to be corrected postoperatively until the animal resumes adequate oral intake. Analgesics should be provided according to the recommendations in Chapter 13. The administration of butorphanol tartrate or other opioids may reduce recurrence. Therapeutic antibiotics are appropriate if peritonitis, abdominal contamination, or severe debilitation is present. Nutritional support via

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an enterostomy tube (see p. 101) may be necessary if the patient is debilitated or vomiting or remains anorectic. Monitor for recurrence and peritonitis.

PROGNOSIS

Prognosis depends on the cause, location, completeness, and duration of the intussusception. Animals with untreated intestinal intussusceptions may die within 3 or 4 days or may live for several months. Those that die acutely usually have high obstructions or enterotoxemia; death is due to hypovolemia and electrolyte and acid-base imbalances. Animals with an intussusception may live for several weeks or months if the obstruction is partial or distal, the vasculature is functional, and an adequate fluid intake is maintained. Prognosis worsens with perforation of the intestine and peritonitis. In rare cases an animal self-cures if the neck of the intussusception seals, a firm adhesion forms to the intussuscipiens, and the intussusceptum sloughs reestablishing luminal patency. The prognosis with surgery is good with aggressive supportive care and early surgical intervention, assuming recurrence is prevented and extensive resections are avoided. Without enteroenteropexy, recurrence may be expected in approximately one-third of affected animals.

Complications following treatment of intussusceptions include recurrence, intestinal obstruction, ileus, anastomosis dehiscence, peritonitis, and SBS. Leakage, dehiscence, peritonitis, and death are possible complications, which occur more frequently in debilitated patients. Stenosis and SBS may occur if large segments of the bowel are removed (see p. 450). Complications of enteroenteropexy include intestinal obstruction and strangulation of intestine between enteroplication sutures; however, these complications are avoidable with meticulous technique.

PYTHIOSIS

DEFINITION

Enteritis is inflammation of the intestine. Organisms or syndromes that cause chronic enteritis may cause obstruction, diarrhea, or vomiting, or all of these.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Pythiosis is a fungal infection that is common in some areas of the United States. *Pythium* spp. are ubiquitous, being found in water, soil, vegetable matter, and feces. *P. insidiosum* is an aquatic organism (oomycete) to which animals in contact with swamp water are commonly exposed. High water temperatures may enhance the growth of *Pythium* organisms and asexual reproduction (motile, biflagellated zoospores), predisposing animals to infection during the summer and fall months. *Pythium* lesions can affect any part of the GI tract (i.e., esophagus, stomach, small and large intestines, rectum, and mesentery), as well as the skin and mesenteric lymph nodes. *Pythium* spp. may invade traumatized tissue (devitalized, necrotic, or ulcerated), but the motile spores may also penetrate intact mucosa. Fungal hyphae invade the intestinal wall, causing infarction, necrosis, and a granulomatous tissue reaction. The intestinal wall thickens, and signs of partial intestinal obstruction or malabsorption occur. Invasion by extension occurs along blood vessels, nerves, lymphatics, and fascial planes to other areas and organs. Invasion into blood vessels can cause thrombosis and visceral infarction.

Other causes of intestinal inflammation include IBD (such as lymphocytic-plasmacytic enteritis, eosinophilic gastroenterocolitis, granulomatous enteritis), antibiotic-responsive enteropathy (which used to be called *intestinal bacterial overgrowth*), and fungal infections (such as *Histoplasma capsulatum* and *Pythium* spp.). In this text, IBD refers to idiopathic inflammation of the intestines, but in veterinary medicine today, there is no clear consensus as to exactly what IBD refers to. IBD is classified according to the predominant inflammatory cell present and the area of intestine affected. The most common form is lymphocytic-plasmacytic enteritis. *Lymphangiectasia is* the dilation of lymph vessels in the intestine and is a major cause of *protein-losing enteropathy*. Laparotomy findings in animals with lymphangiectasia may include thickened small intestine, lipogranulomas in or on the wall of the intestine, dilated lymphatics, lymphadenomegaly, and/or adhesions. Gastroduodenoscopy may be as or more sensitive than laparotomy in diagnosing lymphangiectasia. Intestinal biopsy is important in the diagnosis of IBD, but IBD is not just a histologic diagnosis. IBD is a diagnosis of exclusion, meaning that all other causes of intestinal disease (e.g., parasites, diet, bacteria, cancer, fungi, exocrine pancreatic insufficiency) and other causes of diarrhea (e.g., hyperthyroidism, feline infectious peritonitis, hypoadrenocorticism) must be eliminated. Refer to a medicine text for a discussion of these conditions.

DIAGNOSIS

Clinical Presentation

Signalment

Pythiosis most commonly occurs in large-breed male dogs living in the southern Gulf States, but it has been reported from California to the east coast to the Midwest. The disease is rare in cats but may cause ulcerative gastroenteritis. Young dogs (1–3 years of age) are most frequently affected.

History

Diarrhea and weight loss are the most common complaints. Clinical signs include diarrhea, vomiting, anorexia, depression, and/or progressive weight loss. Intestinal ulceration and necrosis may cause bloody diarrhea.

Physical Examination Findings

Animals with advanced disease are frequently thin. An abdominal mass or marked regional intestinal thickening may be detected on abdominal palpation or digital rectal examination. Sometimes there may be fistulae near the rectum, mimicking perianal fistulae.

Diagnostic Imaging

An abdominal mass, thickened intestine, and/or signs of partial obstruction may be visualized on survey radiographs; however, severe weight loss and loss of abdominal fat may cause poor radiographic contrast. Contrast radiography may delineate thickened, stenotic areas. Intestinal thickening and abnormal wall layering are common findings on ultrasonography. Ultrasonography or fluoroscopy may show a lack of motility in the involved segment.

Endoscopy

A narrowed, nondistensible gastric or intestinal lumen with or without mucosal necrosis and/or ulceration may be seen

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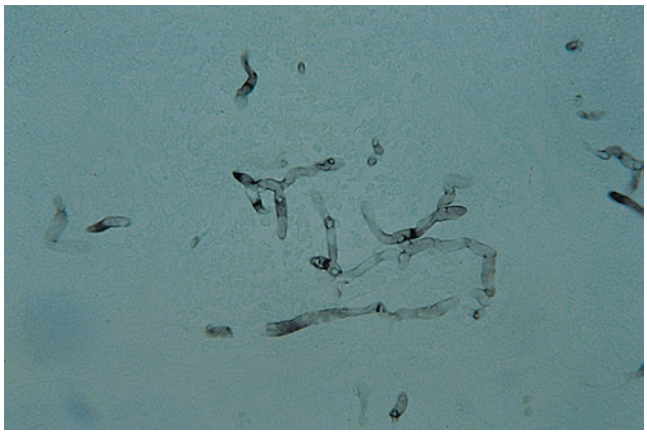


FIG. 18.125 Photomicrograph of *Pythium* hyphae that are broad-branching, nonseptate, or sparsely septate.

endoscopically. Rigid endoscopic biopsies are more likely to be diagnostic than flexible endoscopic mucosal biopsies because the former allows one to obtain a more generous amount of fibrous submucosa, where hyphae are typically found.

Laboratory Findings

A CBC may reveal mild to moderate nonregenerative anemia and mild neutrophilia with or without a left shift. Definitive diagnosis depends on identification of broad-branching, nonseptate, or sparsely septate hyphae in tissues (Fig. 18.125). Hyphae are most easily found in necrotic granulomas of the submucosa and muscularis. Biopsies of enlarged mesenteric lymph nodes generally reveal granulomatous inflammation, often with substantial eosinophilic infiltrates. The intestinal lesion should be cultured to identify the fungal organism more specifically. Serology, which detects anti–*P. insidiosum* antibodies in canine serum, has high sensitivity and specificity for diagnosis and monitoring treatment response.

DIFFERENTIAL DIAGNOSIS

Pythiosis must be differentiated from other causes of partial intestinal obstruction (especially intussusception), neoplasia, other fungal lesions, and regional enteritis. In the perineum, pythiosis may cause lesions similar to perianal fistulae.

MEDICAL MANAGEMENT

Antifungal agents have not proved efficacious; therefore radical surgical excision is the best chance for cure. However, the extensive nature of many of the lesions limits complete resection. A vaccine has resulted in some dogs (approximately one-third) having prolonged remissions.

SURGICAL TREATMENT

Preoperative Management

Fluid, electrolyte, and acid-base abnormalities should be corrected before surgery. Prophylactic antibiotics should be given based on the recommendations provided on p. 435.

Anesthesia

General anesthetic recommendations for compromised patients undergoing GI surgery are provided on p. 435 and Table 18.4.



FIG. 18.126 Multiple small, firm masses causing partial intestinal obstruction associated with pythiosis in a dog.

See Table 19.1 for anesthetic recommendation for stable patients needing abdominal surgery.

Surgical Anatomy

The surgical anatomy of the small intestine is discussed on p. 436.

Positioning

The animal should be positioned in dorsal recumbency for exploratory celiotomy. The ventral abdomen and caudal thorax should be clipped and prepared for aseptic surgery.

SURGICAL TECHNIQUE

Expose the abdomen from the xiphoid process to the pubis. Explore the abdomen; the intestinal lesion may be extensive or multicentric, and the mesenteric lymph nodes are generally enlarged. Intestinal lesions are firm, granulomatous masses with mural thickening (Fig. 18.126). *Biopsy lymph nodes and any other abnormal tissues. Resect the entire intestinal lesion with 4 to 8 cm of grossly normal intestine surrounding it, because fungal hyphae have been found to extend several centimeters into normal-appearing tissue. Reappose intestinal ends with an end-to-end anastomosis (see p. 440).*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Instruments for enterotomy or intestinal resection and anastomosis are discussed on p. 449. Polydioxanone, poliglecaprone 25, polyglyconate, and glycomer 631 (3-0 or 4-0) are the preferred suture materials for these procedures. Nonabsorbable suture (i.e., nylon, polybutester, or polypropylene) generally should be avoided in infected sites. Chromic gut suture should be avoided when infection is present, in young animals, and in those that are debilitated.

POSTOPERATIVE CARE AND ASSESSMENT

Fluid, electrolyte, and acid-base abnormalities should be monitored and corrected after surgery. Analgesics should be provided as needed (see Table 13.2). Therapeutic antibiotics should be given if the animal has evidence of peritonitis, if abdominal contamination occurs during surgery, or if severe debilitation is present. Nutritional support via an enteral feeding tube or parenteral hyperalimentation may be necessary postoperatively if the patient is debilitated or unlikely to eat (see Chapter 10).

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Adjuvant antifungal therapy (e.g., itraconazole, voriconazole, posaconazole, liposomal amphotericin-B, terbinafine) has been tried, but the results have not been encouraging. Anecdotally, vaccines made from fungal cultures have been beneficial as adjuvant therapy. Leakage, dehiscence, peritonitis, stenosis, and SBS are possible complications of surgery. Signs of pythiosis may recur if tissue margins are not free of fungal hyphae.

PROGNOSIS

The prognosis with complete resection is fair to good. Some dogs have recurrence, and others are clinically normal without adjuvant therapy. The prognosis is poor if the disease is advanced and mesenteric vessels have thrombosed or adjacent structures have been invaded. The prognosis for nonresectable lesions is poor to guarded because current antifungal drugs are of questionable efficacy. Marginal excision and medical management with the addition of the pythiosis vaccine was associated with a positive outcome in a single case report in a dog.[43]

INTESTINAL VOLVULUS AND TORSION

DEFINITIONS

Intestinal volvulus is twisting of the intestine that causes obstruction. **Intestinal torsion** is twisting of the intestines about the root of the mesentery. The terms *torsion* and *volvulus* are often used interchangeably. The terms *mesenteric volvulus* and *mesenteric torsion* may also be used for these conditions.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Intestinal volvulus and torsion are uncommon in small animals because they have short mesenteric attachments. The jejunum is most commonly involved. Intestinal volvulus causes mechanical and strangulation obstruction, a medical and surgical emergency (Fig. 18.127). Areas of the intestines that are not fixed in location by attachments to parietal peritoneum or adjacent viscera are suspended by mesentery, which provides greater freedom of movement. Movement and physiologic twisting or turning of suspended intestine occur during physical activity and normal peristalsis. Twisting occurs around the mesenteric axis, or root. If mesenteric attachments fail to prevent excessive rotation, then vascular compromise, tissue ischemia, and luminal obstruction occur. Rotation may exceed 360 degrees in either a clockwise or counterclockwise direction. Predisposing factors in humans include an absence of mesenteric fat, a narrow mesenteric root, excessive mesenteric length, and an increased bowel length.

Twisting compromises the cranial mesenteric artery and all its branches, compromising blood flow to the distal duodenum, jejunum, ileum, cecum, ascending colon, transverse colon, and

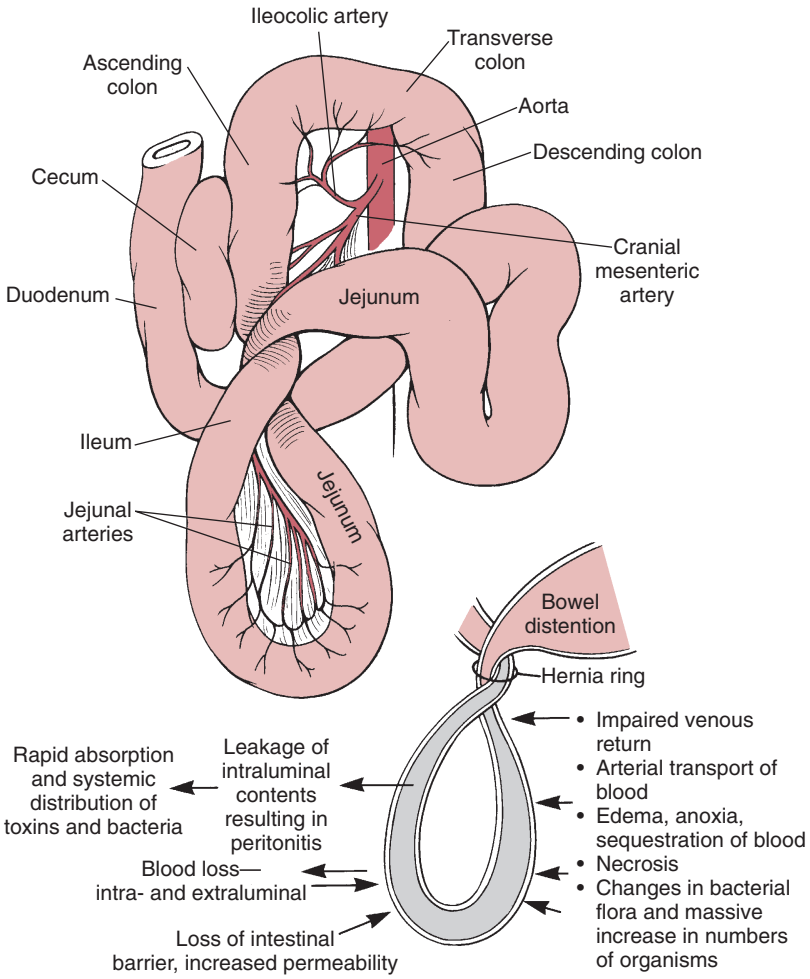


FIG. 18.127 Pathophysiologic events associated with strangulating intestinal obstructions.

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proximal descending colon. The ensuing rapid cascade of vascular obstruction, intestinal anoxia, circulatory shock, endotoxemia, and cardiovascular failure results in death if the condition is not corrected immediately. Mesenteric twisting reduces venous return and arterial perfusion. The arteries and veins may thrombose. Edema and congestion of the intestinal wall lead to anoxia. Blood is lost into both the intestinal lumen and the abdominal cavity. Motility is disrupted, and the normal bacterial flora proliferates rapidly both proximal to and within the strangulated intestine. Small intestinal bacterial concentrations may increase to 10^8 to 10^{11}/mL within 6 hours of strangulation. Endotoxins (primarily from *E. coli*) and exotoxins from *Clostridium* spp. are produced. These toxins and bacteria escape into the abdomen through the damaged mucosal barrier and are absorbed into the systemic circulation. Significant production of inflammatory mediators locally and in other organs such as the liver and lung promote systemic inflammation, leading to systemic compromise and organ dysfunction. Death from strangulation obstruction usually results from a combination of hypovolemic shock, sepsis, and products of tissue necrosis. Reperfusion injury caused by oxygen-derived free radicals after derotation and tissue reoxygenation may be severe and may contribute to mortality.

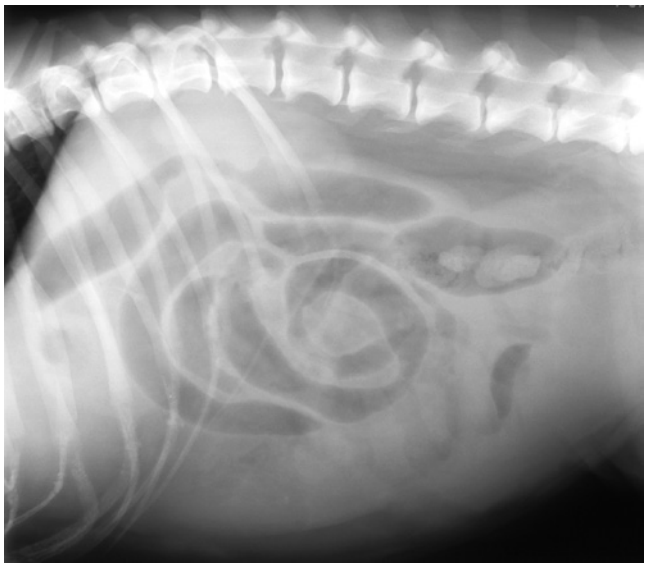


FIG. 18.128 Lateral radiograph of a dog with a mesenteric torsion. Notice the dilated small intestine and decreased visceral detail consistent with effusion.

DIAGNOSIS

Clinical Presentation

Signalment

Male, medium-to-large sporting or working breeds have most commonly been diagnosed with intestinal volvulus and torsion. German shepherds (with pancreatic insufficiency) and English pointers appear to be predisposed to the condition. Young adult dogs (2–4 years of age) are most commonly affected.

History

Vigorous activity, dietary indiscretion, or trauma often precedes volvulus. Other factors that might be associated with intestinal volvulus include recent GI surgery, enteritis, parvoviral infection, parasitism, foreign bodies, intussusception, obstructive masses, exocrine pancreatic insufficiency, and concurrent GDV. Some animals have been ill for several days and then suddenly deteriorate. Others progress from appearing normal to near death in less than 6 hours. Clinical signs range from peracute to acute and are commonly associated with partial obstruction and ischemia. Dogs with colonic volvulus rather than small intestinal volvulus may have a less acute progression of clinical signs. Signs include acute pain, shock (tachycardia, pale to injected mucous membranes, prolonged capillary refill, weak pulses), and mild abdominal enlargement. There is an acute onset of nausea, retching, vomiting, hematochezia, depression, weakness, or recumbency, or all these.

Physical Examination Findings

Affected animals are usually in shock with an acute abdomen. Pain and dilated loops of intestine may be detected by abdominal palpation. Occasionally abdominal fluid is found, the amount varying with the duration of clinical signs.

Diagnostic Imaging

Survey radiographs are often diagnostic, with the entire intestinal tract uniformly distended with gas (Fig. 18.128). The intestines are often severely distended with gas, with the loops lying parallel to each other. These findings, combined with the physical presentation and history, are often highly suggestive of intestinal volvulus. Intestinal fluid, free abdominal fluid, and generalized loss of serosal detail are expected. Definitive diagnosis of intestinal volvulus and torsion is made at surgery or necropsy.

Laboratory Findings

Common laboratory findings are a normal PCV, leukocytosis, hypoproteinemia, hypoalbuminemia, and hypokalemia. Serosanguinous fluid or an exudate may be collected on abdominocentesis. The fluid is initially a transudate from serosal vessel leakage secondary to lymphatic and venous congestion, but once the mucosal barrier becomes incompetent, bacteria (intracellular and extracellular) and degenerative neutrophils will be found.

DIFFERENTIAL DIAGNOSIS

Any systemic or mechanical cause of acute abdomen should be included in the differential diagnosis. Surgical differential diagnoses include GDV, cecocolic volvulus, splenic torsion and rupture, physiologic ileus, mechanical obstruction, abdominal trauma, and peritonitis. Systemic illnesses may include hemorrhagic gastroenteritis, viral enteritis, and pancreatitis.

MEDICAL MANAGEMENT

Shock therapy (fluids and antibiotics, with or without glucocorticoids; discussed later) is essential but not curative. Immediate diagnosis and surgery are necessary if the patient is to survive.

SURGICAL TREATMENT

Preoperative Management

Initial treatment consists of aggressive shock therapy and correction of electrolyte and acid-base abnormalities.[44] Shock doses of fluids (i.e., 90 mL/kg per hour) should be administered rapidly; however, central venous pressure should be monitored if possible to avoid volume overload. As an alternative, hypertonic saline

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or hetastarch may be given (see p. 32). Broad-spectrum antibiotic therapy should be administered. Blood transfusions may be warranted if massive blood loss has occurred. Drugs that block formation of or that scavenge oxygen free radicals (e.g., superoxide dismutase, allopurinol, dimethyl sulfoxide, glucocorticoids, gold compounds) may prove beneficial in the future.

Anesthesia

These patients are extreme anesthetic risks. Anesthetic recommendations for compromised animals undergoing intestinal surgery are provided on p. 435 and Table 18.4.

Surgical Anatomy

The surgical anatomy of the small intestine is provided on p. 436. The cranial mesenteric artery branches to form the caudal pancreaticoduodenal, jejunal, ileocolic, right colic, and middle colic arteries.

Positioning

The patient should be positioned in dorsal recumbency for a ventral midline celiotomy. The caudal thorax and entire abdomen should be prepared for aseptic surgery.

SURGICAL TECHNIQUE

Quickly explore the abdomen to confirm the diagnosis and determine the direction of twisting. The intestine will appear dilated, edematous, and discolored, with the serosal surfaces ranging from red to black. *Decompress the intestine if necessary to allow derotation and reposition the intestines. Allow the intestine to reperfuse and stabilize while the abdomen is more thoroughly explored. Evaluate intestinal viability and resect devitalized tissue. Thoroughly lavage the abdomen with warm physiologic saline or a balanced electrolyte solution. Perform open peritoneal drainage (see p. 534) if intestinal necrosis and peritonitis are identified.* Some have recommended concurrent right-sided gastropexy, gastrocolopexy, and colopexy of the descending colon when the colonic torsion is discovered.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Suction is useful for decompressing the intestine before derotation and repositioning. Suture material for small intestinal surgery is discussed on p. 449.

POSTOPERATIVE CARE AND ASSESSMENT

Fluid, electrolyte, and acid-base abnormalities should continue to be corrected after surgery. These patients may benefit from nasal oxygen postoperatively, and they should be monitored for signs of continued abdominal discomfort. Analgesics should be used if necessary (see Table 13.2). Continuation of perioperative antibiotics is reasonable, particularly if full-thickness necrosis of the intestine was present. Nutritional support should be provided by parenteral hyperalimentation if the animal is likely to be anorectic or to continue to vomit.

PROGNOSIS

Vomiting, diarrhea, shock, intestinal necrosis, dehiscence, and peritonitis are common, and the prognosis for patients with small intestinal volvulus is poor. Patients that survive may develop SBS after massive intestinal resection (see p. 450). Most animals that have survived were incidentally diagnosed during celiotomy for another problem, had rotation limited to 180 degrees, and were operated on within a few hours of occurrence. Large intestinal volvulus is associated with a good prognosis and is treated with repositioning and colopexy (see p. 469).[44]

Surgery of the Large Intestine

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Colopexy is surgical fixation of the colon. **Colectomy** is partial or complete resection of the colon, and **typhlectomy** is resection of the cecum. **Colostomy** is surgical creation of an opening between the colon and the surface of the body. **Tenesmus** is straining to defecate, and **dyschezia** is pain or discomfort on defecation. **Hematochezia** is passage of stools that contain red blood, and **melena** is passage of tarry stools (i.e., digested blood).

PREOPERATIVE CONCERNS

Surgery of the large intestine is indicated for lesions that cause obstruction, perforation, colonic inertia, or chronic inflammation. The most common causes of obstruction are tumors, intussusceptions, and granulomatous masses. Foreign bodies that reach the colon are generally expelled with the feces unless the distal colon or rectum is obstructed or the object has sharp points.

Differentiation of large bowel disease from small intestinal disorders is usually based on the history and physical examination (see Table 18.5). Differentiation of the various causes of large intestinal disorders is based on the history, a physical examination, a fecal examination, endoscopy and biopsy, and/or therapeutic trials. Bacterial culture is rarely appropriate. Careful consideration of past and current medical therapy should be considered, as some medications may alter the integrity and function of the colonic mucosa, predisposing to erosion and ulceration. Most patients with large bowel disease do not have significant weight loss except those with histoplasmosis, pythiosis, protothecosis, histiocytic ulcerative colitis, megacolon, or tumors. Affected patients may have diarrhea or be constipated. Tenesmus, dyschezia, fresh fecal blood, and/or fecal mucus may or may not be present. Other clinical signs may include vomiting, anorexia, abdominal enlargement, abdominal pain, fecaliths, abnormal fecal shape, rectal prolapse, depression, and/or poor hair coat (see Table 18.5).

The physical examination findings vary, depending on the disease and its location in the large bowel (see Table 18.5). The colon is usually palpable in the dorsocaudal abdomen, except in obese animals. Feces can often be differentiated from masses (e.g., tumor) by applying gentle pressure; normal feces deform, whereas the feces of animals with constipation or obstipation are hard and dry. Sublumbar lymph node enlargement can sometimes be detected by palpation of the caudal abdomen or occasionally on rectal examination; lymphandenomegaly is suggestive of metastatic neoplasia. Rectal examination should be done in all patients with large bowel disease. Rectal exam evaluates the shape and symmetry of the pelvis and mucosal thickness. It may also detect masses in the pelvic canal, intraluminal masses, and distal strictures. The anus and anal sacs should

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be checked for thickening, enlargement, and pain. The feces should be examined for the presence of blood or mucus and then examined for parasites.

Most animals with large bowel disease have no laboratory abnormalities. Rarely, dehydration, electrolyte changes, anemia, or hypoalbuminemia is seen. Although nonspecific, elevation of the serum alkaline phosphatase, creatinine phosphokinase, lactic dehydrogenase, and serum glutamic oxaloacetic transaminase may be seen in animals with intestinal ischemia.

Survey colonic radiographs rarely contribute in animals with diarrhea but may help in those with megacolon. Withholding food (24 hours) and evacuating the colon improve visualization. Luminal masses may be identified if the colon contains gas. A "coiled spring" appearance when the colon is filled with air (or barium) indicates a possible cecal-colic intussusception. Contrast barium enemas may identify dilatations, constrictions, wall thickening, filling defects, infiltrative disease, extraluminal compression, intraluminal masses, intussusceptions, volvulus, or cecal inversion. However, barium enemas are labor-intensive procedures and have been almost totally replaced by colonoscopy. Ultrasonography is the preferred imaging modality; it gives information about large bowel wall thickness, wall layers, wall symmetry, peristalsis, and echogenicity of intestinal contents. Biopsies may be obtained with ultrasound guidance. CT and MRI are beneficial in some cases, but these diagnostic techniques are often unavailable. Colonoscopy is safe and more sensitive than radiography for diagnosing masses, ulcers, infiltrates, and intussusceptions. It allows for direct visualization of the lumen, fecal/mucosal culture, mucosal cytology, and mucosal biopsy.

If the patient is not rapidly deteriorating, hydration, acid-base, and electrolyte deficits should be corrected before induction of anesthesia. Cross-matched whole blood should be administered when the PCV drops below 20% or the patient is clinically weak from the anemia. Anemic patients should be given whole blood if hypovolemic and packed red blood cells if normovolemic. Clotting factor deficiencies should be corrected with fresh whole blood (see Box 4.1 and Table 4.5) or fresh or fresh-frozen plasma. Administration of hetastarch (5–20 mL/kg per day) should be considered if albumin levels are below 1.5 g/dL (Box 18.46). There is some evidence that blood transfusions may impair intestinal healing and increase susceptibility to intraabdominal sepsis.

The colon contains more bacteria (i.e., more than 10^{10}/g of feces) than the rest of the GI tract. Preoperative colonic emptying and cleansing are indicated to reduce bacterial numbers unless the colon is perforated or obstructed. Feeding an elemental diet that requires no digestion (e.g., glucose, amino acids) reduces colonic bacterial numbers to 10^3/g of feces. If possible, an elemental diet or a low-residue diet of hamburger and white rice should be fed for 2 to 3 days before surgery. Holding animals off food also reduces bacterial numbers in the colon. Food should be withheld 24 hours before surgery, but free access to water should be allowed. Laxatives, cathartics, and warm water enemas should be given 24 hours before surgery. Colon lavage solutions (i.e., Colyte or GoLytely; Box 18.47) more effectively cleanse the colon than enemas; the only contraindication to their use is obstruction. Bisacodyl, a stimulant laxative, may be administered to facilitate colonic evacuation. Although colonic lavage solutions work well, enemas facilitate complete cleansing. A warm water enema should be given the day before surgery, and a 10% povidone-iodine enema should be given 3 hours before surgery. Enemas given any closer to surgery than 3 hours are contraindicated because they liquefy intestinal contents and may add to dissemination of contaminated material during surgery.

NOTE Be careful; enemas can further deteriorate debilitated, anorectic patients and in rare cases can cause colonic perforation. They may be ineffective in cats with megacolon. Never give hypertonic phosphate enemas to small or constipated patients.

ANESTHETIC CONSIDERATIONS

Anesthetic complications may arise because of uncorrected hydration, electrolyte, or acid-base abnormalities. Large masses or visceral displacement may impair circulation and respiration. Nitrous oxide increases the volume of air trapped in hollow viscera and should be avoided in patients with intestinal obstruction. Atropine (0.02–0.04 mg/kg, given subcutaneously, intramuscularly, or IV) or glycopyrrolate (0.005–0.011 mg/kg given subcutaneously, intramuscularly, or IV) may prevent bradycardia induced by visceral manipulation. Water evaporates from exposed abdominal viscera at an increased rate; therefore fluid administration must be increased to replace this loss. Body heat is lost because of vasodilation and visceral exposure, which cause hypothermia, reducing the need for anesthesia. Patients should be kept dry to minimize the effects of hypothermia. Selected protocols for animals in a stable condition undergoing large intestinal surgery are provided in Table 19.1.

ANTIBIOTICS

The risk of infection after colorectal surgery is high. Although controversial, the use of antibiotics with colorectal surgery seems

BOX 18.46 Colloid Administration (Hetastarch [a], Dextrans)

Total Daily Dose:

Dogs: 10–20 mL/kg/day IV
Cats: 10–15 mL/kg/day IV

Surgical Dose:

Dogs: 0.4–0.8 mL/kg/h CRI
Cats: 0.2–0.4 mL/kg/h CRI

[a]The Food and Drug Administration has issued a black box warning on the use of hetastarch in critically ill people, noting that studies have shown increased mortality and/or severe renal injury when hetastarch was used in critically ill adult patients, including patients with sepsis.
CRI, Constant rate infusion.

BOX 18.47 Bowel Preparation for Large Intestinal and Rectal Surgery

Polyethylene Glycol Electrolyte Solution (Colyte or GoLytely)

25–30 mL/kg PO via stomach tube twice the afternoon before the procedure, approximately 4–6 hours apart (repeat early the next morning if necessary), followed by enema the next morning

Bisacodyl (Dulcolax)

Dogs: 5 mg PO q12–24h
Cats: 2.5–5 mg PO q12–24h

PO, Orally.

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BOX 18.48 Prophylactic Antibiotic Use in Animals Undergoing Perineal, Rectal, or Colonic Surgery

Cefmetazole (Zefazone)
15 mg/kg IV; repeat every 1.5–2 hours for 2 or 3 doses[a]

Cefoxitin (Mefoxin)
15–30 mg/kg IV; repeat every 1.5–2 hours for 2 or 3 doses

Cefotetan (Cefotan)
30 mg/kg IV; repeat q8h for 24 hours

Neomycin (Biosol)
15 mg/kg PO q8h

Metronidazole (Flagyl)
10 mg/kg IV or PO q8h

Erythromycin[b]
11–22 mg/kg PO q8–12h

Amikacin (Amiglyde-V)
Dogs: 15–22 mg/kg IV q24h
Cats: 10–14 mg/kg IV q24h

Ampicillin (Omnipen, Principen, Others)
22 mg/kg IV q6–8h

[a]Dose is anecdotal.
[b]Vomiting and diarrhea are common side effects because of the prokinetic activity of the drug.
IV, Intravenous; *PO,* orally.

to reduce morbidity and mortality from infection. Systemic perioperative antibiotics effective against anaerobes and gram-negative aerobes should be given (Box 18.48). Recommended drugs include the second-generation cephalosporins (e.g., cefoxitin), which are given at the time of induction. Third-generation cephalosporins effective against gram-positive and gram-negative aerobes and some anaerobes are available but expensive. Amikacin plus clindamycin can be given intravenously at induction of anesthesia. Aminoglycosides (e.g., neomycin, kanamycin) and metronidazole can be given orally in combination, beginning 24 hours before surgery. Metronidazole is absorbed from the GI tract and is effective against anaerobes. Aminoglycosides are effective only against aerobic bacteria. GI absorption of aminoglycosides is minimal in normal patients but can be substantial if the bowel is eroded or inflamed. The use of such nonabsorbable antibiotics has been linked with the emergence of resistant infections. A combination of oral neomycin and erythromycin can be given, beginning 24 hours before surgery, to rapidly reduce the number of aerobes and anaerobes. Metronidazole combined with first-generation cephalosporins (cefazolin) or aminoglycosides is also useful.

SURGICAL ANATOMY

The cecum, ascending colon, transverse colon, descending colon, and rectum are segments of the large bowel. The ascending colon and cecum are located at the termination of the ileum. In dogs, the cecum is an S-shaped, blind pouch located to the right of the mesenteric root; in cats, it is a short, straight, blind pouch.

BOX 18.49 Approximate Lengths of Colonic Segments

Ascending Colon
Dogs: 3–9 cm
Cats: 1–2 cm

Transverse Colon
Dogs: 6–8 cm
Cats: 2–4 cm

Descending Colon
10–16 cm; varies with size of animal

The cecum is ventral to the right kidney, dorsal to the small intestine, and medial to the descending duodenum. A short antimesenteric vessel helps identify the ascending colon lying to the right of the mesenteric root. The ascending colon communicates with the ileum via the ileocolic orifice and with the cecum via the cecocolic orifice (approximately 1 cm caudal to the ileocolic orifice). The short ascending colon turns from right to left at the right colic flexure (hepatic flexure) and becomes the transverse colon, traveling cranial to the mesenteric root. The colon turns caudally at the left colic flexure (splenic flexure) and becomes the descending colon. The descending colon is the longest segment of colon (Box 18.49). It begins on the left, where it is dorsal to the small intestine, and continues caudally to the pelvic inlet. The large bowel continues through the pelvic canal to the anus as the rectum. The colorectal junction is difficult to identify. Landmarks include the pubic brim, pelvic inlet, seventh lumbar vertebra, and the seromuscular point of penetration of the cranial rectal artery. The mesocolon is the short mesenteric attachment of the colon to the body wall. The layers of the large intestinal wall are the same as the layers of the small intestinal wall (mucosa, submucosa, muscularis, and serosa).

The blood supply to the large bowel is from the ileocolic artery, a branch of the cranial mesenteric artery, and the caudal mesenteric artery. These major branches run parallel to the intestine, giving off short vasa recta vessels, which penetrate the intestinal wall. Branches of the ileocolic and left colic artery anastomose. The ileocolic artery supplies the ileum, cecum, and ascending and transverse colon. It gives rise to the middle colic and right colic arteries. The right colic artery supplies the cecum, the ascending colon, and part of the transverse colon. The middle colic artery supplies part of the transverse colon and half of the descending colon; it anastomoses with the left colic artery, which supplies the distal half of the descending colon. The left colic and cranial rectal arteries originate from the caudal mesenteric artery. The cranial rectal artery primarily supplies the cranial rectum but also sends several vasa recta to a short segment of the terminal colon. The internal iliac artery supplies branches to the rectum via prostatic or vaginal artery branches. Venous drainage essentially mirrors arterial supply. The caudal mesenteric vein is short and enters the portal vein. The vagus and pelvic nerves supply the colon with parasympathetic innervation. Sympathetic innervation is supplied from the paravertebral sympathetic trunk via the sympathetic ganglia.

SURGICAL TECHNIQUES

The surgical principles for large intestinal surgery are similar to those for small intestinal surgery (Box 18.50), but dehiscence of

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BOX 18.50 Principles of Large Intestinal Surgery

- Reduce colonic bacterial numbers by eliminating oral intake, preparing the colon, and giving antibiotics.
- Early diagnosis and good surgical technique prevent most complications.
- Perform surgery as soon as anesthesia permits in patients with perforation, strangulation, or complete obstruction.
- Optimal healing requires a good blood supply, accurate mucosal apposition, and minimal surgical trauma.
- Systemic factors (e.g., hypovolemia, shock, hypoproteinemia, debilitation, infection) may delay healing and increase the risk of dehiscence.
- Dehiscence is more likely with large bowel surgery than with small bowel surgery.
- Use approximating suture patterns: simple interrupted, Gambee, crushing, or simple continuous.
- Engage submucosa in all sutures.
- Select a monofilament, synthetic absorbable suture: polydioxanone, polyglyconate, poliglecaprone 25, or glycomer 631.
- Cover surgical sites with omentum or a serosal patch.
- Replace contaminated instruments and gloves before closing the abdomen.

large bowel incisions is more likely than it is for small bowel incisions. Bowel viability can be difficult to assess, but it is important that necrotic or avascular areas of the colon be removed at surgery and that unnecessary resection be avoided. Because of the short mesocolon, avulsion of the colonic blood supply is less common than avulsion of the mesenteric blood supply. Techniques for assessing bowel viability are presented on p. 436.

Resection and anastomosis may be performed using sutures or staples. The four stapled anastomosis techniques are (1) triangulating end-to-end anastomosis, (2) inverting end-to-end anastomosis, (3) side-to-side or functional end-to-end anastomosis, and (4) end-to-side anastomosis. End-to-end anastomoses (see p. 470) are most commonly performed during colonic anastomosis. Although more expensive, stapled anastomoses show less tissue reaction, more mature fibrous connective tissue, greater tensile strength, fewer mucoceles and necrotic areas, and less luminal stenosis than suture techniques. The staples in the cartridge are bent against the anvil into a B-shape when fired, providing a degree of hemostasis without collapsing the microcirculation. Thick, inflamed, and edematous tissues may prevent proper firing of the stapler by preventing complete penetration and formation of the staples into the B-shape. Dilatation causes thinning of the visceral walls and may result in tissues that are too thin for the staples to be effective. In such cases, a suture anastomosis should be performed. Circular, inverting, end-to-end staplers are often used to anastomose the colon. It is easier to use end-to-end staplers in the colon than in other areas of the GI tract because the instrument can be introduced through the anus rather than a separate enterotomy or gastrotomy incision. The colon of most adult animals can accommodate the size of available staplers. Transanal introduction of the stapler may not be possible in all cats and small dogs because of the small anus and narrow pelvic canal.

Biopsy Techniques

Intestinal biopsy is indicated to establish a diagnosis for intestinal diseases that have not been diagnosed by other means. The large intestine may be biopsied during endoscopy, ultrasonography, or laparotomy. Full-thickness colonic biopsy should be avoided unless absolutely necessary. Colonoscopy is the preferred method for colonic mucosal biopsy; rigid endoscopes routinely allow biopsies with generous amounts of submucosa to be obtained. Flexible endoscopic biopsies also routinely include the mucosa but are not as large as those obtained with rigid scopes. Rigid proctoscopes typically permit diagnostic biopsies of dense, submucosal rectal lesions.

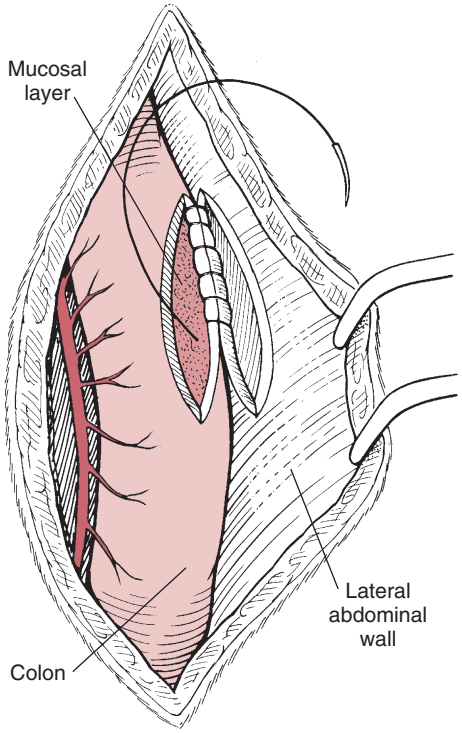


FIG. 18.129 To pexy the colon to the abdominal wall, make a 3- to 5-cm seromuscular incision along the antimesenteric border of the colon. Make a similar incision 2 to 3 cm lateral to the linea alba through the peritoneum and the underlying muscle of the left abdominal wall. Appose the edges of the seromuscular incision to the edges of the abdominal wall incision with two simple continuous suture lines.

Colopexy

Colopexy is performed to create permanent adhesions between the serosal surfaces of the colon and the abdominal wall so as to prevent caudal movement of the colon and rectum (Fig. 18.129). Colopexy is used most often to prevent recurring rectal prolapse. Incisional and nonincisional techniques have been described and are equally effective. Colopexy can be performed laparoscopically using similar techniques. A possible complication is infection resulting from suture penetration of the colonic lumen.

Expose and explore the abdomen. Locate the descending colon and isolate it from the remainder of the abdomen. Pull the descending colon cranially to reduce the prolapse. Verify reduction of the prolapse by having a nonsterile assistant inspect the anus visually and perform a rectal examination. Make a 3- to 5-cm longitudinal incision along the antimesenteric border of the distal descending colon through only the serosal and muscularis layers. Make a similar incision on the left abdominal wall several centimeters (≥2.5 cm) lateral to the linea alba through the peritoneum and underlying

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muscle. Appose each edge of the colonic and abdominal wall incisions with two simple continuous or simple interrupted rows of sutures using 2-0 or 3-0 monofilament, absorbable suture (e.g., polydioxanone, polyglyconate, or poliglecaprone 25), or nonabsorbable suture (nylon, polybutester, or polypropylene) (see Fig. 18.129). Engage the submucosa as each suture is placed. Lavage the surgical site and surround it with omentum before closing the abdomen. As an alternative, scarify an 8- to 10-cm antimesenteric segment of the descending colon by scraping the serosa with a scalpel blade or rubbing it with a gauze sponge. On the left abdominal wall opposite the prepared colon, scarify the peritoneum in the same manner. Preplace and then tie six to eight horizontal mattress sutures between the two scarified surfaces. Roll the colon toward the midline and place a second row of six to eight sutures using 2-0 or 3-0 monofilament, absorbable or nonabsorbable suture. Engage the submucosa with the sutures but do not penetrate the colonic mucosa. Tie the sutures apposing the scarified surfaces.

Colon Resection and Anastomosis

Colectomy and resection are primarily performed to excise colonic masses or to treat megacolon. Other surgical indications include trauma, perforation, intussusception, and cecal inversion. The procedure is much the same as for small intestinal resection and anastomosis (see p. 440) with the exception of vascular ligation.

NOTE Up to 70% of the colon can be resected in animals without adverse side effects; cats tolerate colonic resection better than dogs. Subtotal colectomy (i.e., removal of 90%–95% of the colon) is often done in cats, but should be avoided in dogs. Warn owners that after subtotal colectomy, the cat will probably defecate frequently and have soft stools.

Explore the entire abdomen through a ventral midline celiotomy. Collect nonintestinal specimens before entering the bowel lumen. Carefully isolate the diseased bowel with laparotomy pads or sterile towels. Assess intestinal viability and determine the resection sites. Double ligate all the vasa recta vessels to the diseased segment (Fig. 18.130), but do not ligate the major colic vessels running parallel to the mesenteric border of the bowel unless performing a subtotal colectomy. Gently milk fecal material from the lumen of the isolated bowel. Occlude the lumen at both ends to minimize fecal contamination by having an assistant use a scissor-like grip with the index and middle fingers positioned 4 to 6 cm from the diseased tissue on the colonic wall. Noncrushing intestinal forceps (Doyen) or a Penrose drain tourniquet can also be used to occlude the intestinal lumen. *Place another pair of forceps (either crushing [Carmalt] or noncrushing [Doyen]) across each end of*

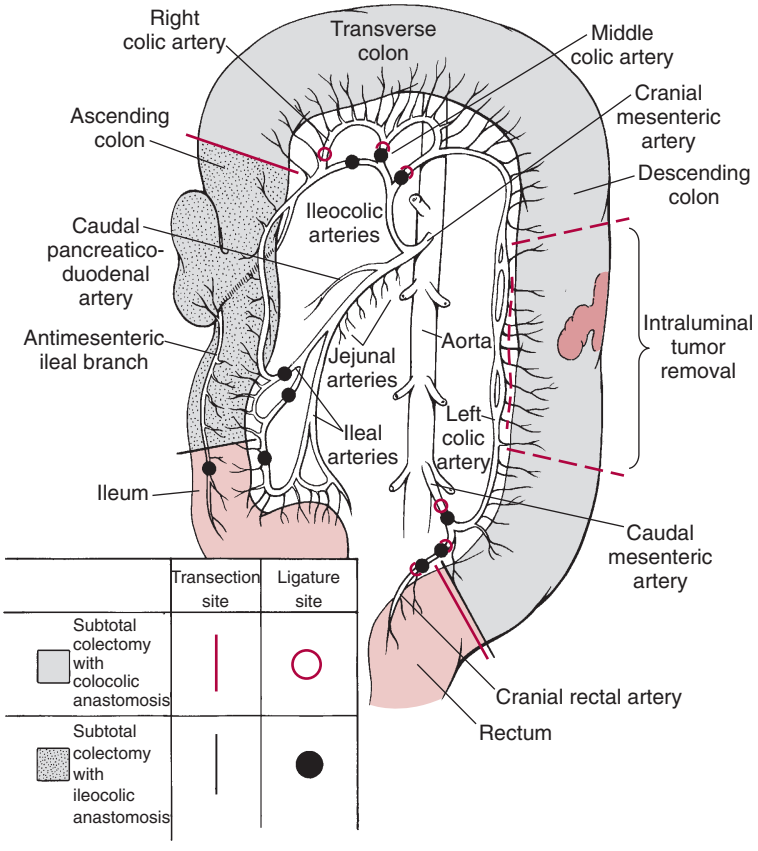


FIG. 18.130 For a partial colectomy *(dashed lines)*, double ligate the vasa recta vessels; preserve the major colonic vessels. Perform a subtotal colectomy, preserving the ileocolic junction by double ligating the paired arteries and veins *(open red circles)*. Perform a subtotal colectomy and ileocolic anastomosis by double ligating the paired arteries and veins *(shaded dark circles)*. Transection sites are identified by the corresponding symbol. *In dogs, do not ligate the cranial rectal artery; ligate the left colic and vasa recta from the cranial rectal artery.*

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the diseased bowel segment. Transect through healthy colon using a scalpel blade or Metzenbaum scissors along the outside of the crushing forceps (see Fig. 18.103). If the lumen sizes are about equal, make the incision perpendicular to the long axis. When the lumen sizes are expected to be unequal, use an oblique incision (45- to 60-degree angle) across the smaller intestinal segment. Angle the incision such that the antimesenteric border is shorter than the mesenteric border. Suction the intestinal ends and remove any debris clinging to the cut edges with a moistened gauze sponge. Trim everting mucosa with Metzenbaum scissors just before beginning the anastomosis or use a modified Gambee suture (see p. 69).

Sutured Anastomoses

Reappose the intestinal ends with a one- or two-layer suture closure. Use 3-0 or 4-0 monofilament, absorbable suture (polydioxanone, polyglyconate, poliglecaprone 25), or nonabsorbable suture (nylon, polypropylene, polybutester) with a taper or tapercut, swaged-on needle. Place simple interrupted sutures through all layers of the wall and position knots extraluminally when a one-layer closure is used. Angle the needle such that slightly more serosa than mucosa is engaged with each bite to help prevent the mucosa from protruding between sutures (see Fig. 18.101). Begin by placing one suture at the mesenteric border and one at the antimesenteric border. If the intestinal ends are of equal diameter, space additional sutures between the first two sutures approximately 2 mm from the edge and 2 to 3 mm apart. Gently appose the tissue edges when tying knots to prevent tissue strangulation and disruption of the blood supply. If minor disparity still exists between the lumen sizes, space sutures around the larger lumen slightly farther apart than the sutures in the intestinal segment with the smaller lumen (see Fig. 18.105). Luminal disparity that cannot be accommodated by the angle of the incisions or suture spacing is usually correctable by resecting a small wedge (1–2 cm long, 1–3 mm wide) from the antimesenteric border of the intestine with the smaller lumen (see Fig. 18.107). This enlarges the stomal perimeter and gives it an oval shape. *After completing the anastomosis, check for leakage by moderately distending the lumen with saline and applying gentle digital pressure. Look for leakage between sutures or through suture holes. Place additional sutures if leakage occurs between sutures. Close the mesenteric defect. Lavage the isolated intestine thoroughly without allowing the fluid to seep into the abdominal cavity. Remove the laparotomy pads, and change gloves and instruments. Lavage the abdomen with sterile, warm saline, then use suction to remove the fluid. Wrap the anastomotic site with omentum or create a serosal patch (see p. 447).*

A two-layer anastomosis is occasionally recommended if there is tension at the anastomotic site. A two-layer anastomosis is performed in the same manner as a one-layer closure except that the serosa and muscularis are apposed in a separate layer. All sutures engage the submucosa. The first layer of simple interrupted sutures is placed to appose the mucosa and submucosa, and the knots are tied within the lumen. The second layer of interrupted sutures apposes the muscularis and serosa, and the knots are positioned extraluminally.

Stapled Anastomoses

The distal colon may be anastomosed to the ileum or jejunum using skin staples, inverting end-to-end, functional end-to-end, or end-to-side stapling techniques. The inverting end-to-end and functional end-to-end techniques are performed in the same way as for small intestinal anastomoses (see p. 440).

For an end-to-side technique, first insert the end-to-end stapling instrument (without anvil) through the open transected end of the colon. Advance the center rod through an antimesenteric stab wound surrounded by a purse-string suture. Tie the suture and place the anvil on the center rod. Introduce the anvil into the lumen of the ileum. Tie the ileal purse-string suture, close the instrument, and fire the staples. Gently rotate and remove the instrument. Inspect the anastomotic site for hemostasis and integrity. Close the transected colon with a transverse stapler. Lavage the surgical sites and place an omental or serosal patch (see p. 447).

Typhlectomy

Typhlectomy, or cecal resection, is performed when the cecum becomes impacted, inverted, perforated, neoplastic, or severely inflamed. *Begin typhlectomy for a noninverted cecum by double ligating the cecal branches of the ileocecal artery in the ileocecal mesenteric attachment (ileocecal fold) (Fig. 18.131A). Dissect the ileocecal fold, freeing the cecum from the ileum and colon (see Fig. 18.131B). Place a clamp across the base of the cecum (see Fig. 18.131C). Milk intestinal contents from the ascending colon and ileum adjacent to the cecocolic orifice and occlude the lumen. Transect the cecum where it joins the ascending colon. Close the defect with simple interrupted sutures. As an alternative, place a transverse or linear cutting stapling instrument across the base of the cecum. Activate the stapler. Transect the cecum before removing the transverse stapling instrument. Lavage, then cover the surgical site with an omental or serosal patch (see p. 447).*

The cecum may be difficult to locate if it is inverted, but its location may be identified by a small indentation where it can be palpated within the colonic lumen (Fig. 18.132). *If possible, manually reduce the cecum before resection. If it cannot be reduced manually, perform an antimesenteric colotomy and exteriorize the cecum (Fig. 18.133). Resect the cecum, and close the cecocolic orifice with sutures or staples as described earlier (Fig. 18.134).*

Bilateral Pubic and Ischial Osteotomy

If additional exposure is needed, a pubic and ischial osteotomy may be performed. *Position the patient in dorsal recumbency with an indwelling urinary catheter to ease urethral identification during dissection. Perform a full exploratory laparotomy via a ventral midline approach. In male dogs, retract the penis laterally. Extend the ventral midline incision along the midline and divide the adductor muscles, taking care to remain on midline. Elevate the adductor muscles subperiosteally from the ventral pelvis, and protect the obturator nerves while exposing approximately two-thirds of the obturator foramina. Incise the prepubic tendon along the cranial aspect of the pubis. Predrill holes on each side of the intended pubic and ischial osteotomy sites and the cranial pubis for reattachment of the prepubic tendon. Use a sagittal saw to perform the pubic and ischial osteotomies, carefully protecting the obturator nerves with a malleable retractor. Elevate the internal obturator muscle from one side of the pelvic floor and retract the ventral pelvic segment laterally. Expose the rectal segment to be excised, taking care to elevate and protect the pelvic nerves on its surface. Isolate the rectal segment for removal and individually ligate penetrating vascular segments. Perform the colorectal resection and anastomosis, and reappose the pubic, ischial, and prepubic tendon with PDS or wire and PDS. Reappose the adductor muscles, subcutis, and skin.*

Colostomy

Colostomies are seldom indicated in animals but may be performed as radical surgical treatment of obstructive colorectal

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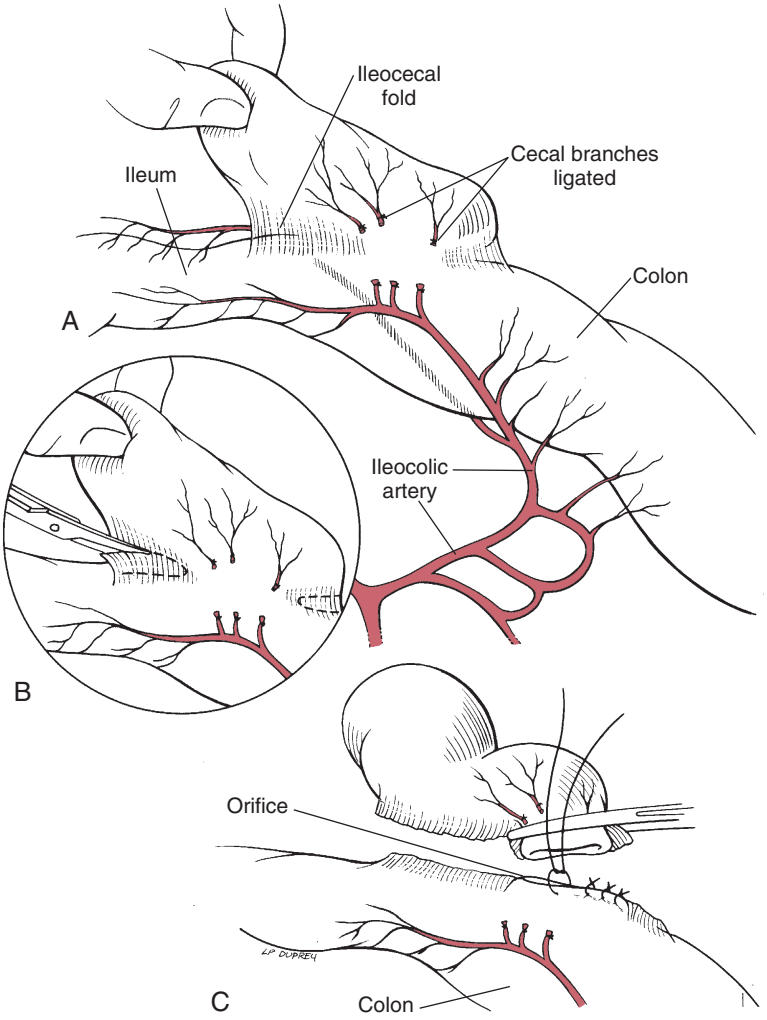


FIG. 18.131 Typhlectomy. (A) Double ligate the cecal branches of the ileocolic vessels. (B) Dissect the ileocecal fold of the mesentery. (C) Place a clamp across the base of the cecum near the cecocolic orifice and transect. Close the colonic defect with simple interrupted sutures.

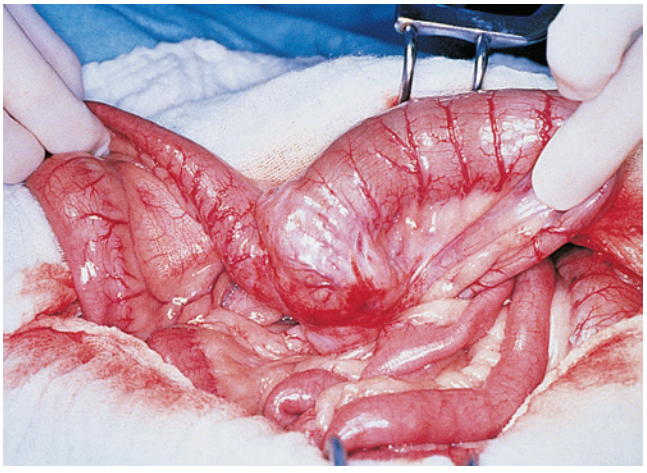


FIG. 18.132 Cecum that is inverted into the colonic lumen.



FIG. 18.133 During typhlectomy, an incision is made on either side of the cecal dimple and the inverted cecum is exteriorized.

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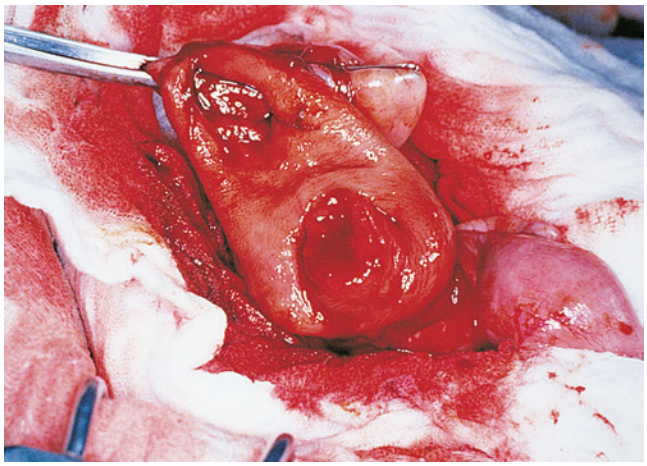


FIG. 18.134 The inverted cecum in Fig. 18.128 has been excised, allowing visualization of the ileal and colonic lumens.

BOX 18.51 Colonic Bacteria

Anaerobes

- *Bacteroides* spp.
- *Bifidobacterium* spp.
- *Lactobacillus* spp.
- *Clostridium* spp.
- *Fusobacterium* spp.
- Anaerobic *Streptococcus* spp.

Aerobes

Gram-negative

- *Escherichia* coli
- *Klebsiella* spp.
- *Proteus* spp.

Gram-positive

- *Staphylococcus* spp.
- *Corynebacterium* spp.
- *Enterococcus* spp.

neoplasia or strictures or for trauma with fecal leakage. However, the fecal incontinence that results from the procedure makes animal management difficult, and client acceptance is often poor. Many of the techniques for end-on or loop colostomies have been developed in animals for use in humans. Loop colostomies are usually temporary; in such cases, the distal colon is intact and allows for closure of the colostomy after resolution of the initial problem. An end-on colostomy is performed after removing the distal colon and temporarily closing the ventral midline incision and rotating the animal into a right lateral oblique recumbent position.

End-on Colostomy

Excise a 2-cm circular segment of skin from a flat area of the left flank. Make a dorsoventrally oriented incision (4 cm) through the lateral abdominal musculature (external abdominal oblique, internal abdominal oblique, and transversus abdominis muscles) and enter the abdominal cavity. Exteriorize the proximal colon by passing it through the flank incision. Suture the circumference of the serosal surface of the colon to the abdominal musculature using 3-0 monofilament, absorbable suture (polydioxanone, polyglyconate, poliglecaprone 25). Complete the stoma by suturing the full-thickness of the transected colon wall to the incised skin edge using 3-0 or 4-0 monofilament, absorbable suture. Then, through the ventral midline incision, perform a colopexy near the stoma to prevent bowel herniation. Attach a colostomy flange and bag to the stoma to collect feces. Replace the colostomy bags and flanges as needed and perform colonic irrigation as desired.

Diverting Loop Colostomy

Perform a diverting loop colostomy after aseptically preparing the left flank. In an area free of skin folds, make a circular incision 4 cm in diameter. Remove the circle of skin. Separate the muscle fibers to allow entry into the abdomen. Grasp the descending colon and exteriorize it through the skin incision. Place a straight plastic loop ostomy rod (90-mm loop ostomy rod) through the colonic mesentery at right angles to the long axis of the bowel. Create subcutaneous pockets dorsal and ventral to the skin incision. Place the ends of the rod in these pockets and suture the rod to the subcutaneous tissue and muscle fascia. Using a simple continuous pattern, suture the seromuscular layers of the colon to the subcutaneous tissue at the border of the skin incision. Create a stoma by making a longitudinal incision into the lumen of the colon and then placing simple continuous sutures to appose the seromuscular layers of the colon to the incised skin edge. Apply an adhesive, 45-mm plastic flange (Surfit Flexible) to the surrounding skin and attach a colostomy bag to the flange. Place a bandage to hold the collection bag against the flank. Close the colostomy when fecal diversion is no longer necessary.

HEALING OF THE LARGE INTESTINE

Colonic healing is similar to that of the small intestine (see p. 448), but delayed. Wound tensile strength lags behind return of strength in the small intestine, and dehiscence is more likely. Optimum healing depends on a good blood supply, accurate mucosal apposition, minimal surgical trauma, and a tension-free closure. A number of factors may delay healing. Collateral circulation to the large intestine is poor compared with the small intestine, and large numbers of anaerobic and aerobic intraluminal bacteria compose up to 10% of the dry fecal weight. Normal colon bacterial counts range from 10^{10} to 10^{11} bacteria per gram of feces. More anaerobes (Box 18.51) than aerobes populate the colon. In addition, high intraluminal pressure develops during passage of a solid fecal bolus. This mechanical stress on the suture line may lead to dehiscence. The risk of dehiscence is high during the first 3 to 4 days because collagen lysis exceeds synthesis. Using antibiotics and placing sutures that do not strangulate tissue may improve healing.

Stapled anastomoses have a higher bursting pressure during the early lag phase of healing and a higher tensile strength after 7 days than hand-sutured anastomoses. Minimal inflammation and a double row of staples increase wound strength. The B-shaped staples provide hemostasis without collapsing the microcirculation, and their alignment ensures equal tension around the circumference of the anastomosis. However, unhealthy tissues should not be stapled.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

The instruments and suture materials for large intestinal surgery are the same as those for small intestinal surgery (see p. 449).

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POSTOPERATIVE CARE AND ASSESSMENT

Postoperative care should be individualized for each patient. Patients should be closely monitored for vomiting or regurgitation during recovery to prevent aspiration pneumonia. Analgesics (e.g., hydromorphone, butorphanol, buprenorphine; see Table 13.2) should be given as needed. Hydration, electrolyte, and acid-base abnormalities should be monitored and corrected. IV fluid therapy should be continued until the animal is eating and drinking normally. Antibiotics may be discontinued 2 to 4 hours after surgery unless peritonitis is present. Small amounts of water may be offered 8 to 12 hours after surgery. If vomiting does not occur, small amounts of food can be given beginning 12 to 24 hours after surgery. A bland, low-fat diet (i/d by Hill's Pet Products or boiled rice, potatoes, and pasta combined with boiled, skinless chicken, yogurt, or low-fat cottage cheese) may be fed three or four times daily. The animal's normal diet can be gradually reintroduced beginning 48 to 72 hours after surgery. Debilitated patients may require enteral or parenteral nutrition (see Chapter 10). Stool softeners or laxatives (or both) should be administered when oral intake begins (Box 18.52). They may be given orally as a supplement (e.g., dioctyl sulfosuccinate-docusate sodium, bisacodyl, lactulose, magnesium salts) or added to food (e.g., psyllium, pumpkin, bran cereal, coarse wheat bran). In some cats, the appetite can be stimulated by oral cyproheptadine (Box 18.53). Mirtazapine is preferred as a feline appetite stimulant over diazepam.

Abdominal palpation and measurement of body temperature should be performed postoperatively to monitor for peritonitis or abscess formation. Depression, high fever, excessive abdominal tenderness, vomiting, and ileus may indicate peritonitis. If peritonitis is suspected, abdominocentesis or a diagnostic peritoneal lavage should be performed (see p. 534). Early ambulation and oral intake should be encouraged to reduce postoperative ileus. The presence or absence of fecal blood (and its color and consistency if present) should be noted after surgery. Tenesmus and hematochezia may be observed.

In cats, subtotal colectomy generally increases the frequency of defecation 30% to 50%. Stools frequently remain loose for days to weeks before becoming more semiformed. After colostomy, the parastomal area must be kept clean and treated as necessary for irritation. Although colonic irrigation is not necessary after colostomy, it reduces fecal production and makes management more practical and cost-effective.

Animals undergoing bilateral pubic and ischial osteotomy for pelvic dissection are expected to become ambulatory within 3 days of surgery but may exhibit signs of lameness prior to becoming fully ambulatory by 7 days after surgery. Towel support when walking on slick floors is recommended due to exposure of and potential compromise of the obturator nerves.

BOX 18.52 Stool Softeners and Laxatives

Dioctyl Sodium Sulfosuccinate or Docusate Sodium (Colace)
Dogs: 50–200 mg PO q8–12h
Cats: 50 mg PO q12–24h

Bisacodyl (Dulcolax)
Dogs: 5 mg PO q8–24h
Cats: 2.5–5 mg PO q12–24h

Lactulose (Chronulac)
Dogs: start with 1 mL/4.5 kg PO q12–24h; titrate to obtain soft, formed feces
Cats: start with 5 mL/cat PO q8h; titrate to obtain soft, formed feces

Psyllium (Metamucil)
Dogs: 1 teaspoon/5–10 kg q12h in food; titrate to obtain desired effect
Cats: 1 teaspoon/5–10 kg q12h in food, titrate to obtain desired effect

Magnesium Hydroxide (Milk of Magnesia) (Cathartic Dose)
Dogs: 15–50 mL/dog PO q24h
Cats: 2–6 mL PO q24h

Canned Pumpkin
Cats: 1–4 tablespoons PO q24h

Coarse Wheat Bran
Dogs: 1–2 tablespoons PO q24h

PO, Orally.

COMPLICATIONS

Hemorrhage and fecal contamination of the abdomen are the most common complications of large intestinal surgery. Other possible complications are shock, leakage, dehiscence, perforation, peritonitis, stenosis, incontinence, and death. Fecal contamination can be prevented by preoperative colon preparation or by manually displacing the contents of the colon away from the resection sites, properly using atraumatic intestinal forceps, and performing copious lavage before closure. The frequency of leakage at the anastomotic site is similar for staple and suture techniques. Leakage may be minimized by placing a serosal patch around the anastomosis (see p. 447). In human beings, long-term use of glucocorticoids is associated with a high incidence of abscess formation after colonic anastomosis and may predispose to dehiscence. Hemorrhage can be prevented with proper ligature placement and careful inspection of the anastomosis.

Postoperative strictures are rare unless inverting or everting suture patterns are used or excessive tension exists at the resection site. Strictures may be managed by incision with an electrosurgical tip or laser ablation. Dilating strictures by balloon or bougienage may also be effective (see p. 382). If these techniques do not alleviate signs of obstruction, surgical resection may be required.

Incontinence is rarely associated with colectomy. Complications associated with colostomy include prolapse of the stoma and skin irritation.

BOX 18.53 Appetite Stimulants in Cats

Mirtazapine (Remeron; Preferred)
1.8–3.7 mg/cat PO q48–72h

Cyproheptadine (Periactin)[a]
2 mg/cat PO q12h

Megestrol Acetate[b]
2.5–5 mg/cat PO for 1–5 days

[a]Give 1 hour before feeding.
[b]Megestrol is a very strong appetite stimulant; however, it can cause diabetes mellitus, pyometra, and mammary problems; therefore it should only be used in extreme cases (e.g., hospice) and with informed owner consent.
PO, Orally.

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SPECIAL AGE CONSIDERATIONS

Young animals more often have rectal prolapse or intussusception as a result of parasite infestation or garbage or foreign body ingestion. They are prone to hypoglycemia and hypothermia during surgery. Neoplasia is more common in older animals.

SPECIFIC DISEASES

NEOPLASIA

DEFINITIONS

Tumors that occur in the colon or rectum are called **colorectal neoplasms. Polyps** are grossly visible protrusions from the mucosal surface of either neoplastic or nonneoplastic cells.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Intestinal tumors occur most often in the rectum or colon of dogs and in the small intestine of cats. Adenomatous polyps and adenocarcinomas are the most common colorectal neoplasms. Other reported tumors include lymphosarcomas, leiomyomas, leiomyosarcomas, plasmacytomas, mast cell tumors, and carcinoids. Intestinal wall tumors usually invade the muscular layer of the intestines, causing obstruction through luminal compromise or interference with peristalsis (or both). Feces, fluid, and/or gas may distend the proximal bowel, which compromises its function. Clinical signs of tenesmus, dyschezia, and hematochezia can be attributed to the presence of a friable luminal mass that bleeds when abraded by the passage of feces. In some animals, tenesmus and dyschezia are caused by partial luminal obstruction by a full-thickness annular mass. Physical examination (especially digital rectal examination), endoscopy, ultrasonography, CT, and MRI are the principal means of assessing abdominal and pelvic structures for neoplasia.

The cause of colorectal polyps is unknown. Most polyps occur in the dog's rectum, near the anorectal junction, although they may occasionally be found in the colon. Most appear dark red or pink, soft, friable, and hemorrhagic. They are usually sessile or slightly pedunculated and may be single or multiple. Single masses may be so large as to suggest a broad-based mass when in fact they have a small stalk. Most polyps are hyperplastic or adenomatous, and epithelial changes do not cross the lamina muscularis; however, some have atypia and are considered carcinoma in situ. Occasionally, rectal lymphoma will present as a large polyp, but these polyps typically have a smooth, relatively normal appearing surface (in distinction to the more common rectal polyps described earlier).

Adenocarcinomas of the colon and rectum are rare in cats; in dogs they are more common, although still relatively rare. In humans, most of these tumors are believed to rise from preexisting adenomatous polyps. Most adenocarcinomas of the large intestine are located in the canine and feline rectum (>50% are midrectum). They may be annular (intramural) or intraluminal. Intraluminal masses may be multiple, pedunculated, nodular, and/or ulcerated. Annular masses typically infiltrate all layers of the intestinal wall, causing circumferential narrowing. These tumors are firm, grayish white, and often ulcerated. Tumors of the large intestine usually grow slowly and spread to adjacent serosal surfaces, mesentery, omentum, and regional lymph nodes by local invasion. Distant metastases may occur to the lymph nodes, lungs, liver, spleen, pancreas, adrenals, and peritoneal surfaces.

Leiomyomas are benign neoplasms of smooth muscle that occur sporadically in the large intestine. They are well encapsulated, circumscribed, and light in color with a smooth, glistening, cut surface. Clinical signs (i.e., tenesmus) may not be seen until the mass reaches an appreciable size. Because the mucosa is usually not involved, melena or hematochezia is uncommon, but some will ulcerate and bleed profusely. They are usually removed by blunt dissection from the colorectal wall via an anal, perineal, or abdominal approach. Recurrence is uncommon, and long-term survival is expected.

Leiomyosarcomas are invasive, malignant, smooth muscle tumors that are usually slow to metastasize. Most reported cecal tumors are leiomyosarcomas. Cecal leiomyosarcomas occur most commonly in old, larger-breed dogs of either sex. Some patients are presented for treatment of cecal rupture and peritonitis, others because of vomiting, diarrhea, GI hemorrhage, polyuria, polydipsia, lethargy, and anorexia. Metastasis to the mesentery, mesenteric lymph nodes, peritoneum, and liver occurs in approximately 50% of those affected. The prognosis for long-term survival is good with early complete resection. Median survival (with or without metastasis) is approximately 2 years.

DIAGNOSIS

Clinical Presentation

Signalment

Colorectal tumors are more common in dogs than cats. The incidence of polyps is equal in males and females, with an increased prevalence suggested in poodles, Airedale terriers, German shepherds, and collies. Colonic carcinomas are two to three times more common in males than females. Mixed-breed dogs, poodles, German shepherds, collies, West Highland white terriers, Airedale terriers, and Lhasa apsos appear to be most commonly affected. Leiomyomas and leiomyosarcomas occur more frequently in medium to large breeds, with no particular breed or sex predilection. Most large intestinal tumors occur in middle-aged and old dogs; however, dogs as young as 2 years of age have been affected. Mean ages for colorectal tumors are 7 years for polyps, 11 years for leiomyomas and leiomyosarcomas, and 8 to 9 years for carcinomas.

History

Most dogs are presented for treatment because the owners noticed blood and mucus in the feces. Common clinical signs include constipation, straining to defecate, passage of blood and mucus with feces, painful defecation, and passage of ribbon-like feces. Vomiting, anorexia, weight loss, depression, septic peritonitis, and/or malabsorption can occur occasionally. Some animals are asymptomatic, and masses are found during a routine physical examination. Tenesmus is often present and can cause prolapse of the mass or rectum. Sometimes the only sign is prolapse of a mass during defecation. Signs involving other organ systems may develop secondary to metastasis and paraneoplastic syndromes.

Physical Examination Findings

Animals with colorectal neoplasia may be thin. Colorectal masses are often identified during abdominal or rectal palpation. More than 60% of anal and rectal canal masses are diagnosed during rectal palpation. Masses may bleed and fragment during palpation, and they may be pedunculated, sessile, nodular, firm, soft, or friable. Prolapse of the mass is often possible, which allows visual inspection. During the rectal examination, it should be determined

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whether the mass is fixed or movable and whether the sublumbar lymph nodes are enlarged.

Diagnostic Imaging

Thoracic and abdominal radiographs should be obtained to evaluate the extent of disease. The rectum and anus are difficult to evaluate on survey radiographs; however, a mass may be identified protruding into the lumen if it is surrounded by intraluminal gas. Sublumbar lymphadenomegaly often indicates metastasis. A barium enema may help delineate a mass, but ultrasound is usually more valuable. In human beings, intrarectal ultrasonography appears to be the most accurate imaging technique for staging rectal cancers. It helps predict the degree of tumor invasion, allowing for better treatment planning; however, its usefulness in animals is uncertain. CT or MRI imaging is helpful to determine tumor extent and invasiveness if radical resection or radiation therapy is anticipated.

Proctoscopy/Colonoscopy

Proctoscopy or colonoscopy should be performed to identify diffuse or multiple lesions and to localize lesions. The size, location, distribution, and multiplicity of lesions should be determined. Examination may reveal a pedunculated or sessile mass, irregular luminal narrowing, and/or ulceration. All lesions should be biopsied, and the submucosa must be included in tissue samples. Presurgical biopsies are useful prognostic tools that can help avoid unnecessary surgery in patients with a poor prognosis. Endoscopic snare polypectomy is often performed in human beings with sessile masses smaller than 15 mm and pedunculated masses smaller than 35 mm; the procedure can occasionally be performed in dogs, but it is hard to obtain all the polyp (and hence many will recur).

Laboratory Findings

The results of CBCs and biochemical profiles are nonspecific for colorectal tumors. In rare cases, anemia and hypoproteinemia are present if chronic bleeding has occurred from an ulcerated mass. Findings consistent with intestinal perforation and peritonitis may be more common in dogs with cecal tumors compared with small intestinal tumors.

DIFFERENTIAL DIAGNOSIS

The differential diagnoses include all other causes of large bowel obstruction or irritation, including intussusception, constipation, obstipation, colitis, perforation, benign stricture, congenital stricture, granulomas, hematomas, or congenital malformation.

MEDICAL MANAGEMENT

Lymphosarcomas may respond to chemotherapy. Rectal tubulopapillary polyps may respond to piroxicam treatment. The response of most other tumors to chemotherapy is unpredictable. Radiation therapy is restricted to masses smaller than 3 cm that are confined to the rectal wall and located in the distal half of the rectum and anal canal. Stenting of the distal colon and rectum has been described for palliation of large intestinal adenocarcinoma in cats.

SURGICAL TREATMENT

Surgical resection is the treatment of choice for intestinal tumors. Unfortunately, many tumors are too advanced for successful resection when diagnosed. Incontinence may occur after resection.

Preoperative Management

Hydration, electrolyte, and acid-base abnormalities should be corrected before surgery. Transfusion should be performed if the patient's PCV is below 20% (see p. 33). If the animal has no obstruction, bacterial numbers may be reduced by evacuating the colon with oral cathartics and enemas and by fasting (see p. 467). Manual evacuation of the rectum is recommended when obstructed. Antibiotics effective against aerobic and anaerobic intestinal bacteria flora should be given (see p. 468).

Anesthesia

General anesthetic recommendations for animals undergoing large intestinal surgery are provided on p. 467 and in Table 19.1.

Surgical Anatomy

The surgical anatomy of the large intestine is presented on p. 468.

Positioning

Tumors of the cecum and colon are approached via a ventral celiotomy with the patient in dorsal recumbency. Rectal tumors may be approached using either a ventral midline celiotomy with pelvic osteotomy, anal eversion, or perineal dissection. Tumors of the cranial to middle rectum are approached with the patient in dorsal recumbency using a ventral celiotomy and pelvic osteotomy. Tumors of the caudal rectum and anal canal are approached with the patient in ventral recumbency using the DePage position and anal eversion or a dorsal (see p. 486) or lateral (see p. 487) perineal approach. For the DePage position, tilting the table, padding the hindquarters, and securing the tail over the back elevate the perineum.

ENDOSCOPIC BIOPSY

The most common mistake made when biopsying submucosal rectal masses is obtaining a sample of mucosa without adequate submucosa. Rigid endoscopic biopsy can reliably obtain large amounts of submucosa, if done correctly. Rigid biopsy forceps that open widely and have a large jaw are preferred. It is often best to remove the proctoscope and guide the biopsy forceps to the lesion digitally. *Once the rigid forceps have been directed to an obviously thickened area (take time and be sure of the placement of the biopsy forceps), forcefully push it into the mass while at the same time closing the jaws of the forceps.* This maneuver typically results in an audible "crunch" sound. When the tissue is retrieved, one should be able to clearly see mucosa and underlying submucosa.

Extension of endoscopic biopsy to polypectomy may be considered for removal of rectal polyps in dogs using a monopolar electrocautery polypectomy snare to excise pedunculated polyps.

SURGICAL TECHNIQUE

Surgical resection is the most common treatment for large intestinal neoplasia. Margins of 4 to 8 cm are recommended for partial colectomy (see p. 470) for malignant tumors. Noninvasive anal or rectal masses are often everted through the anus and excised with limited normal tissue margins. Previously diagnosed benign lesions that cannot be exteriorized may undergo cytoreduction using cautery applied endoscopically using a ball-end

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electrode; the procedure is associated with a risk of rectal perforation, sepsis, and hemorrhage. In human beings, local excision is performed when rectal tumors are limited to the submucosa or muscularis, mobile, 3 cm or smaller, and well or moderately differentiated. Full-thickness resection of the involved colon or rectum is required for large, sessile masses. Tumors invading the serosa or perirectal tissue that are fixed, 3 cm in diameter or larger, poorly differentiated, or atypical should be excised by means of an abdominal incision, rectal pull-through (see p. 485), or perineal resection (see p. 484) with lymph node excision. Abdominal resection may require a pelvic osteotomy to remove a pubic bone flap (see p. 471).

Dilate and retract the anus to allow visualization of the mass. Place stay sutures in the rectal mucosa near the mass to facilitate eversion through the anus. Use an electrosurgical electrode, laser, or scalpel blade to incise the mucosa and submucosa surrounding the mass. Remove the mass and appose the mucosa and submucosa with simple interrupted 4-0 absorbable suture (polydioxanone, polyglyconate, poliglecaprone 25, or glycomer 631). Colostomy may be indicated to allow more aggressive resection or therapy.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Absorbable suture (i.e., polydioxanone, polyglyconate, poliglecaprone 25) is preferred when removing masses from the rectum. Absorbable or nonabsorbable suture may be used for colectomy (see p. 470), but absorbable suture is preferred.

POSTOPERATIVE CARE AND ASSESSMENT

Care and management after surgery should be individualized. Hydration and electrolyte balance are maintained with fluid therapy. Analgesics should be provided as necessary. Therapeutic antibiotics are appropriate when contamination, peritonitis, or severe debilitation is present. Nutritional support (see Chapter 10) may be necessary if the patient is debilitated and refuses to eat. Adjuvant chemotherapy has been recommended for malignant tumors, but its efficacy is unknown.

COMPLICATIONS

Leakage, dehiscence, and peritonitis are possible complications. Tenesmus, dyschezia, and hematochezia are expected after colorectal surgery. Incontinence may be seen in animals after rectal pull-through procedures. Stricture or tumor recurrence may cause signs of intestinal obstruction. One study showed that complications of rectal pull-through are frequent (78%), with the most common being fecal incontinence (56%).[45] Incontinence may be transient (45%) and lasting approximately 2 weeks. The likelihood of permanent incontinence was 13 times greater with complete rectal pull-through surgery compared with a partial procedure. Diarrhea (55%), tenesmus (39%), stricture (27%), constipation (12%), rectal bleeding (14%), dehiscence (10%), and infection (7%) were also reported.

PROGNOSIS

The prognosis is poor for animals with nonresectable tumors because other modes of therapy are ineffective, of questionable value, or unusable because of severe side effects. In some cases, lymphosarcoma responds well to chemotherapy. The prognosis for patients with benign masses is generally good to excellent if excision is complete; however, recurrence is expected when polyp excision is incomplete. Unlike in people, most canine colorectal polyps do not have carcinoma in situ. The prognosis for patients with malignant tumors is fair to guarded, depending on the tumor type, location, stage, and resectability.

Excision is expected to be curative for leiomyoma. Leiomyosarcoma patients live, on average, nearly 2 years before signs of recurrence or metastasis. GIST or GIST-like tumors included in the classification of smooth muscle tumors of the cecum did not alter the recurrence-free period. Complete surgical excision of colorectal adenocarcinomas is curative; however, the prognosis is poor because most cannot be completely excised because of local infiltration, spread to lymph nodes, and the pelvic canal location. Dogs with colorectal adenocarcinomas are usually euthanized because of failure to control dyschezia and hematochezia. Dogs with single, pedunculated, polypoid masses typically survive longer than those with nodular ones. Those with annular masses that cause stricture survived for only a few months. Adjuvant chemotherapy or radiation therapy (or both) is suggested for patients with adenocarcinoma and leiomyosarcoma, but the efficacy of this treatment is unproven.

Cats with colonic adenocarcinoma treated with subtotal colectomy may benefit from adjuvant therapy with carboplatin for a potential median survival time of 269 days.[46] In a 2014 study of dogs with rectal masses, benign tumors treated with rectal pull-through surgery had an overall survival of 1150 days; with malignant tumors the survival was 726 days.[45] Adenocarcinoma was the tumor most commonly treated with rectal pull-through, and these dogs had a median survival time of 696 days (1006 days if the carcinoma was classified as in situ). In another study, dogs with rectal lymphosarcoma treated with surgery and chemotherapy had a median survival time of 2352 days.[47]

COLITIS

DEFINITIONS

Colitis is inflammation of the colon caused by a variety of organisms, diets, and syndromes. **IBD** is idiopathic inflammation of the bowel. Although the terms *colitis, acute colitis, chronic colitis, ulcerative colitis,* and *IBD* often are used interchangeably, they are different.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The underlying causes of acute infectious and inflammatory conditions of the large intestine are often not diagnosed because they are self-limiting. However, with chronic diseases, the cause must often be determined to resolve the condition. A variety of factors may cause colitis, including bacteria, fungi, parasites, and diet. The mucosa may be infiltrated by various inflammatory cells (lymphocytes, plasma cells, eosinophils, neutrophils, or macrophages). Lymphocytic-plasmacytic colitis is the most common form of IBD of the feline colon; IBD is less common in the canine colon. A rare cause of colitis is the late effects of pelvic irradiation. Giving smaller doses per fraction and avoiding systemic radiation potentiators minimize radiation effects on the colon.

Colonic inflammation disrupts the normal secretory and absorptive functions of the colon such that net secretion is increased

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and absorption of sodium, chloride, and water is diminished. Colonic motility patterns change so that segmental contractions, which provide resistance to the flow of feces, are reduced, whereas peristaltic contractions are normal or diminished. The colon is usually hypomotile, and diarrhea results from diminished resistance to flow, accelerated transit, increased secretion of water and electrolytes, and accentuated reflux urge to defecate as a result of mucosal irritation. Mucosal ulceration and smooth muscle spasms may be present, and microbial populations are altered.

DIAGNOSIS

Clinical Presentation

Signalment

Boxers and French bulldogs are predisposed to histiocytic ulcerative colitis. Infiltrative fungal disease is most often found in young and middle-aged dogs.

History

Patients are usually presented for treatment with a history of constant or intermittent large intestinal diarrhea. Failure to lose weight or condition despite chronic diarrhea is the classic finding suggesting large bowel disease. They sometimes void small amounts frequently, have a sense of urgency to defecate, or have accidents in the house (or all three). Hematochezia, fecal mucus, and tenesmus are common but inconsistent findings. Other less common signs include vomiting, depression, weight loss (rare except for fungal infections and neoplasia), dyschezia, and constipation. The animal's diet should be investigated.

Physical Examination Findings

The physical examination findings are often normal. Rectal examination findings may be normal or abnormal (e.g., painful, roughened mucosa).

Diagnostic Imaging

Survey radiographs, contrast studies, and ultrasonography rarely show intestinal wall thickening but are seldom indicated in these patients.

Laboratory Findings

A positive result may be seen on fecal examination for parasites (e.g., whipworms, *Tritrichomonas*). CBCs and serum biochemical profiles are usually normal. Hemograms may reflect chronic inflammation, stress, or anemia. Hypoalbuminemia may be seen in some patients with severe infiltrative disease (e.g., fungal or neoplastic colitis). Colonoscopy with mucosal biopsy provides a definitive diagnosis for fungal infections and neoplasia but is not helpful for dietary and bacterial causes (which are the more common).

DIFFERENTIAL DIAGNOSIS

Food allergy or intolerance, neoplasia, whipworms, *Tritrichomonas*, histoplasmosis, prototheocosis, pythiosis, clostridial colitis, FIV, FeLV, and ileocolic or cecocolic intussusception are important differential diagnoses.

MEDICAL MANAGEMENT

Therapy for acute colitis initially is symptomatic. Food should be withheld for 24 to 48 hours, and then a bland, easily digested, low-fat, and nonallergenic diet should be introduced (e.g., i/d [Hill's Pet Products], or homemade rice, potatoes, and lean meat, cottage cheese, or eggs). Anthelmintics are appropriate if parasites are present or suspected. Clostridial colitis is reasonably common and typically responds promptly to tylosin or amoxicillin therapy. Motility modifiers are rarely used on a short-term basis.

Patients with chronic colonic disease that have normal serum albumin concentrations and have not lost weight may first be given therapeutic trials of anthelmintics, hypoallergenic diets, fiber-supplemented diets, and/or antibiotics effective against *Clostridium* spp. (tylosin or amoxicillin). Glucocorticoids and antimetabolites (azathioprine, chlorambucil; Box 18.54) are typically only given if histopathology reveals severe idiopathic IBD. NSAIDs (sulfasalazine, mesalamine, olsalazine) are typically given after biopsy but can be given without a histologic diagnosis. They can have adverse side effects; consultation of a medical text is recommended. Colonic resection is rarely needed or considered in dogs with colonic inflammatory disease. However, partial or complete colectomy may rarely be required in very severe cases (e.g., subtotal mucosal sloughing).

BOX 18.54 Antiinflammatory Drugs Sometimes Used for Medical Management of Colitis

Prednisolone
1.1–2.2 mg/kg PO q24h

Azathioprine (Imuran)
Dogs: 2.2 mg/kg PO q48h
Cats: Do not administer

Chlorambucil (Leukeran)
Cats: <3 kg: 1 mg PO twice a week
Cats: >3 kg: 2 mg PO twice a week

Sulfasalazine (Azulfidine)
Dogs: 25 mg/kg PO q12h
Cats: 10–20 mg/kg PO q12h

Mesalamine (Asacol, Pentasa, Rowasa, and Others)
5–10 mg/kg PO q8–12h[a]

Olsalazine (Dipentum)
5–10 mg/kg PO q8–12h[a]

[a]Doses are anecdotal.
PO, Orally.

SURGICAL TREATMENT

Biopsy and colectomy may be indicated in affected animals.

Preoperative Management

Preoperative care should be provided as discussed on p. 466. Fluids and transfusions should be given before surgery if indicated. Prophylactic antibiotics should be given before surgery. If surgery is indicated, antibiotics effective against colonic aerobes and anaerobes should be given (see p. 468).

Anesthesia

General anesthetic recommendations for large intestinal surgery are provided on p. 467 and in Table 19.1.

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Surgical Anatomy

The surgical anatomy of the large intestine is described on p. 468.

Positioning

If colectomy is to be performed, patients should be positioned in dorsal recumbency for a ventral midline celiotomy. The entire ventral abdomen should be clipped and prepared for aseptic surgery.

COLONOSCOPIC BIOPSY

Biopsy samples should be obtained by colonoscopy. The techniques for large intestinal biopsy with a flexible scope are similar to those for the small intestine (see pp. 437 and 469). Rigid colonoscopic biopsy provides a better tissue sample, and care must be taken to not perforate the colon. *Deflate the colon and move the scope back and forth to throw up folds of mucosa. Partially open the rigid biopsy forceps and advance them to gently grasp a fold of mucosa, such that the tips of the biopsy cups (not the entire cup) are filled with mucosa. Before closing the forceps completely and cutting off the tissue, gently move the tip back and forth. If just the mucosa moves, then the forceps has been properly placed around a fold of mucosa; completely close the forceps and retrieve the biopsy. However, if the colonic wall moves, then you have grasped too deep a bite and must let go lest you perforate the colon. Then, without reinflating the colon, withdraw the endoscope a few centimeters and repeat the process. The exception is when biopsying dense, submucosal lesions; in that case, do not forcefully but very carefully push the biopsy forceps into the lesion while closing the jaws so as to obtain submucosa in addition to mucosa.* If there is any doubt, it is better to take a conservative biopsy at first and then gradually increase the force used to push into the mass if the first biopsy was inadequate. In general, one should obtain six to eight biopsies of the colon, but only two to three are needed of dense, focal, submucosal lesions.

SURGICAL TECHNIQUE

Surgical biopsy may be required to establish the appropriate medical therapy; however, colonic biopsy during exploratory laparotomy is seldom indicated. Colectomy is described on p. 470.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

See p. 449 for instruments for intestinal surgery. Polydioxanone or polyglyconate suture (3-0 or 4-0) is preferred for colectomy or colonic biopsy; however, nonabsorbable suture (e.g., nylon or polypropylene) may be used in hypoalbuminemic animals. Chromic gut suture should be avoided in young or debilitated animals because the suture may rapidly catabolize and weaken. The use of a biofragmentable anastomotic ring has been evaluated in a small number of cats; however, a larger cohort should be investigated prior to recommending the technique. The ring is inserted into the segments of the large intestine after purse-string suture using a Furness clamp. The unit is closed via digital compression, and a seromuscular oversew may be required to address mild serosal tearing. Serosal tearing was related to the large size of the aborad ring, and use of topical papaverine dilated the segment, decreasing the apparent tearing in subsequent cases.

POSTOPERATIVE CARE AND ASSESSMENT

Severely affected patients may benefit from enteral or parenteral nutritional support after surgery. Analgesics, antibiotics, and fluids should be given as necessary. After surgery, patients should be monitored for evidence of peritonitis (see pp. 449 and 527).

COMPLICATIONS

Colonic perforation is a rare complication of colonoscopic or ultrasonographic biopsy, and leakage may occur from colotomy biopsy or colectomy sites. Strictures may occur after partial colectomy.

PROGNOSIS

Signs of acute colitis are often alleviated by a 24- to 36-hour fast (see the discussion of Medical Management on p. 478). The prognosis for chronic colitis depends on the underlying cause; it is poor for protothecosis, nonresectable pythiosis, and nonresectable malignancy. Lifetime treatment may be needed for some patients with chronic colitis. The prognosis for recovery after surgical biopsy of the large intestines is good if appropriate surgical principles are followed. The patient may require lifetime medical treatment for the underlying cause of colitis.

MEGACOLON

DEFINITIONS

Megacolon is a descriptive term for persistent increased large intestinal diameter and hypomotility associated with severe constipation. A diagnosis of **idiopathic megacolon** is made if mechanical, neurologic, or endocrine causes cannot be identified. **Constipation** is difficult or infrequent defecation with passage of unduly hard, dry fecal material; **obstipation** is extreme constipation (no feces may be passed).

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Megacolon is most often diagnosed in cats (Box 18.55). It is not a specific disease but a clinical sign associated with failure to normally void feces. It may be congenital or acquired, occurring secondary to colonic inertia and outlet obstruction. Causes of colonic inertia may be prolonged distention, neurologic trauma, congenital dysfunction, endocrine disease, metabolic disease, or

BOX 18.55 Megacolon: Key Points

- Most common in cats
- Usually idiopathic; other causes include pelvic obstruction, neurologic, endocrine, behavioral, or congenital
- Diameter of the colon is >1.5 times the length of L7
- Medical management is recommended initially
- Subtotal colectomy is recommended when medical management becomes unsatisfactory
- Cats often handle subtotal colectomy well; dogs tend to not handle subtotal colectomy well
- Postoperatively: frequency of defecation increases, diarrhea improves to soft stool in 4–6 weeks
- Constipation may recur but can usually be managed medically

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behavioral abnormalities, or the condition may be idiopathic. Pelvic fracture malunion, large intestinal strictures or neoplasia, anal atresia or stricture, compressive extraluminal masses, foreign bodies, or improper diet can cause outlet obstruction. Idiopathic megacolon may be associated with other disease processes, such as active colonic inflammation, dysautonomia, and metabolic disorders including severe hypokalemia, marked hypercalcemia, and hypothyroidism. Idiopathic megacolon in cats associated with colonic inertia is thought to be the result of an abnormality of either the intrinsic or extrinsic innervation to the lower large intestine. Cats with idiopathic megacolon are believed to have generalized dysfunction of colonic smooth muscle that involves the activation of smooth muscle myofilaments.

Feces retained in the colon for prolonged periods dehydrate and solidify because of continued water absorption. Fecal concretions are produced that are difficult and painful to eliminate. The fecal mass may become so large and hard that passage through the pelvic canal is impossible. Prolonged, severe colonic distention eventually causes irreversible changes in colonic smooth muscle and nerves, causing inertia. Absorption of bacterial toxins from the retained feces may cause depression, anorexia, and weakness. Vomiting occurs secondary to prolonged obstruction, absorbed toxins, and/or vagal stimulation. Liquid may pass around fecal concretions and cause diarrhea. Blood and mucus from mucosal irritation may be seen in the feces.

Feline longitudinal colonic smooth muscle contraction is calcium-, calmodulin-, and myosin light chain kinase-dependent. Myosin light chain phosphorylation is necessary for the initiation of contraction in feline longitudinal colonic smooth muscle. These facts may be important in feline colonic motility disorders.

Megacolon is uncommon in dogs, but hypertrophic megacolon has been reported in large-breed dogs that were fed bone meal and had minimal exercise. The condition was deemed hypertrophic because of the thickening of both muscle layers of the resected colon.

DIAGNOSIS

Clinical Presentation

Signalment

Idiopathic megacolon is primarily seen in cats but in rare cases occurs in dogs. There is no sex predisposition, but Manx cats may be predisposed. Megacolon that occurs secondary to neurologic, obstructive, or medical disease may be seen in any animal. Middle-aged or older cats are most commonly diagnosed with idiopathic megacolon (range, 1–16 years; mean age, approximately 5–7.5 years).

History

Affected animals are presented for evaluation of constipation or obstipation. They may be depressed and anorexic and have tenesmus, weakness, lethargy, poor hair coat, vomiting, weight loss, and occasionally watery, mucoid, or bloody diarrhea. The clinical signs are often severe and chronic because many clients pay little attention to their pet's elimination habits.

Physical Examination Findings

A lean body condition and poor hair coat may be evident on physical examination. Some animals are depressed and dehydrated. Abdominal palpation reveals a distended colon. Rectal examination reveals hard feces at the pelvic inlet.



FIG. 18.135 Radiograph of a dog with megacolon that occurred as a result of malunion of pelvic fractures and narrowing of the pelvic canal.

Diagnostic Imaging

Abdominal radiographs demonstrate a distended colon impacted with fecal material. Enlargement of colon diameter beyond 1.5 times the length of the body of the seventh lumbar vertebra is considered megacolon. Radiographs should be obtained to rule out obstructive diseases (i.e., pelvic fracture malunions, sacrocaudal spinal trauma or deformities, and intramural or mural colonic or rectoanal obstructive lesions; Fig. 18.135).

Laboratory Findings

Nonspecific changes in the CBC and biochemistry profile may be evident. Histologic examination of colons removed from most cats with idiopathic megacolon usually reveals normal colonic wall ganglion cells.

DIFFERENTIAL DIAGNOSIS

Idiopathic megacolon must be differentiated from congenital, obstructive, neurologic, and systemic causes of megacolon. Causes of obstipation/megacolon are considered idiopathic (>50%) or associated with pelvic canal stenosis (<25%); less common causes are nerve injuries or Manx cat sacral spinal cord deformity. Causes of constipation include drugs (e.g., opiates, anticholinergics, Carafate, barium), severe dehydration, environmental changes, perianal pain (e.g., from perianal fistulae), inappropriate diet, perineal hernia, colorectal masses or strictures, hypercalcemia, hypokalemia, hypothyroidism, and spinal cord or nerve damage.

MEDICAL MANAGEMENT

Constipation is difficult to treat once megacolon develops; however, medical management should typically be attempted before colectomy. Initial management includes correction of hydration, electrolyte, and acid-base abnormalities in severely

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BOX 18.56 Drugs Used for Constipation in Dogs and Cats

Lactulose (Chronulac)

Dogs: Start at 1 mL/4.5 kg PO q8–24h: adjust the dose so that the stools are soft but not watery

Cats: Start at 5 mL/cat PO q8h, adjust the dose so that the stools are soft but not watery

PO, Orally.

affected animals. The colon should be evacuated with stool softeners, enemas, and/or digital evacuation. General anesthesia is typically required for manual evacuation. Because mucosal damage may occur with digital evacuation, antibiotics may be indicated to protect against systemic absorption of bacteria and toxins. To control constipation, long-term use of high-fiber diets, stool softeners, bulk laxatives, and/or enemas may be needed. Osmotic laxatives (e.g., lactulose; ice cream or milk in some cats; Box 18.56) and prokinetic drugs (e.g., cisapride) may help prevent recurrence once the colon has been evacuated by enemas. If recurrent obstipation requires frequent fecal extraction, surgery may be indicated. Some owners find medical therapy intolerable and opt for euthanasia if surgery is not available. However, it must be clear to the owners that dogs do not usually adapt to colectomy as well as cats, and not all cats with a colectomy do well.

SURGICAL TREATMENT

Surgery for megacolon entails removing the entire colon except a short distal segment needed to reestablish intestinal continuity. Megacolon that occurs secondary to pelvic fracture malunion should be treated with subtotal colectomy, pelvic reconstruction, or both. Pelvic reconstruction involves partial pelvectomy and bone repositioning to widen the pelvic canal. Pelvic reconstruction is recommended before irreversible myoneural damage has occurred secondary to chronic colonic distention. Reconstruction should be performed as soon as pelvic narrowing and constipation or obstipation are diagnosed. Signs of obstruction are generally eliminated if the pelvic canal is widened within 6 months of the injury; however, reconstruction alone may not alleviate clinical signs if megacolon is severe. A subtotal colectomy in addition to a pelvectomy may be necessary to alleviate signs in these patients. Care should be taken during pelvectomy to protect adjacent soft tissue structures (i.e., the urethra, rectum, blood vessels, and nerves). After colectomy, the small intestine adapts by increasing stool capacity and water absorption.

Preoperative Management

Preoperative intestinal preparation using multiple enemas to evacuate the large colon is ineffective and unnecessary. Prophylactic antibiotics effective against aerobic and anaerobic colonic bacteria should be given (see p. 466).

Anesthesia

General anesthetic recommendations for large intestinal surgery are given on p. 467 and in Table 19.1.

Surgical Anatomy

The surgical anatomy of the large intestine is described on p. 468.

Positioning

The animal should be positioned in dorsal recumbency with the entire ventral abdomen clipped and prepared for aseptic surgery. The prepared area should extend caudal to the pubic brim.

SURGICAL TECHNIQUE

Subtotal Colectomy

Controversy over whether to remove or preserve the ileocolic junction exists. Removal is thought to allow colonic microorganisms easy access to the small intestine with subsequent malabsorption, as well as to be associated with more severe diarrhea. Preservation is thought to minimize postoperative diarrhea but potentially allow recurrence of constipation. *Explore the abdomen and biopsy abnormal tissues. Isolate the distal small intestine, cecum, and colon from the remainder of the abdomen with several moistened laparotomy pads. Identify resection sites at the distal jejunum or proximal ileum and distal 1 to 2 cm of the colon. Choose sites that will allow apposition without tension. Ligate and transect branches of the ileal artery and vein, ileocolic artery and vein, caudal mesenteric artery and vein, and cranial rectal artery and vein (see Fig. 18.130).*

As an alternative, the ileocolic sphincter can be preserved; however, a tension-free apposition is more difficult to achieve if this is done. Ileocolic anastomosis is technically easier and allows removal of more colon. *If the ileocolic valve is preserved, ligate the right colic, middle colic, and caudal mesenteric vessels. If the ileum is partly or completely removed, also ligate the ileocolic and terminal ileal arcadial vessels.*

NOTE Do not ligate the cranial rectal artery in dogs. Instead, ligate the left colic artery and the vasa recta from the cranial rectal artery.

NOTE Transanal introduction of the stapler may not be possible in all cats because of the small anus and narrow pelvic canal.

Milk feces into the dilated colon, which will be resected. Place intestinal forceps proximal and distal to the planned resection site. Resect the dilated colon at its junction with the small intestine or just distal to the cecum. Perform an end-to-end anastomosis with either a circular stapler or sutures. Correct for luminal size disparity when performing a suture anastomosis by altering the angle of transection (oblique angles on small lumens and perpendicular angles on large lumens), using unequal suture spacing (farther apart on the large lumen), and/or resecting an antimesenteric wedge from the intestine (see p. 471).

If a stapling technique is used, place purse-string sutures at each colonic end before resection. Insert the stapler into the colon transanally or through an antimesenteric incision in the cecum or colon. Lavage the anastomotic site and close the mesenteric defect. Remove the laparotomy pads, lavage the abdomen, and place omentum over the surgical site.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Instruments for large intestinal surgery are discussed on p. 473. Absorbable suture (polydioxanone, poliglecaprone 25, polyglyconate, or glycomer 631) (3-0 or 4-0) is preferred for colectomy; however, nonabsorbable suture (nylon, polybutester, or polypropylene) may be used in debilitated or hypoalbuminemic animals.

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POSTOPERATIVE CARE AND ASSESSMENT

Hydration should be maintained with IV or subcutaneous fluids for 1 to 3 days after surgery with analgesics given as necessary. Prophylactic antibiotics should be continued if gross abdominal contamination occurred or if the patient is extremely debilitated. Patients should be monitored frequently for signs of anastomotic leakage or signs of peritonitis from intraoperative contamination with anaerobic and aerobic bacteria. Food may be offered within 24 hours of surgery, although anorexia may persist for 5 days or longer. Mirtazapine (see Box 18.53) may be used to stimulate eating in some cats. It may be necessary to keep animals on a low-volume, high-caloric diet for 10 to 14 days. Liquid, tarry feces, and tenesmus should be expected immediately after surgery. The character of the feces changes gradually from diarrhea to soft, formed stool in 80% of cats by 6 weeks after surgery. Semiformed stools and, in rare cases, diarrhea persist in some cats. The frequency of defecation usually increases 30% to 50% compared with normal cats; however, most cats are continent. The litter pan should be kept clean to encourage defecation.

COMPLICATIONS

Leakage, dehiscence, peritonitis, ischemic necrosis, stricture, and abscess formation are possible complications of subtotal colectomy. In some cases diarrhea persists, and in other cases constipation recurs. Persistent diarrhea may be the result of antibiotic-responsive diarrhea therapy or hypersecretion in the small intestine, or it may be bile salt and fatty acid mediated. Treatment for persistent diarrhea includes antidiarrheal agents, a low-fat diet, oral antibiotics, and/or bile salt–binding agents. Constipation after subtotal colectomy is often is controlled by dietary management and stool softeners and occasionally by manual extraction. Cats with resistant postoperative constipation may benefit from repeat colectomy, but others are euthanized.

PROGNOSIS

The long-term results of subtotal colectomy for idiopathic megacolon in cats are usually good to excellent. The prognosis is fair to guarded without surgery. Medical management of chronic constipation is possible; however, the frequency of enemas and the need for manual evacuation often become intolerable, prompting euthanasia. Dogs do not do as well with subtotal colectomy as cats; however, dogs with hypertrophic megacolon may respond to subtotal colectomy with preservation of the ileocecocolic junction.

Surgery of the Perineum, Rectum, and Anus

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Rectal resection is removal of a portion of the terminal large intestine. **Rectal pull-through** is resection of the terminal colon or midrectum (or both) using an anal approach, with or without an abdominal approach. **Anal sacculectomy** is removal of one or both of the anal sacs.

PREOPERATIVE CONCERNS

Rectal surgery is usually performed to resect masses or nonfunctional bowel and to repair rectal prolapse, perforation, or fistulae (Box 18.57). Perineal surgery is most often performed to treat perineal hernias, perianal fistulae, anal sac disease, tumors, rectovaginal or rectourethral fistulae, and other traumatic or congenital anomalies (e.g., atresia ani, anovaginal cleft). Scooting, anal licking, constipation, tenesmus, and dyschezia are typical presenting complaints associated with perineal and rectal disease. Fresh blood may be seen in the feces or perianal region. Perianal tumors and perineal fistulae often cause perianal thickening and ulceration, whereas perineal swelling is usually associated with perineal hernias. Pelvic sacroiliac fractures and separations occasionally cause rectal perforation (fewer than 1% of cases). Fresh blood and omentum may be found during rectal examination in animals with rectal laceration or perforation.

Diagnosis of rectal, perianal, or perineal disease is primarily based on the history, clinical signs, physical examination including digital rectal examination, imaging (e.g., radiology, ultrasonography, CT, MRI), endoscopy, and/or histopathologic examination. If impaired anorectal innervation is suspected, myelographic, manometric, and electrodiagnostic evaluations may be required. Many conditions can be diagnosed on physical examination, and a thorough rectal examination is crucial (Box 18.58). Anesthesia may be required for adequate rectal examination of animals that are in pain. Visual inspection of the perineum may reveal unilateral or bilateral swelling, perianal masses, ulceration, fistulae, fecal soiling, or prolapsed mucosa.

The results of CBCs and serum biochemistry tests are nonspecific. Neoplastic masses may be associated with hypercalcemia, anemia, and other paraneoplastic syndromes. Azotemia with or without hyperkalemia may occur with bladder entrapment caused by perineal hernia. Hypercalcemia is common with some anal sac tumors (see p. 490) and may be reduced preoperatively with fluids and diuretics (see p. 491).

BOX 18.57 Possible Indications for Rectal, Anal, or Perineal Surgery

- Diagnostic biopsy
- Anal sac disease
- Colonic obstruction
- Perineal hernia
- Rectal perforation
- Perianal fistulae
- Rectal ischemia
- Rectal prolapse
- Neoplasia
- Fecal incontinence

BOX 18.58 Abnormalities That May Be Noted During Rectal Examination

- Masses
- Strictures
- Perianal thickening
- Anal sac enlargement
- Pain
- Reduced sphincter tone
- Pelvic diaphragm weakness
- Rectal deviation or sacculation
- Sublumbar lymph node enlargement
- Prostatomegaly
- Pelvic canal distortion
- Thickening or irregularity of rectal mucosa

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Rectal and perineal radiographs may confirm the physical examination findings. If possible, the colon and rectum should be evacuated with enemas, laxatives, and/or cathartics before radiographic studies. The rectum should be evaluated for size, location, and masses. Sublumbar lymphadenomegaly suggests metastasis. The prostate may be enlarged and malpositioned. The urinary bladder may be identified within a perineal hernia. A perforated rectum may allow gas into the perineal, intrapelvic, or caudal retroperitoneal soft tissues or into the peritoneal cavity. GI barium studies (enema or oral), urethrograms, and cystograms sometimes help evaluate patients with perineal hernias. Proctoscopy/biopsy helps define rectal disease but should often be combined with colonoscopy because tumors and inflammatory disease may also affect the colon (see p. 479). Samples should be collected for culture, cytology, and histopathology. Normal tissue should be biopsied in addition to thickened folds, masses, strictures, or ulcers. Perforation is an uncommon complication of proctoscopy.

Preoperative patient preparation is similar to that used before colon surgery (see p. 466). Warm compresses should be applied to inflamed or infected areas two or three times a day, and stool softeners should be used if surgery is delayed (see Box 18.52). The locations of fistulae and tumors should be mapped before surgery. Unless the colon is perforated or obstructed, mechanical emptying and cleansing help reduce bacterial numbers. Rectal perforation should be corrected as soon as it is diagnosed. Minimally, the terminal rectum should be evacuated digitally after anesthetic induction but just before surgery in all patients. Some surgeries (e.g., extensive rectal resection) require more complete patient preparation. If possible an elemental diet or a low-residue diet (e.g., hamburger and white rice) should be fed for 2 to 3 days before surgery. Food should be withheld 24 hours before surgery in adult patients (4–8 hours in pediatric patients), but free access to water should be allowed. Bisacodyl, a stimulant laxative, facilitates colonic evacuation (see Box 18.52). Laxatives, cathartics, and warm water enemas should be given 24 hours before surgery (see Box 18.52). Colyte or GoLytely should be administered orally by stomach tube 24 hours and 18 to 20 hours before surgery if the animal is not obstructed (see Box 18.47). Although these colonic lavage solutions work well, enemas facilitate complete cleansing. A warm water enema should be given the day before surgery, and a 10% povidone-iodine enema should be given 3 hours before surgery. Some patients with perianal disease may be in too much pain to allow preoperative enemas. Furthermore, enemas may cause perforation or trauma, further deteriorating debilitated, anorexic patients. Enemas given any closer to surgery than 3 hours are contraindicated because they liquefy intestinal contents and may promote dissemination of contaminated material during surgery. After large bowel evacuation using laxatives, cathartics, and/or enemas, feces may remain in a deviated or dilated rectal area, necessitating manual removal.

After induction of anesthesia, a urinary catheter should be placed to aid intraoperative identification of the urethra, the rectum should be manually cleaned if necessary, and the anal sacs should be expressed. The entire perineum, from dorsal to the tail head and including the ventral tail, should be clipped and aseptically prepared for surgery. If a celiotomy is planned, the ventral abdomen should also be clipped and prepared for aseptic surgery.

ANESTHETIC CONSIDERATIONS

Anesthetic complications may arise from uncorrected hydration, electrolyte, or acid-base abnormalities. If laxatives, cathartics, and/or enemas have been administered preoperatively, the patient may be dehydrated. Fluid administration should replace preoperative as well as intraoperative losses. Recommendations for general anesthesia in the patient undergoing rectal surgery can be found in Table 18.1.

If there are no contraindications (e.g., sepsis, bleeding diatheses, wound infection over epidural site, hypovolemia [for epidurals using local anesthetics]), epidurals (see Chapter 13) may be used in dogs to supplement general anesthesia. Epidural doses should be reduced by at least 25% if cerebrospinal fluid is encountered when using local anesthetics, if the patient is pregnant or obese, or if there are space-occupying vertebral canal lesions. Opioids cause sensory loss without motor block or hypotension. Because bowel preps usually cause some dehydration, an opioid epidural is often a good choice. Local anesthetics in epidurals may cause marked vasodilation and hypotension; therefore dehydration should be corrected before performing the epidural.

ANTIBIOTICS

The risk of infection after colorectal surgery is high. Although controversial, the use of antibiotics in colorectal surgery seems to reduce morbidity and mortality. Systemic perioperative antibiotics effective against anaerobes and gram-negative aerobes should be given (see Box 18.48). Recommended drugs include second-generation cephalosporins (e.g., cefoxitin) given at the time of induction. Third-generation cephalosporins effective against gram-positive and gram-negative aerobes and some anaerobes are available but expensive. Amikacin plus either ampicillin or clindamycin can be given intravenously at induction. Aminoglycosides (i.e., neomycin, kanamycin) and metronidazole can be given orally in combination beginning 24 hours before surgery.

Metronidazole is absorbed from the GI tract and is effective against anaerobes. Aminoglycosides are effective only against aerobic bacteria. GI absorption of aminoglycosides is minimal in normal patients but can be substantial if the bowel is eroded or inflamed. The use of such nonabsorbable antibiotics has been linked with the emergence of resistant infections. A combination of neomycin and erythromycin can be given beginning 24 hours before surgery to rapidly reduce aerobes and anaerobes. Metronidazole combined with first-generation cephalosporins (cefazolin) or an aminoglycosides (see Box 18.48) is usually effective.

SURGICAL ANATOMY

The rectum is the segment of large intestine coursing through the pelvic canal and ending at the anus. The colorectal junction is difficult to identify. Landmarks used to estimate the location of the colorectal junction include the pubic brim, pelvic inlet, seventh lumbar vertebra, and seromuscular penetration point of the cranial rectal artery. The cranial rectum is attached to the sacrum by the mesorectum. The mesorectum does not cover the entire rectum; the terminal rectum is retroperitoneal. At the level of the second caudal vertebra, the mesorectum reflects onto the sides of the pelvis as parietal peritoneum, forming a pararectal fossa on each side. The peritoneal reflection is cranial to the rectococcygeus muscles and contains the autonomic nerve fibers of the pelvic plexus that innervate the rectum. The pelvic plexus is paired, composed of parasympathetic pelvic and sympathetic hypogastric nerves, and lies dorsal to the prostate in males (see

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p. 727). The caudal part of the rectum is supported by the levator ani muscles medially and coccygeus muscles laterally. The external anal sphincter muscle demarcates the caudal limit of the rectum.

The anal canal is a continuation of the rectum to the anus and is only 1 to 2 cm long. It is divided into three zones: the columnar zone, the intermediate zone, and the cutaneous zone. The innermost (columnar) zone has a series of longitudinal mucosal and submucosal ridges called *anal columns* or *pillars*. The pockets between these columns are the anal sinuses, which extend caudally and end in blind pouches under the anocutaneous line. The columnar zone varies from 3 to 25 mm in length. The intermediate zone is usually less than 1 to 2 mm wide but forms a distinct, raised, circumferential ridge called the *anocutaneous line*. Anal glands are found in the columnar and intermediate zones. The outermost (cutaneous) zone has fine hairs but appears as hairless skin. Sebaceous, circumanal, and apocrine sweat glands are found only in the cutaneous zone. The anus is the external opening of the anal canal.

The cranial rectal artery is a branch of the caudal mesenteric artery and is the major blood supply to the rectum. Blood supply from the middle rectal artery (from the internal pudendal branch of the internal iliac artery) and caudal rectal artery (from the middle caudal branch of the median sacral artery or from the internal pudendal branch of the internal iliac artery) varies and is relatively insignificant. To ensure an adequate anastomotic blood supply, the cranial rectal artery in dogs should be preserved unless the intrapelvic rectum is resected. Lymphatics from the anal canal and rectum drain cranially into the medial iliac lymph node.

The internal and external anal sphincter muscles surround the terminal rectum and anal canal to control defecation. The anal sacs lie between these two muscles on each side of the anus (see p. 494). The internal anal sphincter is a caudal thickening of the circular smooth muscle lining the anal canal. It is an involuntary smooth muscle that works with other muscles of defecation to prevent indiscriminate defecation. It is innervated by the parasympathetic branches of the pelvic nerve (S1–S3), which are inhibitory. Motor fibers from the hypogastric nerves are sympathetic to the internal anal sphincter. The external anal sphincter is a large, circumferential band of skeletal (striated) muscle chiefly responsible for fecal continence. It is wider dorsally than ventrally where its fibers decussate and spread to insert on the urethra and bulbospongiosus muscle. The only voluntary nerve supply to the external anal sphincter comes from the caudal rectal branches of the pudendal nerves. The blood supply to the external anal sphincter is from the perineal arteries. See p. 499 for the surgical anatomy of the pelvic diaphragm.

NOTE Fecal incontinence usually occurs if more than 4 cm or the final 1.5 cm of the terminal rectum is resected, if the perineal nerves are damaged, or if more than half the external anal sphincter is damaged.

SURGICAL TECHNIQUES

Rectal Resection

The primary indication for rectal resection is to excise a neoplastic, necrotic, traumatized (e.g., prolapse, fistula, or diverticulum), or strictured segment of rectum. Other indications include congenital anomalies and perforations or lacerations. The rectum may be exposed using a ventral, dorsal, rectal pull-through, lateral, or anal approach. The diseased rectum is resected, and the remaining rectum is reapposed to the rectum, colon, or ileum using the techniques described for colectomy on p. 470.

Ventral Approach

Lesions at the colorectal junction are resected using this approach. A ventral approach to the rectum must be accompanied by a pubic symphysiotomy or pubic osteotomy (Fig. 18.136; also see p. 471) to gain access to the pelvic canal. Pubic symphysiotomy provides more limited exposure than pubic osteotomy. *For pubic*

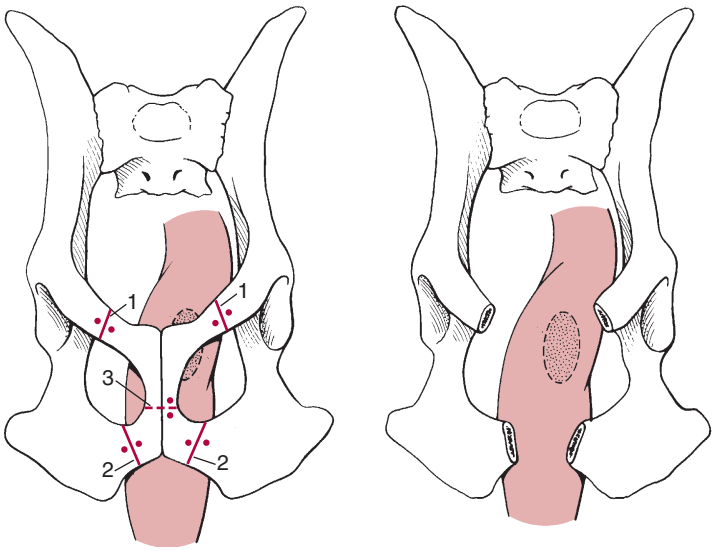


FIG. 18.136 For a ventral rectal approach, extend the ventral celiotomy incision over the symphysis of the pelvis. Incise and elevate the aponeurosis of the adductor and gracilis muscles. Predrill holes on each side of the proposed osteotomy sites. Perform a pubic osteotomy at sites 1 and 2 to expose the entire intrapelvic rectum. Perform an osteotomy at sites 1 and 3 if exposure of the caudal intrapelvic rectum is unnecessary.

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symphysiotomy, incise the entire length of the adductor aponeurosis. Divide the pubis and the ischium on the midline with an osteotome and mallet or an oscillating saw. Separate the pubis and the ischium with a self-retaining retractor (e.g., pediatric Finochietto).

Anal Approach

An anal approach is suitable for excision of small, noninvasive, pedunculated polyps and broad-based rectal masses that can be exteriorized through the anus. Lesions involving the caudal rectum or anal canal can be exteriorized using this approach. Perforations of the terminal rectum may be apposed through this approach, although a lateral approach (discussed later) allows lavage and drainage of contaminated adjacent tissues. The mucocutaneous junction and skin must be resected if they are diseased, but fecal incontinence is a common sequela.

With the patient in ventral recumbency, dilate the anus with three or four stay sutures placed through the mucocutaneous junction (Fig. 18.137A–B). Evert the rectal wall by placing stay sutures (e.g., 3-0 nylon or other monofilament suture) in the rectal mucosa cranial or caudal to the mass or lesion and applying caudal traction. Place additional stay sutures to further retract the mass or lesion if necessary (see Fig. 18.137C). Use electrosurgery, laser, or scalpel incisions to remove masses. Make a partial-thickness or full-thickness incision, depending on malignancy and the need for wide borders. Appose cut edges with simple interrupted sutures (e.g., 3-0 or 4-0 polydioxanone, polyglyconate, poliglecaprone 25, or glycomer 631; see Fig. 18.137D). Remove the stay sutures, and allow the surgical site to retract within the pelvic canal.

Rectal Pull-Through Approach

AP The primary indication for a rectal pull-through is to resect a distal colonic or midrectal lesion not approachable through the abdomen and too large or cranial for an anal approach. When circumferential or near-circumferential lesions are resected using this technique, postoperative stricture is a major concern.

Position the animal in ventral recumbency with the hindquarters elevated. Evert the rectum with stay sutures placed cranial to the mucocutaneous junction (1.5 cm or more if possible; Fig. 18.138A). Using the stay sutures, apply caudal traction to the cranial rectum. Begin a full-thickness, 360-degree incision through the rectum, leaving a 1.5-cm cuff of nondiseased rectal wall attached to the anus if possible. Place three or four stay sutures in the rectal cuff. Mobilize the rectum by bluntly dissecting along the external wall (see Fig. 18.138B). Continue dissection as far cranially as the cranial rectal artery if necessary. If dissection occurs cranial to the second caudal vertebra, the peritoneal cavity will be entered. *Ligate or coagulate rectal vessels as they are encountered. If the lesion is diffuse, split the rectum longitudinally until normal tissue is identified (see Fig. 18.138C). Transect the diseased rectum in stages with*

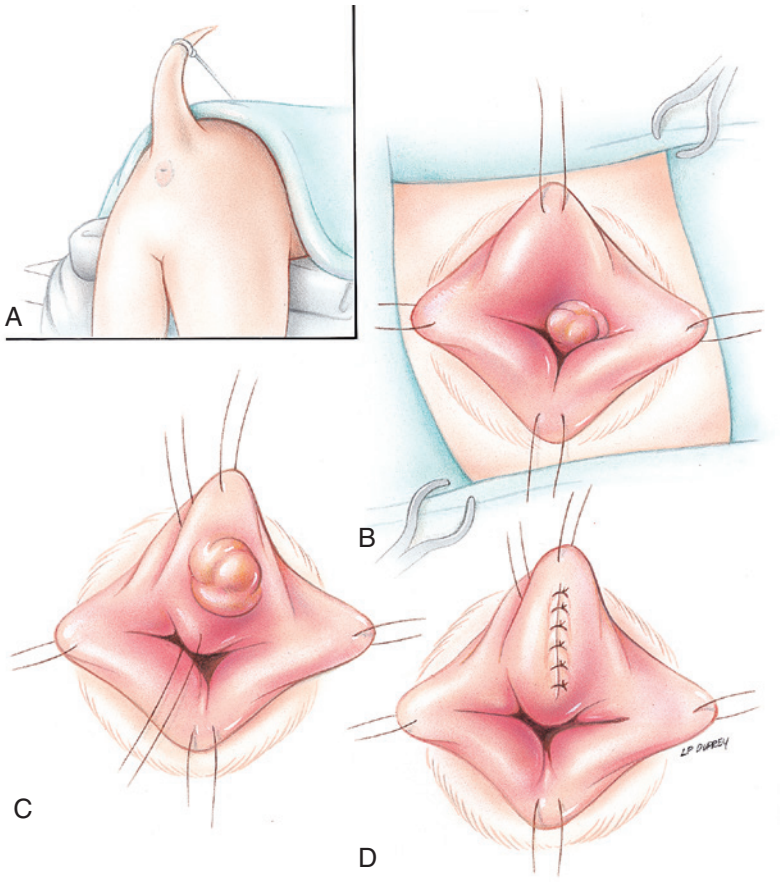


FIG. 18.137 Anal approach to the rectum. (A) Place the patient in a perineal position. (B) Place stay sutures around the anus to dilate it and expose the lesion. (C) Place additional stay sutures near the lesion and apply caudal traction to exteriorize it. (D) Resect the mass and suture the defect.

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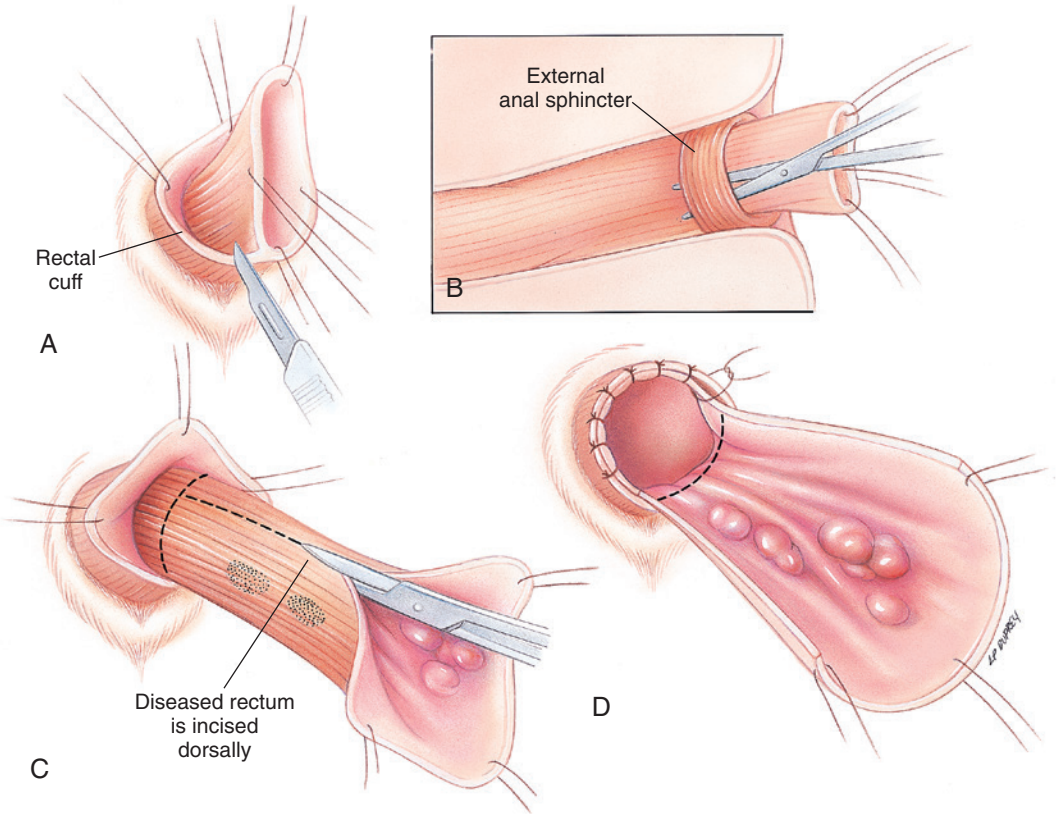


FIG. 18.138 Rectal pull-through procedure. (A) Evert the rectal wall through the anus with stay sutures. Make a full-thickness incision through the rectal wall, preserving a 1.5-cm cuff of distal rectum. (B) Mobilize the rectum by dissecting directly against the rectal wall to separate it from the external anal sphincter and surrounding tissue. (C) Pull the mobilized rectum caudally and incise longitudinally to normal tissue. (D) Appose the cut edge of the normal cranial rectum to the preserved rectal cuff with simple interrupted sutures.

1 to 2 cm of normal tissue at each end. Transect one-fourth to one-third of the circumference, and then appose the cranial end of the rectum to the caudal rectal cuff with simple interrupted sutures (e.g., 3-0 or 4-0 polydioxanone, polyglyconate, poliglecaprone 25, or glycomer 631; see Fig. 18.138D). Continue transecting and apposing until all diseased tissue has been excised.

NOTE Some surgeons prefer a two-layer closure; appose the seromuscular layer first, and then the mucosal-submucosal layer.

AP A Swenson's pull-through procedure is performed when disease extends into the colon. *For this procedure, position the patient in dorsal recumbency so that both a ventral abdominal and an anal approach may be used. Transect the colon proximal to the mass. Oversew the ends of the colon and rectum.* Linear cutting or transverse stapling instruments may be used to reappose the colon and rectum.

Ligate the vessels supplying the distal colon and rectum (see p. 484). Place stay sutures through the end of the remaining colon or ileum and rectum. Grasp the sutures with transanally placed forceps and evert the rectum through the anus. Advance the colon or ileum through the pelvic canal with stay sutures. Resect the lesion and anastomose the end of the colon or ileum to the terminal rectum as described previously. Gently replace the intestine into the pelvic canal.

Expect some dogs to become fecally incontinent following this procedure. Other common complications include self-limiting hematochezia and tenesmus, which should resolve in approximately 2 weeks.

Dorsal Approach

A dorsal approach is used if the lesion involves the caudal or middle rectum and not the anal canal. *Position the patient in ventral recumbency with the pelvis elevated and the tail fixed over the back. Pad the cranial aspect of the hind limbs to prevent pressure on the femoral nerves. Make a curvilinear incision from one ischiatic tuberosity to the other, curving dorsal to the anus (Fig. 18.139A). Incise the subcutaneous fat and perineal fascia. Locate the rectum, external anal sphincter, levator ani, and coccygeus muscles laterally and the rectococcygeus muscles dorsally. Transect the paired rectococcygeus muscles near their origin on the rectal wall or their insertion on the caudal vertebrae (see Fig. 18.139B). Elevate the external anal sphincter and caudal edge of the levator ani to the level of the caudal rectal nerve. For more cranial rectal resections, partly transect the levator ani muscles if necessary. Position a self-retaining retractor (i.e., Gelpi or Weitlaner) to improve visualization if necessary. Gently retract the rectum caudally and mobilize the*

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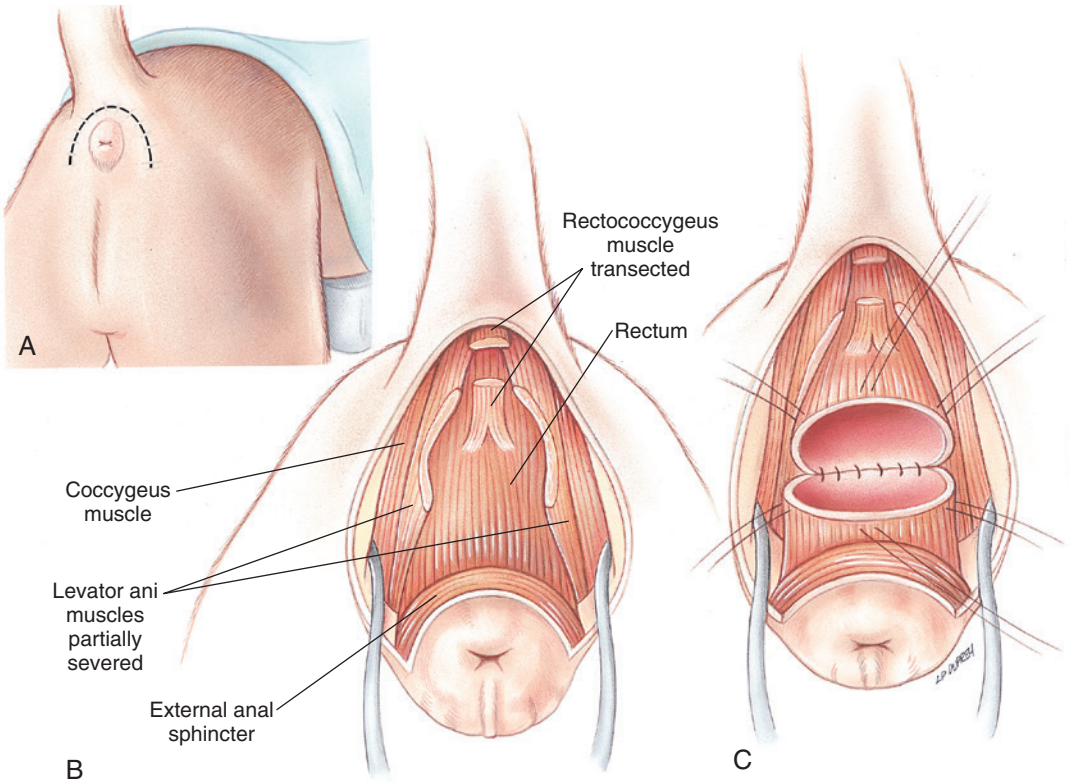


FIG. 18.139 Dorsal rectal approach. (A) Make a curvilinear incision from one ischial tuberosity to the other, curving dorsally to the anus. (B) Identify the rectococcygeus, levator ani, and coccygeus muscles. (C) Transect the rectococcygeus muscle and partly incise the levator ani. Resect the diseased rectum and appose the ends with a suture triangulation technique or end-to-end stapling instrument.

rectum cranially to normal bowel. Repair the lacerated rectum or resect the diseased bowel. Ligate or cauterize vessels to the diseased bowel. Place stay sutures in the cranial bowel before transection. Appose the bowel ends with interrupted appositional sutures (e.g., 3-0 or 4-0 polydioxanone, polyglyconate, poliglecaprone 25, or glycomer 631; see Fig. 18.139C) or with an end-to-end stapling device (see p. 471). Reappose the transected levator ani muscle with appositional cruciate or mattress sutures. Some surgeons reattach the rectococcygeus muscles and external anal sphincter to the rectal wall. *Thoroughly lavage the area and place drains if significant contamination has occurred.* Placing a drain against the anastomotic site may cause dehiscence.

Separately appose the subcutaneous tissue and skin with continuous or interrupted sutures of 3-0 or 4-0 polydioxanone and 3-0 or 4-0 nylon or polypropylene, respectively.

Lateral Approach

The lateral approach limits exposure to one side of the rectum and may be suitable for repairing lacerations or resecting a diverticulum.

Make a curvilinear incision 1 to 3 cm lateral to the anus, beginning dorsal to the tail head and extending ventral to the anus (Fig. 18.140A). Incise the subcutaneous tissues to expose the pelvic diaphragm. Separate the fascia between the external anal sphincter and the levator ani muscle. Preserve the caudal rectal nerve to the external anal sphincter (see Fig. 18.140B). Repair the laceration with a one- or two-layer closure using simple interrupted sutures (e.g., 3-0 or 4-0 polydioxanone, polyglyconate, poliglecaprone 25, or glycomer 631). Thoroughly lavage the area, and place a closed suction drain if soft tissues were contaminated with feces. Resect diverticula with a linear stapling device. Reappose the external anal sphincter and levator ani muscles with interrupted appositional sutures. Place additional sutures between the external anal sphincter and internal obturator muscle if this fascial plane is disrupted. Close the subcutaneous tissues and skin routinely.

Anal Sacculectomy

Anal sacculectomy is performed to remove chronically infected or impacted anal sacs, anal sac fistulae, or neoplasia. Meticulous dissection is required to prevent fecal incontinence by preserving the anal sphincter muscles and nerves. A closed or open technique may be used. The closed technique is preferred because the external anal sphincter muscle is not transected, and the lumen of the anal sac remains closed, preventing contact between secretions and adjacent tissues. See p. 495 for a description of open and closed anal sacculectomy techniques.

NOTE Perform a histopathologic examination to rule out anal sac tumors.

HEALING OF THE RECTUM

Rectal healing is affected by the same factors that affect colonic healing (see p. 473). Optimal healing requires a good blood supply, accurate mucosal apposition, and minimal surgical trauma.

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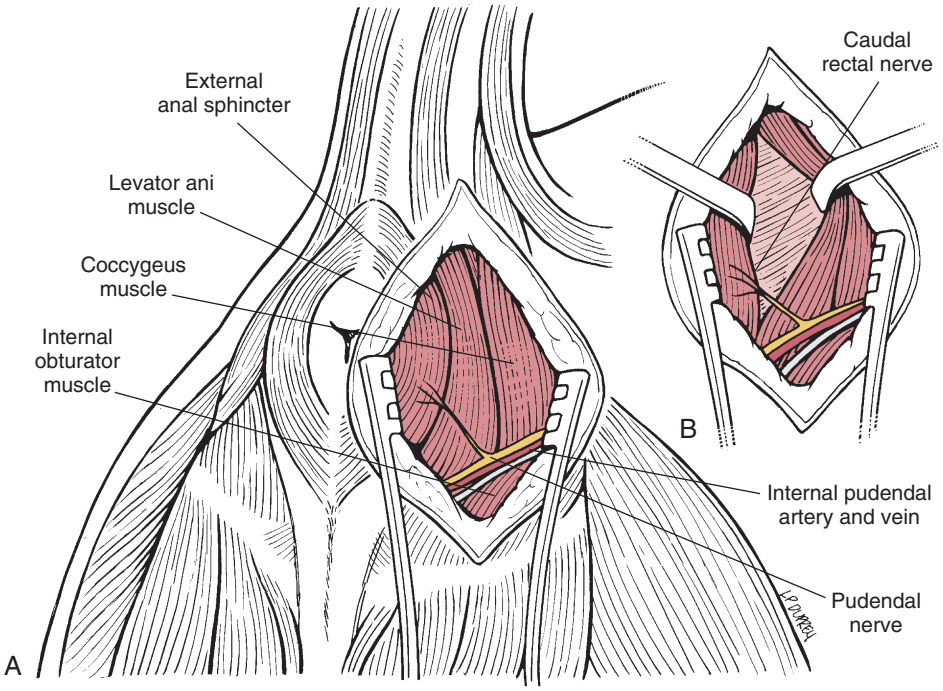


FIG. 18.140 Lateral rectal approach. (A) Make an incision 1 to 3 cm lateral to the anus. Identify the internal pudendal artery and vein and pudendal nerve crossing the internal obturator muscle. (B) Note the caudal rectal nerve to the external anal sphincter. Separate the fascia between the external anal sphincter and levator ani muscles to expose the lateral aspect of the rectum.

Systemic factors that may delay healing and increase the risk of dehiscence include hypovolemia, shock, hypoproteinemia, debilitation, and infection.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

In addition to a general pack, abdominal (i.e., Balfour), perineal (e.g., Gelpi), and pelvic (e.g., Finochietto) retractors are recommended to aid in exposing the surgical field. Doyen, Babcock, and Carmalt forceps may be needed to occlude or retract the intestine. Metzenbaum and iris scissors are indicated for dissection. Closed suction drains are used in contaminated areas. Other special instruments or equipment that may be necessary for rectal or perianal surgery include a probe or groove director, fulguration unit, laser unit, silicone elastomer, and surgical stapling equipment.

For optimal healing, a monofilament, synthetic absorbable suture (e.g., polydioxanone, polyglyconate) and approximating suture patterns (i.e., simple interrupted, Gambee, crushing, or simple continuous) should be used for rectoanal surgery. A monofilament, nonabsorbable suture (e.g., polypropylene, polybutester, or nylon) or absorbable suture material (e.g., polydioxanone, polyglyconate, or poliglecaprone 25) may be used for herniorrhaphy.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative care is individualized for the patient. The animal should be observed closely during recovery for vomiting. Analgesics (see Table 13.2) should be given as necessary. Hydration should be maintained with IV fluids until the patient is eating and drinking normally, and electrolyte and acid-base abnormalities have been corrected. Encouraging early ambulation and eating may minimize ileus. An Elizabethan collar, bucket, or sidebars should be used to protect the surgical site. Purse-string sutures should be removed immediately or within 2 to 3 days after surgery. Prophylactic antibiotics can generally be discontinued 2 to 4 hours postoperatively; however, they should be continued if contamination has occurred (see the previous discussion under Antibiotics on p. 483).

If vomiting does not occur, water should be offered 8 to 12 hours postoperatively. A stool softener should be given when oral intake begins (see Box 18.52) and should be continued for 2 weeks or as needed. After rectal surgery a bland, low-fat food (i/d [Hill's Pet Products] or boiled rice, potatoes, and pasta combined with boiled, skinless chicken, yogurt, or low-fat cottage cheese) should be fed three or four times daily. The animal's normal diet should be gradually reintroduced beginning 48 to 72 hours after surgery. Animals having perineal surgery may resume their normal diet with the first feeding. Debilitated animals may require enteral or parenteral nutrition.

Initially cold compresses are applied to reduce residual bleeding and inflammation; later warm compresses should be applied. Apply compresses two or three times daily for 15 to 20 minutes to minimize postoperative swelling after perianal or perineal surgery. Redness and pain at the incisional site may indicate early infection. Anal sphincter function, continence, perineal swelling, and drainage should be assessed daily. The presence or absence of fecal blood (and its color and consistency if present) should be noted. Increased frequency of defecation may occur after major colorectal resections. Perianal and perineal area surgeries are predisposed to infection because of the high bacterial

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numbers in these areas. Depression, high fever, abdominal tenderness, vomiting, ileus, or perineal inflammation may indicate infection or peritonitis. If peritonitis is suspected, abdominocentesis or diagnostic peritoneal lavage (see p. 534), a chemistry profile, and a CBC should be performed and antibiotic and fluid therapy initiated. If toxic neutrophils, bacteria, or intestinal debris are present in abdominal fluid or lavage solution, the abdomen should be explored. Aggressive treatment of generalized peritonitis by open peritoneal drainage (see p. 534) may be necessary.

COMPLICATIONS

Numerous complications are possible after rectoanal, perianal, and perineal surgery (Box 18.59). Postoperative tenesmus, hematochezia, and fecal incontinence are common. Postoperative tenesmus and hematochezia should resolve in most animals after suture removal or absorption. Incontinence with extensive rectal resection results from loss of rectal afferent nerves or from disruption of the pelvic plexus at the peritoneal reflection. Removal of the distal 1.5 cm of rectum may cause fecal incontinence even if the external anal sphincter is preserved. Fecal incontinence is uncommon when 4 cm or less of the rectum is resected, preserving the terminal rectal cuff. Longer rectal resections (6 cm or longer) disrupt the peritoneal reflection and frequently result in incontinence. Other possible complications are rectal prolapse, perirectal abscesses, dehiscence, and stenosis.

Significant strictures occur more commonly after rectal resection than colonic resection, probably because of excessive tension at the resection site. These strictures can usually be managed by incision with an electrosurgical tip or laser or by balloon dilatation. If these techniques do not alleviate obstruction, surgical resection may be required. Anal stricture is a complication of anal sac disease, perianal fistula, neoplasia, and surgical or nonsurgical trauma. It may be treated by anoplasty; balloon dilatation; incision with electrosurgery, laser, or scalpel; or resection.

Complications associated with anal sacculectomy may be self-limiting (32%) or may affect defecation (14%).[48] In a 2014 study, no dog was permanently incontinent; however, dogs less than 15 kg or those in whom gel was used to distend the anal sac were more likely to have complications including perineal pruritus, bruising, dehiscence, constipation, diarrhea, and perineal soiling.[48] Previous abscessation, recurrent disease, and presurgical treatment with antibiotics did not affect the complication rate.

BOX 18.59 Possible Complications of Perianal and Rectal Surgery

- Infection
- Dehiscence
- Tenesmus
- Rectal prolapse
- Dyschezia
- Hematochezia
- Temporary or permanent incontinence
- Anal stricture
- Flatulence
- Hemorrhage
- Recurrence
- Metastasis
- Nerve damage (pudendal, sciatic, femoral)
- Urethral obstruction
- Stranguria
- Dysuria
- Urinary incontinence
- Bladder atony
- Death

SPECIAL AGE CONSIDERATIONS

Young animals may have a congenital abnormality such as anal atresia or imperforate anus. Neoplasia is more common in older animals.

SPECIFIC DISEASES

ANAL NEOPLASIA

DEFINITION

Perianal glands are modified sebaceous glands. Perianal gland adenomas are also called *circumanal tumors* and *hepatoid tumors;* anal sac tumors are also known as *apocrine gland tumors.*

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The most common perianal tumors are adenomas and carcinomas of the perianal and apocrine glands. Apocrine gland tumors usually involve the anal sacs. Perianal glands are located primarily around the anus and base of the tail; however, they are also found in the thigh, prepuce, and dorsal and ventral midline from the base of the skull to the umbilicus. Perianal tumors may occur at any of these locations. Androgen and estrogen hormone receptors and growth hormone have been identified in both perianal gland adenomas and adenocarcinomas. The most common malignant tumors are perianal gland adenocarcinomas and apocrine gland adenocarcinomas. Other common tumors are listed in Box 18.60.

Perianal adenomas are the most common canine perianal tumors (80%). They are the third most frequent tumor in male dogs. They occur 12 times more often in intact males than in intact females and are more common in ovariohysterectomized females than in intact females. They are hormone dependent and usually diminish in size after castration. They may be single or multiple and are usually small, raised, firm, and well circumscribed; however, some are large and ulcerated. Many dogs with perianal adenomas also have testicular interstitial cell tumors (see p. 773).

Perianal gland adenocarcinomas cannot be grossly differentiated from adenomas. They are usually solitary, ulcerated, and

BOX 18.60 Common Tumors of the Perianal Region

- Perianal gland adenoma
- Perianal gland adenocarcinoma
- Apocrine gland adenocarcinoma
- Lipoma
- Leiomyoma
- Squamous cell carcinoma
- Melanoma
- Lymphoma
- Mast cell tumor
- Miscellaneous skin tumors

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locally invasive and can be confused with perianal fistulae or anal sacs that have ruptured or are impacted. These tumors are not hormone responsive. Both primary and metastatic sites grow more slowly than many other malignancies. They usually metastasize to the intrapelvic and sublumbar lymph nodes. Other metastatic sites include the liver, lungs, kidneys, spleen, bone, and abdominal lymph nodes.

Anal sac apocrine gland adenocarcinomas (anal sac adenocarcinoma, apocrine gland adenocarcinoma) arising in the anal sac account for approximately 2% of skin tumors in dogs. Most patients have unilateral tumors. These tumors can cause hypercalcemia of malignancy with subsequent polyuria, polydipsia, poor appetite, and/or vomiting. Anal sac adenocarcinomas initially grow slowly and are confined to the anal sac; however, invasion into surrounding tissues, the rectum, and the pelvic canal occurs with continued growth. Most show evidence of stromal and lymphatic invasion. Metastasis to the iliac, sacral, and sublumbar lymph nodes may occur. Distant metastasis may occur anywhere, but lungs, liver, and spleen are the most common sites.

NOTE Suspect perianal gland adenomas in male dogs. Suspect anal sac adenocarcinomas if the dog is hypercalcemic.

Anal SCCs arise from the anocutaneous line. They are typically malignant and metastasize quickly. Extensive fistula or mucosal-cutaneous, ulcer-like lesions occur and are often covered with mucus. Anal function is impaired, and pain, tenesmus, and hemorrhage are typical. The prognosis is grave because of their malignant nature. Treatment is often discouraged.

DIAGNOSIS

Clinical Presentation

Signalment

Perianal tumors are common in middle-aged or older male dogs but rare in females. The median age for anal sac apocrine gland adenocarcinoma is approximately 10 years. Adenomas are more prevalent in cocker spaniels, beagles, bulldogs, and Samoyeds. Cats do not have perianal or circumanal glands. Apocrine gland adenocarcinomas usually occur in older dogs. There appears to be no sex predisposition.

History

Tumors in the perianal region cause irritation with subsequent licking, scooting, and tenesmus. Continued growth of the tumor or excoriation of the thin perianal skin causes mild hemorrhage, which may be noted in the feces or where the animal sits. Constipation, obstipation, and dyschezia may occur with large, invasive tumors. Some tumors are asymptomatic and found incidentally on a physical examination. Benign tumors are usually slow-growing and painless. Malignant tumors are usually fast-growing, firm, and invasive and are commonly ulcerated. Perianal tumors in castrated males should be considered malignant until proved otherwise. Paraneoplastic hypercalcemia is common with anal sac adenocarcinomas (Box 18.61); polyuria, polydipsia, anorexia, and/or vomiting secondary to renal failure may be seen, depending on the magnitude of the hypercalcemia. Fecal incontinence may occur with aggressive tumors. Other signs that may be associated with metastatic lesions are chronic cough, limb edema, and urethral and rectal obstruction. Some anal sac adenocarcinomas are asymptomatic, being found incidentally during routine examinations.

! BOX 18.61 Signs of Hypercalcemia

- Anorexia
- Weight loss
- Vomiting
- Polyuria
- Polydipsia
- Muscle weakness
- Constipation

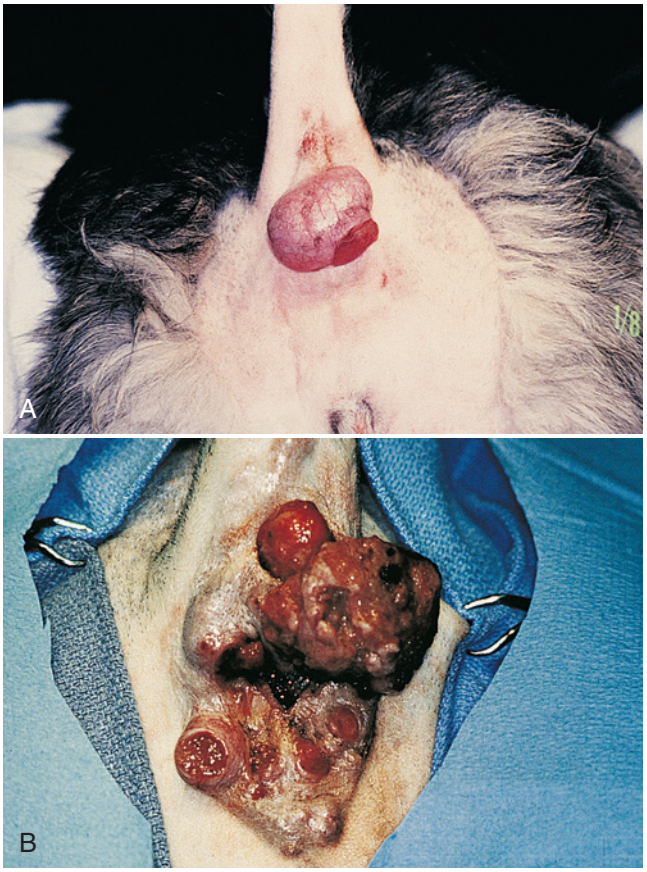


FIG. 18.141 Perianal tumors. (A) A single tumor with partially intact epithelium. (B) Multiple ulcerated perianal tumors.

Physical Examination Findings

Multiple perianal masses are often identified around the circumference of the anus in the hairless area. They may vary in size and may be covered with epithelium or ulcerated, friable, and broad based (Fig. 18.141). Most adenomas are well circumscribed, whereas carcinomas are invasive. Careful palpation of the perianal tissues during rectal examination often identifies masses that are difficult to visually differentiate from normal perianal tissue. Anal sac tumors are not always obvious when the anal sacs are palpated.

NOTE Check the sublumbar and other regional lymph nodes for enlargement and asymmetry.

Diagnostic Imaging

Radiographs of the abdomen and thorax are used to help stage the disease. Enlarged sublumbar lymph nodes suggest

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metastasis. Abdominal ultrasonography allows evaluation of lymph nodes. CT can be used to help determine tumor size and invasiveness.

Laboratory Findings

Cytologic studies help, but histologic examination is necessary to differentiate perianal adenomas from carcinomas. However, it can be difficult to distinguish between benign and malignant tumors, even with histopathologic examination. Anal sac tumors often exfoliate well for cytologic evaluation[49] and may cause hypercalcemia of malignancy (~30%–50%) and renal dysfunction. The total serum calcium concentration is affected by the serum total protein and serum albumin concentrations. Although there are formulas to correct the total serum calcium concentration for changes in the serum albumin concentration, it is much more accurate to measure ionized serum calcium. Renal dysfunction is common in these hypercalcemic patients, and it is important to measure serum creatinine and perform a urinalysis on urine obtained prior to fluid therapy. Some hypercalcemic patients are also hypophosphatemic.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses of anal and perianal irritation include anal sacculitis, dermatitis, endoparasites, perianal fistula, fungal infection, or tumors. Differential diagnoses for perianal swelling include perineal hernia, perianal neoplasia, perianal gland hyperplasia, anal sacculitis, anal sac neoplasia, atresia ani, rectal pythiosis, and vaginal tumors. Differential diagnoses for dyschezia include rectal foreign body, perineal hernia, perianal fistula, anal stricture, rectal stricture, anal sac abscess, rectal or anal neoplasia, anal trauma, anal dermatitis, anorectal prolapse, IBD, histoplasmosis, and pythiosis.

MEDICAL MANAGEMENT

Some perianal tumors respond to chemotherapy or radiation therapy, but reports documenting the effectiveness of these treatments are lacking. Perianal gland adenomas may shrink after a short course of diethylstilbestrol (0.5–1 mg daily for 1–2 weeks). Radiation therapy or chemotherapy is recommended for nonresectable malignancies. Other recommendations include vincristine, doxorubicin, and cyclophosphamide (VAC) or melphalan, mitoxantrone, carboplatin, or cisplatin. Radiation or chemotherapy may convert a marginally operable tumor to an operable tumor. Refer to an oncology text for further information regarding management of perianal tumors.

SURGICAL TREATMENT

Surgical excision is the treatment of choice for perianal tumors. Generally, perianal masses that do not involve the anal sacs are perianal adenomas; therefore castration and resection of small masses or biopsy of multiple or large masses is recommended. Patients should be reevaluated 4 to 6 weeks after castration and biopsy. Adenomas will be smaller at this time and can generally be resected with less trauma to the external anal sphincter. Some adenomas regress completely after castration. Prompt, wide resection of malignancies is recommended.

Preoperative Management

Fluid, electrolyte, and acid-base abnormalities should be corrected before surgery. Mildly to moderately hypercalcemic animals should first be rehydrated and diuresed with physiologic saline solution and furosemide. Hypercalcemia causes renal dysfunction. Although the renal dysfunction is initially mild and reversible, it can become severe, oliguric, and irreversible if therapy is delayed. Therefore it is important to be sure the patient is not oliguric. Prednisolone may be given (Box 18.62) to help lower the serum calcium. Severely affected animals (total serum calcium concentration greater than 16 mg/dL) may also be treated with alkalinizing agents (e.g., sodium bicarbonate) and bone resorption inhibitors (i.e., pamidronate disodium). Pamidronate disodium (1–2 mg/kg IV) has been used in dogs that were hypercalcemic from different causes, and it quickly lowered serum calcium concentrations. However, pamidronate is expensive. Peritoneal dialysis may be performed in oliguric patients but is of uncertain value. Perioperative antibiotics are indicated in old or debilitated patients. The antibiotics should be given intravenously at induction of anesthesia and discontinued within 12 to 24 hours of surgery. Enemas should not be administered on the day of surgery because they may increase contamination of the surgical site. Remaining feces may be removed manually after induction but before preparing the animal for aseptic surgery.

Anesthesia

Patients with perianal tumors may be old and debilitated and may have other serious medical problems requiring special care during anesthesia. Anesthetic recommendations for animals undergoing perianal surgery are given on p. 483 and in Table 18.1.

BOX 18.62 Treatment of Hypercalcemia

Furosemide (Lasix)
2–4 mg/kg IV, PO, SC q8–12h or as CRI (load with 0.66 mg/kg bolus, then give 0.66 mg/kg/h for 4–5 hours; alternatively, estimate the IV or PO dose to be given over the course of the next 24 hours and give that amount as a CRI over the next 24 hours); be sure patient is volume replenished before administering

Prednisolone
1.1–2.2 mg/kg IV, PO, SC q12h

Sodium Bicarbonate
0.5–4 mEq/kg given in IV fluids (check blood gas value first)

Pamidronate Disodium
Dogs: 1–2 mg/kg IV or SC over 2 hours, once weekly
Cats: 1–1.5 mg/kg IV over 2 hours, once weekly

Clodronate
20–25 mg/kg IV over 4 hours

Etidronate Disodium (Didronel)
Dogs: 5 mg/kg/day PO
Cats: 10 mg/kg/day PO

Salmon Calcitonin
4 units/kg IV; then 4–6 U/kg SC q8–12h

Mithramycin[a]
25 µg/kg IV over 4 hours, once every 2–4 weeks

[a]This drug has many potential side effects; a medical text should be consulted before administering it.
CRI, Constant rate infusion; *IV,* intravenous; *PO,* orally; *SC,* subcutaneous.

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Surgical Anatomy

The anatomy of the rectum and perianal region is presented on p. 483. Cats do not have perianal or circumanal glands.

Positioning

Position the patient in ventral (preferred) or dorsal recumbency to allow access to the tumors and scrotal region. Fix the tail over the back, elevate the pelvis, and pad the hind legs when using a perineal position.

SURGICAL TECHNIQUE

Removal of half of the anal sphincter is possible, with some return of fecal continence within a few weeks. Resection of metastatic sites (e.g., lymph nodes) from patients with anal sac adenocarcinomas may help control hypercalcemia.

Begin by performing a prescrotal or perineal castration on intact male dogs with perianal adenomas (see p. 732). Incise the perianal skin surrounding perianal adenomas with minimal margins of normal tissue. Dissect the tumor from the subcutaneous tissues and the external anal sphincter with minimal trauma. Thoroughly lavage the area. Close dead space with monofilament, absorbable suture (e.g., 3-0 or 4-0 polydioxanone, polyglyconate, or poliglecaprone 25), and close the skin with interrupted appositional sutures (e.g., monofilament, 3-0 or 4-0 nylon, polybutester, or polypropylene). Submit the excised masses and testicles for histologic evaluation.

Resect malignant tumors with a minimum of 1 cm of normal tissue on all borders (Fig. 18.142). This includes partial resection of the external anal sphincter, anal canal, and anal sacs in some cases. Appose the epithelial edges to prevent anal stricture.

POSTOPERATIVE CARE AND ASSESSMENT

Give systemic analgesics (see Table 13.2) as necessary for pain. The serum calcium concentration should be monitored at least daily for the first 2 days. Hypercalcemia should be treated until the serum calcium level is normal. Most animals become normocalcemic within 24 hours of primary tumor resection; however, some become hypocalcemic and require calcium supplementation to prevent tetany. If necessary, administer 10% calcium gluconate (0.5–1.5 mL/kg of 10% calcium gluconate IV over 10–20 minutes and monitor the heart; then add 10 mL of 10% calcium gluconate to 250 mL of lactated Ringer's solution and drip at maintenance rate; or give the IV dose diluted in an equal volume of saline solution, subcutaneously). The perianal area should be kept clean, and an Elizabethan collar or similar restraint device should be used to prevent the patient from licking at surgical sites. Animals that are not vomiting may receive water and food within 8 to 12 hours after surgery. A stool softener may be added to the food for 2 to 3 weeks (see Box 18.52). Chemotherapy may slow recurrence and metastatic tumor growth, but its efficacy is unknown. The rectum and perianal area should be palpated for evidence of stricture or tumor recurrence when the sutures are removed at 7 to 10 days. Patients with malignancies should be reevaluated for recurrence or metastasis at 2, 4, and 6 months and then yearly. Rectal palpation, measurement of serum ionized calcium, and imaging (e.g., abdominal radiography, CT, or ultrasonography) are indicated during reevaluation. Recurrence of malignant tumors is often detected by 3 months after surgery. Possible complications of perianal surgery are listed in Box 18.63.

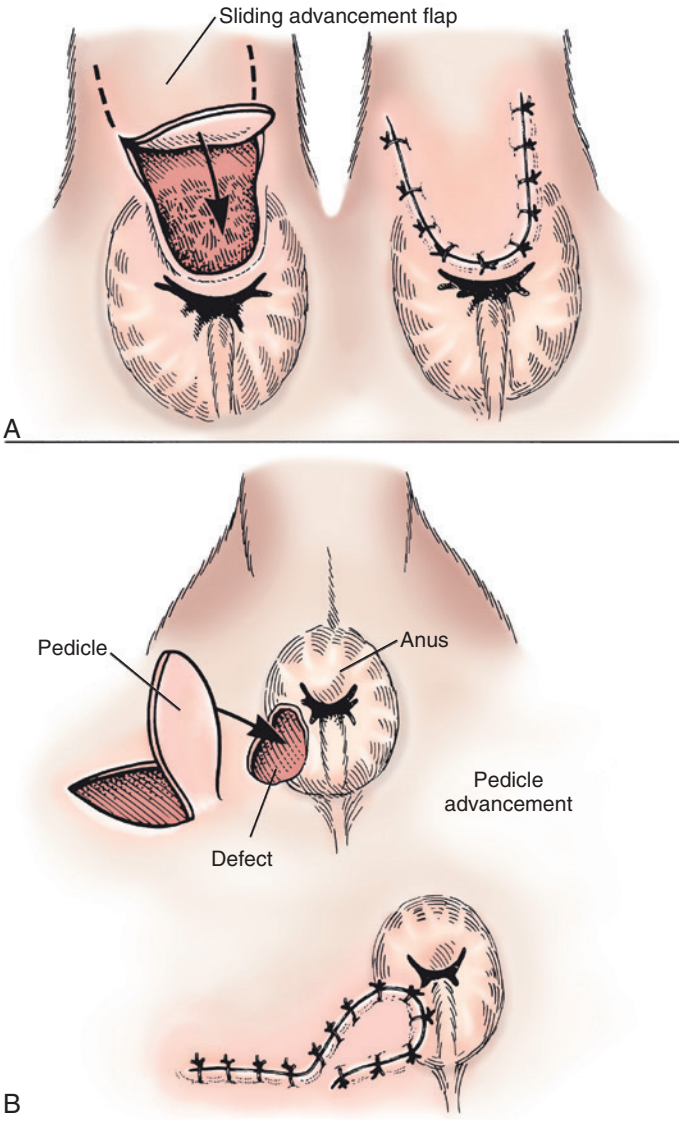


FIG. 18.142 Resect perianal masses with involved muscle. To help prevent anal stenosis, close large defects in the sphincter with (A) a sliding advancement flap or (B) a local pedicle flap.

BOX 18.63 Possible Complications of Surgery for Perianal Tumors

- Infection
- Dehiscence
- Tenesmus
- Rectal prolapse
- Dyschezia
- Hematochezia
- Temporary or permanent incontinence
- Anal stricture
- Tumor recurrence
- Metastasis

NOTE Always administer calcium gluconate to hypocalcemic animals; *never* administer calcium chloride.

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PROGNOSIS

Prolonged estrogen therapy is not recommended because of its myelotoxic effects and its temporary effects on tumor size. Radiation therapy is an option, but surgery is less expensive, faster, and safer. The prognosis after surgery is good for benign perianal tumors but guarded to poor for malignant tumors, although some malignant tumors may be slow-growing and late to metastasize. Palliation for nonresectable malignant tumors may involve partial resection, cryosurgery, chemotherapy, or radiation therapy. The prognosis for perianal gland adenomas is good to excellent after castration. Adenomas occasionally recur (<10%) and should be rebiopsied. Early, complete excision of perianal gland adenocarcinomas can be curative, but most carcinomas are invasive or metastasize to lymph nodes. Recurrence is common, although it may take many months; therefore the prognosis is poor.

Anal sac adenocarcinomas in dogs classically warrant a poor prognosis because they have frequently metastasized by the time of diagnosis. Recurrent hypercalcemia suggests recurrence or metastasis. If metastasis is present, survival of less than a year can be expected; however, in a 2015 study, a median survival time of 531 days was reported, with 62% having disease progression or recurrence at a median of 398 days.[49] Sublumbar lymph node involvement was associated with a shortened survival time (422 days vs. 529 days) in the aforementioned study. In another study, the use of radiation therapy in dogs with grossly visible anal sac adenocarcinoma resulted in 38% of treated dogs having a partial response; for dogs presenting with clinical signs related to the tumor, 63% showed improvement or resolution of clinical signs.[50] For dogs presenting with hypercalcemia of malignancy, resolution was noted in 31% with radiation therapy alone and an additional 46% with radiation, prednisone, and/or bisphosphonates. Median overall survival was 329 days (range, 252–448 days). Median progression-free survival was 289 days (range, 224–469). In this study, there was no difference in survival based on radiation protocol, use of chemotherapy, previous surgery, or advanced stage.

ANAL SAC INFECTION AND IMPACTION

DEFINITION

Anal sac impaction is an abnormal accumulation of anal sac secretions that occurs secondary to inflammation *(anal sacculitis)*, infection *(anal sac abscess)*, or obstruction of the duct. Anal sacs are sometimes erroneously referred to as *anal glands*.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Anal sac diseases include impaction, infection, abscessation, and neoplasia. The anal sacs are a modified adnexal skin structure. They are paired, lying between the fibers of the anal sphincter, and are lined by squamous epithelium with modified apocrine and sebaceous glands. They serve as reservoirs for their malodorous, pastelike secretions. The excretions are expelled through the ducts during normal defecation and extreme excitement. Forceful contractions of the sphincter are necessary for anal sac emptying.

Anal sacculitis affects approximately 10% of dogs and is usually caused by infection or duct obstruction. Ductal obstruction leads to infection and inflammation. Inflammation enhances secretions, which serve as an ideal medium for bacterial growth. Secretions continue to accumulate despite ductal obstruction, and the sacs become impacted and eventually rupture. Distention causes pain. Chronic fistulation may result if infection or duct obstruction persists. Anal sacculitis also occurs without duct obstruction. In these cases, hypersecretion occurs, and the sac is easy to express. Secretions are more liquid than normal with yellowish-white granules. Factors that may cause chronic hypersecretion include infectious, endocrine, allergic, behavioral, and idiopathic mechanisms. Malfunction of the anal sphincter mechanism secondary to chronic diarrhea, anal laxity, constipation, and obesity may contribute to retention of anal sac secretions and the development of anal sacculitis.

DIAGNOSIS

Clinical Presentation

Signalment

Anal sacculitis may occur in an animal of any age, breed, or sex; however, it is most common in small- and toy-breed dogs and rare in cats. In some animals, anal sacculitis may be associated with seborrheic dermatitis or other dermatoses.

History

Many animals have a history of recent diarrhea (1–3 weeks) or soft stools or estrus. They usually evidence anal irritation (e.g., scooting, licking, and biting at the tail head or anus). Other complaints include tail chasing, malodorous perianal discharge, pain or tenderness, and behavioral change. Tenesmus, dyschezia, constipation, and hematochezia occasionally occur. Generalized dermatitis or dermatitis at a secondary site is sometimes recognized.

Physical Examination Findings

The anal sac region may appear swollen and inflamed. Abscesses or impaction may cause the anal sac to rupture and create a draining lesion at the 4 o'clock or 7 o'clock position. Fever may occur with abscesses or severe sacculitis. Palpation of the perianal tissue during rectal examination may identify an enlarged, firm, and sometimes painful anal sac. Digital expression of the anal sac may expel normal secretions (serous, slightly viscid, granular, pale yellow liquid) or abnormal secretions (whitish-gray, brown, yellow, or green, bloody, purulent, gritty, turbid, opaque). It may be impossible to express material from diseased sacs. Animals with untreated anal sac abscesses may be debilitated, may have other perianal or rectal abscesses, or may develop anal stricture. Perineal fistulae occasionally occur.

NOTE Routine palpation and expression of the anal sacs during physical examination may allow early detection of anal sac disease.

Impaction is diagnosed when the sac is distended and mildly painful and cannot be readily expressed. Anal sacculitis is diagnosed when moderate or severe pain is elicited on palpation, and secretions are liquid, yellowish, blood-tinged, or purulent. The diagnosis of anal sac abscessation is made when there is marked distention of the sac with a purulent exudate, cellulitis of surrounding tissues, erythema of overlying skin, pain, and fever. An anal sac rupture is diagnosed by finding a draining tract associated with the anal sac.

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Diagnostic Imaging

Survey radiographs, CT, or MRI are recommended if neoplasia is suspected (see p. 476). A fistulogram may help determine whether a draining tract is associated with the anal sac region or some other perineal location.

Laboratory Findings

Hematologic and serum biochemistry changes are nonspecific. Leukocytosis with/without a left shift may be noted with anal sac abscesses. Cytologic studies from diseased anal sac secretions reveal cellular debris, large numbers of leukocytes, and numerous bacteria. Culture and sensitivity of the anal sac is recommended. The normal bacterial floras of anal sacs include micrococci, *E. coli, Streptococcus faecalis,* and *Staphylococcus* spp. Bacteria typically cultured from diseased anal sacs include *S. faecalis, Clostridium perfringens, E. coli, Proteus* spp., *Staphylococcus* spp., micrococci, and diphtheroids.

DIFFERENTIAL DIAGNOSIS

The primary differential diagnoses for anal sacculitis are flea allergy (from licking and biting), perianal tumor (caused by swelling and ulceration), perianal fistulae, or tail-fold pyoderma (resulting in abscessation and draining tracts). Differential diagnoses for anal or perianal irritation include anal sacculitis, dermatitis, endoparasites, perianal fistulae, vaginitis, or tumors. Differential diagnoses for perianal swelling include perianal hernia, perianal neoplasia, perianal gland hyperplasia, anal sacculitis, anal sac neoplasia, atresia ani, rectal pythiosis, and vaginal tumors. Differential diagnoses for dyschezia include rectal foreign body, perineal hernia, perianal fistulae, anal stricture, rectal stricture, anal sac abscess, rectal neoplasia, anal neoplasia, anal trauma, anal dermatitis, rectal pythiosis, and anorectal prolapse.

MEDICAL MANAGEMENT

Treatment depends on the stage of infection. Manual expression, lavage, topical antibiotics, and dietary change effectively manage most anal sac problems. Treatment of concomitant dermatoses facilitates treatment of anal sacculitis. Mild sacculitis or impaction is treated by expressing, lavaging (with saline), and infusing the glands with an antibiotic-glucocorticoid preparation. Dry secretions may be softened by lavaging with saline or infusing a ceruminolytic agent. If the anal sacs are infected, 0.5% chlorhexidine or 10% povidone-iodine may be added to saline flushes. Adding fiber (e.g., w/d [Hill's Pet Products], pumpkin, bran, or psyllium) to the diet makes the feces bulky, which may stretch the anus during defecation, causing the anal sacs to be compressed and emptied. In more severe cases, weekly evaluation, expression, and lavage with a dilute antiseptic solution or saline may be required. Chronic cases may require antibiotics as determined by sensitivity results. Anal sac abscesses should be lanced, drained, and flushed. Hot compresses, applied two or three times daily for 15 to 20 minutes each, are beneficial for abscesses. Appropriate oral antibiotics should be administered to patients with anal sac abscesses. Chemical cauterization is not recommended, because severe perineal sloughing may result (Fig. 18.143).

SURGICAL TREATMENT

Failure of medical therapy and suspicion of neoplasia are indications for anal sacculectomy. If a draining tract persists after anal sac rupture, surgery should be delayed until inflammation is controlled. Both anal sacs should be removed, even if only one is obviously involved. Either an open or closed technique may be used; however, the open technique carries a greater risk of fecal incontinence and local infection.



FIG. 18.143 Note the extensive perineal sloughing that occurred after chemical cauterization to treat anal sacculitis. This dog was euthanized because of deep vascular erosion and hemorrhage. Chemical cauterization is not recommended for treatment of anal sacculitis.

> **NOTE** Warn owners of the risk of incontinence after anal sacculectomy.

Preoperative Management

Anal sacculitis, abscessation, or fistulation should be treated for several days as described earlier to reduce inflammation before surgery. Inflammation and fibrosis present at the time of surgery increase the risk of damage to the anal sphincter. Temporary or permanent fecal incontinence may result secondary to sphincter damage.

Anesthesia

Anesthetic recommendations for animals undergoing perianal surgery are given on p. 483 and in Table 18.1.

Surgical Anatomy

One anal sac lies on each side of the anus between the internal and external anal sphincters. The anal sac is a cutaneous diverticulum lined by microscopic glands. Secretions of these sebaceous and apocrine glands accumulate in the anal sac and are normally expelled through the ducts during defecation or contraction of the anal sphincter. The ducts of the anal sacs open in the cutaneous zone at approximately the 4 o'clock to 5 o'clock and 7 o'clock to 8 o'clock positions. The duct opening in cats is more lateral to the anocutaneous line than in dogs. They are visible lateral to the anus in the normal contracted state.

Positioning

Position the patient in ventral recumbency with the tail fixed dorsally over the back. Elevate the pelvis and pad the hind legs when using a perineal position.

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SURGICAL TECHNIQUES

Palpate the anal sacs to determine their location and extent by placing the index or middle finger in the rectum and the thumb over the sac. Manually evacuate feces from the rectum if present. Prepare the perineal area for surgery.

Closed Technique

Insert a small probe, hemostat, or balloon-tip catheter into the orifice of the anal sac duct (Fig. 18.144A). Advance the instrument or inflate the balloon with saline until the lateral extent of the sac is identified. As an alternative, wax or synthetic resin may be infused to distend the sac before resection.

Make a curvilinear incision over the anal sac. Dissecting directly against the anal sac, separate the internal and external anal sphincter muscle fibers from the sac's exterior with small Metzenbaum or iris scissors. Avoid excising or traumatizing the muscles or the caudal rectal artery medial to the duct. Continue dissecting to free the sac and duct to its mucocutaneous junction at the anal canal (see Fig. 18.144B). Perforation of the sac may occur during dissection, and tissues may be contaminated with secretions. *Place a ligature around the duct at the mucocutaneous junction using 4-0 monofilament absorbable suture (see Fig. 18.144C). Excise the anal sac and duct, then inspect for completeness of removal. Control hemorrhage with ligatures, electrocoagulation, or pressure. Lavage the tissues thoroughly. Appose subcutaneous tissues with 4-0 interrupted, monofilament absorbable sutures (polydioxanone, polyglyconate, or poliglecaprone 25), and appose the skin with 3-0 or 4-0 monofilament nonabsorbable (nylon, polypropylene, or polybutester) sutures.*

Open Technique

Place a scissors blade or groove director into the duct of the anal sac (Fig. 18.145A). Apply medial traction on the duct while incising through the skin, subcutaneous tissue, external anal sphincter, duct, and sac. Continue the incision to the lateral extent of the anal sac. Elevate the cut edge of the sac and use small Metzenbaum or iris scissors to dissect the sac free of its attachments to muscle and surrounding tissue (see Fig. 18.145B). Complete the procedure as for closed sacculectomy (see Fig. 18.145C).

NOTE The lining of the anal sac is grayish and glistening; it is easily distinguished from surrounding tissue.

POSTOPERATIVE CARE AND ASSESSMENT

Systemic analgesics (see Table 13.2) should be given as necessary. The perianal area should be kept clean, and an Elizabethan collar or similar restraint device should be used to prevent the animal from licking the sites. Food and water may be offered 8 to 12 hours postoperatively if no vomiting has been noted. A stool

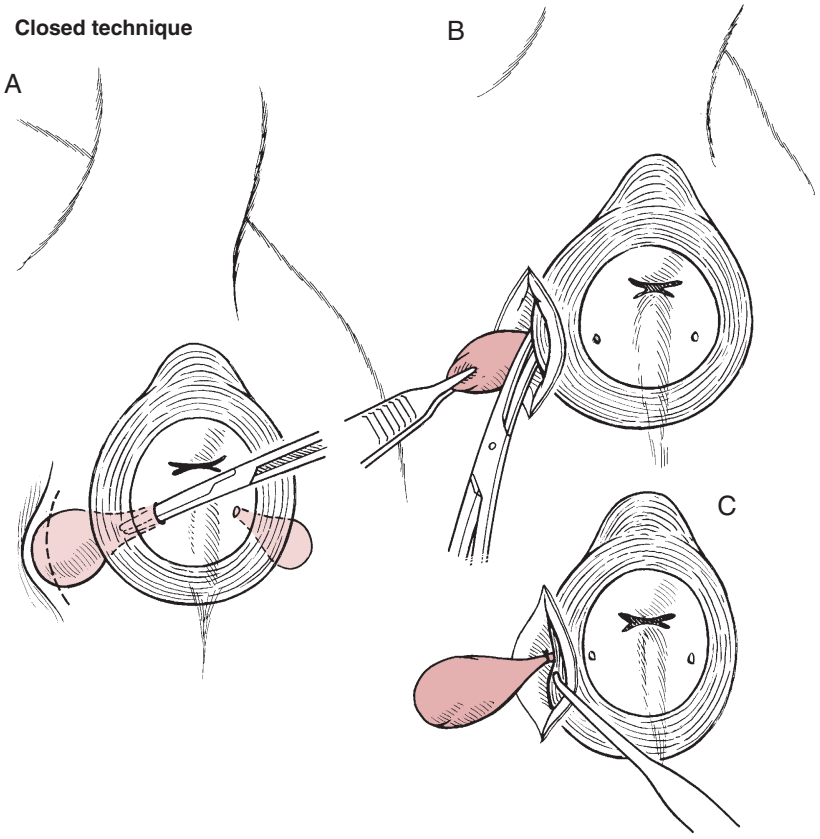


FIG. 18.144 Closed technique for anal sacculectomy. (A) Locate the anal sacs at the 4 o'clock to 5 o'clock and 7 o'clock to 8 o'clock positions between the internal and external anal sphincter muscles. Insert a small probe, hemostat, or balloon-tip catheter into the anal sac. *Dashed line* indicates incision location. (B) Make an incision at the lateral aspect of the anal sac and carefully dissect the sac from the sphincter muscle fibers. (C) Ligate the duct near the orifice.

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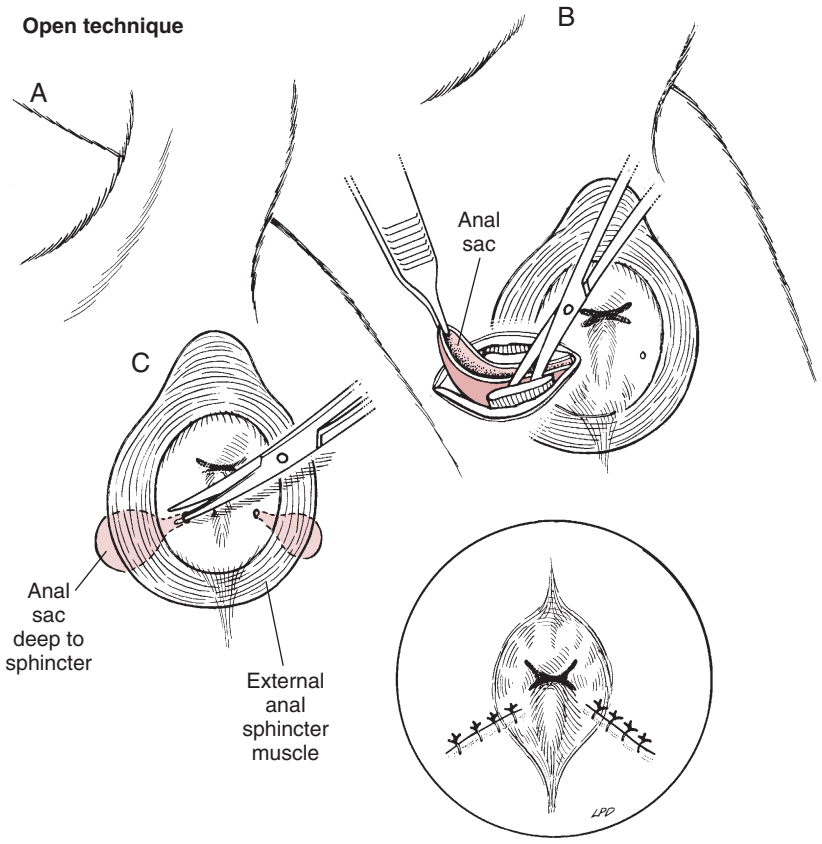


FIG. 18.145 Open technique for anal sacculectomy. (A) Insert the blade of the scissors into the sac and incise through the skin, subcutaneous tissues, external anal sphincter, and anal sac. (B) Elevate the cut edge of the sac and dissect it from the anal sphincter. (C) Appose the sphincter, subcutaneous tissues, and skin.

softener may be added to the food for 2 to 3 weeks (see Box 18.52). The surgical site should be monitored for signs of infection or drainage, and the rectum and perianal area should be palpated for evidence of stricture when sutures are removed at 7 to 10 days. Fecal continence may be impaired during the healing process but usually returns to normal within several weeks.

COMPLICATIONS

Short-term complications (within 14 days) may include excessive drainage, scooting, inflammation, and seroma formation. Long-term complications include continued licking of the surgical site, fecal incontinence, fistulation, and stricture formation. Fecal incontinence after anal sacculectomy may be temporary or permanent. A draining tract after surgery suggests that a piece of anal sac was left at the surgical site. This occurs more commonly with inexperienced surgeons or inflamed and fibrotic tissues. Surgical excision is necessary or drainage will continue. Other complications include infection, dehiscence, tenesmus, rectal prolapse, dyschezia, and hematochezia.

PROGNOSIS

The prognosis for nonneoplastic anal sac disease is good if it is not associated with perianal fistulae. Most cases of anal sacculitis can be treated medically if they are recognized early, treated appropriately, and not associated with neoplasia or perianal fistulae.

PERINEAL HERNIA

DEFINITIONS

Perineal hernias occur when the perineal muscles separate, allowing rectum, pelvic, and/or abdominal contents to displace perineal skin. Depending on their location, they may be referred to as a *caudal hernia, sciatic hernia, dorsal hernia,* or *ventral hernia.*

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Perineal hernia occurs when pelvic diaphragm muscles fail to support the rectal wall, allowing persistent rectal distention and impaired defecation. The cause of pelvic diaphragm weakening is poorly understood but believed to be associated with male hormones, straining, and congenital or acquired muscle weakness or atrophy. The pelvic diaphragm is stronger in female dogs than in males. Atrophy of the pelvic diaphragm muscles, possibly of neurologic origin, has been identified in some animals with hernias. Any condition that causes straining may stress the pelvic diaphragm (Box 18.64).

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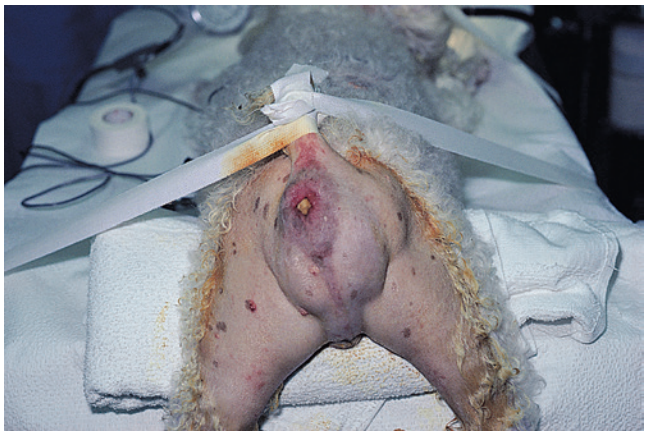


FIG. 18.146 Bilateral perineal hernias in a dog.

BOX 18.64 Conditions That Cause Straining and May Predispose to Perineal Herniation

- Prostatitis
- Cystitis
- Urinary tract obstruction
- Colorectal obstruction
- Rectal deviation or dilatation
- Perianal inflammation
- Anal sacculitis
- Diarrhea
- Constipation

Herniation may be unilateral or bilateral (Fig. 18.146). Most herniations occur between the levator ani, external anal sphincter, and internal obturator muscles (caudal hernia); however, some occur between the sacrotuberous ligament and coccygeus muscle (sciatic hernia), levator ani and coccygeus muscles (dorsal hernia), or ischiourethralis, bulbocavernosus, and ischiocavernosus muscles (ventral hernia). Hernial contents are surrounded by a thin layer of perineal fascia (hernial sac), subcutaneous tissue, and skin. The hernial sac may contain pelvic or retroperitoneal fat, serous fluid, a deviated or dilated rectum, a rectal diverticulum, prostate, urinary bladder, or small intestine (Fig. 18.147). Cats usually have only rectum within the hernial sac. Organs displaced into the hernia may become obstructed and strangulated. Visceral obstruction or strangulation is associated with rapid deterioration unless the obstruction or entrapment is corrected.

DIAGNOSIS

Clinical Presentation

Signalment

Perineal hernias are common in dogs and rare in cats. They occur almost exclusively in intact male dogs (93%). Perineal hernias in female dogs are often related to trauma. Feline perineal hernias usually occur in neutered males; however, female cats are more prone to perineal hernias than female dogs. Dogs with short tails may be predisposed to herniation. Breeds most commonly affected are Boston terriers, boxers, Welsh corgis, Pekingese, collies, poodles, kelpies, dachshunds, Old English sheepdogs, and mongrels. Most perineal hernias occur in dogs over 5 years of age. The median age is approximately 10 years in both dogs and cats. The risk of occurrence increases with age until 14 years in intact male dogs.

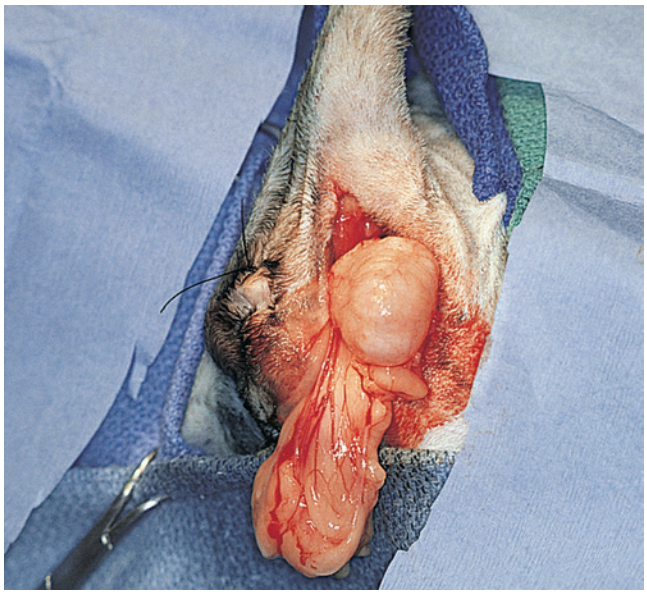


FIG. 18.147 Perineal hernia in a dog. Note the urinary bladder and pelvic fat occupying this hernial sac.

History

Affected animals are usually presented for treatment because of difficulty defecating. Some owners notice a swelling lateral to the anus. Occasionally animals are presented as emergencies because of postrenal uremia associated with bladder entrapment or shock associated with intestinal strangulation. Clinical signs may include perineal swelling, constipation, obstipation, dyschezia, tenesmus, rectal prolapse, stranguria, anuria, vomiting, flatulence, and/or fecal incontinence.

Physical Examination Findings

The diagnosis is based on finding a weakened pelvic diaphragm during digital rectal examination, with or without a perineal swelling lateral to the anus (see Fig. 18.146). Not all dogs with perineal hernias have perineal swelling. When present, the swelling may appear to surround the anus and cause it to bulge. A rectal deviation often contains impacted feces. Some reports indicate a right-sided predominance. Cats typically have bilateral hernias, which seldom cause obvious perineal swelling. The prostate is sometimes found in the hernia. Severe straining can cause rectal prolapse. Some animals are systemically ill and have cardiovascular instability because of visceral strangulation. If ballottement suggests liquid is present and the animal is dysuric, ultrasound or perineal centesis should be performed to determine if fluid (i.e., urine) is present. A hernia filled with a full bladder may have a tight, resilient feel when palpated, instead of a fluid wave. Concurrent inguinal hernias have been identified in some dogs with perineal hernia.

NOTE A fluctuant swelling in the perineal region may indicate that the bladder is entrapped in the hernia. Prompt therapy may be necessary to relieve urinary obstruction in these animals.

Diagnostic Imaging

Survey radiographs are seldom needed for diagnosis; however, they are useful in ascertaining whether the urinary bladder, prostate, or small intestines are in the hernia (Figs. 18.148 and

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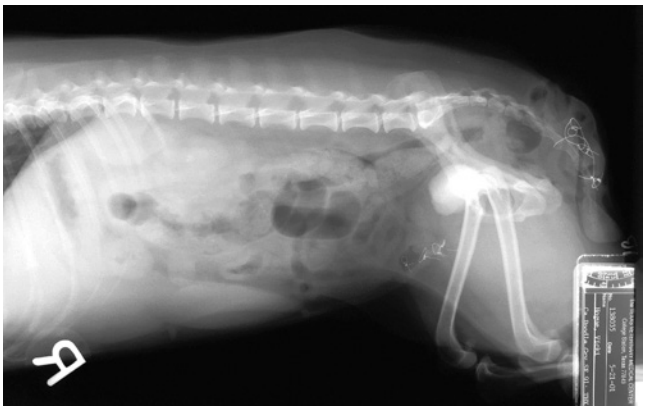


FIG. 18.148 Lateral radiograph of a dog with a perineal hernia. Note the nonstrangulated intestines in the hernial sac.

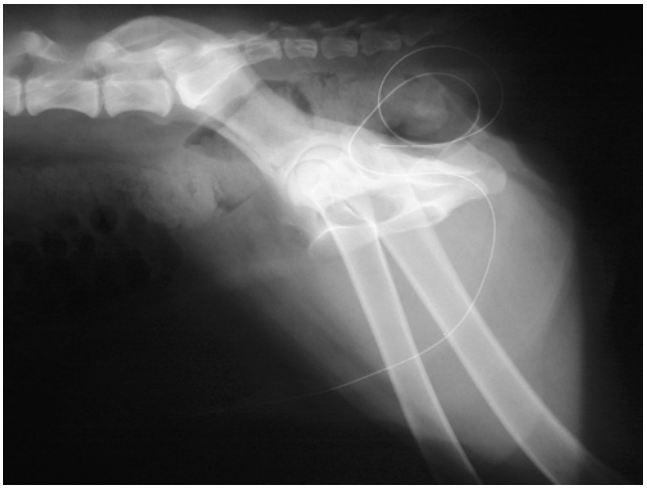


FIG. 18.149 Lateral radiograph of a dog with its bladder retroflexed into the hernia. Note the urinary catheter in the bladder.

18.149). Radiographically documenting retroflexion of the urinary bladder often requires a urethrogram, cystogram, or both or the use of ultrasonography. Administration of oral or rectal barium demonstrates the position of the colon and rectum. Most dogs have rectal deviation and some also have rectal dilatation. Diverticula are typically not documented radiographically or at surgery.

Laboratory Findings

Patients with bladder retroflexion often have azotemia, hyperkalemia, hyperphosphatemia, and neutrophilic leukocytosis.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for perianal swelling include perineal hernia, perianal neoplasia, perianal gland hyperplasia, anal sacculitis, anal sac neoplasia, atresia ani, and vaginal tumors. Differential diagnoses for dyschezia include rectal foreign body, perineal hernia, perianal fistula, anal stricture, rectal stricture, anal sac abscess, rectal neoplasia, anal neoplasia, anal trauma, anal dermatitis, rectal pythiosis, and anorectal prolapse.

MEDICAL MANAGEMENT

The goal of treatment is to relieve and prevent constipation, dysuria, and organ strangulation. Causative factors (i.e., urinary tract obstruction or infection, megacolon, prostatic hypertrophy, prostatitis) should be corrected. Normal defecation can sometimes be maintained using laxatives, stool softeners, dietary changes, periodic enemas, and/or manual rectal evacuation. The urinary bladder can be decompressed by centesis or catheterization. However, long-term use of these treatments is contraindicated because life-threatening visceral entrapment and strangulation may occur.

SURGICAL TREATMENT

Herniorrhaphy should always be recommended. Retroflexion of the urinary bladder and visceral entrapment require emergency surgery. Castration, although controversial, is recommended during herniorrhaphy because it has been reported to reduce recurrence. Intact male dogs have a recurrence rate 2.7 times greater than castrated dogs.

The two most commonly used techniques are (1) the traditional, or anatomic reapposition, and (2) the internal obturator roll-up, or transposition technique. It is more difficult to close the ventral aspect of the hernia using the traditional technique. Temporary deformity of the anus occurs and is especially pronounced after bilateral herniorrhaphy. Postoperative tenesmus and rectal prolapse may be more common in these cases. The internal obturator transposition technique is more difficult, especially if internal obturator muscle atrophy is severe. However, it causes less tension on sutures, brings about less deformity of the anus, and creates a ventral patch or sling for the defect. Other herniorrhaphy techniques have included using the superficial gluteal, semitendinosus or semimembranosus muscles, fascia lata, placement of synthetic mesh, small intestinal submucosa, dermal collagen, or a combination of techniques. Bilateral herniorrhaphy is possible, but postoperative discomfort and tenesmus may be greater than after unilateral procedures. If accessible, an enlarged prostate should be biopsied. Either a caudal or prescrotal castration may be performed (see p. 732), neither of which has a higher complication rate.[51] Rectal imbrication or sacculectomy is rarely indicated and significantly increases the risk of postoperative infection. Colopexy may help prevent recurrent rectal prolapse after herniorrhaphy. Fixation of the ductus deferens may help prevent recurrence when the bladder or prostate has been displaced into perineal hernias. Cystopexy has also been performed but is not routinely recommended as retention cystitis may occur.

NOTE Some surgeons prefer waiting 4 to 6 weeks before performing the second herniorrhaphy in dogs with bilateral disease.

Preoperative Management

Stool softeners (see Box 18.52) should be given 2 to 3 days before surgery. The large intestine should be evacuated with laxatives, cathartics, enemas, and manual extraction (see p. 466). Prophylactic antibiotics effective against gram-negative and anaerobic organisms (see Box 18.48) should be given intravenously after induction of anesthesia. If the urinary bladder is retroflexed into the hernia, a urinary catheter should be placed or cystocentesis

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performed via the perineum to relieve distress and prevent further physiologic deterioration.

Anesthesia

Anesthetic recommendations for animals undergoing perineal surgery are given on p. 483 and in Table 18.1. Epidural analgesia may reduce the occurrence of postoperative rectal prolapse. Many affected animals are geriatric and have concurrent abnormalities that may influence drug selection.

Surgical Anatomy

The pelvic diaphragm is composed of the paired medial coccygeal and levator ani muscles. The paired levator ani muscle originates from the floor of the pelvis and medial shaft of the ilium, fans out around the sides of the rectum, and then narrows and inserts ventrally on the seventh caudal vertebra. The paired coccygeus muscle is a thick muscle lying lateral to the thin levator ani. The coccygeus originates from the ischiatic spine on the pelvic floor and inserts ventrally on the second to fifth caudal vertebrae.

The paired rectococcygeus muscle arises from the external longitudinal musculature of the rectum caudal to the levator and coccygeus muscles and inserts on the ventral surface of the fifth to sixth caudal vertebrae. The rectococcygeus muscle shortens the rectum when the tail is raised during defecation. The peritoneal reflection is cranial to the rectococcygeus muscles. The sacrotuberous ligament in the dog is a fibrous band running from the transverse process of the last sacral and first caudal vertebrae to the lateral angle of the ischiatic tuberosity rostral to the pelvic diaphragm. Cats do not have a sacrotuberous ligament. The sciatic nerve lies just cranial and lateral to the sacrotuberous ligament. The internal obturator muscle is a fan-shaped muscle covering the dorsal surface of the ischium. It originates from the dorsal surface of the ischium and pelvic symphysis. Its tendon of insertion passes over the lesser ischiatic notch, ventral to the sacrotuberous ligament. The internal pudendal artery and vein and the pudendal nerve run caudomedially through the pelvic canal on the dorsal surface of the internal obturator muscle, lateral to the coccygeus and levator ani muscles. The pudendal nerve is dorsal to the vessels and divides into the caudal rectal and perineal nerves. The obturator nerve passes through the ventral aspect of the levator ani in a caudolateral direction.

NOTE Perineal vessels and nerves may be displaced from their normal anatomic location by the hernial contents. Careful observation and dissection is required to preserve these structures.

Positioning

Clip and aseptically prepare the perineum for surgery. The prepared area should extend 10 to 15 cm cranial to the tail base, laterally beyond the ischial tuberosity, and ventrally to include the scrotum. The animal should be positioned in ventral recumbency with the tail fixed over the back, the pelvis elevated, and the hind legs padded. As an alternative, a well-padded perineal stand may be used.

SURGICAL TECHNIQUES

Approach

Make a curvilinear incision beginning cranial to the coccygeus muscles, curving over the hernial bulge 1 to 2 cm lateral to the anus, and extending 2 to 3 cm ventral to the pelvic floor (Fig. 18.150). Incise the subcutaneous tissue and hernial sac. Identify and reduce the hernial contents by dissecting subcutaneous and fibrous attachments. Biopsy any abnormal structures within the hernia (e.g., prostate, masses). Maintain hernial reduction by packing the defect with a moistened, tagged sponge. Identify the muscles involved in the hernia, the internal pudendal artery and vein, the pudendal nerve, the caudal rectal vessels and nerve, and the sacrotuberous ligament. Repair the hernia with one of the described techniques. After herniorrhaphy, perform a caudal castration through a median perineal incision (see p. 734).

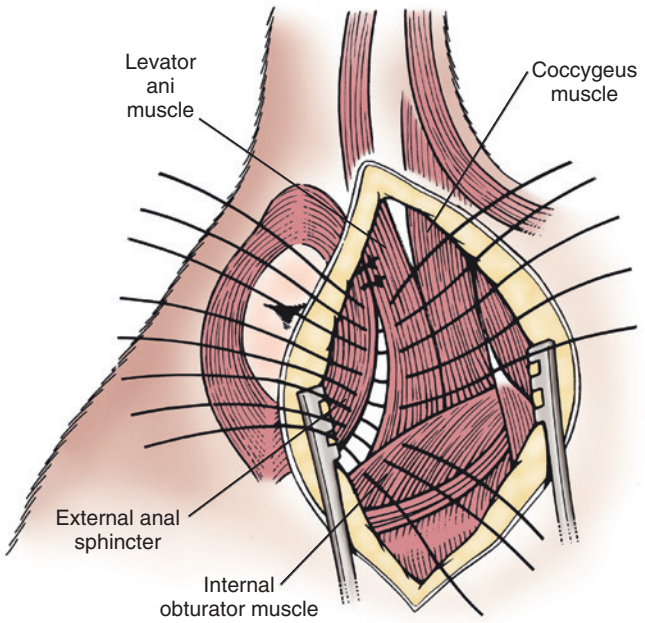


FIG. 18.150 For perineal hernia repair using the traditional technique: make a curvilinear incision 1 to 2 cm lateral to the anus from dorsal to the tailhead to ventral to the anus. Appose the external anal sphincter to the combined levator ani and coccygeus muscles (with or without the sacrotuberous ligament) laterally and the external anal sphincter and internal obturator muscles ventrally.

NOTE Do not mistake the prostate for a mass and attempt to excise it.

Traditional (Anatomic) Herniorrhaphy

Preplace simple interrupted 0 or 2-0 monofilament sutures using a large, curved needle (see Fig. 18.150). Begin suture placement between the external anal sphincter and the levator ani, coccygeus, or both muscles. Space sutures less than 1 cm apart. As placement progresses ventrally and laterally, incorporate the sacrotuberous ligament for a secure repair if necessary. To avoid entrapping the sciatic nerve, place sutures *through* rather than around the sacrotuberous ligament. *Direct ventral sutures between the external anal sphincter and the internal obturator muscle. Be cognizant of the pudendal vessels and nerves at all times to avoid traumatizing these structures. Tie sutures beginning dorsally and progressing ventrally. Remove the sponge used to maintain reduction before tying the last few sutures. Evaluate the repair; place additional*

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sutures if weaknesses or defects persist. Lavage the area. Close the subcutaneous tissues in an interrupted or continuous appositional pattern with 3-0 or 4-0 monofilament absorbable suture (polydioxanone, polyglyconate, poliglecaprone 25, or glycomer 631), and close the skin in an appositional interrupted pattern with nonabsorbable suture (e.g., 3-0 or 4-0 nylon).

Internal Obturator Transposition Herniorrhaphy

Incise the fascia and periosteum along the caudal border of the ischium and origin of the internal obturator muscle. Using a periosteal elevator, elevate the periosteum and internal obturator muscle from the ischium (Fig. 18.151A). Transpose dorsomedially or roll up the muscle into the defect to allow apposition between the coccygeus, levator ani, and external anal sphincter. Transect the internal obturator tendon of insertion, if necessary, to get adequate coverage of the defect. The internal obturator tendon often is difficult to visualize, making transection difficult. *Take care to avoid transection of the caudal gluteal vessels and perineal nerve. Preplace simple interrupted sutures as with the traditional technique. Begin by apposing the combined levator ani and coccygeus muscles with the external anal sphincter muscle dorsally. Then place sutures between the internal obturator and external anal sphincter medially and the levator ani and coccygeus muscles laterally (see Fig. 18.151B).*

Ductus Deferopexy

After castration and herniorrhaphy in dogs with bladder or prostate retroflexion, the ductus deferens can be secured to the abdominal wall to prevent recurrent caudal organ displacement.

Approach the abdomen through a caudal ventral midline incision. Retroflex the urinary bladder caudally through the incision to expose the ductus deferens. Separate the ligated ductus deferens from the testicular artery and vein and gently pull it through the inguinal ring. Dissect each ductus deferens from its peritoneal attachments to the level of the prostate. Pull the urinary bladder and prostate forward by applying moderate traction on the ductus deferens. At an adjacent site on the ventrolateral abdominal wall, make two incisions (1.5–2 cm apart) through the peritoneum and transversus abdominis muscle. Tunnel between these incisions and draw the ductus deferens through the tunnel. Suture the ductus deferens to itself and the abdominal wall with three or four 3-0 monofilament sutures. Repeat the procedure on the opposite side to fix the bladder and prostate in a more cranial position.

POSTOPERATIVE CARE AND ASSESSMENT

Analgesics (see Table 13.2) should be given as necessary to minimize straining and rectal prolapse. If rectal prolapse occurs, a purse-string suture should be placed. Recurrent rectal prolapse may be prevented by performing a colopexy. Fluid therapy should be continued in uremic patients. Cold compresses applied immediately after surgery and two to three times daily for 15 to 20 minutes during the first 48 to 72 hours minimize hemorrhage and inflammation. After 48 to 72 hours, warm compresses applied to the surgical site two or three times daily for 15 to 20 minutes reduce swelling and perianal irritation. Antibiotics may be discontinued within 12 hours of surgery unless ischemic, necrotic, or contaminated tissues were present before surgery or the patient is debilitated. After herniorrhaphy, patients should be monitored for signs of wound infection (i.e., redness, pain, swelling, discharge). Stool softeners (see Box 18.52) should be continued for 1 to 2 months. The animal should be fed a canned diet high in fiber.

COMPLICATIONS

The overall complication rate is approximately 40%.[52] Most postoperative complications can be prevented by meticulous surgical technique. Castration during herniorrhaphy is believed to reduce hernia recurrence or contralateral herniation. Recurrence (20%) is related to the expertise of the surgeon; inexperienced surgeons have higher recurrence rates. Infection and dehiscence

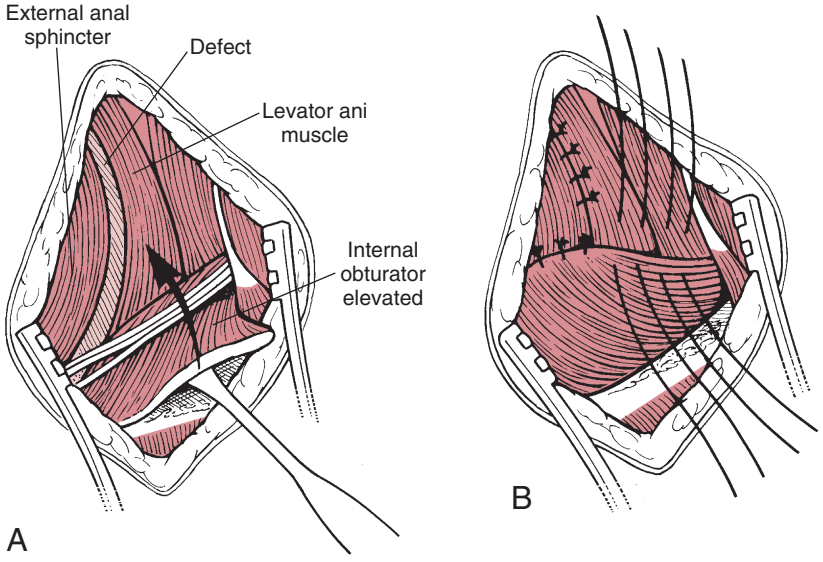


FIG. 18.151 Internal obturator transposition technique. (A) Elevate the internal obturator muscle from the ischium. (B) Appose the external anal sphincter muscle and combined levator ani and coccygeus muscles dorsally. Transpose the internal obturator muscle dorsomedially to fill the ventral defect and suture it to the external anal sphincter muscle medially and the coccygeus muscle and sacrotuberous ligament laterally.

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BOX 18.65 Possible Complications of Herniorrhaphy

- Hemorrhage
- Depression
- Anorexia
- Tenesmus
- Dyschezia
- Flatulence
- Hematochezia
- Rectal prolapse
- Anal sacculitis
- Fecal incontinence
- Urethral damage
- Dysuria
- Stranguria
- Bladder atony
- Bladder necrosis
- Urinary incontinence
- Intestinal necrosis
- Rectocutaneous or perineal fistula

can usually be prevented by appropriate antibiotic prophylaxis and surgical technique. Marked pain, non–weight-bearing lameness, and knuckling after surgery suggest sciatic nerve entrapment. If this is suspected, the offending suture should be removed immediately via a caudolateral approach to the hip. Other possible complications are listed in Box 18.65.

The use of synthetic, allograft, or autologous tissues to replace or augment perineal herniorrhaphy have been reported.[53,54]

PROGNOSIS

Medical and dietary management facilitate defecation in affected patients. The danger of prolonged medical therapy is that the bladder, intestine, or prostate will become trapped in the hernia, causing life-threatening consequences. The prognosis is fair to good when an experienced surgeon performs surgery. Patients with bladder retroflexion have the poorest prognosis. Preexisting neurologic abnormalities (i.e., anal sphincter incompetence or compromised urinary bladder innervation) are not corrected by the herniorrhaphy.

PERIANAL FISTULAE

DEFINITION

Perianal fistulae are chronically relapsing suppurative, progressive, deep ulcerating tracts in the perianal tissues. They have also been referred to as perianal sinuses, perineal fistulae, perianal fissures, furunculosis, pararectal fistulae, anusitis, fistulae-in-ano, and anorectal abscesses.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The etiology of perianal fistulae is unknown. The current theory is a multifactorial immune-mediated disease process. Other theories include poor conformation, crypt fecalith impaction and abscessation, and spread of infection from anal sacs or trauma. The combination of infection and abscessation of glands and hair follicles around the anus, moist contaminated anal environment, and a broad-based low-set tail conformation are believed to contribute to the formation of perianal fistulae. Colitis and enteral triggers such as dietary antigens, bacterial antigens, or superantigens may initiate the condition. German shepherds have a greater density of apocrine glands in the cutaneous zone of the anal canal than other breeds, which may predispose them to perianal fistulae. Bacterial infection may occur after development of cutaneous lesions. Endocrine studies have found no abnormalities in affected dogs.

Fistulae first appear as small, draining holes in perianal skin that is inflamed and hyperpigmented. As the disease progresses, these punctate holes enlarge and coalesce, forming large areas of ulceration and granulation. Tracts may extend into the deep perirectal tissues and anal sacs. These tracts are typically lined by squamous epithelium and are infiltrated with a mixture of lymphocytes, plasma cells, macrophages, neutrophils, and eosinophils. Hidradenitis, chronic necrotizing pyogranulomatous inflammation of the skin and hair follicles, cellulitis, dilated and inflamed lymphatics, necrosis, and fibrosis occur. Partial rectal stricture resulting from inflammatory infiltrates may occur.

DIAGNOSIS

The diagnosis is based on history, physical examination findings, and ruling out other differentials primarily by histopathology findings.

Clinical Presentation

Signalment

Perianal fistulae occur most commonly in German shepherds, but Irish setters also are predisposed. Various other breeds have been diagnosed with this condition, including collies, border collies, Old English sheepdogs, Labrador retrievers, English bulldogs, beagles, Bouvier des Flandres, spaniels, and mixed breeds. The disease appears to be more common in males than females (approximately 2:1), with a predominance in intact animals, but not all studies have identified a sex predilection. Perianal fistulae are extremely rare in cats. Perianal fistulae may occur at any age; however, the mean age of affected dogs is approximately 5 years.

History

Dogs with perianal fistulae are usually presented for treatment because of anal discomfort, constipation, diarrhea, odor, licking, scooting, tenesmus, dyschezia, ulceration, and/or purulent perianal discharge. Clinical signs of discomfort, licking, and scooting may be present before the fistulae are evident externally. Many owners have noticed perianal ulcers or tracts. Pain may cause dogs to become vicious when the tail or perineum is examined or manipulated. Weight loss, diminished appetite, and lethargy may occur.

Physical Examination Findings

Dogs with perianal fistulae often appear normal; however, some are thin with a poor hair coat (Box 18.66). The perianal area should be examined for fistulae that are recognized as dissecting ulcerative tracts usually with a malodorous mucopurulent discharge. The perineum is often painful, and affected dogs may snap, bite, or cry when the tail is lifted. Sedation or general anesthesia is often required for thorough perineal examination. Tracts may be single but typically are multiple, of varying depths, and epithelial lined (Fig. 18.152). Fistulae can be difficult to identify if there are only a few punctate lesions and minimal ulceration. The condition becomes more evident as tracts coalesce and swelling and inflammation develop. In severe cases, the entire circumference of the anus may be ulcerated. Previously unidentified tracts often become obvious when the dog is anesthetized and clipped. A rectal examination should determine the depth of involvement, the degree of fibrosis, and the relationship of

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BOX 18.66 Clinical Signs of Perianal Fistulae

- Tenesmus
- Dyschezia
- Hematochezia
- Constipation or obstipation
- Ribbon-like stool
- Increased defecation frequency
- Weight loss
- Perianal discharge: purulent, hemorrhagic
- Offensive odor
- Perianal licking
- Perianal pain
- Scooting
- Self-mutilation
- Low tail carriage



FIG. 18.152 Coalescing ulcers that characterize moderately severe perianal fistulae.

the anal sacs to fistulae. The tracts should be probed with a sterile instrument to determine their extent. Cannulating and flushing the anal sacs with sterile saline may demonstrate communication with adjacent fistulae. Anal stenosis and rectocutaneous fistulae may be identified during rectal examination.

NOTE Examination of the perineal area establishes the tentative diagnosis; however, histologic examination is necessary to rule out SCC, pythiosis, and other erosive conditions.

Diagnostic Imaging

Imaging is generally not necessary unless there is a suspicion of neoplastic involvement.

Laboratory Findings

Laboratory findings are nonspecific. Cytology reveals pyogranulomatous inflammation with a mixed bacterial population. Commonly isolated bacteria from deep, intraoperative perianal fistulae samples are *E. coli, Staphylococcus pseudintermedius,* α-hemolytic streptococci, and *Proteus mirabilis.* Bacterial contamination is believed to occur after ulceration. Tissue biopsy rules out neoplasia and pythiosis. Acute and chronic inflammation with fibrosis and granulation tissue are expected. Some tracts have an epithelial lining, whereas others involve the anal crypts and rectal mucosa. Histopathologic diagnosis of mild to moderate colitis may be identified if the colon is biopsied. A restrictive diet trial helps rule out adverse food reactions. Other diagnostics that may be performed are fecal parasite exams, fungal culture, and serology for pythiosis.

BOX 18.67 Perianal Fistula Treatment Goals

Goal 1: Alleviate signs of tenesmus, dyschezia, hematochezia, constipation, diarrhea, and pain. Reduce defecation frequency. Obtained by induction phase treatment lasting 8 to 20 weeks.

Goal 2: Reduce diameter, depth, extent, and recurrence of fistulae. Obtained by maintenance therapy at the lowest effective dose every 24–72 hours; may be required for life.

DIFFERENTIAL DIAGNOSIS

The early stages of anal SCC may resemble perianal fistulae. Other important differential diagnoses include perianal tumors, anal sac or rectal tumors, anal sac fistulae, atypical bacterial infections, pythiosis, and fistulae associated with tail-fold pyoderma.

MEDICAL MANAGEMENT

Medical management includes immunosuppression, hygiene, and dietary therapy. Management of perianal fistulae requires diligence, is frustrating for veterinarians and clients, and is uncomfortable for patients. Stool softeners may reduce dyschezia (see Box 18.52). Regular perianal cleansing and antibiotic therapy reduce inflammation but seldom allow the fistulae to heal and may allow the disease to progress. However, the administration of immunosuppressives (e.g., tacrolimus, prednisolone, cyclosporine, azathioprine) and antibiotics can effectively treat most perianal fistulae. Initial treatment is necessary for several months, and lifelong treatment may be required. If one drug combination is not achieving the treatment goal, then another drug protocol can be initiated (Boxes 18.67 and 18.68). Medical treatment can be expensive; however, continence is maintained. Treatment of concurrent diseases (e.g., hypothyroidism, IBD) facilitates treatment of perianal fistulae. Dogs with perianal fistulae and concomitant infiltrative bowel disease or colitis may improve with initial high-dose prednisolone therapy (i.e., 2 mg/kg PO given daily for 2 weeks). The prednisolone dose then is reduced (1 mg/kg PO given daily) and continued for 4 weeks. Prednisolone therapy directed at fistula is at a higher dose and usually combined with azathioprine (see Box 18.68).

Cyclosporine is effective in most dogs. The drug affects the immune response by blocking proliferation of activated T lymphocytes (T-helper cells). Adverse side effects have included hyporexia, vomiting, diarrhea, gingival hyperplasia, papilloma-like skin lesions, hypertrichosis, and increased hair shedding. Microemulsion formulations of cyclosporine are recommended because they are more bioavailable (Atopia, Neoral); however, absorption is erratic between dogs, and therapeutic drug monitoring is essential in dogs that do not respond to therapy. The recommended initial cyclosporine dosage is shown in Box 18.68. Trough blood cyclosporine concentrations should be measured 7 to 12 days after initiating therapy. Treatment is continued for at least

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BOX 18.68 Management of Perianal Fistulae in Dogs

Immunosuppressive/Immunomodulatory Therapy

Cyclosporine (Atopica, Neoral)[a] (± Ketoconazole)

3–5 mg/kg (initial dose) PO 2 hours before or after feeding, q12h; adjust this dose based on therapeutic drug monitoring to achieve a whole blood cyclosporine trough measurement of 300–600 ng/mL

Ketoconazole (Nizoral)

3–10 mg/kg PO, q24h (in this case, ketoconazole is being used to inhibit metabolism of cyclosporine and thereby increase blood levels; it is very important to measure trough blood levels of cyclosporine to determine further adjustments to dose of cyclosporine and ketoconazole)

Azathioprine (Imuran) (plus Metronidazole)

2.2 mg/kg/day EOD[b]

Metronidazole (Flagyl) (plus Azathioprine)

10–15 mg/kg PO q12h

Prednisolone (± Azathioprine ± Metronidazole)

2 mg/kg PO q24h for 2 weeks, 1 mg/kg PO qd for 4 weeks, then 1 mg/kg PO q48h maintenance

Tacrolimus 0.1%

Topical, thin film q12h for induction, then q24–72h for maintenance; apply with gloved hand

Hygiene Therapy

Clipping and cleaning
Keep area free of hair
Lavage once or twice daily and dry
Antimicrobial therapy:

- Oral—pending sensitivity use amoxicillin-clavulanic acid or metronidazole
- Topical—mupirocin

Stool Softener

(see Box 18.52)

Dietary Therapy

Trial diets with alternative protein source:

- Lamb and rice
- Fish and potato
- Kangaroo and oats
- Vegetarian

Surgical Therapy

Deroof and fulgurate or laser ablate
Excise persistent fistulae
Removed involved anal sacs

[a]Do not use Sandimmune.
[b]Giving the drug every other day is safer than giving it every day, but it will take longer to see beneficial effects.
EOD, Every other day; *PO,* orally.

16 weeks or 2 weeks beyond resolution of fistulae. The dosage and cost of cyclosporine can be reduced by oral administration of ketoconazole (see Box 18.68), which alters hepatic metabolism of cyclosporine. Lower doses of cyclosporine (5 mg/kg q24h) may be less expensive yet may diminish but not completely abolish clinical signs. Therapeutic drug monitoring becomes even more important when combining ketoconazole and cyclosporine.

Combination therapy using azathioprine and metronidazole (see Box 18.68) reduces perianal irritation and the severity and extent of lesions. Azathioprine suppresses both humoral and cell-mediated immunity. Side effects include GI upset, bone marrow suppression, hepatotoxicity, and pancreatitis. Metronidazole reduces fecal bacterial colonization and is antiprotozoal. Its potential side effects include anorexia, GI upset, and central vestibular signs. Surgery is recommended if improvement plateaus after 4 to 6 weeks of therapy. Azathioprine and metronidazole are continued for 3 to 6 weeks after surgery.

Topical tacrolimus is initially coadministered with other drugs unless the animal has a very mild case of perianal fistula. Tacrolimus is a calcineurin inhibitor with pharmacologic actions similar to, but 10 to 100 times more potent than cyclosporine. Potential side effects are stinging and burning. Application is initially twice daily but is then reduced to the lowest frequency that controls inflammation.

Monitoring medical therapy requires reevaluation every 3 to 5 weeks. Monitoring signs and mapping fistula help to assess the effectiveness of treatments and dosages. Dosages are modified according to the response noted. Hematologic and biochemical monitoring may be required, depending on the drugs used.

SURGICAL TREATMENT

Perianal fistulae were once considered a surgical disease, but now medical management is the initial treatment of choice. Surgery is recommended for fistulae resistant to medical therapy and those associated with the anal sacs. The goals of surgery are to eliminate necrotic or unhealthy tissue and stimulate second intention healing without causing fecal incontinence or anal stenosis. Numerous surgical procedures have been used to treat perianal fistulae, including superficial or radical excision, cryotherapy, fulguration, laser ablation, chemical cautery, and tail amputation. Staged procedures may be necessary during the initial months of treatment and may need to be repeated intermittently for life. Dogs with mild to moderate disease usually respond better to treatment than do those with severe disease.

Radical resection is the excision of all diseased skin, subcutaneous tissue, muscle, and fascia. The rectum is apposed to remaining skin with widely spaced interrupted sutures. The remainder of the defect is allowed to heal by second intention. Fecal incontinence is a common postoperative problem. Superficial resection (i.e., excision of all skin involved in the inflammatory process) is recommended with severe or unresponsive perianal fistulae. Debridement and fulguration of fistulae are less likely to cause fecal incontinence than extensive resection but tend to be ineffective in severe cases. Some surgeons recommend concurrent tail amputation. Anal sacculectomy (see p. 495) is needed when fistulae involve the anal sacs. Debridement and chemical cauterization (resection of epithelium overlying coalescing fistulous tracts followed by application of an irritant chemical to the underlying granulation tissue) may be performed using a strong iodine solution. This technique is less effective than debridement and fulguration or ablation but may be selected for patients with mild disease, when healing begins to lag, or small fistulae are identified after debridement and fulguration or ablation. Use of chemical cauterization at this time may eliminate the need for general anesthesia and reoperation.

Cryotherapy of perianal fistulae involves the application of a cryogen to destroy diseased tissue. Tissue that has been frozen necroses and then sloughs off during the subsequent 1 to 2

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weeks. The wounds heal by second intention. Appropriately controlling the freeze by using thermocouples is helpful. However, cryosurgery is not recommended because it is difficult to control the freeze; muscles and nerves are often inadvertently destroyed. Up to half of the patients have severe anal stenosis after cryosurgery. Other complications include flatulence, tenesmus, incontinence, diarrhea, and constipation.

Preoperative Management

Initiate medical management and only operate on persistent fistulae. Owners must be informed of the expected results of treatment, and their important postoperative role should be thoroughly explained. Owners must make a firm commitment of ability and willingness to provide long-term postoperative care. Fistula location should be mapped on a chart of the perianal region. Administration of stool softeners should be initiated several days before surgery (see Box 18.52). The colon should be evacuated and food withheld the day before surgery. Hot compresses should be applied to the perineum to help remove exudate and debris. Analgesics may be necessary if the patient objects to perianal manipulations. Prophylactic antibiotics effective against gram-negative and anaerobic bacteria should be given during induction of anesthesia.

Anesthesia

Anesthetic recommendations for animals undergoing perianal surgery are given on p. 483 and in Table 18.1.

Surgical Anatomy

The surgical anatomy of the perianal region is on p. 483.

Positioning

The patient should be positioned in ventral recumbency with the hind legs over the end of the table. The pelvis should be elevated with padding and the tail secured over the back. The end of the table should be padded to prevent pressure on the femoral nerves. As an alternative, a padded perineal stand may be used.

SURGICAL TECHNIQUES

Surgery is seldom indicated for treatment of perianal fistulae.

POSTOPERATIVE CARE AND ASSESSMENT

Systemic (see Table 13.2) or epidural analgesics (see Table 13.5) should be used as needed. The perineum should be cleaned three or four times daily, especially after defecation, with warm saline (water) or a dilute antiseptic solution. Using a hose and warm tap water is a convenient and acceptable method of cleaning. An Elizabethan collar, bucket, or sidebars should be used to prevent self-mutilation, and stool softeners should be given to facilitate fecal passage during the first 3 to 4 weeks (see Box 18.52). The stool softener should make the stool soft but not sticky or pasty. A low-bulk diet should be fed. Giving antibiotics effective against gram-negative and anaerobic bacteria is helpful although not essential. The size of the debrided areas should be mapped immediately after surgery and at each reevaluation to allow accurate monitoring. The patient should be reevaluated every 2 to 4 weeks, and nonhealing or new fistulae should be treated as needed. After the fistulae have resolved, owners should keep the perineum clipped and clean. They should check for new fistulae monthly.

BOX 18.69 Possible Complications of Wide Resection of Fistulae

- Fecal incontinence
- Flatulence
- Diarrhea
- Tenesmus
- Dyschezia
- Constipation
- Anal stenosis
- Recurrence

COMPLICATIONS

Fecal incontinence, anal stenosis, and recurrence sometimes precipitate euthanasia. These are common complications with some surgical techniques. Flatulence, tenesmus, constipation, and diarrhea may also occur. Complications are more common and severe after radical resection (Box 18.69) than after superficial resection or fulguration and ablation. Hair loss and lameness may occur with cyclosporine therapy, but these resolve when therapy is discontinued. Recurrence often may be controlled with a 7- to 14-day course of cyclosporine.

PROGNOSIS

Medical therapy with cyclosporine and tacrolimus or other immunomodulating drugs is effective in resolving most fistulae and reducing the severity of others. Mild perianal fistulae may be controlled if owners are diligent about daily perianal care. The area must be kept clean and dry to prevent progression of the disease. The prognosis after surgery alone is fair to poor, depending on the severity of the disease at the time of surgery and the owner's postoperative compliance. Early diagnosis and preoperative medical therapy allow less radical surgical procedures with fewer postoperative complications. Recurrence is common with either medical or surgical therapy. Many animals are euthanized because of pain, lack of response to treatment, recurrence, and/or client frustration.

RECTAL PROLAPSE

DEFINITION

Rectal prolapse *(anal prolapse)* is a protrusion or eversion of the rectal mucosa from the anus.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Rectal prolapse is principally associated with endoparasitism or enteritis in young animals, and tumors or perineal hernias in middle-aged and older animals. However, any condition that causes tenesmus may cause rectal prolapse (Box 18.70). Weakness of perirectal and perianal connective tissues or muscles, uncoordinated peristaltic contractions, and inflammation or edema of rectal mucous membranes predisposes patients to rectal prolapse.

Rectal prolapse may be complete or incomplete. Incomplete prolapse involves only mucosa. Any part of the entire anorectal circumference may be affected. Complete prolapse involves all layers of the rectal wall and the entire circumference. The amount of eversion increases with continued straining, varying from a few millimeters to many centimeters. Everted tissue becomes edematous, preventing spontaneous retraction into the pelvic

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BOX 18.70 Conditions Associated With Rectal Prolapse

- Endoparasitism
- Enteritis
- Intestinal foreign bodies
- Dystocia
- Urolithiasis
- Constipation
- Congenital defects
- Sphincter laxity
- Prostatic disease
- Perineal surgery



FIG. 18.153 Rectal prolapse. Note the large amount of everted rectum that must be differentiated from an intussusception.

canal. Continued exposure causes excoriation, bleeding, desiccation, and necrosis.

DIAGNOSIS

Clinical Presentation

Signalment

Rectal prolapse occurs in dogs and cats, with no documented breed predisposition. However, it may occur more often in Manx cats because of their anal laxity. It may occur at any age but is more common in young animals.

History

Straining or recent perineal surgery is common. Constipation, diarrhea, prostatitis, urinary tract infections, dyspnea, and dystocia may produce tenesmus. Perineal or perianal irritation from trauma or surgery may also cause straining and rectal prolapse.

Physical Examination Findings

The patient's physical status is unpredictable because of the numerous possible causes of rectal prolapse. Protrusion of anorectal mucosa is obvious on physical examination. The degree of prolapse may vary from a few millimeters to several centimeters (Fig. 18.153). Rectal prolapse must be differentiated from an ileocolic intussusception that is protruding from the anus (discussed later).

Diagnostic Imaging

Imaging may help identify the cause of the prolapse.

Laboratory Findings

Laboratory tests are nonspecific for rectal prolapse but may identify the cause and define the patient's physiologic status. Parasites and acute enteritis are common in young animals.

DIFFERENTIAL DIAGNOSIS

The primary differential diagnosis for rectal prolapse is intussusception. Insertion of a finger (preferred) or probe (i.e., thermometer or smooth tube) alongside the prolapsed mass is possible with an intussusception but not with a rectal prolapse.

NOTE Be sure to differentiate prolapse from intussusception. Palpation of the outer surface of the prolapse identifies a fornix.

MEDICAL MANAGEMENT

The treatment and prognosis depend on the cause, degree of prolapse, chronicity, and whether it is a recurrent prolapse. Acute rectal prolapse is easily treated, but chronic disease may require resection. Manual reduction and placement of a purse-string suture around the anus are recommended for acute prolapses with minimal tissue damage and edema (Fig. 18.154A–B). Warm saline lavages, massage, and lubrication (e.g., with a water-soluble gel) should be applied to the everted tissue before digital reduction. Giving a retention enema of several milliliters of Kaopectate or barium sulfate will help alleviate further straining. A purse-string suture tight enough to maintain prolapse reduction without interfering with passage of soft stool should be placed. Epidural anesthesia may also help prevent additional straining and reprolapse. Most rectal prolapse patients respond well to manual reduction when the cause is treated and resolved.

SURGICAL TREATMENT

Nonreducible or severely traumatized prolapses require amputation. Colopexy should be performed when rectal prolapse repeatedly recurs after manual reduction or amputation (see p. 469).

NOTE Be sure to identify potential underlying causes of the prolapse and treat them.

Preoperative Management

Surgery should be prompt to prevent further trauma to the everted tissues. Extensive colorectal preparation is unnecessary. Prophylactic antibiotics effective against gram-negative and anaerobic bacteria (see Box 18.48) should be given at induction of anesthesia. The exposed tissue should be lavaged with warm sterile saline and lubricated with a water-soluble gel.

Anesthesia

Anesthetic recommendations for animals undergoing rectal and perineal surgery are given on p. 483 and in Table 18.1.

Surgical Anatomy

The surgical anatomy of the rectum and perineum are presented on p. 483.

Positioning

After the perianal area has been clipped and aseptically prepared for surgery, the everted tissue should again be lavaged and lubricated. The patient should be positioned in ventral recumbency with the hind legs over the end of the table. The pelvis

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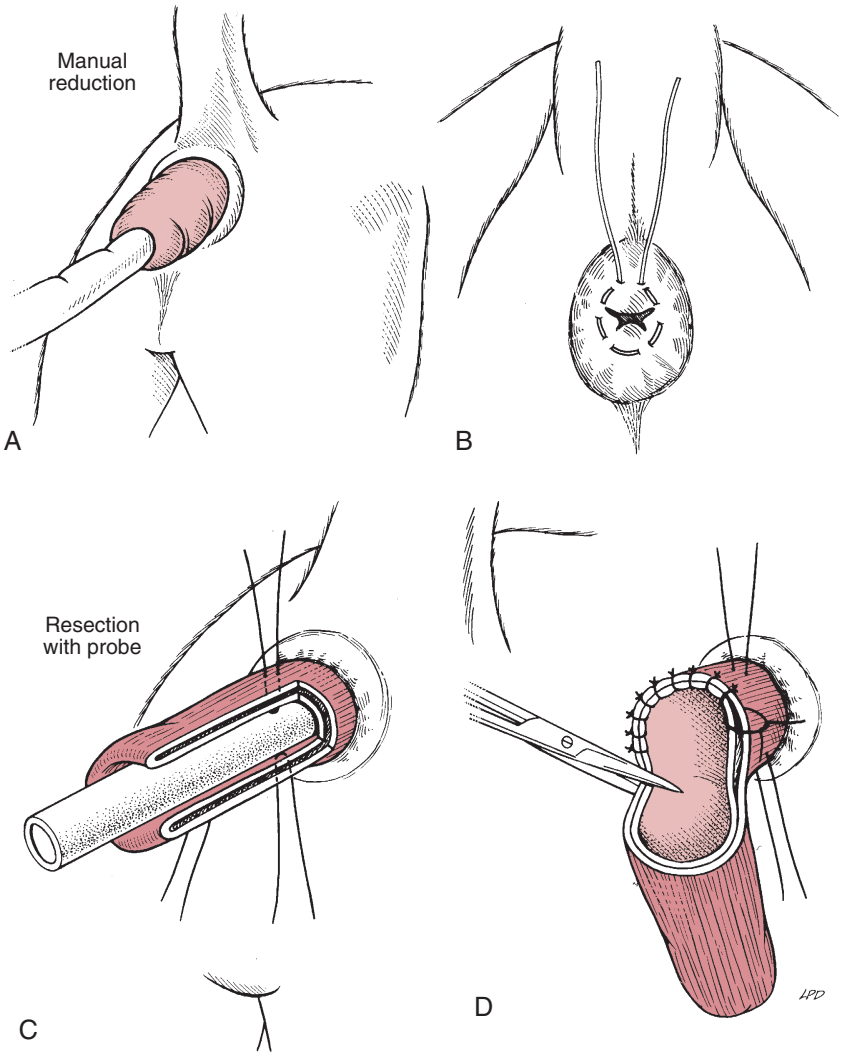


FIG. 18.154 (A–B) Digitally reduce small prolapses with healthy mucosa and place a purse-string suture around the rectum. (C–D) Resect irreducible or traumatized prolapses. Place a probe in the rectal lumen and three or four stay sutures in the rectal wall. Make a full-thickness incision through the prolapsed tissue one-third to half the distance around the circumference. Appose the edges with simple interrupted sutures. Then complete the resection.

should be elevated with padding and the tail secured over the back. The end of the table should be padded to prevent pressure on the femoral nerves. As an alternative, a perineal stand may be used.

SURGICAL TECHNIQUE

Place a probe into the rectal lumen to serve as a guide (see Fig. 18.154C). Place three horizontal mattress stay sutures (at the 12 o'clock, 5 o'clock, and 8 o'clock positions) through all layers of the prolapse just cranial to the proposed transection site. These sutures should enter the rectal lumen with the needle being deflected by the probe before being passed through the rectal tissues again. *Transect the traumatized tissue in stages caudal to the stay sutures. After each stage of the resection, anatomically appose the transected edges with simple interrupted sutures (e.g., 3-0 or 4-0 monofilament absorbable; see Fig. 18.154D). Space the sutures approximately 2 mm apart and 2 mm from the cut edge. Inspect the anastomosis for gaps between sutures. Remove the stay sutures and gently replace the anastomotic site in the pelvic or anal canal. Place a purse-string suture around the anus if postoperative tenesmus is expected.*

POSTOPERATIVE CARE AND ASSESSMENT

The cause of the prolapse must be treated to prevent recurrence. Retention enemas of Kaopectate or epidural opioids may eliminate postoperative tenesmus for several hours (see p. 505). Systemic analgesics should be given if necessary (see Table 13.2). A low-fiber diet should be fed while the purse-string suture is in place. The purse-string suture can generally be removed 3 to 5 days after manual reduction and 1 to 2 days after resection. Stool softeners should be given for 2 to 3 weeks after resection (see Box 18.52). Amputees must be monitored for leakage from the surgical site.

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COMPLICATIONS

Possible complications of manual reduction of rectal prolapses are tenesmus, dyschezia, hematochezia, and recurrence. Additional complications of resection include hemorrhage, leakage, anal stenosis, infection, dehiscence, and fecal incontinence.

PROGNOSIS

Incomplete prolapses that occur during defecation may reduce spontaneously. The prognosis for chronic rectal prolapse without manual reduction or surgery is poor. Chronically exposed rectal mucosa is traumatized by licking, sitting, and environmental exposure, ultimately becoming necrotic with secondary sepsis. The prognosis for most animals treated surgically is good provided the primary cause of tenesmus or irritation is appropriately treated.

FECAL INCONTINENCE

DEFINITIONS

Fecal incontinence is the inability to voluntarily control defecation. **Reservoir incontinence** results from a failure of the large bowel to adapt to and contain the colorectal contents. **Sphincter incontinence** is a failure of the sphincter mechanism to resist propulsive forces in the rectum, so that feces are involuntarily passed.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Fecal incontinence is uncommon in dogs and cats. Fecal continence depends on maintenance of colonic reservoir function and anal sphincter control. Muscles involved in fecal continence include the internal anal sphincter, external anal sphincter, rectococcygeus, levator ani, and coccygeus muscles. As fecal material is propelled distally into the terminal rectum, the rectum distends and the internal anal sphincter dilates while the external anal sphincter and caudal portion of the levator ani muscles contract. Subsequently, propulsive contractions diminish, and normal resting tone is restored in 2 to 3 minutes. Thus the rectum distends and adapts with each new bolus to increase its storage capacity, and a resting anorectal high-pressure zone is created by the internal anal sphincter, external anal sphincter, and caudal portion of the levator ani muscles. The internal anal sphincter contributes 50% to 80% of the resting tone in the high-pressure zone. The external anal sphincter is tonically active but contributes minimally to the resting tone; its short contractions resist peristaltic waves. The role of the levator ani is uncertain. Animals posture and increase abdominal pressure by closing the glottis, fixing the diaphragm, and contracting the abdominal wall when defecation is appropriate. This causes the external anal sphincter to relax and the rectococcygeus, levator ani, and coccygeus muscles to contract.

Reservoir incontinence (Box 18.71) is usually characterized by frequent, conscious defecation. Loss of reservoir continence results in abnormally soft, unformed, or liquefied feces. Loss of reservoir continence may be caused by diffuse colonic disease resulting in diminished distensibility, or it may occur secondary to reduced colonic length after resection (e.g., two-thirds or more). The small intestine increases water absorption and capacity after subtotal colectomy; therefore many animals regain reservoir continence.

Sphincter incontinence (see Box 18.71) may be neurogenic or nonneurogenic. Partial fecal incontinence may occur if only one muscle group malfunctions. Loss of sensory receptors and the afferent limb of the continence mechanism can be secondary to rectal resection. An adequate cuff of rectal muscularis must be preserved to maintain sphincter continence. Efferent neural control is lost when caudal rectal nerves are damaged. Sacral spinal cord lesions of the S1–S3 cord segments (L5 vertebral level in dogs and L6 in cats) damage the cell bodies of the pudendal nerve. Peripheral pudendal nerve damage can occur anywhere from the cauda equina distally. Unilateral pudendal nerve damage causes fecal incontinence for only 3 to 4 weeks because of cross-innervation and muscle fiber decussation. Bilateral pudendal nerve damage causes permanent incontinence. Nonneurogenic sphincter incontinence occurs secondary to physical disruption of the external anal sphincter after anorectal trauma, rectal prolapse, severe perianal disease (inflammation, tumors), or surgical resection. The incidence of incontinence increases as external anal sphincter resection approaches 180 degrees. Resection of more than half of the external anal sphincter usually results in fecal incontinence.

BOX 18.71 Causes of Fecal Incontinence

Reservoir Incontinence

- Diffuse colonic disease resulting in decreased distensibility
- Reduced colonic length after resection (e.g., two-thirds or more)

Sphincter Incontinence

- Rectal resection (>4 cm of terminal rectum)
- Inadequate cuff of terminal rectal mucosa (less than approximately 1.5 cm)
- Damage to the caudal rectal nerves
- Sacral spinal cord lesions of S1–S3 cord segments (L5 vertebral level in dogs and L6 in cats)
- Peripheral pudendal nerve damage
- Physical disruption of the external anal sphincter after:
  - Anorectal trauma
  - Rectal prolapse
  - Severe perianal disease (e.g., inflammation, tumors)
  - Surgical resection
- Resection of more than half the external anal sphincter

DIAGNOSIS

Clinical Presentation

Signalment

Any breed or sex of dog or cat may have fecal incontinence. Manx cats may be predisposed because of anal laxity resulting from abnormal innervation. Dogs with perineal fistulae (see p. 501) and those with surgery of the anal region (e.g., anal sac ablation) may develop fecal incontinence. Fecal incontinence may occur in any age animal, although 50% of affected animals are 11 years or older.

History

The history is important for differentiating reservoir incontinence from sphincter incontinence and for determining possible causes. Animals are presented for treatment because of inappropriate defecation. Some affected animals posture and void normally

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despite having inappropriate defecation. A fecal bolus may be voided when barking, coughing, or rising from a recumbent position, without posturing or recognition of the event. It is important to find out if the onset of incontinence is associated with recent colorectal or perineal surgery, trauma, perianal disease, or neurologic disease. Signs of incontinence vary from occasional incontinence to perineal soiling and fecal dribbling. Reservoir incontinence may be associated with frequent defecation, tenesmus, hematochezia, and mucoid stools.

Physical Examination Findings

Patients with fecal incontinence often appear normal. Rectal and perineal examination may reveal colorectal or perianal disease. Anal sphincter tone may be diminished, the anus dilated, and/or the rectal mucosa prolapsed. The abdomen should be palpated to determine the size of the colon and bladder tone, as well as how difficult it is to express the urinary bladder, and a thorough neurologic examination should be performed. Self-inflicted skin trauma suggests paresthesia. Hyperesthesia may be detected in the lumbosacral area. Hind limb paresthesia, urinary incontinence, and hyperesthesia suggest cauda equina syndrome. A Foley catheter can be inflated in the rectum to determine if the dog has a normal anal reflex.

Diagnostic Imaging

Survey radiographs should be evaluated for colorectal or pelvic canal masses, vertebral abnormalities (e.g., lumbosacral stenosis or fracture), or pelvic fractures. Myelography, epidurography, CT, or MRI helps diagnose spinal cord or cauda equina lesions. Endoscopic biopsy of the colon and anus are indicated if reservoir incontinence is suspected.

Laboratory Findings

A hemogram, serum biochemistry profile, urinalysis, and fecal examination are recommended. Hematologic and serum biochemistry values may suggest the etiology. Electromyography and manometry may help assess the anorectal sphincter complex in some cases. These examinations help differentiate reservoir incontinence from sphincter incontinence and neurogenic incontinence from nonneurogenic incontinence. Electromyography can reveal denervation or myopathy. Manometry gives anal and colorectal pressure profiles and evaluates the degree of impairment of anorectal tone and internal and external anal sphincter function. The presence of an intact reflex arch may be determined more simply by inflating a Foley catheter in the rectum and observing the response.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses and possible causes of fecal incontinence include anal disease (e.g., anal sacculitis, dermatitis, endoparasites, perianal fistulae, or tumors), colorectal disease (e.g., colitis, neoplasia), poor diet, trauma to or denervation of the muscles of continence, damage to somatic peripheral nerves (i.e., pudendal, or S2-S3-Cd1 spinal nerves), damage to autonomic peripheral nerves (i.e., pelvic plexus, pelvic nerves), cauda equina syndrome, central nervous system injury (e.g., sacral spinal segments, spinothalamic tracts, frontal cortex), and behavioral abnormalities. Fecal incontinence must also be differentiated from partial or type 3 atresia ani and congenital or traumatic rectovaginal or rectourethral fistula. With these conditions feces may mix with urine, causing perineal soiling and inappropriate passage of feces.

BOX 18.72 Pharmacologic Therapy to Slow Bowel Transit Time

Loperamide Hydrochloride (Imodium)
Dogs: 0.1 mg/kg PO q8–12h
Cats: 0.08–0.16 mg/kg PO q12h

PO, Orally.

MEDICAL MANAGEMENT

The causative disease or condition should be treated if possible. Owner acceptance may require that the pet be kept outdoors. Many patients are euthanized because owners are unable or unwilling to tolerate incontinence. The goals of medical management are to reduce fecal water content and fecal bulk, slow transit time, and increase anal sphincter tone. Symptomatic medical management includes dietary change, pharmacologic therapy, and induced defecation. A low-residue diet (i.e., cottage cheese and rice) reduces fecal volume by up to 85% and lessens the frequency of defecation. Opioids (Box 18.72) promote segmental contractions, slowing bowel transit time and increasing water absorption. Enemas and rectal stimulation can promote colonic evacuation at appropriate times and help prevent inappropriate defecation.

SURGICAL TREATMENT

Sphincter-enhancing procedures for treating fecal incontinence in animals have been inadequately investigated; described surgical techniques must be considered investigational. Anal sphincter function has been enhanced by using a fascial sling, silicone elastomer (Silastic sheeting #501-3), or dynamic myoplasty.

Preoperative Management

The colon should be evacuated with laxatives, oral cathartics, and/or enemas, and any remaining fecal material should be manually evacuated after induction of anesthesia. Antibiotics effective against gram-negative and anaerobic bacteria (see Box 18.48) should be given after induction.

Anesthesia

General anesthesia is recommended. Anesthetic recommendations for animals undergoing rectal and perineal surgery are given on p. 483 and in Table 18.1.

Surgical Anatomy

The surgical anatomy of the rectum and perineum is presented on p. 483.

Positioning

Clip and aseptically prepare the perineum or ventral abdomen. The patient should be positioned in ventral recumbency with the hind legs over the end of the table. The pelvis should be elevated with padding, and the tail should be secured over the back. The end of the table should be padded to prevent pressure on the femoral nerves. As an alternative, a perineal stand may be used.

SURGICAL TECHNIQUES

Fascial Sling

Harvest two strips of tensor fasciae latae (6 × 0.5 cm) from the lateral thigh, suture them together, and transfer to the anus. Close

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the fascial defect and appose the subcutaneous tissues and skin. Make a 3- to 4-cm incision on each side of the anus just lateral to the tail base. Connect the two incisions by undermining the tissue ventral to the anus with a curved hemostat. Direct the fascial strip around the ventral anus using curved hemostats. Suture the fascia to the coccygeus muscle at the base of the tail on one side, pull it snug, and suture it to the tail base on the opposite side using 2-0 or 3-0 monofilament, nonabsorbable suture (nylon, polybutester, or polypropylene). As an alternative, secure one end to the coccygeus muscle and direct the other end of the fascial strip over the base of the tail, pull it snug, and suture it to itself at the tail base. Lavage the area and reappose the subcutaneous tissues and skin.

Implantation of a Silicone Elastomer

Make two incisions lateral to the anus as described previously (Fig. 18.155A). Make a tunnel ventral and dorsal to the anus with curved hemostats. Insert the implant through the tunnels (see Fig. 18.155B). Overlap the implant ends and pull it snug around the anus. To prevent overtightening, place a 1-cm probe in the rectum while the implant is tightened (see Fig. 18.155C). Suture the overlapping ends of the implant together with nonabsorbable, monofilament suture (e.g., 2-0 or 3-0 nylon, polybutester, or polypropylene). Lavage the area and reappose the subcutaneous tissues and skin.

POSTOPERATIVE CARE AND ASSESSMENT

Analgesics should be given as needed, and antibiotics should be continued for several days to minimize the risk of implant infection. Stool softeners should be administered (see Box 18.52), and a low-residue diet fed. The animal should be monitored for infection and effectiveness of the procedure. Tenesmus is expected for several days.

COMPLICATIONS

The major complications associated with these procedures are failure or recurrence of incontinence. Slings often loosen slightly after implantation. Correction of incontinence may be only partial, and some signs may persist. Infection, dehiscence, and sloughing of the implant are risks. Persistent tenesmus and obstruction may occur if the sling is too snug.

PROGNOSIS

The prognosis depends on the type and extent of the incontinence. Complete lower motor neuron incontinence is incurable. Improved sphincter function is expected after surgery, although

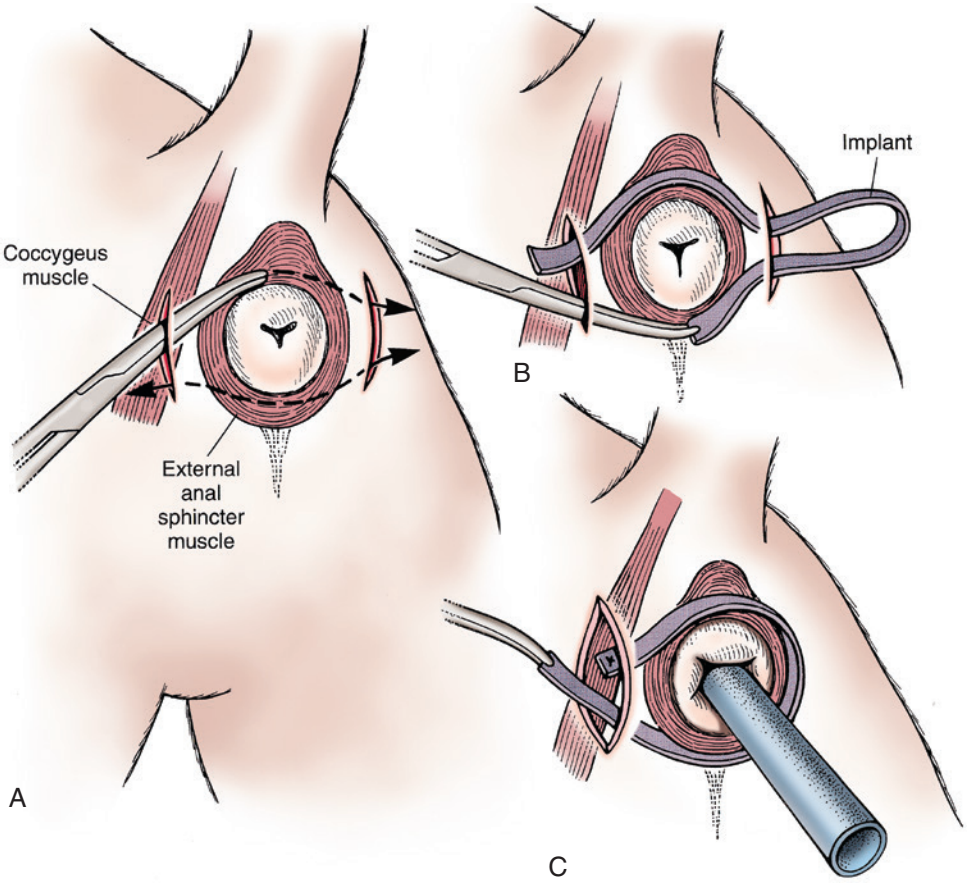


FIG. 18.155 Fecal continence may be improved by enhancing the function of the anal sphincter with an implant. (A) Make 3- to 4-cm incisions on each side of the anus. Connect the incisions with a tunnel dorsal and ventral to the anus *(dashed lines)*. (B) Direct the implant through the tunnels. (C) Secure the implant to the coccygeus muscle, pull it snug around a probe, and secure it to itself.

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signs may worsen with loosening of the implant. Muscle trauma or irritation often causes partial or temporary incontinence, which improves as the muscles heal.

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19

Surgery of the Abdominal Cavity



GENERAL PRINCIPLES AND TECHNIQUES

DEFINTIONS

Celiotomy is a surgical incision into the abdominal cavity; the term **laparotomy** is often used synonymously, although it technically refers to a flank incision. A sudden onset of substantial clinical signs referable to the abdominal cavity (e.g., abdominal distention, pain, vomiting) is called an **acute abdomen**. **Abdominal evisceration** is the herniation of the peritoneal contents through the body wall with exposure of the abdominal viscera.

PREOPERATIVE CONCERNS

Celiotomy is performed for various reasons; it may be indicated for diagnostic (e.g., biopsying an organ) and/or therapeutic reasons. Many animals undergoing abdominal exploratory surgery have chronic disease, but some patients require emergency abdominal surgery because of acute clinical signs. Some conditions are life-threatening (e.g., gastric dilatation-volvulus, intestinal perforation, severe hemorrhage), and appropriate therapy must be started promptly. Conditions that require surgery must be differentiated from those that can be managed medically. Although obviously unnecessary surgery must be avoided, surgery cannot always be delayed until one is certain the patient will benefit from it.

The decision to operate is based on the history, physical examination findings, imaging studies, and laboratory analyses. Physical examination can be unreliable in predicting severity of abdominal trauma. Examining patients with acute abdominal disease, particularly that associated with trauma, is often inaccurate because of the patient's condition at the time of examination and the fact that there is typically a delayed development of clinical signs associated with traumatic injuries. Depressed or lethargic animals may not show pain during abdominal palpation. Clinical signs of hemorrhage are often inapparent immediately after trauma; delays of 3 to 4 hours between injury and development of shock and collapse are common in patients with hepatic or splenic lacerations. Therefore animals that have suffered traumatic injuries should be closely observed for at least 8 to 12 hours. Life-threatening hemorrhage becomes apparent before this time in most cases. However, animals with traumatic bilious peritonitis may not have overt clinical signs for days or even weeks. Likewise, traumatic mesenteric avulsion is seldom associated with clinical signs until peritonitis develops, usually several days after injury. Diagnostic tests such as abdominal ultrasound (focused assessment with sonography for trauma [FAST], see pp. 536 and 29) may help identify patients with significant abdominal trauma and hemoperitoneum before overt clinical signs develop.

NOTE Be aware that overt clinical signs associated with mesenteric avulsions or rupture of the biliary tract may not become evident for days to 2 weeks after injury.

Preoperative management of most animals undergoing exploratory laparotomy is dictated by the underlying abdominal disease. General observations include noting the animal's attitude and posture, temperature, respiratory rate and effort, and heart rate and rhythm. Abdominal auscultation, percussion, and palpation plus rectal examination are indicated. Serial examinations are important to detect trends or deterioration in the patient's status. An intravenous catheter should be placed for fluid and drug administration, and blood samples should be drawn. Useful initial bloodwork in an animal with acute abdomen includes complete blood count, platelet count, serum total protein and glucose concentrations, and blood urea nitrogen. Other laboratory tests (e.g., serum biochemistry profile, clotting parameters) can be performed, depending on the animal's condition and the suspected underlying disease. Urine may be collected by means of cystocentesis or catheterization for urinalysis. An indwelling urinary catheter may be used to quantitate urinary output if necessary. Abdominal radiographs/ultrasound may detect peritoneal fluid (e.g., uroabdomen, peritonitis) or abnormal accumulations of air. Animals with acute abdominal signs of uncertain origin may have diagnostic peritoneal lavage (DPL; see p. 534) performed if radiographs, ultrasonography, and computed tomography (CT) (if available) are nondiagnostic. DPL may be most useful in the hemodynamically unstable patient who has an equivocal or negative FAST examination. Electrolyte and hydration abnormalities should be corrected before surgery.

NOTE If you note free air in the abdominal cavity of an animal that has suffered a recent traumatic injury, consider exploratory surgery; this finding may indicate rupture or perforation of the gastrointestinal (GI) tract.

Major abdominal evisceration may occur in dogs secondary to postsurgical dehiscence or trauma. In affected animals, the intestines are typically eviscerated and may have gross contamination with dirt or other debris (e.g., cat litter). Regardless of the inciting cause, exposure and contamination of the abdominal viscera warrants immediate surgical intervention. Postoperative

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major abdominal evisceration is most commonly associated with ovariohysterectomy, which is likely due to the fact that it is one of the most frequently performed abdominal procedures in small animals.

ANESTHETIC CONSIDERATIONS

The anesthetic management of animals with abdominal disease depends on the underlying disease. Animals that are stable can be premedicated with a benzodiazepine plus an opioid; an alpha-2 agonist or alfaxalone plus an opioid (cats and small dogs); or ketamine, an opioid, and a benzodiazepine (Table 19.1). They can be induced with propofol, ketamine, alfaxalone, or etomidate given intravenously to effect (see Table 19.1). Table 19.2 provides suggested anesthetic protocols for animals that are in shock or are debilitated.

ANTIBIOTICS

The appropriate use of antibiotics in patients undergoing abdominal surgery depends on the underlying disease, the animal's overall general health, and length and type of surgical procedure (see Chapter 9).

SURGICAL ANATOMY

The rectus sheath is composed of an external and internal leaf (Fig. 19.1). The external leaf is formed by the aponeurosis of the external abdominal oblique muscle and a portion of the aponeurosis of the internal abdominal oblique muscle. The aponeurosis of the transversus abdominis muscle joins the external leaf near the pubis (see Fig. 19.1). The internal leaf consists of a portion of the aponeurosis of the internal abdominal oblique muscle, the aponeurosis of the transversus abdominis muscle, and the transversalis fascia. The internal leaf disappears in the caudal third of the abdomen where the aponeurosis of the internal abdominal oblique muscle joins the external leaf, leaving the caudal rectus abdominis muscle covered only by a thin sheet of transversalis fascia and peritoneum (see Fig. 19.1).

NOTE The linea alba is easier to locate near the umbilicus because it becomes thinner near the pubis.

SURGICAL TECHNIQUES

The abdomen is generally explored by means of a ventral midline incision. In most animals the entire abdomen, including the inguinal areas and caudal thorax, should be prepared for aseptic surgery to allow extension of the incision into thoracic or pelvic cavities if necessary. Prepping too small an area is a common mistake, particularly for abdominal exploration in trauma patients. To visualize all abdominal structures adequately, the incision must extend from the xiphoid process to the pubis. If only a specific abdominal structure will be examined, a shorter incision can be made. A caudal abdominal incision extending from umbilicus to pubis is adequate for bladder exploration; similarly, a cranial abdominal incision (i.e., umbilicus to xiphoid process) allows evaluation of liver and stomach. Occasionally, the midline incision is extended laterally at the xiphoid process (1 cm caudal to the last rib) to facilitate exposure of the liver, biliary system, and diaphragm. A paracostal (paralumbar) celiotomy can be used to expose the kidneys and adrenal glands; it is most commonly used for unilateral adrenalectomy.

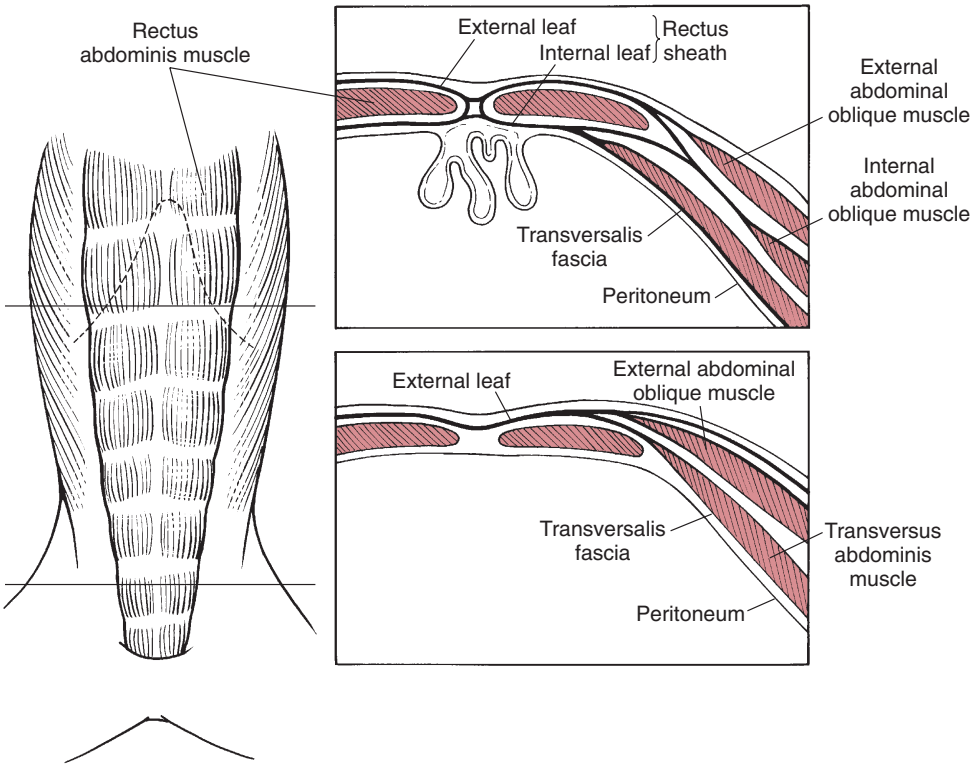


FIG. 19.1 Anatomy of the rectus sheath.

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TABLE 19.1 Anesthetic Considerations in the Stable Patient Undergoing Abdominal Surgery

Preoperative Considerations

Associated Conditions	• Minimal; usually healthy
Bloodwork	• HCT • TP • In patients older than 5–7 y, consider electrolytes, liver enzymes, BUN, and Cr
Physical Exam	• May be painful if incarcerated hernia

Premedications	• If patient is anxious give:
	  • Diazepam (0.2 mg/kg IV), *or*
	  • Midazolam (0.2 mg/kg IV, IM), *plus*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM in dogs and 0.05–0.2 mg/kg IV, IM in cats) *or*
	  • Oxymorphone (0.1–0.2 mg/kg IV, IM), *or*
	  • Morphine[b] (0.1–0.5 mg/kg IV, IM), *or*
	  • Buprenorphine (0.005–0.02 mg/kg IV, IM) (use if only moderate pain anticipated)

Intraoperative Considerations

Induction	• If premedicated, give:
	  • Propofol (2–4 mg/kg IV), *or*
	  • Alfaxalone (2–3 mg/kg IV)
	• If no premeds given, give:
	  • Propofol (4–8 mg/kg IV), *or*
	  • Alfaxalone (2–5 mg/kg IV), *or*
	  • Ketamine (5 mg/kg IV with diazepam or midazolam as above)

Maintenance	• Isoflurane or sevoflurane, *plus*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV PRN in dogs and 0.05–0.2 mg/kg IV PRN in cats), *or*
	  • Morphine (0.1–0.5 mg/kg IV PRN), *plus PRN*
	  • Fentanyl (2–30 μg/kg IV PRN) for short-term pain relief or as a CRI, *plus*
	  • Ketamine low dose (0.5–1 mg/kg IV), *or*
	  • Ketamine CRI (0.5 mg/kg IV loading dose, then 10 μg/kg/min IV), *plus*
	  • Lidocaine CRI (1–2 mg/kg IV loading dose, then 1–3 mg/kg/h IV in dogs)
	NOTE: each CRI can be given alone or in combination

Fluid needs	• 5–10 mL/kg/h plus 3 × EBL
Monitoring	• Blood pressure • HR • Respiratory rate • SpO_2 • Temperature • End-tidal CO_2
Blocks – Epidurals	• Morphine (0.1 mg/kg preservative free), *or* • Buprenorphine (0.003–0.005 mg/kg diluted in saline) NOTE: can give bupivacaine (0.5 mg/kg) in combination with either morphine or buprenorphine to provide a longer duration of action

Postoperative Considerations

Analgesia	• Fentanyl CRI (2–30 μg/kg/h IV), *or*
	• Hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs), *or*
	• Morphine (0.1–1 mg/kg IV, IM q3–4h in dogs and 0.1–0.5 mg/kg IV, IM q3–4h in cats), *or*
	• Oxymorphone (0.1–0.2 IV, IM), *or*
	• Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q3–4h in dogs and 0.05–0.2 mg/kg IV, IM q3–4h in cats), *plus if necessary*
	• Ketamine CRI (2–10 μg/kg/min IV), *or*
	• Dogs:
	  • Carprofen (2.2 mg/kg q12h PO), *or*
	  • Deracoxib (3–4 mg q24h PO for <7 days), *or*
	  • Meloxicam (0.1–0.2 mg/kg once SC or PO then 0.1 mg/kg PO q24h)
	• Cats:
	  • Meloxicam (0.05–0.1 mg/kg PO q24h for 1–3 days), *or*
	  • Robenacoxib (2 mg/kg SC after surgery), then for:
	  • ≤3 days:
	  if 2.5–6 kg; 6 mg/cat PO q24h;
	  if 6.1–12 kg; 12 mg/cat PO q24h
	  • 3–11 days: 1 mg/kg q24h PO

Monitoring	• SpO_2 • Blood pressure • HR • Respiratory rate • Temperature
Bloodwork	HCT and TP if large blood loss
Estimated pain score	Can be severe depending on etiology and/or procedure. Some of these patients have been in chronic pain.

[a]Monitor for hyperthermia in cats.
[b]Give slowly to prevent histamine release.
BUN, Blood urea nitrogen; *Cr,* creatinine; *CRI,* constant-rate infusion; *EBL,* estimated blood loss; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscularly; *IV,* intravenous; *PO,* orally; *PRN,* as needed; *q,* every; *SC,* subcutaneously; *SpO_2,* oxygen saturation via a pulse oximeter; *TP,* total protein.

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TABLE 19.2 Anesthetic Considerations in the Septic Patient

Preoperative Considerations

Associated conditions	• Dehydration
	• Electrolyte abnormalities
	• Hypotension
	  • Abnormal blood glucose
	  • Anemia

Bloodwork	• HCT • Electrolytes • BUN • Cr • TP • Lactate • Blood glucose, often serial glucose checks • Urinalysis
Physical exam	• Often a younger patient that was previously healthy • May be dehydrated, tachycardic or bradycardic, hypotensive, vomiting, and/or hypothermic • Painful abdomen may be present
Other diagnostics	• Blood pressure • ECG

Premedicants	• Rehydrate over 4–6 h if possible
	• Correct electrolyte and glucose abnormalities
	• Avoid sedatives in depressed patients
	• If patient is anxious, give midazolam (0.2 mg/kg IV or IM) or diazepam (0.2 mg/kg IV)
	• If patient is not depressed, give:
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM in dogs and 0.05–0.2 mg/kg IV, IM in cats), *or*
	  • Oxymorphone (0.1–0.2 mg/kg IV, IM), *or*
	  • Morphine (0.1–0.5 mg/kg IV, IM), *or*
	  • Buprenorphine (0.005–0.02 mg/kg IV, IM)

Intraoperative Considerations

Induction	• If dehydrated, give IV:
	  • Etomidate (0.5–1.5 mg/kg) (give benzodiazepine prior to etomidate), *or*
	  • Propofol (1–4 mg/kg)
	• If hydrated, give IV:
	  • Propofol (2–6 mg/kg), *or*
	  • Alfaxalone (2–5 mg/kg)

Maintenance	• Isoflurane or sevoflurane, *plus* • Hydromorphone[a] (0.05–0.2 mg/kg IV PRN in dogs and 0.05–0.2 mg/kg IV PRN in cats), *or* • Morphine (0.1–0.5 mg/kg IV PRN), *plus PRN* • Fentanyl (2–10 µg/kg IV PRN for short-term pain relief or as CRI 2–30 µg/kg/h IV), *or* • Ketamine (low dose: 0.5–1 mg/kg IV or as CRI 0.6–1.2 mg/kg/h IV), *or* • Lidocaine (1–2 mg/kg loading dose then CRI at 1–3 mg/kg/h in dogs) NOTE: *CRIs can be given as a single infusion or can be combined with the other CRIs for additional analgesia.* • For hypotension (to keep MAP 60–80 mm Hg) give phenylephrine, ephedrine, norepinephrine, vasopressin, or dopamine as needed
Fluid needs	• 5–10 mL/kg/h if minimal EBL and minimal evaporative losses, or 10–20 mL/kg/h if open abdomen with higher evaporative losses, plus 3 × EBL. Higher rates of fluids are necessary if dehydration was not corrected preoperatively. • Consider colloids if persistent hypotension • Consider blood transfusion if anemia or severe blood loss • Consider plasma if coagulopathy • Dextrose if hypoglycemic
Monitoring	• Blood pressure • ECG • Respiratory rate • SpO_2 • End-tidal CO_2 • Temperature • U/O
Blocks (epidural)	• Morphine (0.1 mg/kg preservative free), or • Buprenorphine (0.003–0.005 mg/kg diluted in saline) • Avoid local anesthetics for spinal or epidural as they will enhance hypotension via vasodilation

Continued

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TABLE 19.2 Anesthetic Considerations in the Septic Patient—cont'd

Postoperative Considerations	
Analgesia	• Fentanyl CRI (2–30 µg/kg per hr IV, IM), *or* • Morphine (0.1–1 mg/kg IV, IM q3–4h in dogs and 0.01–0.5 mg/kg IV, IM q3–4h in cats), *or* • Oxymorphone (0.1–0.2 mg/kg IV, IM), *or* • Hydromorphone[a] (0.1–0.5 mg/kg IV, IM q3–4h in dogs and 0.05–0.2 mg/kg IV, IM q2–4h in cats), *or* • Hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs), *or* • Ketamine CRI (2–10 µg/kg/min IV)
Monitoring	• SpO_2 • Blood pressure • HR • Respiratory rate • Temperature • U/O • ECG if electrolyte abnormalities
Bloodwork	• HCT if significant blood loss • Repeat abnormal preoperative bloodwork • Serial blood glucose checks if necessary
Estimated pain score	Moderate to severe if open abdominal surgery or if underlying pancreatitis

[a]Monitor for hyperthermia in cats.
BUN, Blood urea nitrogen; *Cr,* creatinine; *CRI,* constant-rate infusion; *EBL,* estimated blood loss; *ECG,* electrocardiogram; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscularly; *IV,* intravenous; *MAP,* mean arterial pressure; *PRN,* as needed; *q,* every; *SpO_2,* oxygen saturation via a pulse oximeter; *TP,* total protein; *U/O,* urine output.

NOTE Always count surgical sponges before making the incision and before abdominal closure to help ensure that none are inadvertently left in the abdominal cavity.

Ventral Midline Celiotomy in Cats and Female Dogs

With the patient in dorsal recumbency, make a ventral midline skin incision beginning near the xiphoid process and extending caudally to the pubis (Fig. 19.2A). Sharply incise the subcutaneous tissues until the external fascia of the rectus abdominis muscle is exposed. Ligate or cauterize small subcutaneous bleeders and identify the linea alba. Tent the abdominal wall and make a sharp incision into the linea alba with a scalpel blade. Palpate the interior surface of the linea for adhesions. Use scissors to extend the incision cranially or caudally (or both) to near the extent of the skin incision. Digitally break down the attachments of one side of the falciform ligament to the body wall, or excise it and remove it entirely if it interferes with visualization of cranial abdominal structures. Clamp the cranial end of the falciform ligament and ligate or cauterize bleeders before removing it.

Ventral Midline Celiotomy in Male Dogs

With the patient in dorsal recumbency, place a towel clamp on the prepuce and clamp it to the skin on one side of the body (see Fig. 19.2B). Drape the tip of the prepuce and clamp outside the surgical field. Make a ventral midline skin incision beginning at the xiphoid process and continuing caudally to the prepuce. Curve the incision to the left or right of the penis and prepuce (i.e., the side opposite the clamped prepuce) and extend it to the level of the pubis (see Fig. 19.2B). Incise the subcutaneous tissues and fibers of the preputial muscle to the level of the rectus fascia in the same plane as the skin incision. Ligate or cauterize large branches of the caudal superficial epigastric vein at the cranial aspect of the prepuce. Retract incised skin and subcutaneous tissues laterally and locate the linea alba and external fascia of the rectus abdominis muscle. Do not attempt to locate the caudal linea alba until subcutaneous tissues have been incised and the abdominal musculature fascia identified. Tent the

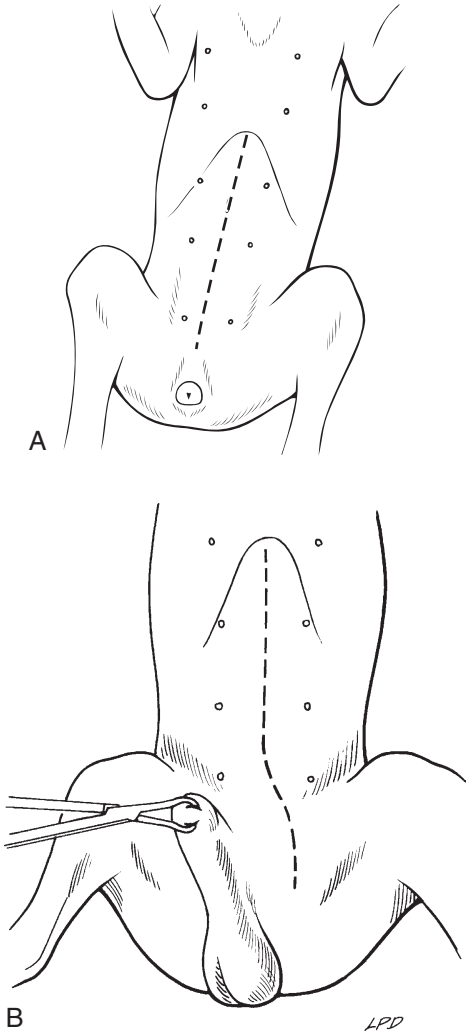


FIG. 19.2 Ventral midline celiotomy in (A) cats and female dogs and in (B) male dogs.

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abdominal wall and make a sharp incision into the linea alba with a scalpel blade. Palpate the interior surface of the linea for adhesions. Use scissors to extend the incision cranially or caudally (or both) to near the extent of the skin incision.

Paracostal Celiotomy

Position the animal in lateral recumbency and place a rolled towel or sandbag between the animal and the operating table. Make a skin incision from the ventral vertebral column to near the ventral midline. Center the incision halfway between the wing of the ilium and the last rib. Extend the incision through the external abdominal oblique muscle with scissors. Separate internal abdominal oblique and transversus abdominis muscle fibers and expose the peritoneal and transversalis fascia. Tent the peritoneum and sharply incise it with scissors.

ABDOMINAL EXPLORATION

Systematically explore the entire abdomen. Various techniques may be used; however, every surgeon should develop a consistent pattern to ensure that the entire abdominal cavity and all structures are visualized and/or palpated in each animal (Box 19.1). *Use moistened laparotomy sponges to protect tissues from drying during the procedure. If generalized infection is present or if diffuse intraoperative contamination has occurred, flush the abdomen with copious amounts of warmed, sterile saline solution.*

Historically, many different antiseptics (e.g., povidone iodine, chlorhexidine) and antibiotics have been added to lavage fluids. Povidone-iodine is the most widely used antiseptic; however, this practice has not shown a beneficial effect in repeated experimental and clinical trials and may be detrimental in animals with established peritonitis because the carrier, polyvinylpyrrolidone, inhibits macrophage chemotaxis. Although adding antibiotics to abdominal lavage fluid is recommended by some, sufficient data to determine the value of doing so in patients being given appropriate systemic antibiotics are lacking. Room temperature lavage fluids should not be used in anesthetized patients. Heated lavage fluids are useful for increasing the temperature in dogs.

Remove the lavage fluid and blood and inspect the abdominal cavity before closure to ensure that all foreign material and surgical equipment have been removed. Perform a sponge count and compare it with the preoperative count to ensure that surgical sponges have not been left in the abdominal cavity.

Abdominal Wall Closure

The linea alba may be closed with simple interrupted sutures or a simple continuous suture pattern. The simple continuous technique does not increase the risk of dehiscence when properly performed (i.e., secure knots, appropriate suture material), and it allows for rapid closure. Preferably strong, absorbable suture material (e.g., polydioxanone [PDS], polyglyconate [Maxon], poliglecaprone 25 [Monocryl], or glycomer 631 [Biosyn]) should be used for continuous suture patterns, and six to eight knots should be placed at each end of the incision line. Monofilament, nonabsorbable suture material (e.g., polybutester [Novafil], polypropylene [Prolene], or nylon) has been associated with suture sinus formation and should be avoided. Surgical gut and stainless steel wire should not be used for continuous suture patterns.

On each side of the incision, incorporate 4 to 10 mm of fascia in each suture. Place interrupted sutures 5 to 10 mm apart, depending on the animal's size. Tighten sutures sufficiently to appose but not enough to strangulate tissue, because sutures that are too tight adversely affect wound healing. Incorporate full-thickness bites of the abdominal wall in the sutures if the incision is midline (i.e., through the linea alba; Fig. 19.3). Do not incorporate the falciform ligament between the fascial edges. If the incision is lateral to the linea alba and muscular tissue is exposed (i.e., paramedian incision), close the external rectus sheath without including muscle in the sutures. Do not attempt to include peritoneum in the sutures. Close subcutaneous tissues with a simple continuous pattern of absorbable

BOX 19.1 Systematic Exploration of the Abdominal Cavity

1. Explore the cranial quadrant.
   - Examine the diaphragm (including the esophageal hiatus) and the entire liver (palpate the liver).
   - Inspect the gallbladder and biliary tree; express the gallbladder to determine its patency.
   - Examine the stomach, pylorus, proximal duodenum, and spleen.
   - Examine both pancreatic limbs (palpate gently!), the portal vein, hepatic arteries, and caudal vena cava.
2. Explore the caudal quadrant.
   - Inspect the descending colon, urinary bladder, urethra, and prostate or uterine horns.
   - Inspect the inguinal rings.
3. Explore the intestinal tract.
   - Palpate the intestinal tract from the duodenum to the descending colon and observe the mesenteric vasculature and nodes.
4. Explore the gutters.
   - Use the mesoduodenum to retract the intestine to the left and examine the right "gutter." Palpate the kidney and examine the adrenal gland, ureter, and ovary.
   - Use the descending colon to retract the abdominal contents to the right. Examine the left kidney, adrenal gland, ureter, and ovary.

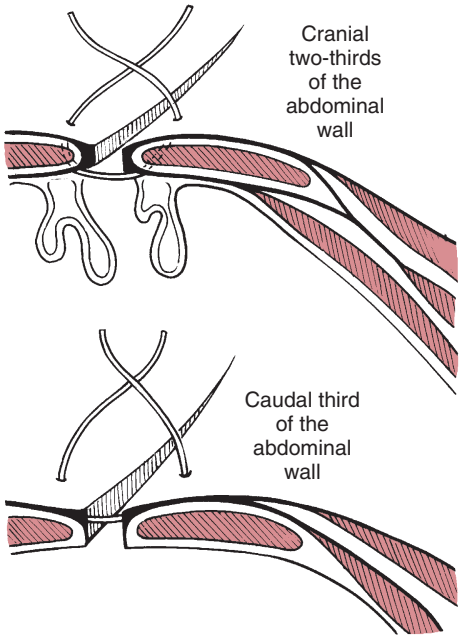


FIG. 19.3 To close a midline incision, incorporate full-thickness bites of the linea alba (or the external sheath only) in the sutures.

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suture material and reappose the preputial muscle fibers. Use nonabsorbable sutures (simple interrupted or continuous appositional pattern; see Chapter 8) or stainless steel staples to close the skin. Place skin sutures without tension.

For paracostal celiotomy, close the individual muscle layers with synthetic absorbable suture material in a continuous or interrupted pattern. Attempt to eliminate dead space between muscle layers. Appose subcutaneous tissue with absorbable suture in a continuous or interrupted pattern and close the skin with nonabsorbable suture in a simple interrupted or continuous pattern.

HEALING OF THE ABDOMINAL WALL

The ability of tissues to hold sutures without tearing depends on the tissue's strength and the orientation of collagen fibrils. Skin and fascia are strong, whereas muscle and fat are weak. Peritoneum heals rapidly across the incision and does not contribute to wound strength; therefore closure of this layer is not beneficial. Experimental and clinical studies in dogs suggest that suturing peritoneum may increase the incidence of postoperative intraabdominal adhesions.

NOTE Make sure to incorporate fascia in the linea closure. Because the holding layer of abdominal incisions is fascia rather than muscle, dehiscence is common if the rectus fascia is not incorporated in sutures.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Useful instruments for celiotomy include Balfour abdominal retractors, Poole or Yankauer suction tips, malleable retractors, and Mixter (right angle) forceps. Laparotomy pads and 4 × 4 sponges should have radiopaque markers. See the previous discussion on abdominal wall closure for choice of suture material.

POSTOPERATIVE CARE AND ASSESSMENT

The abdominal incision should be checked twice daily for redness, swelling, or discharge. If the animal licks or chews at the incision, a conventional Elizabethan collar or a collar that may be more comfortable for the patient (e.g., ProCollar, Soft-E-Collar, BiteNot collar, Comfy Cone, Kong EZ Collar) should be used to prevent iatrogenic suture removal (Fig. 19.4). Early signs of altered wound healing are inflammation and edema. Swelling and serosanguineous drainage from the incision are consistent signs of acute incisional dehiscence. Dehiscence usually occurs 3 to 5 days after surgery, when minimal healing has occurred and the sutures have weakened; however, it may occur earlier if knots were tied improperly or if fascia was not incorporated into the sutures. Evisceration usually causes sepsis and severe blood loss secondary to mutilation of exposed intestine; it must be treated promptly. Evisceration requires that the abdomen be bandaged, fluid therapy initiated, and broad-spectrum antibiotics given while the animal is prepared for surgery. If technical failure is suspected, such as poor knot tying or improper suturing, the entire suture line should be removed and replaced. Debridement of the wound edges is unnecessary and delays wound healing. The intestine should be closely inspected for viability and damaged sections resected if appropriate (see p. 440). The abdominal cavity should be lavaged copiously with warmed, sterile saline. Open abdominal drainage (OAD) (see p. 534) or suction drainage may be considered in animals with generalized peritonitis. Wound disruption after 10 to 21 days usually causes hernia formation rather than evisceration. Hernia repair in these animals may require excision of fibrotic tissues. Subsequent closure requires that tissue layers be accurately apposed.



FIG. 19.4 A soft collar used to prevent dogs from chewing on sutures. (Courtesy Laurie Miller, Midwestern University, Glendale, AZ.)

COMPLICATIONS

Dehiscence (incisional hernias) and abdominal evisceration may occur if improper surgical technique is used (see the previous discussion). The most common causes of wound dehiscence in the early postoperative period are suture breakage, knot slippage or untying, or sutures cutting through tissue. A higher rate of dehiscence may be seen in animals with wound infections, fluid or electrolyte imbalances, anemia, severe hypoproteinemia, select metabolic diseases (e.g., hyperadrenocorticism, diabetes mellitus), immunosuppression (e.g., feline immunodeficiency virus, feline leukemia virus), or abdominal distention, or in those that have been treated with corticosteroids, chemotherapeutic agents, or radiation. Suture sinus formation has been reported with nonabsorbable suture material. Such cases require surgical resection of affected tissues and removal of offending sutures.

SPECIAL AGE CONSIDERATIONS

Healing may be delayed in debilitated, very young or very old, or hypoproteinemic animals; chromic gut suture should not be used for abdominal wall closure in these patients. Survival after major abdominal wall evisceration following ovariohysterectomy is high. The low mortality may be due to the fact that it most commonly occurs in young, otherwise healthy animals and is promptly recognized and treated.

SPECIFIC DISEASES

UMBILICAL AND ABDOMINAL HERNIAS

DEFINITIONS

External abdominal hernias are defects in the external wall of the abdomen that allow protrusion of abdominal contents;

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internal abdominal hernias are those that occur through a ring of tissue confined within the abdomen or thorax (i.e., diaphragmatic hernia, hiatal hernia). External abdominal hernias may involve the abdominal wall anywhere other than the umbilicus, inguinal ring, femoral canal, or scrotum. **Umbilical hernias** occur through the umbilical ring. The contents of **true hernias** generally are enclosed in a peritoneal sac; **false hernias** allow protrusion of organs outside a normal abdominal opening, and therefore the contents are seldom contained in a peritoneal sac. **Omphaloceles** are large, midline, umbilical, and skin defects.

Abdominal hernias may be defined according to their location: ventral, prepubic, subcostal, hypochondral, paracostal, or lateral. The cranial pubic ligament was formerly called the *prepubic tendon.*

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Abdominal hernias generally occur secondary to trauma, such as vehicular accidents or bite wounds; however, they occasionally occur as congenital lesions. Congenital cranial abdominal hernias (i.e., cranial to the umbilicus) have been reported in association with peritoneopericardial diaphragmatic hernias in dogs and cats. Abdominal hernias are false hernias because they do not contain a hernial sac. When associated with blunt trauma, they arise as a result of rupture of the wall from within caused by an increase in intraabdominal pressure while the abdominal muscles are contracted. The most common sites for traumatic abdominal hernias are the prepubic region and the flank. Cranial pubic ligament hernias often occur in association with pubic fractures (Fig. 19.5). Paracostal hernias may allow migration of abdominal contents along the thoracic wall (see Fig. 19.5). In rare cases the abdominal contents enter the chest through defects in the intercostal muscles. Nearly half the animals that suffer from traumatic abdominal hernias also have serious concurrent injuries, including orthopedic (e.g., pelvis) and soft tissue injuries; thus a thorough physical examination of all affected animals should be performed.

Umbilical hernias are usually congenital; they are caused by flawed embryogenesis (see Fig. 19.5). Umbilical vessels, the vitelline duct, and the stalk of the allantois pass through the umbilical ring in the fetus, but this aperture closes at birth, leaving an umbilical cicatrix. If the aperture fails to contract or is too large or improperly formed, a hernia results. These hernias are lined by a peritoneal sac and are considered true hernias. The cause of umbilical hernias is seldom known, but most are thought to be inherited. Many male dogs with umbilical hernias are also cryptorchid. Omphaloceles allow abdominal organs to protrude externally (eviscerate). The abdominal contents are initially covered by amniotic tissue, but this membrane covering is easily ruptured. Most affected neonates either die or are euthanized at birth.

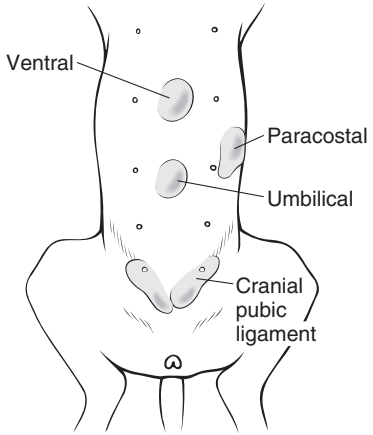


FIG. 19.5 Location of abdominal and umbilical hernias.

DIAGNOSIS

Clinical Presentation

Signalment

Most animals with umbilical or abdominal hernias are young. Umbilical hernias are believed to be heritable in some breeds (e.g., airedale, basenji, and Pekingese). Cranial ventral abdominal hernias associated with peritoneopericardial diaphragmatic hernias may be inherited in Weimaraners.

History

A history of trauma is common with abdominal hernias. The hernia may initially may overlooked while more obvious or life-threatening injuries are treated. Small umbilical hernias are often not noticed until the animal is examined for neutering. If strangulation or intestinal obstruction occurs, the animal may be presented for treatment of vomiting, abdominal pain, anorexia, and/or depression.

Physical Examination Findings

Abdominal structures (organs or omentum) in the subcutaneous space or between muscle layers usually cause asymmetry of the abdominal contour. The size of the swelling may not correspond to the size of the hernia, particularly if intestine has migrated into the hernia. The swelling should be palpated carefully to discern the contents of the hernia (e.g., intestine, bladder, or spleen) and to locate the abdominal defect. These patients should be thoroughly examined to determine whether a concurrent abdominal or thoracic injury or abnormality exists. Rupture of the cranial pubic ligament is often difficult to palpate because of subcutaneous swelling and pain.

Umbilical hernias usually manifest as a soft ventral abdominal mass at the umbilical scar. Deep palpation of the swelling reveals the size of the umbilical ring and helps characterize hernial contents. The hernial ring is not palpable in some animals because the ring closes subsequent to herniation of falciform fat or omentum. Occasionally intestines or other abdominal structures can be palpated; they can generally be reduced into the abdominal cavity. If the umbilical sac is warm or painful and the contents are irreducible, intestinal strangulation or obstruction should be suspected.

> **NOTE** Be sure to evaluate dogs with congenital hernias for other defects (e.g., cryptorchidism with umbilical hernias, ventricular septal defects, and pericardial diaphragmatic hernias with cranial abdominal hernias).

Diagnostic Imaging

Radiographs should be taken in animals with abdominal hernias. Routine ventral dorsal and lateral views may show an associated abdominal or thoracic injury (e.g., abdominal fluid, diaphragmatic hernia). Abdominal radiographs may help confirm a hernia (e.g.,

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subcutaneous intestinal loops and loss of the ventral abdominal stripe) when the abdominal wall defect cannot be palpated because of swelling or pain. Radiographs are generally not indicated in small umbilical hernias. Ultrasound examination may also help define contents of hernias.

Laboratory Findings

Laboratory abnormalities are uncommon with umbilical hernias unless strangulation or intestinal obstruction is present. Abnormalities associated with abdominal hernias vary depending on the severity of concurrent internal injuries.

DIFFERENTIAL DIAGNOSIS

Most hernias are diagnosed on physical examination. Differential diagnoses for abdominal swellings include abscesses, cellulitis, hematomas or seromas, and neoplasia.

MEDICAL MANAGEMENT

Initial treatment of animals with abdominal hernias is directed toward diagnosing and treating shock and concurrent life-threatening internal injuries.

SURGICAL TREATMENT

Most abdominal hernias can be repaired by suturing torn muscle edges or apposing disrupted abdominal wall edge to the pubis, ribs, or adjacent fascia. Synthetic mesh must be used to repair the defect in rare cases. Some hernias (e.g., intestinal strangulation, urinary obstruction, concurrent organ trauma) require emergency surgical correction. However, the extent of devitalized muscle may not be apparent initially, and delaying surgery in stable patients until muscle damage can be accurately assessed facilitates surgical correction. The most common surgical complications are hernia recurrence and wound infection. Abdominal hernias secondary to bite wounds are usually contaminated; wound infection and dehiscence of the skin or hernial repair (or both) are common. Mesh should not be placed in these hernias, and the wounds should be drained. Treatment of infected wounds includes cultures, drainage, antibiotics, and/or flushing. Abdominal exploration should be performed at herniorrhaphy to diagnose concurrent abdominal organ injury (e.g., mesenteric avulsion, gastric or intestinal perforation, diaphragmatic herniation, bladder rupture).

Many umbilical hernias resolve spontaneously in young animals or are small and are not corrected until the animal is neutered. Spontaneous closure may occur as late as 6 months of age. Intestinal strangulation is most likely when the hernial defect is approximately the size of the intestine and the hernial sac is large. Strangulation is unlikely in very small or large defects. If abdominal viscera in the hernia cannot be reduced, surgery should be performed as soon as possible.

Preoperative Management

Preoperative care depends on the animal's status and concurrent injuries. Hydration and electrolyte abnormalities should be corrected before surgery.

Anesthesia

If there are no concurrent abdominal injuries or disease, a variety of anesthetic protocols can be used to anesthetize the animal (see Table 19.1). See Table 19.2 for anesthetic management of animals that are in shock or are debilitated. See also subsequent chapters for detailed information about anesthetic management of patients with specific diseases (e.g., renal, pancreatic, hepatic).

Surgical Anatomy

The abdominal wall is composed of four muscle layers (the external and internal abdominal oblique muscles, the rectus abdominis muscle, and the transversus abdominis muscle). Abdominal hernias may occur at insertions or attachments of these muscles or through muscle bellies themselves. In the dog, the cranial pubic ligament (prepubic tendon) is a band of transverse fibers that serves to attach the ventral abdominal muscles to the cranial border of the pubis. In cats, no distinct prepubic tendon exists; rather, the abdominal muscles attach directly to the pelvic brim (Fig. 19.6).

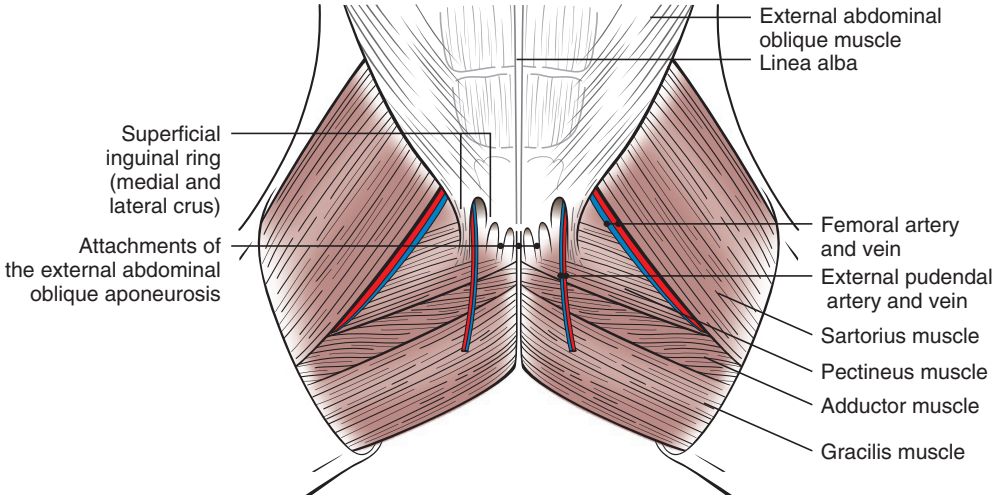


FIG. 19.6 Normal anatomy of the prepubic musculature attachments in the cat. (Modified from Beittenmiller MR, Mann FA, Constantinescu GM, et al. Clinical anatomy and surgical repair of prepubic hernia in dogs and cats. *J Am Anim Hosp Assoc.* 2009;45:284–290.)

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Positioning

For ventral hernias the animal is placed in dorsal recumbency and the area around the hernia is prepared for aseptic surgery. Repair of ruptures of the cranial pubic ligament may be facilitated by placing the animal in dorsal recumbency with the rear limbs flexed and pulled cranially.

SURGICAL TECHNIQUES

Abdominal Hernias

For most abdominal hernias, perform a ventral midline abdominal incision to allow the entire abdomen to be explored. Assess the extent of visceral herniation. Reduce the herniated contents and amputate or excise necrotic or devitalized tissue around the hernia. Close the muscle layers of the hernia with simple interrupted or simple continuous sutures. If a large area of devitalized tissue is removed, use synthetic mesh such as Marlex or Prolene to close the defect (do not place mesh in infected sites). Fold the edges of the mesh over and suture the folded edges to viable tissue using simple interrupted sutures. Injuries to the cranial pubic ligament can be difficult to repair. *If necessary, drill holes in the pubic bone to anchor the sutures.*

Paracostal Hernias

Make a midline incision or make one directly over the hernia. Explore the hernia and suture the torn edges of the transverse, internal, and external abdominal oblique muscles. Incorporate a rib in the suture if muscle has been avulsed from the costal arch.

Cranial Pubic Ligament (Prepubic) Hernias

In dogs, make a ventral midline skin incision and identify the ruptured tendon and its pubic insertion. Evaluate the inguinal rings and vascular lacuna; these hernias may extend into the femoral region as a result of rupture of the inguinal ligament. In cats, inspect the crura of the superficial inguinal ring and the aponeurosis of the external abdominal oblique muscle for damage. Reattach the free edge of the abdominal wall to the cranial pubic ligament with simple interrupted, cruciate, or mattress sutures of nonabsorbable monofilament suture (e.g., polybutester [Novafil], polypropylene [Prolene], or nylon) or absorbable suture material (see below under Suture Materials and Special Instruments). If an inguinal or femoral hernia is present, reattach the prepubic tendon to the pubis first, before repairing the other defects.

As an alternative, suture the tendon remnant to the muscle fascia and periosteum covering the pubis or anchor it to the pubis by drilling holes in the pubic bone through which sutures can be placed (Fig. 19.7). If the hernia extends into the femoral region, it may be necessary to suture the body wall to the medial fascia of the adductor muscles. When doing so, take care to avoid damaging the femoral vessels or nerves.

A cranial sartorius muscle flap may also be used to repair prepubic tendon injuries. *Separate the cranial portion of the muscle from its caudal belly and the quadriceps femoris muscle. Then transect the cranial portion of the muscle at its distal insertion on the medial aspect of the patella and elevate it to the level of the proximal vascular pedicle (Fig. 19.8). Rotate the flap 180 degrees to the defect and secure it using simple interrupted sutures. Use the flap to repair concurrent inguinal or femoral hernias, if present.*

Umbilical Hernias

For umbilical hernias, palpate the hernial ring, reduce the abdominal contents if possible, and incise the skin over the umbilicus. If the hernia contains only fat or omentum, ligate the hernial neck and excise the sac and its contents. As an alternative, if adhesions are not present, invert the sac and its contents into the abdominal cavity. Do not debride the wound margins. Suture the edges of the defect with monofilament, synthetic, absorbable sutures (e.g., polydioxanone [PDS], polyglyconate [Maxon], poliglecaprone 25 [Monocryl], or glycomer 631 [Biosyn]) in a simple interrupted pattern. If the hernial contents cannot be reduced, make an elliptic incision around the swelling to avoid damaging the contents. Incise the hernial sac and replace the contents in the abdominal cavity. If the contents are irreducible or if strangulation or intestinal obstruction is present, extend the abdominal defect on the midline. Explore the abdomen and inspect the intestines for viability before closing the defect. Umbilical hernia repair seldom requires mesh implantation.

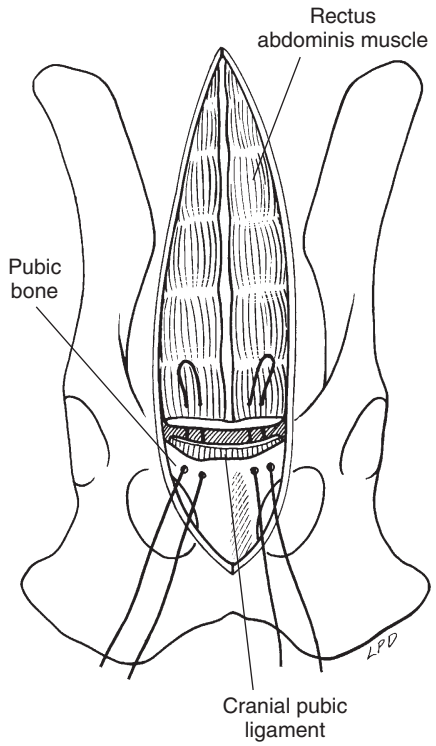


FIG. 19.7 To repair injuries to the cranial pubic ligament, it may be necessary to anchor the ligament to the pubis by drilling holes in the pubic bone through which sutures can be placed.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Strong, absorbable suture (polydioxanone [PDS], polyglyconate [Maxon], poliglecaprone 25 [Monocryl], or glycomer 631 [Biosyn]) or nonabsorbable suture (e.g., polybutester [Novafil], polypropylene [Prolene], or nylon) suture should be used to repair abdominal or ventral hernias. Nonabsorbable suture materials may be preferred over absorbable suture materials because they maintain tensile strength for greater than 1 year (see p. 62); however, successful repair with absorbable suture materials has been reported. Marlex and Prolene synthetic mesh may be used to repair some large defects.

POSTOPERATIVE CARE AND ASSESSMENT

The postoperative care of these patients is dictated by the presence of concurrent injuries or disease. The patient should be kept

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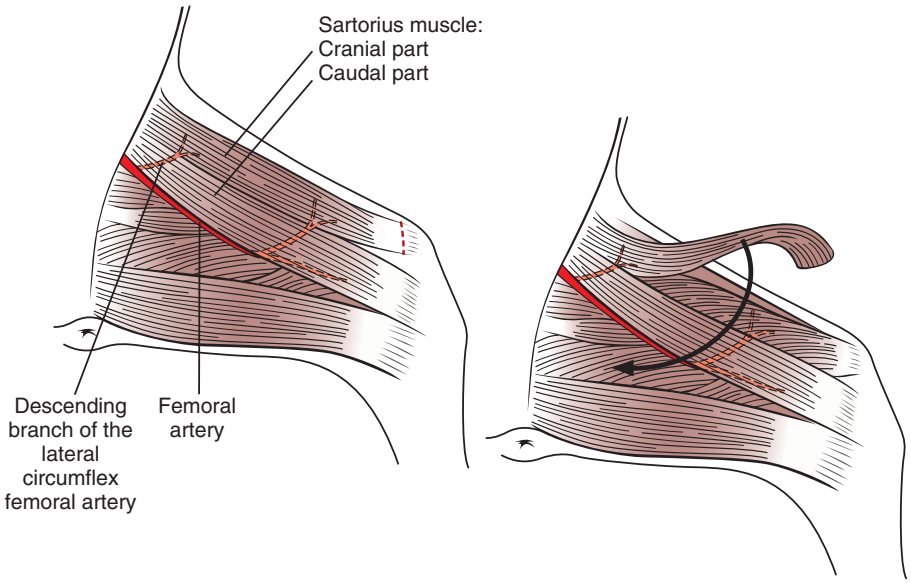


FIG. 19.8 Sartorius muscle flap in a cat. Transect the sartorius muscle distally at its insertion on the medial aspect of the patella and elevate it to the level of the proximal vascular pedicle. (Modified from Beittenmiller MR, Mann FA, Constantinescu GM, et al. Clinical anatomy and surgical repair of prepubic hernia in dogs and cats. *J Am Anim Hosp Assoc.* 2009;45: 284–290.)

quiet, and the wound should be checked frequently for infection or dehiscence. Vomiting, fever, and/or leukocytosis may indicate peritonitis (see p. 527).

PROGNOSIS

The prognosis is generally good, and recurrence is uncommon. When recurrence occurs, it is generally noted within a few days of surgery. Most animals have excellent long-term results when appropriate techniques are used.

INGUINAL, SCROTAL, AND FEMORAL HERNIAS

DEFINTIONS

Inguinal hernias are protrusions of organs or tissues through the inguinal canal adjacent to the vaginal process. **Scrotal hernias** occur when inguinal ring defects allow abdominal contents to protrude into the vaginal process adjacent to the spermatic cord. **Femoral hernias** occur through a defect in the femoral canal.

GENERAL CONSIDERATIONS AND CLINCIALLY RELEVANT PATHOPHYSIOLOGY

Inguinal hernias may arise from a congenital abnormality of the inguinal ring or may be caused by trauma (Fig. 19.9). An inguinal ring defect allows abdominal contents (e.g., intestine, bladder, uterus) to enter subcutaneous spaces. Congenital hernias may be associated with other abnormalities, such as umbilical hernias, perineal hernias, and cryptorchidism. Whether inguinal hernias are heritable in most breeds is unknown; neutering is recommended in dogs with nontraumatic hernias until the genetics of this condition become known.

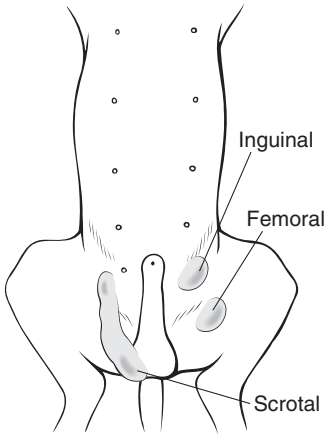


FIG. 19.9 Location of scrotal, inguinal, and femoral hernias.

Causes of inguinal herniation in small animals are poorly understood. Both neutered and intact male and female dogs may develop nontraumatic inguinal hernias. They may be unilateral or bilateral; unilateral inguinal hernias occur more commonly on the left side. Sex hormones have been incriminated in the formation of inguinal hernias in mice, but their role in dogs is unclear. Pregnancy and obesity may be associated with the formation of an inguinal hernia. Traumatic inguinal hernias may occur as a result of a congenital weakness of the musculature or abnormality of the inguinal ring.

Scrotal hernias are rare, indirect hernias (see Fig. 19.9). They are usually unilateral, and strangulation of abdominal contents is common. Little is known about the cause and heritability. A congenital defect or trauma may predispose some dogs to hernia formation. An increased incidence of testicular tumors has been reported in conjunction with scrotal hernias.

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Femoral hernias are rare in dogs and cats. They occur when abdominal contents or fat protrude through the femoral canal, caudomedial to the femoral vessels (see Fig. 19.9). They may be mistaken for inguinal hernias. Femoral hernias may occur after trauma and avulsion of the cranial pubic ligament, or they may result if the origin of the pectineus muscle is transected from the pubis during subtotal pectineal myectomy.

DIAGNOSIS

Clinical Presentation

Signalment

Nontraumatic inguinal hernias are most often reported in intact, middle-aged female dogs or young male dogs (<2 years of age). Inguinal hernias presumably arise in young male dogs because late testicular descent delays closure of the inguinal ring. Breeds that are predisposed to this condition include Pekingese, Cairn terrier, basset hound, Basenji, and West Highland white terrier. Older bitches may be predisposed to develop inguinal hernias because they have a relatively large-diameter ring with a short canal. Inguinal hernias are rare in cats. Scrotal hernias have been reported most commonly in chondrodystrophic dogs. No breed or sex predisposition has been reported for femoral hernias.

History

Animals with inguinal hernias may be presented because of a painless swelling in the inguinal region or for vomiting, lethargy, pain, and/or depression if the hernial contents are incarcerated. Small hernias often go unnoticed unless organ entrapment or incarceration occurs. Omentum is the organ most commonly present in canine inguinal hernias. The uterus is often in hernias of affected intact females. These hernias are often chronic and do not cause clinical signs until pregnancy or pyometra develops.

Animals with scrotal and femoral hernias are usually presented for evaluation of scrotal or medial thigh swelling, respectively, or for vomiting and pain if intestinal incarceration occurs.

Physical Examination Findings

Physical characteristics of the swelling vary according to hernial contents and degree of associated vascular obstruction. Often a soft, painless, unilateral or bilateral swelling is noted in the inguinal region. If intestinal strangulation has occurred or if a gravid uterus or urinary bladder is in the hernia, the swelling may be large, fluctuant, and painful. Finding nonviable small intestine is more common in young male dogs (<2 years of age) with nontraumatic hernias than in older animals. Associated vascular or lymphatic obstruction (or both) may cause testicular and spermatic cord edema. Concurrent abnormalities may be noted, such as perineal hernia or cryptorchidism. Unilateral inguinal hernias are more common than bilateral hernias. Bilateral hernias occur more commonly in young dogs, and careful palpation of the contralateral inguinal region for occult hernias is recommended in all dogs.

A scrotal hernia usually appears as a firm, cordlike mass that extends into the caudal aspect of the scrotum. Pain and bluish-black tissue discoloration may be noted if intestinal strangulation has occurred. Femoral hernias cause swelling on the medial aspect of the thigh that may extend into the inguinal region. The swelling is located caudal to the inguinal ligament and ventrolateral to the pelvic brim.

Diagnostic Imaging

Abdominal radiographs may help identify herniation of a gravid uterus, intestine, or bladder in an inguinal hernia. Loss of the caudal abdominal stripe may be noted in affected animals. Ultrasonography is useful with scrotal hernias to assess the viability of testicular blood flow and to help determine if spermatic cord torsion or a hydrocele is present.

Laboratory Findings

Laboratory abnormalities are uncommon unless intestinal incarceration has occurred.

DIFFERENTIAL DIAGNOSIS

Differentiation of mammary tumors, lipomas, lymphadenopathy, hematomas, abscesses, and/or mammary cysts from inguinal hernias is facilitated by placing the animal on its back and attempting to reduce the contents of the swelling. Incarceration of intestine may prevent reduction and makes differentiation of these abnormalities difficult. Differential diagnoses for scrotal hernias include trauma, testicular or scrotal neoplasia, orchitis, and severe scrotal inflammation or swelling. Differential diagnoses for femoral hernias include neoplasia, abscesses, and lymphadenopathy. Femoral and inguinal hernias may be difficult to distinguish from each other before surgery.

NOTE Do not mistake the caudal abdominal fat pad in obese cats for an inguinal hernia.

MEDICAL MANAGEMENT

The animal's condition should be stabilized before surgery.

SURGICAL TREATMENT

Prompt surgical correction is recommended to prevent complications associated with intestinal strangulation or pregnancy. Undescended testicles should be removed during repair of an inguinal hernia. Necrosis of ipsilateral descended testicles may occur secondary to vascular obstruction and requires orchiectomy. If a gravid uterus is contained in the inguinal hernia, the animal can be spayed, or if the fetus is viable and termination of the pregnancy is not desired, an attempt can be made to reduce the uterus and close the inguinal ring. However, parturition or uterine enlargement may be associated with recurrence.

NOTE Neuter animals with inguinal hernias. Warn owners of intact female dogs that the hernia may recur if the dog is bred or develops pyometra.

Preoperative Management

If intestinal incarceration or strangulation is suspected, antibiotics should be given before surgery.

Anesthesia

If the animal is healthy, a variety of anesthetic protocols can be used safely (see Table 19.1). For patients in which nonviable intestine is present, see Table 19.2. See p. 513 for additional anesthetic recommendations for animals undergoing intestinal

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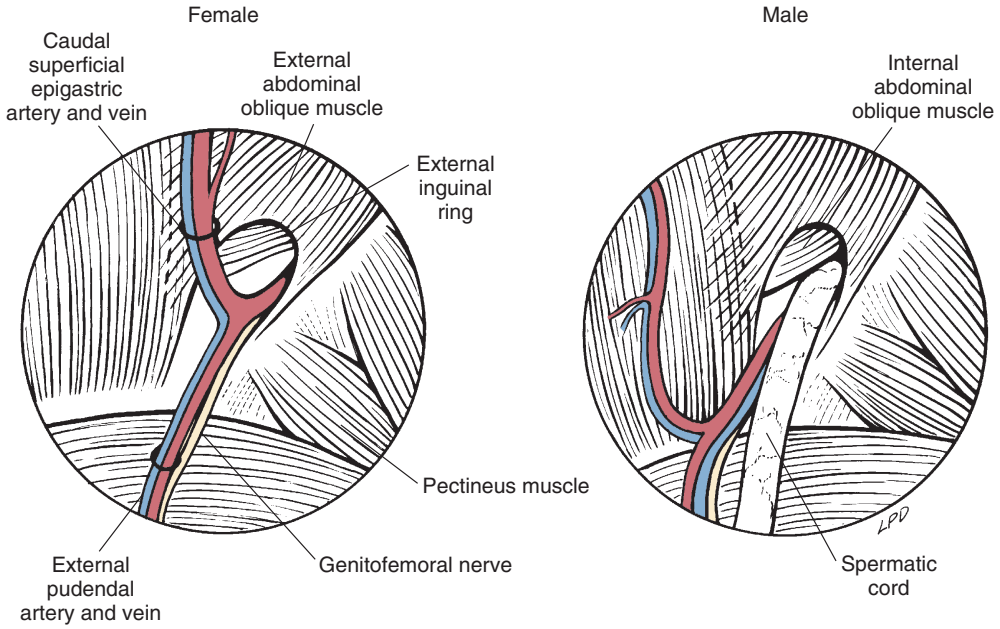


FIG. 19.10 Components of the inguinal canal.

surgery. Anesthetic protocols for pregnant animals can be found on p. 725.

Surgical Anatomy

The inguinal canal is a sagittal slit in the caudoventral abdominal wall through which pass the genital branch of the genitofemoral nerve, artery, and vein, the external pudendal vessel, and the spermatic cord (males) or round ligament (females) (Fig. 19.10). The vascular structures are located in the caudomedial aspect of the canal. The inguinal canal is bounded by the internal and external inguinal rings. The internal inguinal ring is formed by the caudal edge of the internal abdominal oblique muscle (cranial), the rectus abdominis muscle (medial), and the inguinal ligament (lateral and caudal); the external inguinal ring is a longitudinal slit in the aponeurosis of the external abdominal oblique muscle. Direct hernias occur when peritoneal evagination occurs as a separate, distinct outpocketing from the vaginal process; indirect hernias are protrusions through the normal evagination of the vaginal process.

Positioning

The animal is positioned in dorsal recumbency, and the caudal abdominal and inguinal areas are prepared for aseptic surgery.

Surgical Techniques

The goal of surgery is to reduce the abdominal contents and close the external inguinal ring so that herniation of abdominal contents cannot recur. The approach for inguinal hernias depends on whether the hernia is unilateral or bilateral; if the contents can be reduced; and if intestinal strangulation or concurrent abdominal trauma is a factor. Although an incision can be made parallel to the flank fold directly over the lateral aspect of the swelling, a midline incision is usually preferred in female dogs because it allows palpation and closure of both inguinal rings through a single skin incision. Inguinal hernias can usually be closed without using prosthetic materials. Occasionally repair of recurrent or large traumatic defects requires placement of synthetic mesh (see p. 521) or a cranial sartorius muscle flap (see p. 229). Bilateral orchiectomy is recommended with scrotal hernias to lessen recurrence.

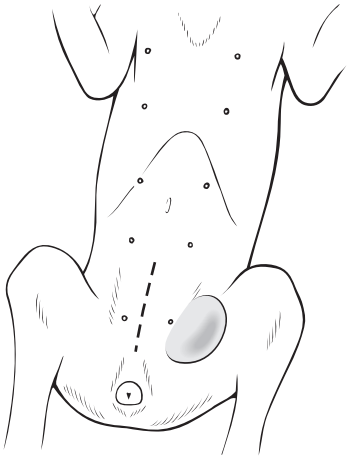


FIG. 19.11 Incision for repair of an inguinal hernia.

Inguinal Hernias

Make a caudal abdominal midline skin incision in female dogs cranially from the brim of the pelvis (Fig. 19.11). Deepen the incision through subcutaneous tissues to the ventral rectus sheath. Expose the hernial sac by bluntly dissecting beneath mammary tissue and identify the hernial sac and ring (Fig. 19.12A). Reduce the abdominal contents by twisting the sac and milking the contents through the ring; or, if necessary, incise the hernial sac and make an incision in the craniomedial aspect of the ring to enlarge it (see Fig. 19.12B). After reducing the abdominal contents, amputate the base of the hernial sac and close it with horizontal mattress sutures in a simple

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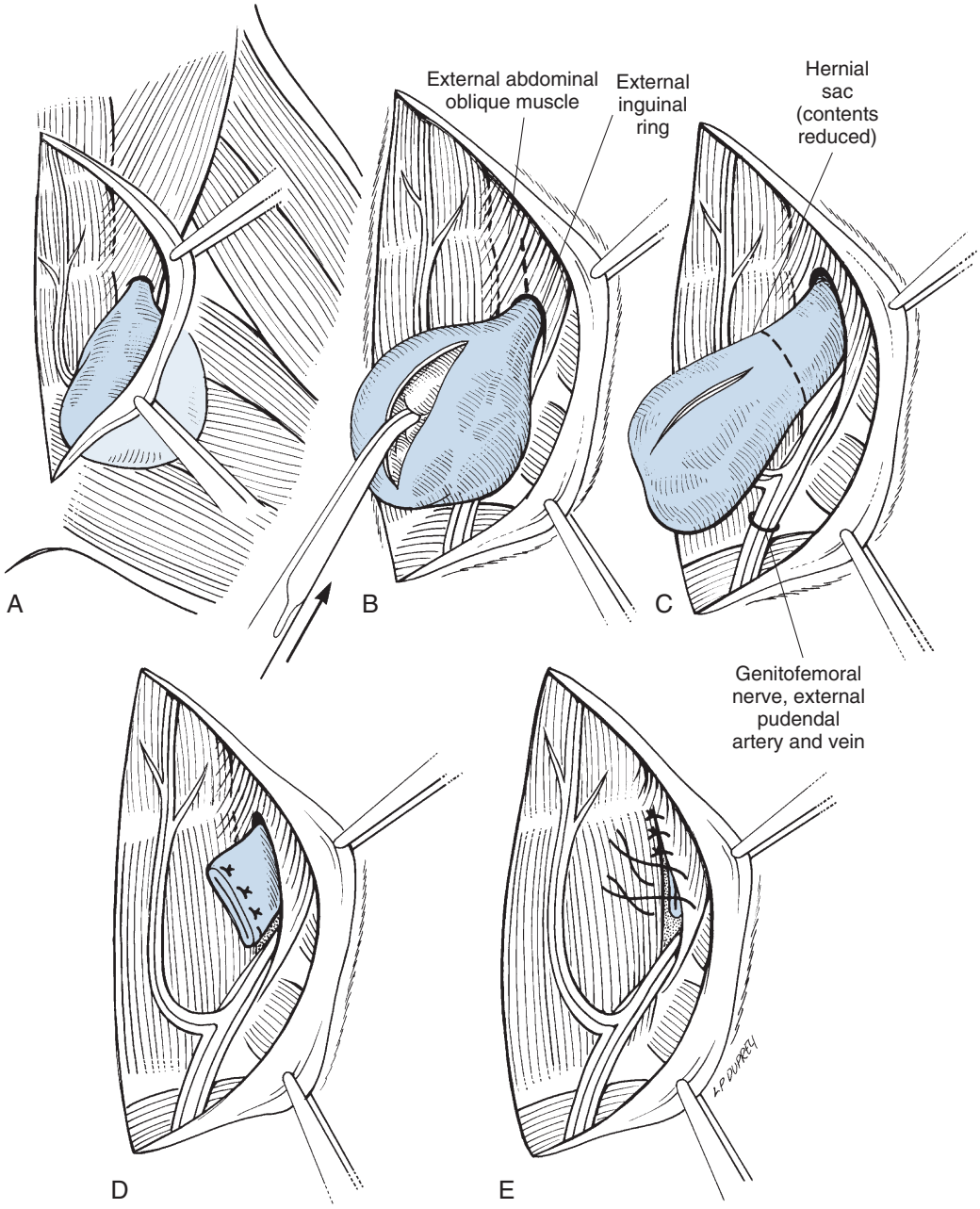


FIG. 19.12 Inguinal hernia repair. (A) Bluntly dissect beneath mammary tissue and identify the hernial sac and ring. (B) If necessary, incise the hernial sac. (C) Reduce the hernial contents and amputate the base of the sac. Close (D) the sac and (E) the inguinal ring.

continuous suture pattern or an inverting suture pattern (i.e., Cushing plus Lembert) (see Fig. 19.12C and D). Close the inguinal ring with simple interrupted sutures of absorbable or nonabsorbable synthetic suture material (see Fig. 19.12E). Avoid compromising the external pudendal vessels and genitofemoral nerve, which exit from the caudomedial aspect of the ring (or the spermatic cord in intact male dogs). Palpate the contralateral ring and close it if necessary before skin closure.

If the hernial contents cannot be reduced, perform a celiotomy and explore the abdominal contents. Expose the inguinal ring as described earlier and reduce the hernial contents (enlarge the inguinal ring if necessary). Resect nonviable intestine or perform an ovariohysterectomy and close the inguinal ring (or rings).

Scrotal Hernias

Incise the skin over or lateral to the inguinal ring and parallel to the flank fold (Fig. 19.13). Expose the hernial sac and reduce the abdominal contents (incise the hernial sac if necessary). If hernial repair is performed in conjunction with orchiectomy (preferred), open the hernial sac and ligate the contents of the spermatic cord (Fig. 19.14A). Remove the testicle after disrupting the ligament of the tail of the epididymis and ligate the hernial sac at the level of the internal inguinal ring (see Fig. 19.14B). If castration is not performed, make an incision into the hernial sac (parietal vaginal tunic) and evaluate the hernial contents (see Fig. 19.14C). Reduce the herniated contents and place a transfixing ligature or several horizontal mattress sutures in the hernial sac to reduce the size of

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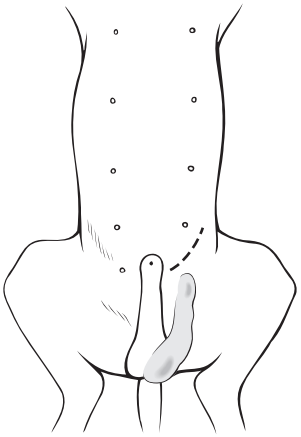


FIG. 19.13 Incision for Scrotal hernia repair.

the vaginal orifice (see Fig. 19.14D). Partly close the external inguinal ring with interrupted sutures (see Fig. 19.14E). Do not compromise the spermatic cord or vascular structures at the caudomedial aspect of the ring.

If the hernial contents cannot be reduced or if viscera are strangulated and necrotic, perform a midline celiotomy as described previously. After resecting the intestine, expose the inguinal ring and repair the hernia. Perform a scrotal ablation if the vaginal process and scrotum have been severely contaminated.

Femoral Hernias

Incise the skin parallel to the inguinal ligament and expose the hernial sac. Reduce the contents and ligate the hernial sac as high in the femoral canal as possible. If the inguinal ligament is intact, close the femoral canal by placing sutures between the inguinal ligament and the pectineal fascia. Do not damage or compromise

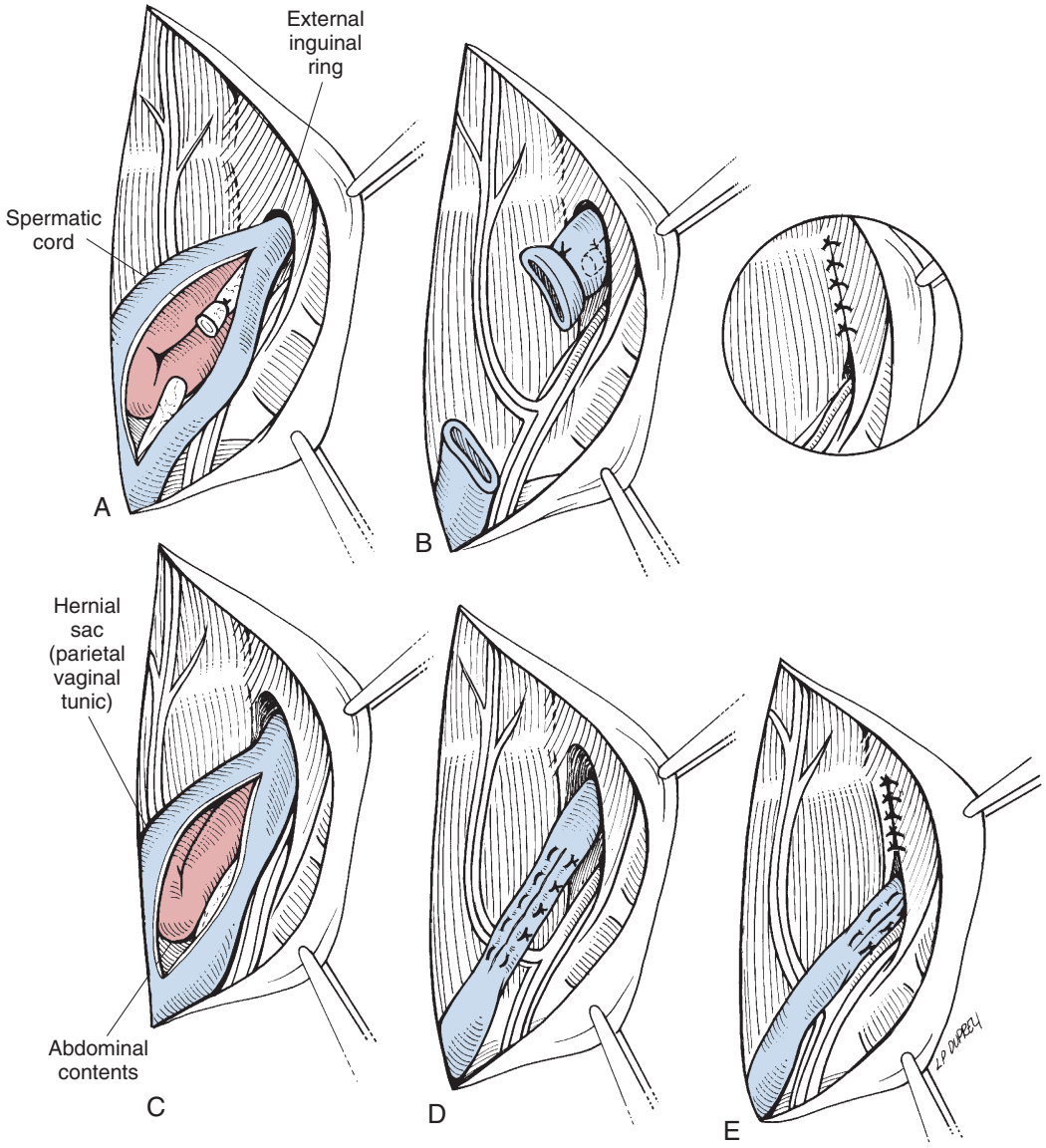


FIG. 19.14 Scrotal hernia repair in conjunction with orchiectomy. (A) Open the hernial sac and ligate the contents of the spermatic cord. (B) Remove the testicle and ligate the hernial sac at the level of the internal inguinal ring. If castration is not performed, (C) make an incision into the hernial sac, reduce the contents, and (D) reduce the size of the vaginal orifice. (E) Partly close the external inguinal ring.

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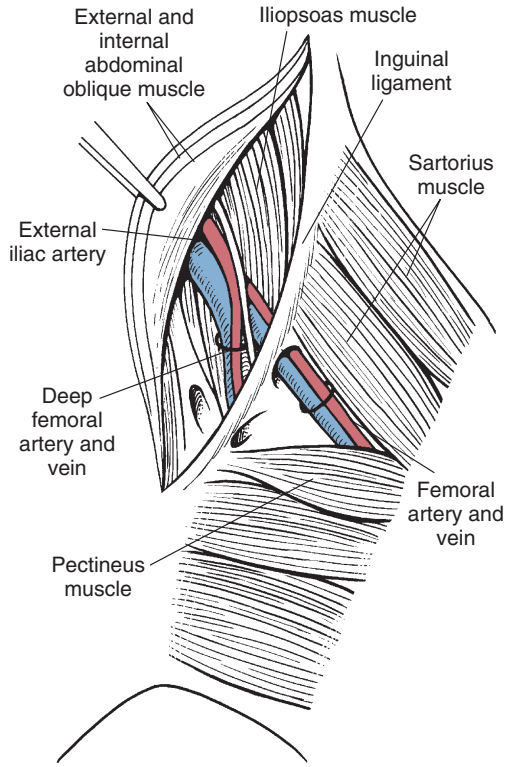


FIG. 19.15 Neurovascular structures of the femoral canal.

the neurovascular structures of the femoral canal (Fig. 19.15). Close the subcutaneous tissues and skin. If abdominal organs have strangulated, perform a midline celiotomy. Reduce the abdominal contents, then invert and ligate the sac. Dissect laterally from the skin incision to the femoral canal and close the femoral canal defect as described earlier.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Monofilament absorbable (e.g., polydioxanone [PDS], polyglyconate [Maxon], poliglecaprone 25 [Monocryl], or glycomer 631 [Biosyn]) or nonabsorbable (e.g., polybutester [Novafil], polypropylene [Prolene], or nylon) suture material should be used to close the hernial ring. Multifilament nonabsorbable suture may be associated with a higher incidence of wound infection. Mesh can be used as an overlay to reinforce the primary hernia repair (see p. 521).

POSTOPERATIVE CARE AND ASSESSMENT

Routine use of drains is not recommended; however, hernial sites should be assessed postoperatively for evidence of infection or the formation of a hematoma or seroma. If abscessation occurs, prompt removal of skin sutures, drainage, and topical therapy are indicated to prevent dehiscence of the hernia repair. Exercise should be restricted to leash walks for several weeks. An Elizabethan collar may be necessary to prevent the animal from licking at the surgical site. Postoperative testicular swelling may indicate compromise of testicular lymphatic or vascular drainage (or both). With femoral hernias, hobbles may be necessary during healing to prevent limb abduction, and femoral nerve function should be assessed postoperatively. Nerve deficits or severe pain may indicate compromise of the femoral nerve during the repair; reoperation is warranted in such cases.

PROGNOSIS

The prognosis is excellent unless intestinal leakage and perforation occur. The overall complication rate with inguinal hernias is generally very low with little or no associated mortality.

PERITONITIS

DEFINITIONS

Primary generalized peritonitis refers to spontaneous inflammation of the peritoneum without any obvious intraabdominal reason for leakage of bacteria. **Secondary generalized peritonitis** occurs in conjunction with an intraabdominal reason for the inflammation/infection and may be further classified as infectious or noninfectious.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Secondary generalized peritonitis is the predominant form of peritonitis in dogs and is usually caused by bacteria. Most cases arise through contamination from the GI tract, often secondary to surgical wound dehiscence or GI neoplasia. Other important causes include gallbladder perforation, rupture, or necrosis; perforation due to gastric or intestinal foreign bodies; intussusception; mesenteric avulsion; gastric dilatation-volvulus; necrotizing cholecystitis; prostatic abscesses; iatrogenic GI rupture due to gastroscopy, or foreign body penetration of the body wall. In a 2014 study, GI perforation was found to have occurred in 1.6% of cats and 0.1% of dogs that underwent endoscopy during a 17-year study period (1993–2010).[1] Risk factors for perforation were suggested to be small intestinal infiltrative disease in cats and preexisting GI ulceration in both cats and dogs. *Candida peritonitis* has been reported in dogs associated with duodenal perforation due to nonsteroidal antiinflammatory drug therapy; leakage after intestinal resection and anastomosis or intestinal foreign body removal; and after cholecystectomy due to gallbladder rupture and dehiscence of intestinal biopsy sites.[2]

Primary peritonitis is defined as an infection of the peritoneal cavity with no identifiable intraperitoneal source of infection or history of a peritoneal penetrating injury. Primary, septic peritonitis of unknown origin (despite exploratory surgery) is occasionally seen in cats and dogs. Gram-negative organisms are most commonly cultured from septic abdomens in dogs and cats with secondary peritonitis and reflects the high incidence of GI rupture in these animals. Gram-positive organisms are more commonly cultured from the abdomens of dogs and cats with primary peritonitis. Whereas infections associated with secondary peritonitis are often polybacterial, infections associated with primary peritonitis are more likely to be monobacterial. Differentiating primary and secondary peritonitis is important because surgery is not routinely indicated in the former but is requisite in the latter; however, it is often very difficult to make this distinction without surgery.

Sclerosing encapsulating peritonitis (SEP) is a clinical syndrome associated with an irreversible sclerosis of the peritoneal membrane. In people it may occur secondary to ambulatory peritoneal dialysis, severe acute bacterial or fungal peritonitis, chronic infections, blunt abdominal trauma, and intraperitoneal administration of drugs

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and chemicals such as chlorhexidine. Reported causes in dogs and cats include abdominal foreign bodies, steatitis, fiberglass ingestion, bacterial peritonitis, and leishmaniasis. The condition is typically life-threatening in dogs and is associated with chronic weight loss and peritoneal effusion. At surgery a thick, fibrous membrane may be found encapsulating the abdominal organs. There is no expert agreement on whether the treatment of choice should be surgical or conservative, and there are no clinical studies comparing the different therapeutic approaches. Medical treatment based on glucocorticoids, methotrexate, and tamoxifen have been recommended. Tamoxifen is used because it has shown efficacy in other fibrotic diseases, including retroperitoneal fibrosis. Notwithstanding that there are minimal data supporting a positive effect of any of the cited medical treatment options, successful treatment of this condition in a German shepherd dog was reported using enterolysis, open abdominal lavage, antibiotics, methylprednisolone (1 mg/kg PO, bid intermittently for 3–4 months), GI protectants, and tamoxifen (1 mg/kg PO, qd for 5 months; see below under Medical Management).[3]

Multiorgan failure is common in patients with peritonitis, with the lung being the organ most commonly affected. In humans, severe peritonitis has been found to produce an early pulmonary expression of chemoattractants in the lung, creating a gradient for pulmonary neutrophil sequestration and activation. These parameters are associated with expression of apoptosis in the lung causing acute lung injury and acute respiratory distress syndrome. Pulmonary neutrophil influx and activation is increased in nonsurviving peritonitis patients when compared to those who survive the insult.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, gender, or breed of dog or cat may develop peritonitis. It is particularly common in young animals that have perforating foreign bodies and in those that incur abdominal injury, such as vehicular trauma or bite wounds. The median age of cats with peritonitis is approximately 3 years, with the majority being under 5 years of age.

History

The history is often nonspecific. The animal may not show signs of illness for several days after the traumatic episode. Mesenteric avulsions often do not cause clinical signs of peritonitis for 5 to 7 days after the injury. Animals with traumatic bile peritonitis may be asymptomatic for days to several weeks after injury. Previous GI surgery with associated dehiscence is a common historical finding in affected animals. Most animals are presented for treatment of lethargy, anorexia, vomiting, diarrhea, and/or abdominal pain. In cats with peritonitis, lethargy, depression, and anorexia may be more common presenting signs than abdominal pain or vomiting. Clinical signs of SEP are vague and nonspecific (e.g., anorexia, vomiting, lethargy, weight loss, ascites).

NOTE Be sure to evaluate any sick intact female dog for pyometra.

Physical Examination Findings

Abdominal palpation often causes pain in affected dogs. The pain may be localized, but generalized pain is more common, and the animal often tenses or "splints" the abdomen during palpation. Cats with septic peritonitis may be less likely to show evidence of pain on abdominal palpation than similarly affected dogs. Vomiting and diarrhea may be noted. Abdominal distension may be noted if sufficient fluid has accumulated. Pale mucous membranes, prolonged capillary refill times, and tachycardia may indicate that the animal is in shock. Dehydration and arrhythmias may also occur.

Diagnostic Imaging

Critically ill patients are often difficult to evaluate because they may have distracting injuries, be obtunded, have respiratory or cardiac failure, or other significant pathology that makes imaging difficult. CT has been determined to be the most useful diagnostic test for human patients with intraabdominal infections, with the exception of injuries of the biliary tract, where ultrasonography is generally more useful. A diagnostic guide for managing animals with peritonitis is presented in Fig. 19.16.

The classic radiographic finding in animals with peritonitis is loss of visceral detail with a focal or generalized "ground glass" appearance (Fig. 19.17). The intestinal tract may be dilated with air or fluid or both. Free abdominal air may occur due to rupture of a hollow organ and sometimes from infection with gas-producing anaerobic bacteria. Localized peritonitis may occur secondary to pancreatitis and can cause the duodenum to appear fixed and elevated (a sentinel loop). Ultrasonography is useful for localizing fluid accumulation and assisting in obtaining a sample for analysis. Abdominal radiography and ultrasonography should be performed before DPL (see p. 534). Ultrasonography is also a valuable tool to evaluate the abdomen and surgical site in dogs. Most abdominal organs can be assessed ultrasonographically 24 hours postoperatively; the limiting factor to evaluating affected animals is often pain.

Laboratory Findings

The most common laboratory finding in animals with peritonitis is a marked leukocytosis; however, the neutrophil count may be normal or low in some cases. The predominant cell type is the neutrophil, and a left shift is often, but not always, present. Other abnormalities may include anemia, thrombocytopenia, dehydration, hypoglycemia or hyperglycemia, hyperbilirubinemia, and/or electrolyte and acid-base abnormalities. Lactate levels at the time of diagnosis may be a useful prognostic indicator in cats with peritonitis as nonsurvivors often have a significantly higher blood lactate concentration than survivors. The activity of circulating anticoagulant proteins (total protein C and antithrombin) are often significantly lower in dogs with sepsis.[4] C-reactive protein and procalcitonin are biomarkers that have been studied extensively in humans with septic peritonitis; procalcitonin may be a superior biomarker to C-reactive protein in humans with sepsis. Deficiencies of total protein C and antithrombin activities and hypercoagulability are consistent features of sepsis in dogs.[4] Thomboelasotograms of dogs with septic peritonitis that survive were found to be different from nonsurvivors in that the preoperative maximum amplitude, α angle, and coagulation index were significantly greater (indicating hypercoagulability) in nonsurvivors than in survivors.[4] In this study the maximum amplitude was the most specific predictor of survival. Lactate concentrations greater than 2.5 mmol/L in dogs with septic peritonitis was associated with mortality in a 2015 study; the inability to normalize plasma lactate concertation is also associated with nonsurvival.[5] In the aforementioned study, a lactate clearance less than 21% at 6 hours had a sensitivity of 54% and a specificity of 91% for

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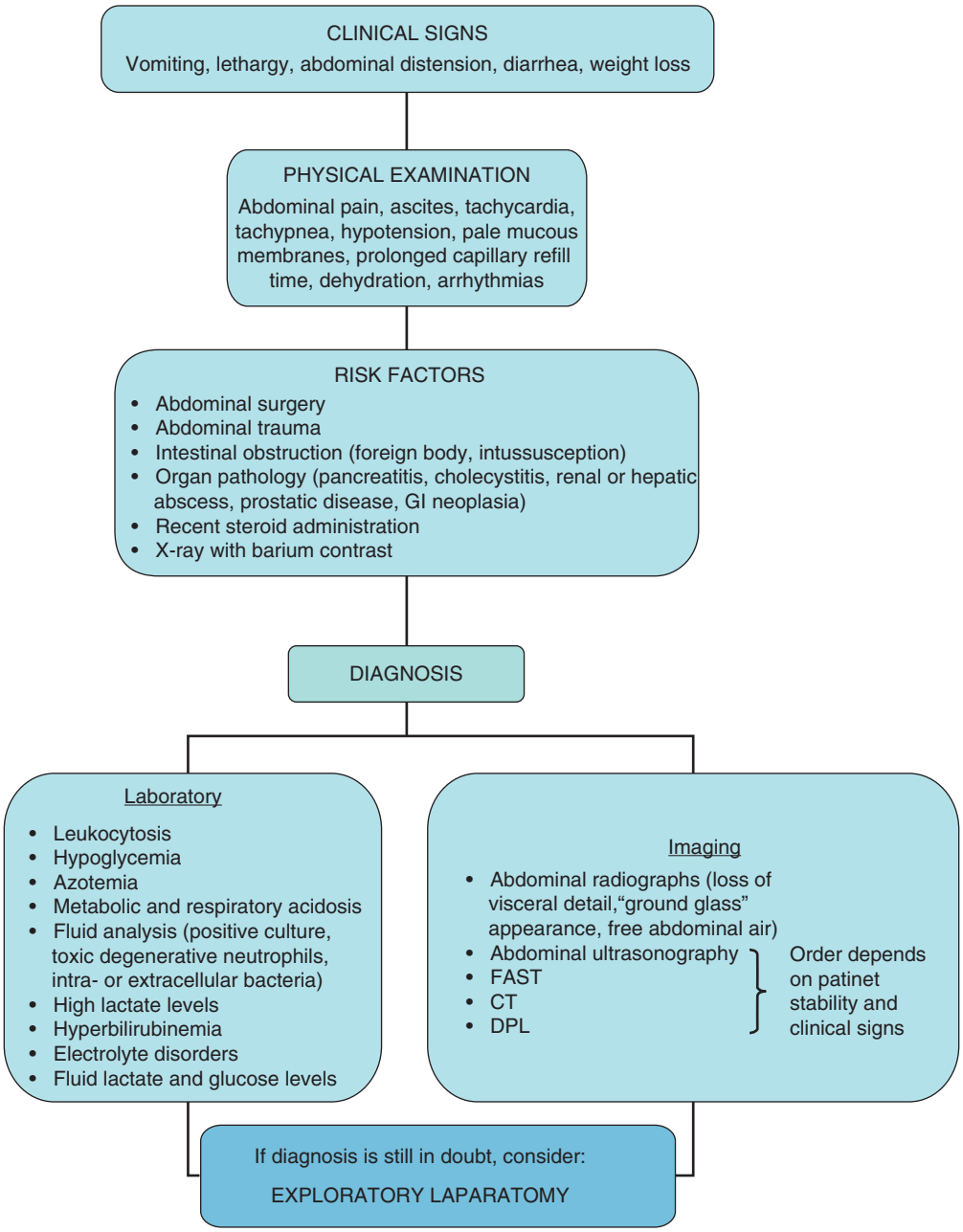


FIG. 19.16 Guide for managing animals with peritonitis. *CT,* Computed tomography; *DPL,* diagnostic peritoneal lavage; *FAST,* Focused Assessment with Sonography for Trauma; *GI,* gastrointestinal.

nonsurvival; lactate clearance less than 42% at 12 hours had a sensitivity of 82% and a specificity of 100% for nonsurvival. Bile peritonitis usually causes elevations of alkaline phosphatase, alanine transaminase, and total bilirubin concentrations (see p. 582). See p. 699 for a description of laboratory abnormalities associated with uroabdomen.

Little or no abdominal effusion may be seen in early cases of peritonitis. When effusion is present, abdominocentesis should be performed (see p. 534) and fluid retrieved for analysis. See pp. 698 and 581 for fluid analysis of animals with uroabdomen and bile peritonitis, respectively. Toxic degenerative neutrophils with intracellular or extracellular bacteria are indicative of bacterial peritonitis, as is plant material (from feces). Leukocyte morphology and the presence of bacteria are more important than leukocyte numbers. If total nucleated cell counts are performed, they should be done on fluid that has been placed in an ethylenediaminetetraacetic acid (lavender top) tube (Box 19.2). Bacteria are sometimes very hard to find in patients with bacterial peritonitis, especially if they have been receiving antibiotics. Differentiating between these effusions and those due to pancreatitis may be difficult because large numbers of degenerative white blood cells can be seen with both. A concentration difference greater than 20 mg/dL between blood and peritoneal fluid glucose concentration has also been reported to reliably differentiate between septic peritoneal effusions and nonseptic peritoneal effusions in dogs and also in cats. Peritoneal fluid lactate levels greater than 2.5 mmol/L or a difference less than –2 mmol/L between lactate levels in blood and peritoneal fluid is indicative

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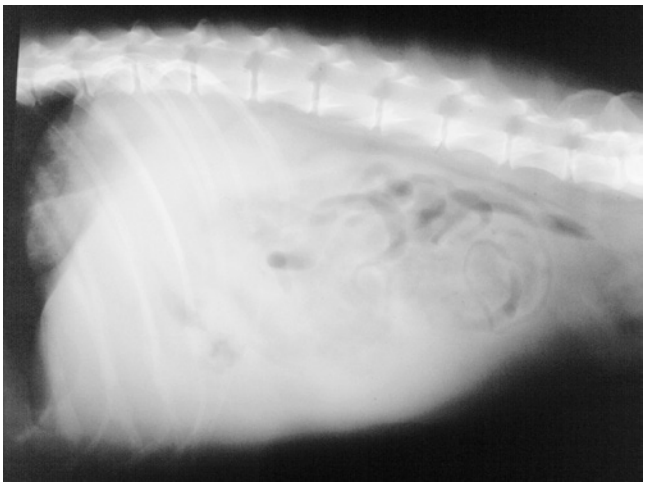


FIG. 19.17 Lateral radiograph of an animal with peritoneal effusion. notice the lack of visceral detail.

BOX 19.2 Estimating the TNCC and Amount of Blood in Abdominal Fluid

The total nucleated cell count (TNCC) of abdominal fluid can be estimated using the following formula:

$$X = NC \times OP^2$$

where:
X = TNCC
NC = number of nucleated cells per microscopic field
OP = objective power of the lens. The objective power chosen should allow visualization of 1–10 cells per microscopic field.

The amount of blood in the abdominal cavity can be estimated using the following formula:

$$X = \frac{L \times V}{P - L}$$

where:
X = Amount of blood in the abdominal cavity
L = Packed cell volume (PCV) of the returned lavage fluid
V = Volume of lavage fluid infused into the abdominal cavity
P = PCV of the peripheral blood before intravenous infusion of fluids

of septic peritonitis in dogs. Lactate levels are not accurate tests for detecting septic peritoneal effusions in cats. Determination of the glucose concentration difference for whole blood versus peritoneal fluid using a veterinary point-of-care glucometer was not useful in identifying all dogs with septic peritonitis.[6] However, in the same study, a glucose concentration difference of 38 mg/dL or higher for plasma versus peritoneal fluid or plasma versus peritoneal fluid supernatant was found to support an accurate diagnoses of septic peritonitis in dogs with peritoneal effusion. When peritoneal fluid from dogs with causes other than bacterial peritonitis (e.g., neoplasia, hepatic or renal disease) are compared to those with bacterial peritonitis, statistically significant differences are found in the values of pH, partial pressure of O_2, and partial pressure of CO_2.[7] The increase in fibrin accumulation that occurs with bacterial peritonitis leads to a decreased oxygen supply causing the partial pressure of CO_2 to be higher and the pH and partial pressure of O_2 to be lower than in noninfected fluid. Ultimately, nonoxidative glucose metabolism predominates, causing a decrease in pH, acidosis, and growth of anaerobic bacteria. Use of a leukocyte esterase test pad on a urine dipstick may assist clinicians in making a tentative identification of cases

TABLE 19.3 Intravenous Potassium Supplementation Guidelines

Serum Potassium[a] (mEq/L)	Meq KCl/L of Fluid	Maximal Infusion Rate[b] (mL/kg/h)
<2.0	80	6
2.1–2.5	60	8
2.6–3.0	40	12
3.1–3.5	28	16

[a]If serum potassium is not available, add potassium to a total concentration of 20 mEq/L.
[b]Do not exceed 0.5 mEq/kg/h.

BOX 19.3 Sodium Bicarbonate Therapy

1–2 mEq/kg given intravenously; repeat only if indicated based on assessment of acid-base balance and potassium concentration.

The following formula can also be used:
0.3 × Base deficit (mEq) × Body weight (kg)

Give half of this dose intravenously over 10–15 minutes and reevaluate. If the remainder is needed, administer over 4–6 hours.

in which bacterial infection is unlikely (a negative test may suggest an alternative diagnosis is likely, whereas a positive result suggests the necessity for further diagnostic tests).[8]

DIFFERENTIAL DIAGNOSIS

Advanced peritonitis with significant accumulation of abdominal fluid is not difficult to diagnose. The difficulty usually arises in determining the etiology of the effusion or infection. Early peritonitis, before the onset of overt clinical signs, is difficult to diagnose and may require DPL (see p. 534). If a diagnosis of primary peritonitis can be made preoperatively, surgery is not indicated and may result in worsening of the underlying disease process and subsequently increase morbidity.

MEDICAL MANAGEMENT

The goals of management of animals with peritonitis are to eliminate the cause of the contamination, resolve infection, and restore normal fluid and electrolyte balances. Food should be withheld if the animal is vomiting. Intravenous fluid replacement therapy should be initiated as soon as possible, particularly if the animal is dehydrated or appears to be in shock (in dogs, 60–90 mL/kg per hour; in cats, 40–60 mL/kg per hour). Synthetic colloids such as Hetastarch and pentastarch (see p. 32) may be beneficial, particularly if vasculitis is present. Hypokalemia (Table 19.3) and hyponatremia may be present and require intravenous supplementation. Hypoglycemia is common in animals with systemic inflammatory response syndrome (SIRS; formally called *septic shock*), and glucose may need to be added to the fluids (i.e., 2.5%–5% dextrose). Standard shock therapy should be initiated (i.e., fluid replacement and antibiotics). Bicarbonate therapy can be detrimental and should only be used if acidosis is so severe as to be immediately life-threatening (Box 19.3). Twenty-five percent human serum albumin was found to increase albumin, total protein, and colloid osmotic pressure in one study in dogs with septic peritonitis, and higher postoperative

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BOX 19.4 Antibiotic Therapy in Animals With Peritonitis

Ampicillin
22 mg/kg IV tid or qid

Enrofloxacin (Baytril)
10–20 mg/kg IV qd; give diluted in IV fluids slowly over 30 min

Amikacin (Amiglyde-V)
20–25 mg/kg IV qd (dilute and give slowly)

Clindamycin (Antirobe)
11 mg/kg IV tid

Metronidazole (Flagyl)
10 mg/kg IV tid

Cefoxitin (Mefoxin)
30–40 mg/kg IV qid

bid, Twice a day; *IV,* intravenous; *qd,* once a day; *qid,* four times a day; *tid,* three times a day.

BOX 19.5 Adjunctive Therapy in Dogs With Peritonitis[a]

Heparin
50–70 U/kg SC bid to tid

Heparin (Low Molecular Weight) (Dalteparin)
100–150 U/kg SC qd to tid

Heparin-Activated Plasma
Incubate 5–10 U heparin/kg of body weight with 1 U fresh plasma for 30 min. Give 10 mL/kg intravenously.
Put the first heparin dose (75–100 U/kg) in the plasma and incubate for 30 min before administration. Once antithrombin III (ATIII) levels are above 60%, continue heparin subcutaneously. If additional plasma is needed, incubation with heparin is not necessary.

[a]See Box 22.7 for therapy of disseminated intravascular coagulation.
bid, Twice a day; *qd,* once a day; *SC,* subcutaneous; *tid,* three times a day.

albumin levels were found to be associated with increased survival.[9] Canine-specific albumin may be safer than human serum albumin and was found to result in a persistent increase in albumin at 24 hours in dogs with septic peritonitis.[10] Steroid therapy was once considered standard for SIRS patients. Then it was almost universally rejected. Currently, there appears to be a place for judicious use of steroids in septic patients in SIRS that are not responding to standard resuscitative therapy.

Broad-spectrum antibiotic therapy should be initiated as soon as the diagnosis is made. *Escherichia coli, Clostridium* spp., and *Enterococcus* spp. are commonly isolated from animals with peritonitis, and ampicillin plus enrofloxacin (Box 19.4) typically is an effective antimicrobial combination. However, amikacin plus either clindamycin or metronidazole (see Box 19.4) may be necessary. A second-generation cephalosporin (e.g., cefoxitin [see Box 19.4]) also has a reasonable gram-negative and anaerobic spectrum. If renal compromise is present in an animal with a resistant bacterial infection, imipenem may be considered but should only be given as a last resort. The initial antibiotic therapy should be altered according to the aerobic and anaerobic culture results of lavage fluid or cultures obtained at surgery. Septic peritonitis typically causes disseminated intravascular coagulation, and plasma administration to replace clotting factors is probably one of the most beneficial therapies in such patients.

Low-dose heparin (Box 19.5) increases survival and significantly reduces abscess formation in experimental peritonitis. The inflammatory process in peritonitis is associated with an outpouring of fibrous exudate that causes intraabdominal loculation of bacteria. The loculated bacteria are protected from host defense mechanisms and antibiotics that may not be able to penetrate the fibrin clots. Although the exact mechanism of its beneficial effect is still unknown, heparin appears indicated in patients with severe peritonitis. Heparin may also be incubated with plasma and given to animals with disseminated intravascular coagulation (see Box 19.5). Low molecular weight heparin (enoxaparin, dalteparin) differs from unfractionated heparin in its mechanism of action and may be more effective (see p. 887); however, large clinical studies are lacking. Intraoperative administration of a lidocaine infusion to dogs with septic peritonitis may improve the short-term (48 hours) survival after surgery.[11] In a recent retrospective study of 75 dogs with septic peritonitis, survival of dogs that received an opioid only and those that received lidocaine (50 µg/kg per minute IV) in addition to an opioid during surgery were compared. The proportion of dogs that survived for 48 hours after surgery was significantly greater for the group receiving lidocaine than for the group that received only opioids.

Tamoxifen may be a useful adjunctive therapy in dogs with SEP. In breast tissue, tamoxifen acts as an antiestrogen to control tumor cell growth. It also appears to cause reexpression of a tumor suppressor gene known as *maspin* that inhibits tumor invasion and metastasis. Its mechanisms of action in SEP is unknown; however, it stimulates transforming growth factor beta 1, which has a stimulatory effect on metalloproteinases 2 and 9. Metalloproteinase 9 degrades type IV and denatured collagens; thus transforming growth factor β-1 production may favor mesothelial healing by facilitating the removal of denatured collagen.

SURGICAL TREATMENT

Abdominocentesis (see below) is the percutaneous removal of fluid from the abdominal cavity, usually for diagnostic purposes, although it may also be therapeutic. Indications include shock without apparent cause, undiagnosed disease with signs involving the abdominal cavity, suspicion of postoperative GI dehiscence, blunt or penetrating abdominal injuries (e.g., gunshot wounds, dog bites, vehicular injury), abdominal effusions, and undiagnosed abdominal pain. A multifenestrated catheter will enhance fluid collection. Physical and radiographic examinations should precede abdominocentesis to detect instances in which it may be unsafe and to guide needle placement. Four-quadrant paracentesis may be performed if simple abdominocentesis is unsuccessful in retrieving fluid. It is similar to simple abdominocentesis except that multiple abdominal sites are assessed by dividing the abdomen into four quadrants through the umbilicus and tapping each of these four areas. Alternatively, ultrasound may be used to identify small amounts of fluid and allow collection of a sample for evaluation. DPL should be performed in animals suspected of having peritonitis if the previous methods are unsuccessful in obtaining fluid for analysis (see p. 534) and ultrasonography (FAST; see p. 536) or CT imaging are not available.

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Exploratory surgery is indicated when the cause of peritonitis cannot be determined or when bowel rupture, intestinal obstruction (e.g., bowel incarceration, neoplasia), or mesenteric avulsion is suspected. Surgical therapy for the underlying disease is focused on either repair or removal of the inciting cause. Serosal patching and plication may reduce the incidence of intestinal leakage, dehiscence, or repeated intussusception (see p. 447). Animals that require surgery and have peritonitis secondary to intestinal trauma (disruption of mesenteric blood supply, bowel perforation, chronic intussusception, foreign body) often are hypoproteinemic. The role that protein levels play in healing intestinal incisions is not understood. However, most surgeons believe that hypoproteinemic patients may not heal as quickly as patients with normal protein levels despite the fact that similar complication rates are typically seen among euproteinemic and hypoproteinemic animals. Most experimental evidence has shown that retardation of wound healing is only seen with severe protein deficiencies (<1.5–2 g/dL).

Although the practice of lavaging the abdominal cavity of animals with peritonitis is controversial,[12] lavage is generally indicated with diffuse peritonitis. Lavage should be done with care in animals with localized peritonitis to avoid dissemination of infection. When lavage is performed, as much of the fluid as possible should be removed because fluid inhibits the body's ability to fight off infection, probably by inhibiting neutrophil function (opsonization and chemotaxis). Historically many different agents have been added to lavage fluids, especially antiseptics and antibiotics. Povidone-iodine is the most widely added antiseptic; however, its use may be contraindicated with established peritonitis. Furthermore, no beneficial effect of this agent has been shown in repeated experimental and clinical trials in animals. Although a great many antibiotics have been added to lavage fluids over the years, there is no substantial evidence that their addition benefits patients being treated with appropriate systemic antibiotics. Warmed isotonic saline is the most appropriate lavage fluid (~200 mL/kg). Peritoneal lavage has not been shown to be of benefit in patients with severe pancreatitis.

Controversy exists regarding whether postoperative drainage is beneficial in animals with septic peritonitis. OAD may be useful for managing animals with peritonitis. Reported advantages include improvement in the patient's metabolic condition secondary to improved drainage, fewer abdominal adhesions and abscesses, and access for repeated inspection and exploration of the abdomen. Disadvantages include hypoalbuminemia, hypoproteinemia, anemia, and nosocomial infections. With this technique, a small section of the abdomen is left open, and sterile wraps are placed around the wound. The frequency of wrap changes depends on the amount of fluid drained and external soiling. Complications of OAD include persistent fluid loss, hypoalbuminemia, weight loss, adhesion of abdominal viscera to the bandage, and contamination of the peritoneal cavity with cutaneous organisms. Because of the effort and time involved in OAD, it is rarely performed; although with highly exudative effusions it may be more effective than closed-suction drainage. Nonetheless vacuum-assisted-suction drainage (e.g., Jackson-Pratt) is often effective in dogs and cats with generalized peritonitis (particularly if the effusion is serous in nature), and it involves far less time and effort. Disadvantages of active drains are similar to those of OAD and include ascending nosocomial infection, hypoproteinemia, and electrolyte imbalances. Additionally, premature removal and occlusion of the drain may occur.

Preoperative Management

Animals with peritonitis that are in shock should be stabilized before surgery. Preoperative management of peritonitis is similar to that described in the previous discussion on medical management. The nutritional management of animals with peritonitis is extremely important; if the patient is debilitated, vomiting, or unlikely to resume eating for several days after surgery, enteral or parenteral hyperalimentation should be considered (see Chapter 10). Early nutritional support was associated with decreased length of hospitalization in dogs with septic peritonitis in one study.[13]

Anesthesia

Animals with peritonitis often are endotoxic and hypotensive. Small amounts of endotoxins are normally absorbed from the intestine and transported via the portal system to the liver where they are removed and destroyed by hepatocytes; however, hypotension in dogs is associated with intense portal vasoconstriction. This vasoconstriction causes breakdown of the intestinal mucosal barrier, allowing more intestinal endotoxin to be absorbed. If hepatic function is impaired (a common condition in septic animals), small doses of endotoxin that would normally be harmless may be lethal. Therefore hypotension should be corrected before and prevented during and after surgery in animals with peritonitis.

Animals with less than 4 g/dL total protein or 1.5 g/dL albumin may benefit from perioperative colloid administration. Colloids (e.g., hetastarch) may be given preoperatively, intraoperatively, or postoperatively for a total dose of 20 mL/kg per day (dog) or 10 to 15 mL/kg per day (cat). Colloids such as hetastarch are less apt to cause coagulation abnormalities than older products (e.g., dextrans). However, if large volumes of either crystalloid or colloid are given, clotting factors may be diluted. Clotting profiles, packed cell volume (PCV), and total solids should be checked frequently, regardless of which volume replacement therapy is used, when treating severe dehydration, hypotension, fluid loss, and blood loss. Fresh-frozen plasma should be considered when clotting tests are prolonged (see Table 4.5). Blood should be considered when PCV is in the low 20s (see Box 4.1).

NOTE The Food and Drug Administration has issued a black box warning on the use of hetastarch in critically ill people, noting that studies have shown increased mortality and/or severe renal injury when hetastarch was used in critically ill adult patients, including patients with sepsis.

Dopamine (Box 19.6) stimulates dopaminergic, β-adrenergic, and α-adrenergic receptors at various infusion rates from 2 to 10 μg/kg per minute IV, making it useful in patients with low cardiac output, hypotension, increased atrial filling pressures, and low urine output. This combination occurs after cardiac bypass; thus dopamine is still used in this situation in human medicine. However, it is seldom used alone after bypass surgery but is administered with other vasopressors (e.g., norepinephrine, phenylephrine, and/or vasopressin). If cardiac output remains insufficient, then dopamine is not increased. In that case, a phosphodiesterase inhibitor (e.g., milrinone or amrinone) is added. Dopamine is used intraoperatively during the ischemic phase in kidney transplants, but its use is controversial as it has never been proven to protect the kidneys. In patients that receive

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BOX 19.6 Inotropic and Vasopressor Support of Hypotensive Animals

Dobutamine
2–10 µg/kg/min IV

Dopamine
2–10 µg/kg/min IV

Ephedrine
0.06–0.2 mg/kg bolus IV

Epinephrine
0.1–1 µg/kg/min IV

Norepinephrine
0.05–2 µg/kg/min IV

Phenylephrine
0.5–5 µg/kg/min IV
20–200 µg bolus IV

IV, Intravenous.

dopamine prior to the "renal insult," there is a clear diuretic effect but no evidence that creatinine clearance is changed. One of the biggest disadvantages to using dopamine as a vasopressor is that high doses need to be used. This results in a significantly higher incidence of cardiac arrhythmias. In septic patients that already have tachycardia, the beta effects of dopamine further increase the heart rate, causes arrhythmias, and increases cardiac work and oxygen requirements. A study of human intensive care unit patients showed dopamine as an independent predictor of intensive care unit mortality regardless of whether shock was due to sepsis or other causes. Interestingly, epinephrine was also associated with a greater 30-day mortality, yet norepinephrine and dobutamine were not.

Dobutamine, at infusion rates of 2 to 10 µg/kg per minute IV, is a β-1 agonist with a small amount of β-2 activity (see Box 19.6). As such, it increases cardiac output by increasing cardiac contractility and heart rate, but the β-2 activity prevents it from causing a significant rise in blood pressure. Dobutamine is the drug of choice for patients with circulatory failure due to decreases in contractility. However, it is not recommended as the first-line single therapy in septic patients with hypotension. Like dopamine, it is often used with a vasopressor, such as norepinephrine. When used with norepinephrine for human patients with SIRS (septic shock), it has been shown to increase gastric mucosal perfusion. Controversy exists over the use of high doses of dobutamine; detrimental effects have been noticed when cardiac outputs increase to supra-normal states. Therefore it is generally recommended to use dobutamine as a second-line agent at lower doses and with norepinephrine after fluid resuscitation to improve perfusion.

Norepinephrine is the drug of choice for resuscitation (see Box 19.6). Norepinephrine is a very potent α-1 agonist with moderate β-1 and no β-2 activity at infusion rates of 0.1 to 1 µg/kg per minute IV. As an α-agonist it produces arterial and venous vasoconstriction in all vascular beds. Septic patients have systemic hypotension that is often unresponsive to aggressive fluid therapy. It is the generalized vasodilation that causes these dangerously low blood pressures; norepinephrine therapy is targeted at these vascular beds. High doses of phenylephrine and norepinephrine may cause renal artery constriction; however, therapeutic doses of norepinephrine have been shown to increase renal blood flow in multiple studies. Thus these drugs should not be given at high doses without adequate volume resuscitation.

Phenylephrine, a potent α-1 agonist, is one of the most common drugs used to counteract the vasodilation caused by inhalant agents (see Box 19.6). It is easy to use in small boluses or as a continuous infusion at rates of 0.5 to 3 µ/kg per minute IV. Phenylephrine can usually be discontinued at the end of the surgery when the inhalant and its vasoactive properties disappear. Sometimes phenylephrine is used to support blood pressure until volume can be restored. It is not meant to be a replacement for volume resuscitation, but used instead as a bridge until the bleeding stops and the volume reexpansion is complete. One of the properties of phenylephrine that makes it such a useful therapeutic medication is its effects on the venous vasculature. It causes greater venous vasoconstriction than arterial vasoconstriction, resulting in a greater increase in preload than afterload. Because of phenylephrine's lack of beta stimulation, a temporary reflex bradycardia occurs when it is administered. It must be used cautiously as intense vasoconstriction can lead to decreased tissue perfusion.

Ephedrine is an α- and β-agonist that acts directly and indirectly to increase blood pressure and heart rate, through norepinephrine (see Box 19.6). Tachyphylaxis occurs with repeated doses due to a depletion of norepinephrine stores. It can be given as either an IV bolus (0.06–0.2 mg/kg) or IM injection. When used as an IM injection (0.1 mg/kg), it has a greatly delayed absorption and thus a slow and prolonged effect. Ephedrine is not used as a continuous infusion.

Hepatic necrosis occurs during sepsis, reducing hepatic function. The pathogenesis of hepatic necrosis is uncertain, but it may be associated with hypotension and hypoxia. Patients with hepatic necrosis may have diminished ability to metabolize drugs resulting in prolonged duration of action or altered function of these drugs. Acepromazine should not be used in animals with peritonitis if hypotension or hepatic dysfunction is present. Midazolam administered with an opioid is a useful IV premedicant in patients with hepatic dysfunction (see p. 131). Benzodiazepines (diazepam, midazolam) undergo hepatic metabolism, and therefore they may have a prolonged effect in patients with hepatic dysfunction; however, they have the benefit that their effect can be reversed. In addition, they should be used cautiously in hypoalbuminemic patients because this may increase the concentration of free active drug. Most opioids have little or no adverse effect on the liver; however, morphine may cause hepatic congestion secondary to histamine release when given rapidly IV. If hypotension occurs with morphine, small boluses of either phenylephrine or ephedrine may be given to reverse the drop in blood pressure; an antihistamine may be administered if the hypotension is unresponsive. Although some opioid analgesics may have prolonged action when hepatic function is reduced, their effects can be antagonized.

Preoxygenation prior to induction is recommended to reduce hypoxia secondary to respiratory depression. Anesthetic protocols for relatively healthy patients with peritonitis are provided in Table 19.1. See Table 19.2 for recommendations for septic patients. Propofol may cause hypotension secondary to vasodilation and decreased cardiac output. Boluses of phenylephrine and ephedrine need to be available at any induction with propofol, especially with sick patients prone to hypotension; dopamine and norepinephrine are also useful as they can be titrated to effect and therefore one can avoid potential reflex bradycardia that can be seen with bolus doses of phenylephrine. A balanced induction approach with IV premeds prior to induction allows a smaller dose of propofol to be used, minimizing its hypotensive effects. Medetomidine is contraindicated because it causes vasoconstriction and secondarily decreases perfusion to vital organs, even at very low doses. Ketamine may have direct cardiodepressant effects associated with poor contractility, decreased cardiac output,

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and hypotension in debilitated patients. Etomidate should be used cautiously in the critical care patient because a single induction dose of this agent may cause prolonged adrenal suppression. Etomidate may also cause hemolysis and pain on injection due to the propylene glycol. These may be reduced by administering etomidate via a large vein with concurrent fluids, and by not using large or repeated doses. Hemolysis may be a greater problem in cats than in dogs with this drug.

Surgical Anatomy

The surgical anatomy of the abdominal cavity is described on p. 513.

Positioning

For abdominocentesis and diagnostic lavage, the abdomen should be clipped and prepared aseptically. These procedures may be performed with the animal in lateral recumbency or standing.

SURGICAL TECHNIQUES

Abdominocentesis

Insert an 18- or 20-gauge, 1.5-inch (38 mm) plastic over-the-needle catheter (with added side holes) into the abdominal cavity at the most dependent part of the abdomen. Do not attach a syringe; instead, allow the fluid to drip from the needle and collect in a sterile tube. If sufficient fluid is obtained, place it in a clot tube and an EDTA tube, submit samples for aerobic and anaerobic culture, and make four to six smears for analysis. If fluid is not obtained, apply gentle suction using a 3-mL syringe.

It is difficult to puncture bowel by this method because mobile loops of bowel move away from the tip of the needle as it strikes them. Perforations created by a needle this size usually heal without complications. The major disadvantage of needle paracentesis is that it is insensitive in finding small volumes of intraperitoneal fluid; a negative result can be meaningless. At least 5 to 6 mL of fluid per kilogram of body weight (BW) must be present in the abdominal cavity of dogs to obtain positive results in most cases using this technique.

Diagnostic Peritoneal Lavage

Make a 2-cm skin incision just caudal to the umbilicus and ligate any bleeders to avoid false positive results. Spread loose subcutaneous tissues and make a small incision in the linea alba. Hold the edges of the incision with forceps while the peritoneal lavage catheter (e.g., Stylocath) without the trocar is inserted into the abdominal cavity (Fig. 19.18). Direct the catheter caudally into the pelvis. With the catheter in place, apply gentle suction. If blood or fluid cannot be aspirated, connect the catheter to a bottle of warm sterile saline and infuse 20 mL/kg of fluid into the abdominal cavity. When the calculated volume of fluid has been delivered, roll the patient gently from side to side, place the bottle on the floor, vent it, and collect the fluid by gravity drainage. Do not be surprised if you do not retrieve all the fluid, particularly in dehydrated animals.

Alternatively, place an over-the-needle catheter into the abdomen as described previously and infuse a crystalloid solution. Remove the catheter and gently palpate the abdomen. Thirty minutes later, perform single or four-quadrant abdominocentesis.

NOTE This technique does not reliably exclude significant retroperitoneal injury or hemorrhage.

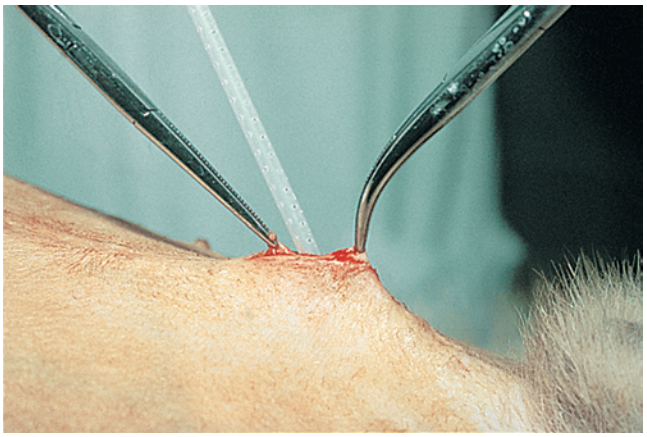


FIG. 19.18 Diagnostic peritoneal lavage.

Exploratory Laparotomy

Perform a ventral midline incision from the xiphoid process to the pubis (see pp. 513–517). Obtain a sample of fluid for culture and analysis. Explore and inspect the entire abdomen. Find the source of infection and correct it. Break down adhesions that may hinder drainage. Lavage the abdomen with copious amounts of warm, sterile saline if the infection is generalized. Remove as much necrotic debris and fluid as possible. Close the abdomen routinely or perform OAD.

Open Abdominal or Closed-Suction Drainage

OAD and closed-suction drainage have both been advocated for treatment of generalized peritonitis in dogs and cats. The latter has the advantage of a reduced risk of nosocomial infection and reduced postoperative care with a potentially shortened hospitalization stay. Disadvantages are that the drains may become occluded. Septic fluid that is more serous in nature may best be managed by closed-suction drainage, whereas fluids that are highly exudative in nature, with a large amount of fibrinous material, may best be managed by open peritoneal drainage.

Open Abdominal Drainage

After completing the abdominal procedure, leave a portion of the abdominal incision (usually the most dependent portion) open to drain. In general, make the opening just large enough to allow a gloved hand to be inserted. Close the cranial and caudal aspects of the incision with monofilament suture using a continuous suture pattern. Place a sterile laparotomy pad over the opening, then place a sterile wrap over the laparotomy pad. Change the wrap at least twice daily initially with the animal standing; sedation is seldom necessary (use sterile bandage materials and wear sterile gloves). The volume of drainage dictates the number of wrap changes needed. *Break down adhesions to the incision that may interfere with drainage.* Abdominal lavage may be attempted but is seldom necessary. *Place a diaper over the wrap to reduce contamination from urine. Assess the fluid daily for bacterial numbers and cell morphology. When the bacterial numbers have declined and the neutrophil morphology is normal (nondegenerative), close the incision (generally in 3–5 days). If the opening is small, it may be left to heal by second intention.*

Closed-Suction Drainage

Place a drain (e.g., Jackson-Pratt; Fig. 19.19) into the abdomen prior to closure. In cats and small dogs place one drain between

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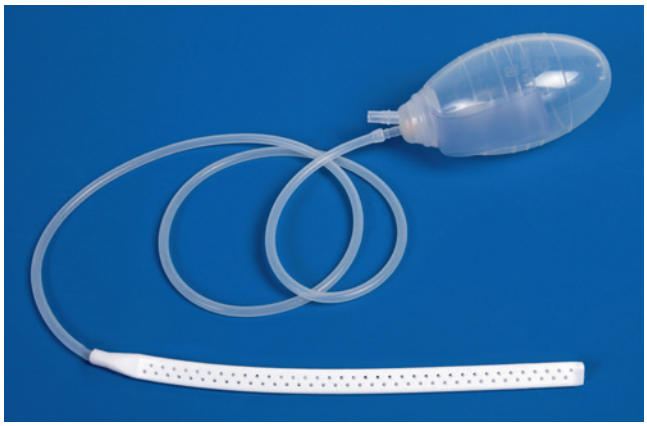


FIG. 19.19 Jackson-pratt drainage catheter.

the liver and diaphragm; in large dogs place a second drain into the caudal ventral abdomen. Exit the drain tube(s) through the body wall via a paramedian stab incision and suture them to the abdominal skin with a Roman sandal or Chinese finger-trap suture (see p. 923). Close the abdomen routinely and attach to the suction reservoir bulb to the tubing with vacuum applied. Place a sterile, protective bandage around the tube-skin interface. Empty the bulb using aseptic technique and record the volume of fluid. Remove the drain by cutting the suture and applying gentle traction.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Monofilament synthetic nonabsorbable suture (e.g., polybutester [Novafil], polypropylene [Prolene], or nylon) or slowly absorbable suture should be used to close the abdomen in animals with peritonitis. Braided suture (Dacron, silk, braided nylon) or suture that may be rapidly degraded (chromic gut) should not be used.

POSTOPERATIVE CARE AND ASSESSMENT

Fluid therapy should be continued postoperatively in most animals with peritonitis and is mandatory in those being managed with an open abdomen. Electrolytes, acid-base, and serum protein should be assessed in the postoperative period and corrected as necessary. Nasal oxygen may benefit septic animals. Ensuring that patients with peritonitis have an adequate caloric intake postoperatively is often difficult. An animal's energy requirement is much greater after injury or illness than at rest. Generally, the basal energy requirement (BER) for dogs and cats weighing less than 2 kg is BER = 70 × (BW in kg)$^{0.75}$; for animals weighing greater than or equal to 2 kg, the BER = (30 × (BW in kg) + 70. Postoperatively, the metabolic rate of dogs and cats increases 25% to 35% over resting levels. With mild trauma, the increase in the energy requirement is 35% to 50%; with sepsis, 50% to 70% more calories can be required. The factor 1.5 has been used to estimate the energy requirement of ill or injured dogs and cats. Meeting these caloric requirements in dogs with intestinal disease is particularly difficult and may require enteral (or rarely parenteral) nutritional support (see Chapter 10). If hypoproteinemia becomes severe, plasma transfusions might be considered. Postoperative analgesia is recommended (see Chapter 13).

PROGNOSIS

The prognosis for animals with generalized peritonitis is guarded; however, many survive with appropriate, aggressive therapy. The overall prognosis depends on a number of factors including the underlying cause and whether it can be treated effectively, the patient's health, and whether medical and surgical management are aggressive and appropriate. In a 2013 retrospective study of 44 dogs and 11 cats operated on for septic peritonitis because of GI perforation, the overall mortality was 63.6%.[14] In another study of 20 dogs with septic peritonitis of confirmed GI origin treated with closed suction drainage, 85% of the dogs survived to discharge.[15] A number of veterinary studies have looked at factors that might predict survival. In the aforementioned study, no association was found between age, duration of clinical signs, or prior abdominal surgery and outcome.[15] Animals with a history of prior antiinflammatory drugs were significantly more likely to have perforation of the pylorus (73.3%) than animals that had not received these drugs. In a retrospective study of 55 dogs with secondary septic peritonitis, no association was found between outcome and the time from hospital admission to surgical source control.[16] Factors that were found to influence survival in this study were age, needing vasopressors, lactate levels, preoperative PCV, serum alkaline phosphatase, serum total bilirubin, and postoperative serum albumin. The survival rate for dogs with recurrent septic peritonitis (n = 41) in a 2016 study was 43.9%.[17] Evaluation of peripheral blood and abdominal fluid variables as predictors of intestinal surgical site failure in dogs with septic peritonitis following celiotomy and closed-suction abdominal drain placement failed to identify any objective predictive indicators for those dogs that would develop postoperative septic peritonitis versus those that would have an uneventful recovery.[18] Dogs with septic peritonitis treated with vacuum-assisted, closed suction, or OAD are likely to develop nosocomial infection and hypoproteinemia. Administration of albumin and/or early nutritional intervention is often necessary in these cases after surgery (see Postoperative Care and Assessment).[19] Septic bile peritonitis has a worse prognosis than septic peritonitis of nonbiliary origin (see p. 581). Sterile bile peritonitis is associated with a good prognosis.

HEMOPERITONEUM

DEFINITIONS

Hemoperitoneum or hemoabdomen is the abnormal accumulation of blood in the peritoneal cavity.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Hemoperitoneum is associated with many diseases. Determination of the cause of the effusion requires a careful examination of the animal, coupled with consideration of potential diagnoses. Trauma (vehicular accidents, falls, kicks) is a common cause in both dogs and cats. Neoplasia is the most common cause of nontraumatic hemoabdomen in dogs and cats. Splenic neoplasia (see p. 645) is particularly common in dogs, although rupture of other tumors (e.g., hepatocellular carcinoma) may also cause hemoabdomen. Hemoperitoneum is strongly associated with splenic hemangiosarcoma in dogs (see p. 646). Nontraumatic rupture of adrenal gland tumors has been reported in dogs and

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may be associated with life-threatening hemorrhage. Nonmalignant disease associated with nontraumatic hemoperitoneum includes benign neoplasms, primary hepatobiliary disease, gastric dilation-volvulus (see p. 419), splenic torsion (see p. 643), liver lobe torsion (see p. 568), and intoxication with vitamin K antagonists.

DIAGNOSIS

Clinical Presentation

Signalment

Young male mixed breed dogs are more likely to be presented because of trauma; older dogs are more likely to have underlying neoplasia. Similar findings are true in cats.

History

A history of trauma may be present. With neoplasia, the history is often nonspecific. It should be ascertained whether there is a previous history of hemorrhage. Other important historical findings include previous surgery or diagnostic procedures, presence of previously diagnosed abdominal masses, access to toxins or rodenticides, and administration of medications. Cats with spontaneous hemoabdomen typically present for lethargy, anorexia, and vomiting.[20]

Physical Examination Findings

The animal should be carefully observed for external signs of trauma. Other findings may include abdominal distension (possibly with the presence of a fluid wave), abdominal tenderness, discoloration or bruising of the abdominal wall, and/or bulging of the umbilicus. Intraabdominal hemorrhage may cause severe hemorrhagic shock, or the animal may appear clinically normal. Common findings on physical examination in cats with spontaneous hemoabdomen are inadequate hydration and hypothermia.[20]

Diagnostic Imaging

The classic radiographic finding in animals with hemoabdomen is loss of abdominal detail with a focal or generalized "ground glass" appearance. Both lateral views and a ventrodorsal view are recommended to help detect abdominal masses. Ultrasonography is generally more sensitive than radiographs when small quantities of fluid are present. Ultrasonography is also useful for localizing fluid accumulation, and it may assist fluid retrieval and evaluation of abdominal organs for enlargement (e.g., neoplasia) or evidence of trauma. The presence of metastatic disease may also be noted ultrasonographically.

A rapid sonographic technique to detect the presence of free abdominal fluid after motor vehicle accidents has been reported. FAST examination has been advocated in hemodynamically unstable blunt abdominal trauma patients to determine the severity of their disease and whether they are surgical candidates because it gives a rapid assessment of injuries (see Fig. 4.1 and Box 4.2). AFAST is a term used to describe abdominal FAST examinations in veterinary medicine and is analogous to the FAST examination in human medicine. It is an ideal initial screening test for early recognition of intraperitoneal blood because it is rapid, noninvasive, safe, portable, and can be repeated if the patient's status changes.[21] The definition of AFAST has been expanded in veterinary medicine to AFAST[3] in which the "T" stands for trauma, triage, and tracking. Therefore repeated scanning abdominal ultrasound can be a valuable tool to see if abdominal fluid develops, particularly after a patient's volume status has been addressed. It has been recommended that AFAST with the abdominal fluid score (AFS) be repeated at 4 hours in stable patients and sooner in unstable patients. Transverse and longitudinal views are obtained at each of four sites (just caudal to the xiphoid process, on the midline over the urinary bladder, and at the left and right flank regions). The technique is simple and rapid and can be performed by veterinary clinicians with minimal previous ultrasonographic experience. The AFS is calculated based on the number of positive bleeding sites: AFS 0 (no positive sites), AFS 1 (positive at any single site), AFS 2 (positive at any two sites), AFS 3, (positive at any three sites), and AFS 4 (the maximum score, positive at all four sites). An increase in the AFS over time suggests ongoing intraabdominal hemorrhage. Readers are referred to reviews on AFAST for additional information on the technique.

Thoracic radiographs should be taken in animals with suspected or confirmed neoplasia. In animals with trauma, thoracic radiographs should be taken to look for diaphragmatic hernia (see p. 926) or other thoracic trauma. CT is useful for detecting neoplasia and is considered the gold standard for diagnosis of carcinomatosis in humans; however, ultrasonography may actually detect masses that are smaller than can be visualized with CT.

It has been suggested that CT may be more sensitive in diagnosing injury in blunt abdominal trauma than AFAST and should be used when the patient is hemodynamically stable. The efficacy of CT in the diagnosis of bowel injury (vs. hemorrhage) is controversial (Fig. 19.20). The CT "sentinel clot sign" may be helpful in identifying the source of bleeding in dogs with hemoabdomen.[22]

Laboratory Findings

Hemoperitoneum is diagnosed by finding nonclotting bloody fluid in the abdomen that has been retrieved by abdominocentesis (see p. 534) or DPL (see p. 534). Hemorrhage may be suspected based on an AFAST exam or CT of patients with blunt trauma (see Fig. 19.20). If fluid is collected, the PCV and total solids of the fluid should be determined and the fluid carefully examined for bacteria or neoplastic cells. If bacteria or degenerative neutrophils are noted, microbial culture (and exploratory surgery) is warranted. Other parameters that may be useful include serum creatinine, bilirubin, lactate, glucose, and lipase. Nonneoplastic effusions typically have lower lactate concentrations and higher glucose concentrations than neoplastic effusions.

Peripheral blood should be evaluated for anemia, but the complete blood count may not reflect the degree of bleeding for several hours after trauma. The majority of dogs presenting with acute nontraumatic hemoabdomen are anemic and hypoalbuminemic. A clotting profile should be performed in animals in with nontraumatic hemorrhage. Dogs with spontaneous hemoperitoneum have evidence of hypocoagulability, protein C deficiency, and hyperfibrinolysis.[23] Abnormal hemostatic profiles are common in dogs with splenic hemangiosarcoma (see p. 645) and may be seen in animals with other types of malignancies, as well as coagulopathies.

After abdominocentesis or DPL, the amount of blood in the abdominal cavity can be estimated by observing the lavage sample. A red color reflects the presence of red blood cells, and a deep red color usually indicates severe hemorrhage. If newsprint cannot be read through the plastic tubing, hemorrhage is significant; if print can be seen through the tubing, only moderate or minimal

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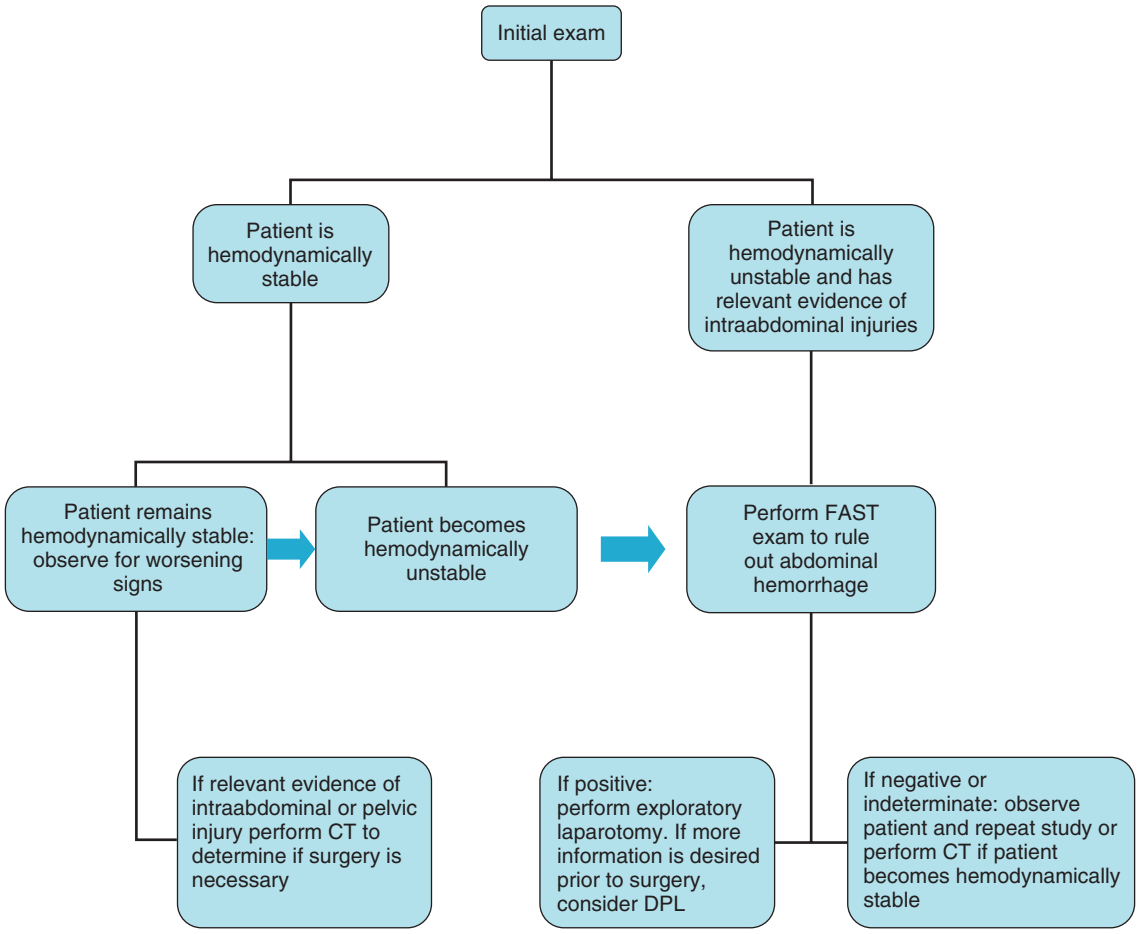


FIG. 19.20 Evaluation of the blunt trauma patient. *CT,* Computed tomography; *DPL,* diagnostic peritoneal lavage; *FAST,* Focused Assessment with Sonography for Trauma.

BOX 19.7 Indications for Abdominal Surgery Following Blunt Trauma

- Hemodynamically significant intraabdominal hemorrhage
- Evidence of intraabdominal bile leakage
- Septic peritonitis
- Body wall herniation; prepubic tendon rupture[a]
- Diaphragmatic herniation[a]
- Uroabdomen[a]

[a]Emergency surgery may not be indicated.

hemorrhage is present. The amount of blood in the abdominal cavity can be estimated using the equation shown in Box 19.2. Surgical intervention is indicated when the PCV of lavage samples taken within 5 to 20 minutes of each other increases substantially or if an animal in shock does not respond to aggressive fluid therapy (Box 19.7).

NOTE Clinicopathologic abnormalities in dogs with hemoabdomen are typically similar regardless of the cause of the abdominal bleeding.

DIFFERENTIAL DIAGNOSIS

Hemoabdomen must be differentiated from other causes of peritoneal effusion (e.g., peritonitis; see earlier) based on results of fluid analysis (see earlier and p. 529). Once hemoabdomen has been diagnosed, the underlying cause must be determined. DPL, which was introduced in 1965, is a rapid and sensitive test for intraabdominal hemorrhage following trauma, but because of its invasiveness and low specificity, it results in a high rate of nontherapeutic laparotomies. Therefore its use is declining. Common reasons for false negative results include injuries confined to the retroperitoneum and ruptured diaphragms.

Ultrasonography (AFAST) is rapidly replacing DPL as a diagnostic test for hemorrhage associated with blunt trauma. This test has a high sensitivity, specificity, and accuracy; however, the drawback is the lack of specificity about where the blood initiates from and how severe the organ damage is. Although AFAST is an expedient tool for the primary assessment of polytraumatized patients to rule out high grade intraabdominal injuries, the overall diagnostic sensitivity of AFAST may lead to underestimation of injury and delayed complications. Hence, in hemodynamically stable patients with abdominal trauma, an early CT scan (see Fig. 19.20) should be considered.

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MEDICAL MANAGEMENT

Rapid action must often be taken to save an animal's life that presents with a hemoabdomen. Intravenous fluid replacement therapy (see p. 32) should be initiated as soon as possible, particularly if the animal is in shock (in dogs, 60–90 mL/kg per hour; in cats, 40–60 mL/kg per hour). In one study, limited fluid volume replacement with hypertonic saline and hyperoncotic fluids (hydroxyethyl starch) was found to result in more rapid cardiovascular stabilization in dogs with spontaneous hemoperitoneum versus conventional resuscitation with large volume (90 mL/kg) isotonic crystalloid therapy.[24] Application of a tight abdominal bandage or compress may help attenuate or arrest the bleeding; however, arterial or hepatic hemorrhage is unlikely to be stopped by this method. Abdominal compresses should only be used short term while the animal is being stabilized and other definitive measures are being taken. Oxygen therapy via a nasal catheter or oxygen cage should be considered to optimize tissue oxygenation (see p. 29).

Blood transfusions may be required if the PCV falls below 20%. Typed and cross-matched blood should be available for transfusion, if needed (see p. 33). In an emergency situation where blood typing is not possible, dogs may be given dog erythrocyte antigen 1.1 negative blood. All cats should be blood typed before transfusion because of the risk of fatal reactions when nonspecific blood is given (see p. 34). If other sources of blood are not available, autologous blood transfusion may be considered.[25] Complications of autologous blood transfusion may include hypocalcemia, prolonged coagulation times, and hemolysis. Trauma patients with hemoabdomen that stabilize after medical management often do not require surgery.

SURGICAL TREATMENT

Surgery is indicated when the source of bleeding cannot be determined or effectively controlled or when evaluation and removal of intraabdominal neoplasia is required (see Box 19.7). A routine ventral abdominal incision should be performed. Suction is useful to remove the blood and help identify any sites of active bleeding. Although transfusion of suctioned blood back into the patient has been done, use of a cell saver system is recommended because it removes clots and activated blood components from red cells allowing blood to be salvaged and safely autotransfused. The kidneys, liver, and spleen should be carefully examined for trauma. Severe bleeding from the spleen or evidence of splenic neoplasia is an indication for splenectomy (see p. 637). Nephrectomy (see p. 655) may be required if severe bleeding from the kidney is noted. Clotting agents such as platelet gel or commercial products may help control bleeding from the liver. If these are not successful, partial hepatectomy may be required (see p. 544).

Preoperative Management

Animals with hemoabdomen that are in shock should be stabilized before surgery (see earlier). Every effort should be made to correct fluid, acid-base, electrolyte, and cardiovascular abnormalities before inducing anesthesia. Blood transfusions should be given if the PCV is less than 20%, the animal is hypoxic due to anemia, or ongoing bleeding is expected.

Anesthesia

Preoxygenation of these patients is recommended to reduce hypoxia secondary to respiratory depression or anemia. Anesthetic principles are similar to those given under peritonitis (see p. 513).

Surgical Anatomy

The surgical anatomy of the abdominal cavity is described on p. 513.

Positioning

The animal is placed in dorsal recumbency for a midline exploratory celiotomy. The entire abdomen and caudal chest should be prepped for aseptic surgery.

SURGICAL TECHNIQUES

The procedure for an exploratory laparotomy is given on p. 534. If neoplasia is suspected, biopsies or the entire mass should be submitted for histopathology.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Monofilament synthetic, absorbable suture should be used to close the abdomen in these animals.

POSTOPERATIVE CARE AND ASSESSMENT

Fluid therapy should be continued postoperatively in most animals. Electrolytes, acid-base, and serum protein should be assessed in the postoperative period and corrected as necessary. Postoperative analgesia is recommended (see Chapter 13).

PROGNOSIS

The prognosis for animals with hemoabdomen depends on the underlying disease. Factors associated with death and failure to be discharged from the hospital in dogs include tachycardia, the need for massive transfusion with blood products, and the development of respiratory disease secondary to pulmonary thromboembolism or acute respiratory distress syndrome. Surgical intervention for treatment of hemoperitoneum, regardless of etiology, resulted in discharge from the hospital for 70 of the 83 (84%) dogs in a 2013 study.[26] The source of bleeding was isolated to the spleen in 75 of 83 (90%) dogs in this study; a splenic source of hemorrhage was determined to be a positive predictor of survival to discharge from the hospital. Cats with spontaneous hemoperitoneum are as likely to have neoplastic (particularly splenic hemangiosarcoma) as nonneoplastic disease.[20]

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20

Surgery of the Liver



GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Hepatectomy refers to removal of the entire liver (**total hepatectomy**) or of a portion of the liver (**partial hepatectomy**).

PREOPERATIVE CONCERNS

The liver is the largest gland in the body. It is the primary site for detoxification of many substances and plays a central role in the metabolism of protein, fat, and carbohydrates. Unfortunately, clinical signs of hepatic disease may not be apparent until the disease is advanced and irreversible. Hepatic failure may affect many other organ systems, including the central nervous system (CNS), kidneys, intestines, and heart. Scoring animals undergoing hepatic biopsy for chronic hepatitis based on the presence or absence of ascites, hypoglobulinemia, hepatic encephalopathy, hypoalbuminemia, hyperbilirubinemia, and clinical signs may relate to survival. Ascites in Labrador retrievers presenting for chronic hepatitis may also be associated with a shorter survival time.

The liver produces most of the plasma proteins (i.e., albumin, α- and β-globulins, fibrinogen, prothrombin). Hypoalbuminemia and hypocholesterolemia are each common in patients with hepatic insufficiency. Fluid therapy may further dilute serum albumin; thus severely affected patients may need colloid infusions (see Chapter 4). Albumin levels below 2 g/dL may be associated with delayed wound healing. Potassium abnormalities are common in patients with hepatic disease. Coagulopathies may occur because of diminished synthesis of clotting factors or consumption. Preoperative evaluation of clotting function, especially mucosal bleeding time, is warranted; fresh whole blood transfusions may reduce intraoperative hemorrhage in selected patients. Some patients with hepatic disease are anemic because of nutritional deficiencies, coagulation abnormalities, and/or gastrointestinal hemorrhage. Animals with hematocrits below 20% and anemic animals that are clinically hypoxic or weak should be given preoperative blood transfusions (see Table 4.5 and Box 4.1). Many patients with hepatic disease are anorexic, and some require preoperative nutritional supplementation (see Chapter 10). Hypoglycemia sometimes occurs with severe hepatic insufficiency; blood glucose levels may need to be monitored before, during, and after the procedure. Patients with massive ascites may be dyspneic owing to pressure on the diaphragm that restricts lung expansion; removing some abdominal fluid before induction of anesthesia is sometimes indicated to prevent hypoventilation and hypoxemia. If ascites prevents the patient from lying in dorsal recumbency, ventilation during surgery will be required and may be difficult. Because high ventilator pressures reduce hepatic blood flow, it is important to reduce this pressure by removing some fluid; if time allows, it may be best to do this gradually over a couple of days. Laparoscopic liver biopsy can be done in the presence of ascites, and removal of all the fluid is generally not required. Animals with ascites will reform the fluid after removal. Patients with severe hepatic encephalopathy should be treated symptomatically (e.g., controlled protein diet, antibiotics, cleansing enemas, fluids, lactulose; see p. 555) to diminish or eliminate clinical signs before surgery.

NOTE The cranial location of the liver may make hepatic biopsy somewhat difficult, particularly in large, deep-chested breeds, or when the liver is abnormally small. Laparoscopic sampling can be done in these animals; alternatively, the incision can be extended cranially by incising the xiphoid cartilage and one or two caudal sternebrae to facilitate hepatic exposure during laparotomy.

ANESTHETIC CONSIDERATIONS

The goal of managing patients with hepatic dysfunction is to preserve what liver function is present while avoiding drugs or other factors that might be detrimental. Many medications normally administered during anesthesia undergo hepatic metabolism. In patients with moderate to severe dysfunction, such drugs should be avoided or used in markedly reduced doses. Furthermore, many medications are highly protein bound. With hypoalbuminemia, the percentage of bound drug is decreased and more is free in the plasma, resulting in more profound effects with normal drug dosages. In addition, in patients with severe disease, alterations in the blood-brain barrier heighten the depressant effects of anesthetic medications (Table 20.1). Drugs to be avoided in animals with hepatic dysfunction include (but are not limited to) acepromazine, alpha-2 agonists, pancuronium, vecuronium, and telazol. Premedications should not be administered to patients that are depressed. Midazolam (see Chapter 12) is the preferred benzodiazepine in alert patients in which some sedation is warranted. Diazepam has active metabolites with a potentially very prolonged duration of action. It should be used only in patients with mild to moderate liver dysfunction and with no CNS depression. Because etomidate and propofol rely on redistribution rather than liver metabolism for the initial recovery from the drug effect, they are the induction agents of choice. Etomidate, methohexital, and ketamine lower the seizure threshold. If recurrence of seizures is a risk, propofol should be administered for induction because of its antiepileptic

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ABSTRACT

Surgery is commonly required to diagnose and treat hepatic disease. Chronic liver value abnormalities often require hepatic biopsy to properly diagnose and treat these conditions. Solitary mass lesions of the liver may respond favorably to resection; however, the surgery can be complex and hemorrhage is a possible significant complication. Vascular anomalies associated with the liver may be congenital or acquired, singular or multiple, and correction requires knowledge of the normal anatomy. Congenital portosystemic shunts are the most common form, and when extrahepatic in lcoation may be treated with a number of techniques. Intrahepatic shunts are more challenging to diagnose and often require specialized techniques for treatment. Intrahepatic shunts are typically associated with a higher morbidity and mortality than extrahepatic shunts.

KEYWORDS

Hepatic
liver
portosystemic shunt
neoplasia
arteriovenous fistulae

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TABLE 20.1 Anesthetic Considerations for Animals With Hepatic Disease[a]

Preoperative Considerations

Associated conditions	• Dependent on severity and origin • Anemia • Coagulopathy • Altered renal function • Urinary calculi • Electrolyte abnormalities • Gastritis/gastric erosions • Malnutrition and weight loss • Hypotension • Ascites (due to hypoalbuminemia) • Dyspnea (due to hypoalbuminemia) • Seizures
Bloodwork	• HCT • TP • Electrolytes • BUN • Cr • Glucose • Albumin • +/– Bilirubin • +/– Bile acids • +/– Protein C • +/– Clotting profile • +/– Blood gas • +/– Calcium (if blood transfusions)
Physical examination	• May have jaundice, palpable ascites, hypothermia, CNS depression, thin body condition, respiratory distress • Younger animals may have stunted growth, thin body condition, small liver, and signs of hepatic encephalopathy
Other diagnostics	• Blood pressure • ECG • X-rays (abdominal) • Ultrasound (abdominal) • +/– Liver biopsy

Premedications	• Remove abdominal fluid if patient is dyspneic or if patient unable to tolerate ventrodorsal position while awake
	• Blood transfusion if HCT <20% in dogs and <18% in cats
	• FFP if coagulopathy
	• Correct electrolyte abnormalities
	• Avoid sedatives in sick or depressed patients
	• Administer GI protectant, preferably proton pump inhibitor
	• If patient is anxious, administer midazolam (0.1–0.2 mg/kg IV, IM)
	• If patient is not depressed, then administer:
	  • Hydromorphone[b] (0.05–0.2 mg/kg IV, IM in dogs; 0.025–0.05 mg/kg IV, IM in cats) *or*
	  • Oxymorphone (0.1–0.2 mg/kg IV, IM) *or*
	  • Buprenorphine[c] (0.005–0.01 mg/kg IV, IM)
	• If using etomidate for induction, give a benzodiazepine immediately prior to administration of etomidate to avoid myoclonus
	  • Diazepam 0.25 mg/kg IV
	  • Midazolam 0.25 mg/kg IV

Intraoperative Considerations

Induction	• Preoxygenate 3–5 minutes • Propofol (2–6 mg/kg IV) *or* • Etomidate (0.5–1 mg/kg IV); give a benzodiazepine prior as noted above to prevent myoclonus

Maintenance	• Isoflurane or sevoflurane *plus*
	  • Fentanyl (1–10 µg/kg IV PRN in dogs; 1–4 µg/kg IV PRN in cats), *plus* PRN
	  • Fentanyl CRI (2–15 µg/kg/h IV after 1–5 µg/kg IV loading dose), *or*
	  • Hydromorphone[b] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Oxymorphone (0.1–0.2 mg/kg IV, IM), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV PRN), *plus*
	  • Ketamine (low dose; 0.5–1 mg/kg IV), *or*
	  • Ketamine CRI (0.5 mg/kg IV loading dose, then 10 µg/kg/min IV)
	• For hypotension (to keep MAP 60–80 mm Hg), give phenylephrine, ephedrine, dopamine, norepinephrine, or vasopressin as needed
	• For muscle relaxation, give (need to provide positive-pressure ventilation)
	  • Cisatracurium (0.1 mg/kg IV), *or*
	  • Atracurium (0.1–0.25 mg/kg IV)

Fluid needs	• 5–20 mL/kg/h to replace evaporative losses. Place two forelimb IV catheters if large blood loss is a possibility. Have blood and colloids available to replace blood loss • Give dextrose if hypoglycemic

Continued

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TABLE 20.1 Anesthetic Considerations for Animals With Hepatic Disease[a]—cont'd

Monitoring	• Blood pressure • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature • U/O • +/– Arterial line • +/– CVP • Serial HCT if moderate to high blood loss • Blood glucose if hypoglycemic
Blocks	• Not recommended if coagulopathy

Postoperative Considerations

Analgesia	• Fentanyl CRI (1–10 µg/kg IV loading dose, then 2–10 µg/kg/h IV), *or* • Morphine[d] (0.1–0.5 mg/kg IV or 0.1–1 mg/kg IM q2–8h in dogs; 0.025–0.2 mg/kg IV or 0.05–0.3 IM q2–8h in cats) if no hypotension, *or* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q3–8h in dogs; 0.025–0.1 mg/kg IV, IM q3–8h in cats), *or* • Hydromorphone CRI (0.025–0.0.5 mg/kg/h IV in dogs), *or* • Oxymorphone (0.1–0.2 mg/kg IV, IM), *or* • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM q4–8h or 0.01–0.02 mg/kg OTM q6–12h in cats) Note: NSAIDs are contraindicated
Monitoring	• SpO_2 • Blood pressure • HR • Respiratory rate • Temperature • U/O
Bloodwork	• HCT • Electrolytes • Glucose • Abnormal preoperative laboratory work • +/– Blood gas
Estimated pain score	• Moderately severe

[a]Patients with hepatic disease may have hypoalbuminemia with decreased binding as well as decreased clearance of many drugs. It is recommended to start with low doses and titrate slowly to effect.
[b]Monitor for hyperthermia in cats.
[c]Buprenorphine is a better analgesic than morphine in cats.
[d]Give slowly to prevent histamine release.
BUN, Blood urea nitrogen; *CNS,* central nervous system; *Cr,* creatinine; *CRI,* constant-rate infusion; *CVP,* central venous pressure; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *FFP,* fresh-frozen plasma; *GI,* gastrointestinal; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *MAP,* mean arterial pressure; *NSAID,* nonsteroidal antiinflammatory drug; *OTM,* oral transmucosal; *PRN,* as needed; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein; *U/O,* urine output.

effect. Alfaxalone can be used for induction but does undergo hepatic metabolism; metabolites are eliminated by hepatic, fecal, and renal routes. The sympathetic stimulation of ketamine makes it a less desirable induction agent, but its use at low doses may assist in lowering opioid requirements. Barbiturates should be used only in markedly reduced dosages. Likewise, the opioids are CNS depressants and must be used at reduced dosages. The opioid of choice during surgery and in the postoperative period is fentanyl because it can be titrated easily to effect as a continuous infusion (see Chapter 13). Early studies of morphine at doses 5 to 10 times normal recommended analgesic doses showed decreased hepatic portal blood flow. However, at normal doses and at the reduced doses required in hepatic dysfunction, this is not a concern. Morphine does cause histamine release with subsequent hypotension, usually occurring within 5 minutes of intravenous (IV) administration. If morphine is administered, hypotension should be anticipated and treated with a vasopressor. Usually, one to two doses is adequate because the hypotension is of short duration. Hydromorphone and oxymorphone are also good analgesics and do not cause histamine release when given intravenously. In addition, if the depressant effects of mu-agonist opioids become pronounced, they can be partially reversed with butorphanol (0.1 mg/kg IV).

Because all anesthetics are CNS depressants, fewer IV agents should be used in patients with hepatic dysfunction. The inhalant anesthetics sevoflurane and isoflurane are generally considered the maintenance drugs of choice. Both maintain hepatic arterial blood flow while increasing portal blood flow, thereby increasing hepatic oxygenation. Conversely, halothane reduces hepatic blood flow by vasoconstriction and should not be used. Nitrous oxide has minimal hepatic effect and may be used in these patients. Its use should be balanced against the need to give a higher inspired oxygen concentration. It is not uncommon for it to be necessary to use reduced amounts of the inhalant. Depending on the patient, sometimes only one-half to three-quarters minimum alveolar concentration is needed.

Because preservation of hepatic oxygenation has been shown to be extremely important in patients with liver dysfunction, hepatic blood flow must be maintained. Factors known to reduce hepatic blood flow, such as hypotension, excessive sympathetic stimulation (e.g., inadequate pain control), and high airway pressures, should be avoided. If possible, removal of ascites

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preoperatively is beneficial because it allows improved venous return associated with lessened venous compression. Venous compression from ascites is exaggerated when the animal is anesthetized and placed in dorsal recumbency; sudden removal of ascites with surgical suction can lead to profound hypotension due to rapid venous decompression. To increase oxygenation, preoxygenate before induction, keep inspired oxygen high during surgery, and do not rush extubation. Patients often need supplemental oxygen in the postoperative period. Hypotension needs to be treated promptly with vasopressors and/or inotropes (see Box 19.6). In patients with chronic liver disease and decreased liver mass, the circulatory system may be in a hyperdynamic state. Cardiac output is increased and is dependent on preload and systemic/pulmonary vasodilation. If chronic ascites has been present, some underlying renal dysfunction and impaired free water clearance may occur. Coupled with hypoalbuminemia, colloids are often warranted instead of large boluses of crystalloids. Fluid maintenance needs to be individualized based on underlying causes, severity of hepatic and renal dysfunction, degree of anemia and hypoalbuminemia, blood loss in surgery, and evaporative losses due to incision size. As a general rule, replace evaporative losses with crystalloids and blood loss with colloids to assist in maintaining adequate blood pressure.

Monitoring of patients with moderate to severe hepatic dysfunction is extremely important. If the surgery time is expected to be long or high blood loss a risk, it is important to measure arterial pressures. In addition, central venous pressure monitoring can assist in volume replacement. Patients are at risk of decompensation in the postoperative period. Therefore close monitoring needs to be continued for 24 to 48 hours. Monitor patients with severe hepatic dysfunction for potential coagulopathies and ascertain the need for administration of fresh-frozen plasma.

See recommendations on p. 556 for anesthetic management of animals with portosystemic shunts (PSSs).

ANTIBIOTICS

Aerobic and anaerobic bacteria normally reside in the liver but may proliferate with hepatic ischemia or hypoxia. Therefore prophylactic antibiotics are warranted in patients with severe hepatic disease that are undergoing hepatic surgery (other than simple biopsy). The pharmacokinetics of antibiotics may be altered in these patients by depressed hepatic metabolism, alterations in hepatic arterial or portal blood flow, hypoalbuminemia, or reductions in biliary excretion. Antibiotics are specifically indicated in the treatment of hepatic encephalopathy (see p. 555), bacterial cholangitis/cholangiohepatitis, and hepatic abscess. Broad-spectrum antibiotics effective against anaerobes (e.g., penicillin derivatives, metronidazole, clindamycin) are appropriate and relatively safe in patients with hepatocellular compromise (Box 20.1). Potentially hepatotoxic antibiotics (e.g., doxycycline, chlortetracycline, erythromycin) should be avoided if possible.

NOTE Metronidazole, when administered at doses above 40 to 50 mg/kg of body weight per day, can cause severe neurologic signs (e.g., central vestibular signs such as ataxia, nystagmus, head tilt, and seizures) in dogs and cats.

SURGICAL ANATOMY

The diaphragmatic surface (parietal surface) of the liver is convex and lies mainly in touch with the diaphragm. The visceral surface faces caudoventrally and to the left and contacts the stomach, duodenum, pancreas, and right kidney. Six hepatic lobes are present (Fig. 20.1). The borders of the liver are normally sharp but appear more rounded in young animals and in those with infiltrated, congested, or scarred livers. The liver has two afferent blood supplies: a low-pressure portal system and a high-pressure arterial system. The portal vein drains the stomach, intestines, pancreas, and spleen and supplies four-fifths of the blood that enters the liver. The remainder of the afferent blood supply is derived from the proper hepatic arteries. These arteries are branches of the common hepatic artery and may number between two and five. Efferent drainage of the liver occurs through the hepatic veins. In the fetal pup, the ductus venosus shunts blood from the umbilical vein to the hepatic venous system. The ductus venosus becomes fibrotic after birth and is known as the *ligamentum venosum.* Bile, formed in the liver, is discharged into bile canaliculi lying between the hepatocytes. These canaliculi unite to form interlobular ducts that ultimately merge to form lobar or bile ducts (see p. 572). The portal vein, bile ducts, hepatic artery, lymphatics, and nerves are contained in the lacelike and nonsupporting portion of the lesser omentum known as the *hepatoduodenal ligament.*

BOX 20.1 Antibiotics in Animals With Hepatocellular Compromise

Ampicillin
22 mg/kg IV, IM, or SC q6–8h

Metronidazole (Flagyl)
10–12 mg/kg PO or IV q12h

Cefazolin (Ancef, Kefzol)
22 mg/kg IV or IM q8–12h

Clindamycin (Antirobe)
Dogs: 11–33 mg/kg q12h PO, or 11 mg/kg IV (dilute and give slowly when administering IV)
Cats: 11–33 mg/kg PO or SC q12–24h

Ampicillin Sodium and Sulbactam Sodium (Unasyn)
15–30 mg/kg IV q6–8h off-label use

IM, Intramuscular; *IV,* intravenous; *PO,* orally; *SC,* subcutaneous.

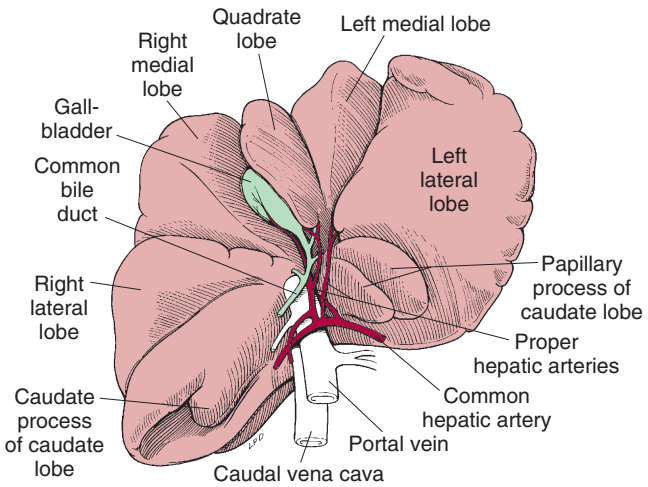


FIG. 20.1 Anatomy of the liver.

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NOTE Use caution when dissecting around the pylorus to avoid damaging the common bile duct.

SURGICAL TECHNIQUES

The standard approach for hepatic surgery is a cranial ventral midline abdominal incision. The caudal aspect of the sternum can be split if additional exposure is needed. Surgery of the liver is complicated by the fact that hepatic tissue is friable. Because of the sparsity of fibrous protein in the liver, sharp dissection is difficult and results in retraction of blood vessels and bile ducts in the friable stroma. Ligation of structures (e.g., blood vessels, bile ducts) after they have been cut is extremely difficult. Packing the liver firmly enough to obtain hemostasis may cause ischemia and necrosis. Maintaining hepatic blood supply is important because the liver normally harbors potentially pathogenic anaerobes. For these reasons, surgery of the liver requires techniques different from those used in surgery on most other abdominal organs.

Hepatic biopsies are commonly indicated in patients known to have or suspected of having hepatic disease. Biopsies may be obtained percutaneously, with laparoscopy (see p. 545), or at surgery. Partial hepatectomies are less commonly performed but may be indicated for focal neoplasms or trauma. Several breeds have been associated with hepatopathies, including the Bedlington terrier, Doberman pinscher, cocker spaniel, West Highland white terrier, Dalmatian, Skye terrier, and Labrador retriever. Chronic hepatitis of Labrador retrievers occurs in middle-aged to older dogs; it causes no or vague clinical signs and is likely associated with copper accumulation. It is more common in female Labradors than males and may be associated with a genetic abnormality in liver copper metabolism, although the exact cause is unknown. Anorexia, hypoglobulinemia, and prolonged partial thromboplastin time (PTT) may be associated with shorter survival; however, the median survival is approximately 1 year, with some affected dogs surviving as long as 8 years.

NOTE Liver biopsy is appropriate in patients with clinical signs of hepatic disease and in clinically normal animals with laboratory abnormalities or imaging changes consistent with hepatic disease.

Percutaneous Liver Biopsy

Percutaneous core biopsies and fine-needle aspirations are relatively inexpensive and easy techniques that can be sensitive and specific for **focal** lesions (e.g., tumors such as carcinomas and lymphosarcoma) when used with ultrasound guidance. However, with the exception of feline hepatic lipidosis, these percutaneous techniques are insensitive and unreliable in patients with **diffuse** hepatic disease (e.g., inflammation, fibrosis, cirrhosis, necrosis) and in those that may have congenital vascular shunts; their use is not recommended when such diseases are suspected. Properly obtained laparoscopic or surgically obtained samples are superior to those obtained percutaneously and are recommended for the diagnosis of diffuse liver disease.[1] Animals with clinical bleeding, severe thrombocytopenia (i.e., fewer than 20,000 platelets/μL), prolonged mucosal bleeding time, highly vascular lesions (determined with ultrasound), or cavitary lesions generally should not have percutaneous core biopsies because of the risk of uncontrollable hemorrhage or abdominal infection. Caution is also recommended with fine-needle aspiration in these patients.

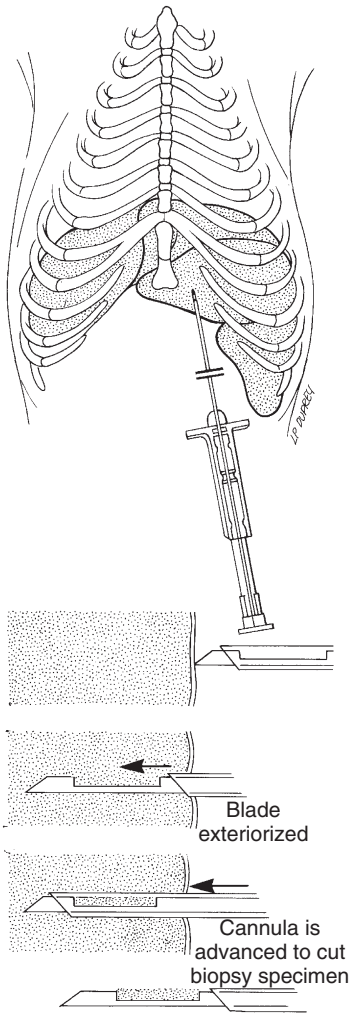


FIG. 20.2 Tissue core biopsy.

Tissue core biopsies may be obtained with a Tru-Cut biopsy (Cardinal Health) (Fig. 20.2), a large-bore needle, or an automated biopsy device (e.g., Bard Biopsy instrument). The latter should not be used in cats because of the potential for death associated with the shock wave caused by triggering the device. For histopathologic specimens, the Tru-Cut needle should be removed from the syringe or gun and placed in formalin. Once the sample has been fixed, it should be removed from the needle for processing. A core biopsy should use the largest gauge needle that may be used safely in the patient, typically a 14-gauge needle. If core biopsies are performed, at least two or three samples, each 2 cm long, are recommended. Core biopsies are generally taken from only one liver lobe (i.e., the left liver lobe, so as to minimize the chance of lacerating the bile ducts or gallbladder, both of which are on the right side). However, this is a significant limitation because lesions may be present in only a few of the liver lobes. The throw of the needle is at least 2 cm, which can lacerate relatively large vessels, and extreme care must be taken to ensure that the core biopsy needle will not pass through the liver and lacerate structures (e.g., veins, stomach, intestines, diaphragm, lungs, heart) under the hepatic lobe being biopsied.

Fine-needle aspirates may be obtained using two different techniques. *In the first technique, attach a syringe or an aspiration gun with a syringe to a 20- to 25-gauge, 1- to 3-inch (2.54–7.62 cm)*

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needle. Insert the needle into the area to be sampled, aspirate 5 to 10 mL two or three times (i.e., quickly and briskly pull the plunger back and immediately release it, repeat this two or three times), remove the needle from the syringe, aspirate 5 mL of air, reattach the needle, and then blow the contents of the needle onto a glass slide. If blood or fluid is seen entering the hub during the aspiration process, immediately stop aspirating. In the second technique, repeatedly pass the needle (without the syringe attached) through the liver with no suction applied. After three to five passes, attach the needle to a syringe and blow the contents out onto a slide.

Fine-needle aspiration is most likely to be diagnostic in patients with diffuse hepatic neoplasia (e.g., lymphosarcoma), infectious causes (e.g., histoplasmosis), and feline idiopathic hepatic lipidosis. However, inability to diagnose such conditions on a fine-needle aspirate does not preclude the disease or even make it unlikely enough to essentially eliminate. Furthermore, even if one of these conditions is found, other undiagnosed diseases could be present. This latter possibility is particularly important in cats because almost all sick, anorexic cats will have some fat vacuoles in the hepatocytes. Therefore, before clinical illness due to hepatic lipidosis can be diagnosed, one must determine that fat in the hepatocytes is sufficient to cause hepatic dysfunction. Furthermore, diagnosing hepatic lipidosis does not guarantee that there is *not* another hepatic disease present that was not found by the fine-needle technique. Cytologic evaluation of ultrasound-guided fine-needle aspirates is most likely to agree with histopathologic findings when the animal has vacuolar hepatopathy; however, this condition is typically not clinically important as a cause of hepatic failure or insufficiency. Overall agreement between cytology and histopathology in dogs and cats is usually low. Concurrence with wedge biopsy specimens is similarly low. The limitations of diagnosing hepatic disease in dogs and cats by percutaneous techniques should be recognized by clinicians.

NOTE False-negative results (missing a diagnosis) are far more common than false-positive results (categorizing a healthy animal as diseased); therefore, if your clinical index of suspicion is high, consider using another technique to biopsy the liver.

Percutaneous biopsies may be obtained under tranquilization or heavy sedation. Approximately half of cats require injectable anesthesia for needle biopsy to be performed. Samples are obtained using a transthoracic or transabdominal approach; however, to avoid laceration of the liver during respiration, the former should be utilized only if the latter (described here) is not possible.

Percutaneous Blind Biopsy

With the animal in dorsal recumbency, clip the hair from the area surrounding the xiphoid process and prepare it for aseptic surgery. Make a small incision in the skin on the left side between the costal arch and the xiphoid process. Insert the biopsy needle through the skin incision in a craniodorsal direction, angling it slightly toward the left of midline. Advance the needle until ultrasound guidance shows the needle to be positioned at the surface of the liver. Advance the biopsy needle into the hepatic tissue and obtain the biopsy sample (see Fig. 20.2).

Percutaneous Ultrasound-Guided Biopsy

Three ultrasound-guided methods can be used to biopsy or aspirate hepatic structures or lesions. Any of these techniques may be used when samples of the liver are obtained. If diffuse disease is suspected, the left liver should be sampled to avoid the main biliary structures. Extreme care should be taken to ensure that no large biliary or vascular structures lie within the 2-cm area required for needle advancement. The first technique is called the *indirect guidance method* and is not generally recommended. It entails using ultrasound to find the structure of interest, then the ultrasound probe is removed. The needle is passed blindly in the area of interest. This technique is applicable only when the target is extremely large and direct visualization of the needle is not required as it is passed into the structure to be biopsied or aspirated.

The other two techniques allow direct visualization of the needle as it is passed into the liver. It is critical that the needle pass at an oblique angle to the ultrasound beam for it to be seen on the image. A needle that passes parallel to the beam will not create useful echoes. The second technique is the *freehand technique*. This method requires good hand-eye coordination and takes practice to master. Many sonographers prefer this technique because it allows the operator more choices in approaching the structure. *Handle the ultrasound probe with one hand (usually the nondominant hand) and the needle with the opposite hand. Use the probe to visualize the structure of interest, then pass the needle into it and obtain the biopsy.*

The last technique is the *needle guide technique*. Many ultrasound machines have a manufactured needle guide that fits onto the transducer. This equipment will guide the needle at a preset angle and will maintain the orientation to the transducer. Software on the machine will project the needle path onto the screen, allowing accurate placement of the needle. One problem with this approach is that the needle guide is often bulky and thus the choice of approaches may be limited, particularly in small animals. Because the needle is passed through the needle guide, sterile technique is essential. *Sterilize the guide before use. Apply a sterile covering to the transducer (a sterile surgical glove works well). Place coupling gel into the glove, then fit it over the transducer. Attach the guide. Pass the needle through the needle guide and obtain the biopsy.*

Complications associated with automated needle biopsy devices occur, particularly in cats. The strong increase in vagal tone due to the force of the automated device may lead to severe bradycardia, weak peripheral pulses, respiratory impairment, loss of consciousness, and cardiac arrest. A semiautomated biopsy needle device for sample procurement may be preferable.

Laparoscopic Liver Biopsy

Laparoscopy (see Chapter 14) offers several advantages over other hepatic biopsy techniques. First, laparoscopy can find lesions missed by ultrasound, allowing the clinician to biopsy clearly abnormal areas that would have been missed if a percutaneous technique had been used (Video 20.1). Second, laparoscopy allows one to obtain better tissue samples than is possible with percutaneous techniques (sufficient hepatic tissue can readily be obtained for histopathology, mineral analysis, and culture), and it allows multiple liver lobes to be sampled (as opposed to just one lobe, as commonly occurs with core needle techniques). Laparoscopic biopsy using a 5-mm "double-spoon" biopsy forceps should obtain approximately 45 mg of tissue. In comparison, an 18-gauge Tru-Cut will obtain approximately 5 mg of tissue, and a 14-gauge Tru-Cut will obtain approximately 15 to 20 mg of tissue. At the same time, the endoscopist can quickly examine the abdomen and peritoneum, omentum, stomach, pancreas, intestines, and/

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or kidneys to evaluate whether other, unsuspected disease is present. Finally, laparoscopy can be done quickly by an experienced laparoscopist (in less than 15 minutes), and the patient is routinely able to be discharged within hours of completion of the procedure. Coagulopathies are not an absolute contraindication unless they are severe; electrocautery and coagulation-enhancing materials can be used but are seldom needed. The amount of collateral tissue damage should be considered when determining the sample size needed if coagulatory methods are used because there will be coagulative and cavitational fragmentation of the samples.

Laparoscopic hepatic biopsy is best obtained with double-spoon type forceps. *If no focal abnormalities are found, open the forceps and place them around the edge of the liver lobe (Video 20.2). Push the forceps into the lobe until the entire cup of the biopsy forceps is filled with hepatic tissue. Do not insert the sampling forceps beyond the point at which the two jaws come together, as this can result in tearing of the liver lobe. Tightly close the jaws, hold for 10 seconds, and pull the sample off the lobe. If a biopsy of a different area of the liver lobe is desired, open the forceps and thrust the lower jaw into the liver lobe at the desired spot. Once the lower jaw is thrust as far as desired into the liver lobe, close the upper jaw over the lower jaw and retrieve the tissue sample.* Samples can also be similarly obtained from central portions of the liver lobe. *Before removal of carbon dioxide insufflation, evaluate each site and be certain that any blood present is coagulated by passing the forceps across the area of blood adjacent to each site sampled.*

NOTE It is important that the endoscopist always be able to see everything that is being grabbed by the forceps; never grab anything unless you can see it.

Alternatively, use a harmonic scalpel to harvest liver samples. Collect a 2 × 1 cm sample of liver using a harmonic scalpel with a working blade length of 15 mm and set to a power level of 3.

Hemorrhage routinely occurs when the sample is torn off the liver, but one must be aware that the laparoscope will magnify any hemorrhage that occurs. Typically, less than 1 to 5 mL of blood is lost with a biopsy. Less hemorrhage may occur when the harmonic scalpel is used; however, significantly more coagulation necrosis, cavitational fragmentation, and fibrosis necessitate that slightly larger samples be obtained to offset the tissue damage. Sampling from central regions of the liver with the harmonic scalpel may rely on natural coagulation rather than vessel sealing. Needle biopsies often result in inadequate samples for histopathologic evaluation.

Surgical Liver Biopsy

Biopsies of the liver should be done routinely during exploratory laparotomy in animals known to have or suspected of having liver disease. Surgical biopsy allows the entire liver to be thoroughly inspected and palpated and focal lesions to be biopsied for histopathologic examination, culture, or copper analysis. Furthermore, hemorrhage from the biopsy site can be readily identified and controlled with proper technique. If generalized hepatic disease is present, the sample can be taken from the most accessible site (marginal biopsy samples). With focal disease, the entire liver should be palpated carefully for intraparenchymal nodules or cavities, and representative samples obtained.

A centrally located sample of liver parenchyma may be taken with a Baker biopsy punch. The recommended size for collection of at least six to eight portal triads is 6 mm in diameter. *Press the punch against the area to be sampled and advance it using a clockwise and counterclockwise twisting motion. Use Metzenbaum scissors to separate the deep margin of the sample from the parenchymal attachment. If needed, insert hemostatic gelatin sponge material into the defect to aid in hemostasis.*

A biopsy of the hepatic margin may be obtained by the "guillotine" method. *Place a loop of suture around the protruding margin of a liver lobe (Video 20.3). Pull the ligature tight and allow it to crush through the hepatic parenchyma before tying it (Fig. 20.3A).* As the suture tears through the soft hepatic tissue, vessels and biliary ducts are ligated. *Hold the liver gently between the fingers if necessary, and with a sharp blade, cut the hepatic tissue approximately 5 mm distal to the ligature (allowing the stump of crushed tissue to remain with the ligature). To avoid crushing the biopsy sample and causing artifacts, do not handle it with tissue forceps. Use the pack from which the suture was removed as a "cutting board" to reduce handling of the sample. Place a portion of the sample in formalin for histologic examination; reserve the remainder for culture and cytologic study. Check the biopsy site for hemorrhage. If hemorrhage continues, place a pledget of absorbable gelatin foam over the site. As an alternative, if a focal (nonmarginal) area of the liver is to be sampled, use a punch biopsy or a Tru-Cut biopsy (see Fig. 20.2), or place several overlapping guillotine sutures around the margin of the lesion and excise it (see Fig. 20.3B). Use caution with a punch biopsy to avoid penetrating more than half the thickness of the liver with each biopsy. Apply pressure to the site until bleeding stops. If hemorrhage continues, place a pledget of absorbable gelatin sponge over the site.*

Partial Lobectomy

Partial lobectomy may be indicated in some cases in which disease involves only a portion of a liver lobe (e.g., peripheral hepatic arteriovenous [AV] fistula, focal neoplasia, hepatic abscess, trauma). Partial lobectomy may be challenging because of the difficulty in obtaining hemostasis and should be done with extreme caution in animals with bleeding disorders. Stapling instruments have been used for partial and complete lobectomies, but discretion should be exercised in their use because hemorrhage may occur if the staples do not adequately compress hepatic tissue. This is particularly important in cats; in one study, 14 out of 18 required additional methods to establish hemostasis.[2]

Determine the line of separation between normal hepatic parenchyma and that to be removed, and sharply incise the liver capsule along the selected site (Fig. 20.4A). Bluntly fracture the liver with the fingers (see Fig. 20.4B) or the blunt end of a Bard-Parker scalpel handle, and expose the parenchymal vessels. Ligate large vessels (hemoclips may be used), and electrocoagulate small vessels encountered during the dissection (see Fig. 20.4C). As an alternative, place a stapling device (e.g., Autosuture TA 90, 55, or 30) across the base of the lobe and deploy the staples. Excise the hepatic parenchyma distal to the ligatures or staples. Before closing the abdomen, make sure the raw surface of the liver is dry and free of hemorrhage. In small dogs and cats, several overlapping guillotine sutures (as described previously) may be placed along the entire line of demarcation (Fig. 20.5). Be sure that the entire width of the hepatic parenchyma is included in the sutures. After tightening the sutures securely, use a sharp blade to cut the hepatic tissue distal to the ligature, allowing a stump of crushed tissue to remain with the ligature.

Partial lobectomy in dogs up to 50 kg may be done using a single pretied loop ligature of 0 glycolide-lactide copolymer, a

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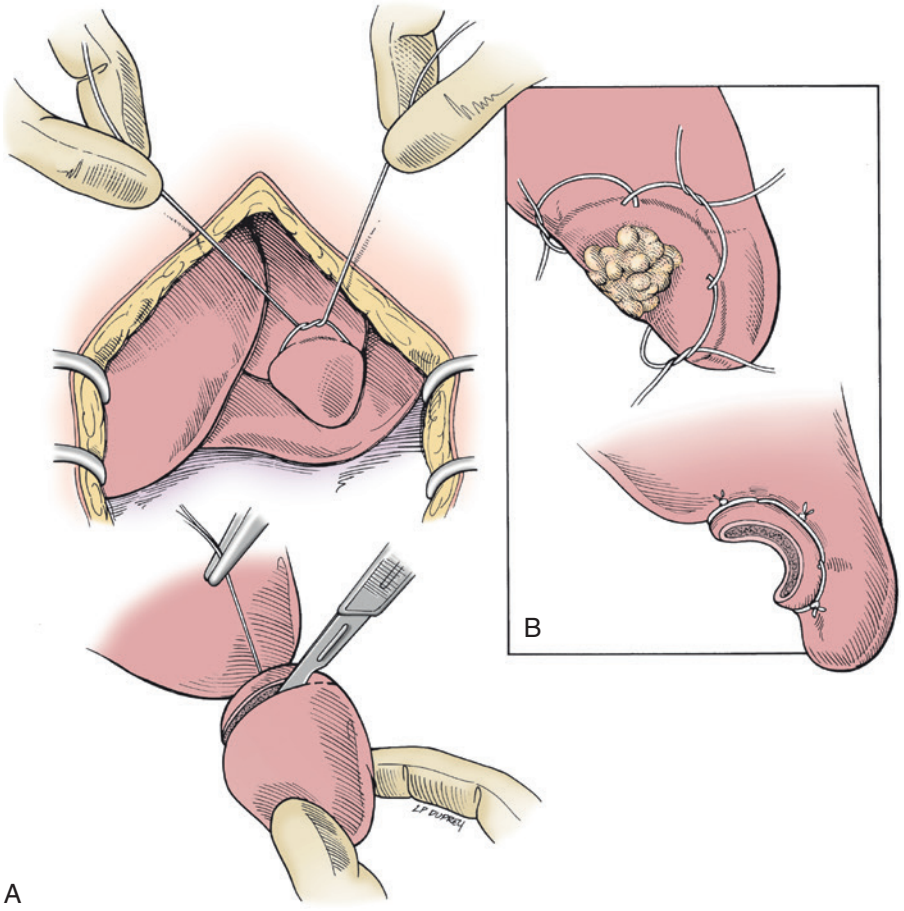


FIG. 20.3 Biopsy of the hepatic margin by the guillotine method. (A) Place a loop of suture around the protruding margin of a liver lobe. Pull the ligature tight, and allow it to crush through the hepatic parenchyma before tying it. Using a sharp blade, cut the hepatic tissue approximately 5 mm distal to the ligature. (B) As an alternative, place several overlapping guillotine sutures around the margin of the lesion and excise it.

vessel-sealing device (see p. 78), or a harmonic scalpel.[3] Intraoperative hemorrhage may occur with pretied loops and the harmonic scalpel, necessitating additional ligation or clip application. None of the three methods results in clinically evident hemorrhage.[3]

Complete Lobectomy

Complete lobectomy may be indicated for some focal lesions involving one or two hepatic lobes (e.g., traumatic lacerations of the liver, hepatic AV fistulae). The vascular anatomy of the liver is variable within and between species[4,5]; however, the left lobes of the liver (i.e., left lateral and left medial lobes) maintain their separation near the hilus to a greater degree than the other lobes do. Therefore the left lobes can often be removed in small dogs and cats by placing a single encircling ligature around the base of the lobe.

For the right lateral and caudate lobes, careful dissection around the hepatic caudal vena cava is usually necessary. *Before performing the dissection, pass umbilical tape around the portal vein, celiac artery, cranial mesenteric arteries, and caudal vena cava in front of and behind the liver.* The tape is passed through rubber tubing, which can be used to occlude the hepatic blood supply if uncontrollable hemorrhage occurs. *For the left lobes in small dogs and cats, crush the parenchyma near the hilus with the fingers or a forceps. Place an encircling ligature around the crushed area, and tie. For the left lobes in larger dogs and for the right and caudate lobes, carefully dissect, if necessary, the lobe from the caudal vena cava. Isolate the blood vessels and biliary ducts near the hilus, and ligate them. Double ligate or oversew the ends of large vessels. Resect the parenchymal tissue, leaving a stump of tissue distal to the ligatures to prevent retraction of hepatic tissue from the ligatures and subsequent hemorrhage.*

Dissection of individual liver lobes can be achieved for selective excision. Each may be drained by more than one hepatic vein, and careful identification of the venous drainage can be achieved using the inner cannula of a Poole suction tip. The hepatic vein(s) associated with the left lateral or left medial liver lobe may be closed using a thoracoabdominal stapling device after ligation and transection of the hepatic artery and biliary duct associated with the lobe to be removed. The general process involves first identifying and ligating the hepatic arterial supply and biliary drainage, followed by ligation or stapling of the veins draining the dissected lobe(s).

Left Lateral Liver Lobe

For specific dissection of the left lateral liver lobe isolate, ligate and divide the left lateral lobar hepatic artery and biliary duct. Retract the left lateral lobe laterally, and retract the left medial lobe medially.

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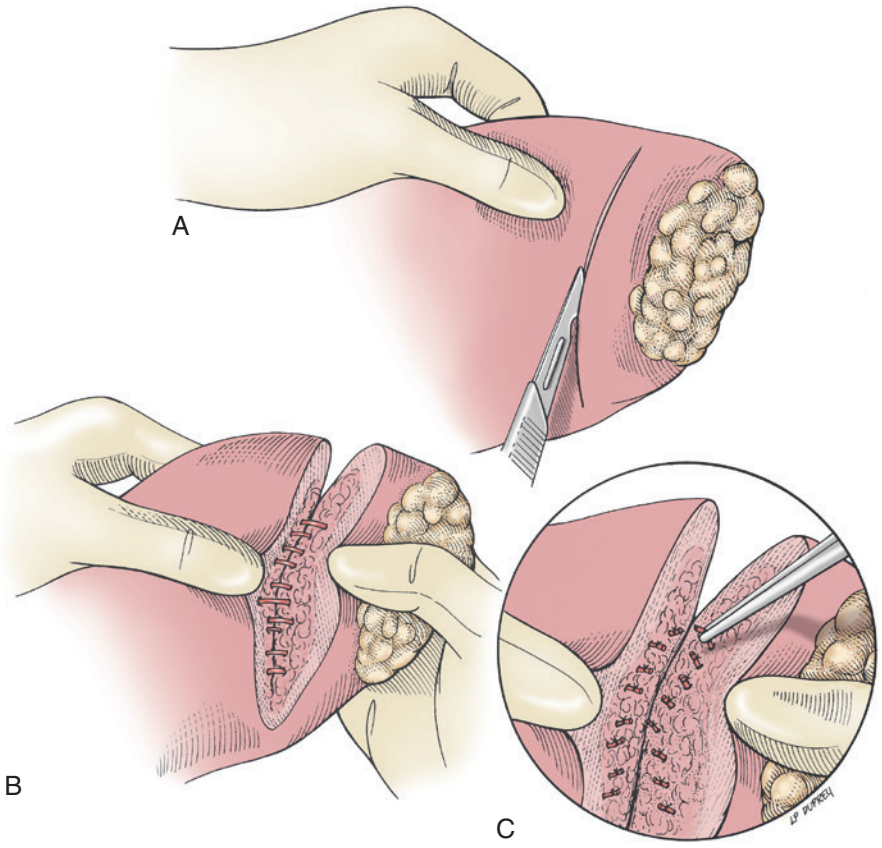


FIG. 20.4 Partial lobectomy. (A) Determine the line of separation between normal hepatic parenchyma and that to be removed, and sharply incise the liver capsule along the selected site. (B) Bluntly fracture the liver and expose the parenchymal vessels. (C) Ligate large vessels and electrocoagulate small bleeders.

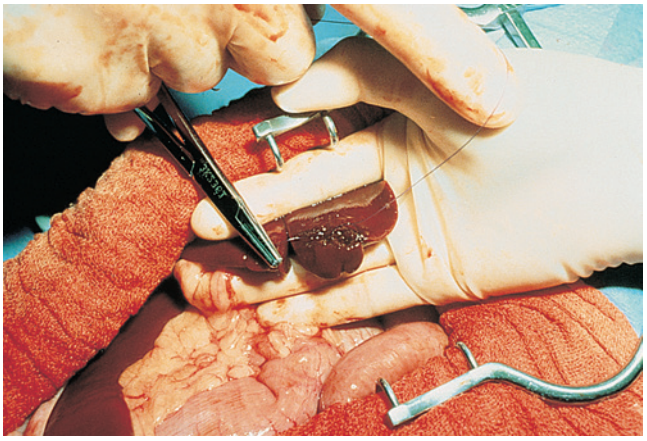


FIG. 20.5 Partial hepatectomy or biopsy may be performed by placing a suture around the liver section to be removed and pulling it tight, such that the parenchyma is crushed and vessels are ligated.

Expose the left lateral portal vein and hepatic vein(s), the latter being cranial and to the left. Isolate, ligate, and divide the left lobar portal vein, then separate the parenchyma of the left lateral lobe from the left hepatic vein using the inner cannula of a Poole suction tip. Divide the triangular ligament, and ligate and divide the left lateral lobar hepatic vein.

Left Medial Liver Lobe

For specific dissection of the left medial liver lobe, use cranial retraction for hilar exposure. Isolate, ligate, and transect the left medial lobar portal vein. Isolate, ligate, and transect the left medial lobar hepatic artery and biliary duct individually where they are located, in the fibrous tissue dorsal to the transected portal vein. Retract the left medial liver lobe to the left and the quadrate lobe to the right to dissect the parenchyma from the hepatic veins using the inner cannula of a Poole suction tip. Use ligation and cauterization to separate vessels extending from the left lateral liver lobe. Retract the left medial liver lobe cranioventrally and continue to separate the hepatic parenchyma. Ligate the hepatic vein(s) as they are identified.

Central Division of the Liver

The central division of the liver may be removed following cholecystectomy or without it. *Use Lahey gallbladder forceps to bluntly dissect the base of the quadrate lobe. Isolate and divide the quadrate lobar portal vein, biliary duct, and artery, and ligate and divide them en bloc. Perform the same dissection and transection of the right medial liver lobe portal vein, artery, and biliary duct. Bluntly separate the parenchyma between the quadrate and right medial liver lobes using the inner cannula of a Poole suction tip. Retract the lobes to expose the diaphragmatic surface; division of the triangular ligamentous attachments will aid in the exposure.*

The accessory right hepatic vein is identified to the right of the right medial lobar portal vein. *Isolate the accessory right hepatic*

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vein with blunt dissection on the diaphragmatic surface of the liver and ligate it. Retract the quadrate lobe to the right, and separate the parenchyma joining it and the left medial liver lobe to locate, isolate, and transect the central hepatic vein. If the gallbladder has not been removed, suture it to the diaphragm or the left medial liver lobe to prevent it from twisting.

Right Lateral Liver Lobe

Expose the right lateral liver lobe by retracting the duodenum and the central division of the liver to the left. Transect the left triangular ligament and create a window in the peritoneum; isolate, ligate, and transect the right lateral lobar portal vein, hepatic artery, and biliary duct, taking care to avoid the portal vein of the central division. Alternatively, the right lateral lobe portal vein, artery, and biliary duct may be individually isolated, ligated, and divided. *Approach the right lateral lobe hepatic vein(s) in the cranioventral liver for ligation.*

Caudate Process of the Caudate Liver Lobe

Approach this lobe via incision of the hepatorenal ligament. Create a window in the overlying hilar peritoneum and ligate the supplying artery and draining biliary duct. Isolate, ligate, and transect the portal vein after its branching from the right portal vein. Identify the caudate lobar hepatic vein cranial and dorsal to the portal vein and isolate it using blunt dissection for suture or thoracoabdominal stapling device ligation. Approach the papillary process of the caudate liver lobe after opening the omental bursa. Retract the stomach caudally and ligate the papillary lobar artery ventral to the papillary lobar portal vein. Carefully identify, isolate, and ligate the papillary lobar portal vein, and avoid the left portal vein as it crosses over the papillary process. Identify, ligate, and transect the papillary lobar biliary duct, then bluntly separate parenchymal attachments to the caudate process. Expose and ligate the associated hepatic vein.

HEALING OF THE LIVER

The liver is unique among the visceral organs in its healing properties. It has relatively little connective tissue stroma, is highly susceptible to small changes in blood flow, and has an enormous regenerative capacity. With regeneration, adequate liver function is possible in patients even after 80% of the organ has been removed or destroyed. Lacerations of the liver should be closed only when bleeding is profuse. If lacerations are sutured, they should be closed in a manner that does not create an internal pocket of bile or blood, nor cause ischemia of surrounding cells. The proper hepatic artery can be ligated as an emergency measure to control hemorrhage from extensive liver lacerations. Complex fractures and severe contusions should be treated by hepatic lobectomy if ligation of the hepatic artery does not result in hemostasis.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Guillotine biopsies often are performed with large (0 or 2-0) chromic gut suture or polyglactin 910. Suture with good knot security (e.g., silk suture) may facilitate partial hepatectomy. Absorbable suture may be used for vessel ligation in complete and partial lobectomies.

POSTOPERATIVE CARE AND ASSESSMENT

Recovery from anesthesia should be monitored closely in animals with severe hepatic dysfunction. Because of the increased half-life of some drugs in patients with hepatic dysfunction, recovery may be prolonged. IV fluids should be provided until the patient is able to maintain hydration, but care must be taken to avoid overhydrating hypoalbuminemic patients. Blood glucose levels should be monitored; transient hypoglycemia is common after removal of large portions of the liver. Hypophosphatemia may occur after lobectomy and may require supplementation of potassium phosphate in the IV fluids. Albumin levels should be monitored. If the patient becomes severely hypoalbuminemic (i.e., <2 g/dL) or has substantial worsening of third space fluid accumulation, one should consider administering plasma, whole blood, or synthetic colloids (e.g., hetastarch). Clotting times may be assessed if hemorrhage or petechiation occurs. Antibiotics given during surgery should be continued for 2 to 3 days if partial hepatectomy has been performed. Nutritional supplementation may be necessary in some patients during the early postoperative period, particularly if the animal is anorexic or has severe vomiting or diarrhea (see Chapter 10).

Analgesics (e.g., hydromorphone, oxymorphone, butorphanol, buprenorphine) should be provided to patients after surgery (see Table 13.2). For severe pain, a fentanyl-lidocaine-ketamine combination given as a constant-rate infusion may be indicated (see Box 13.3). Dogs with acute hepatitis may progress to chronic hepatitis and cirrhosis; repeat liver sampling should be considered to assess response to therapy.

COMPLICATIONS

Biopsy samples may be useless for histopathology if the tissue sample is crushed, fragmented, or too small, or if the specimen contains predominantly blood or necrotic portions of mass lesions. Bile peritonitis may occur if the gallbladder or bile ducts are inadvertently penetrated. The complication rate in animals undergoing ultrasound-guided biopsy of abdominal structures is less than 1.5%. The most important problem is the propensity for an incorrect diagnosis when this technique is used (see earlier).

The most common and serious complication of hepatic surgery is hemorrhage. This may result from ligatures slipping off friable hepatic tissue. Care should be taken to ensure that a stump of tissue remains distal to the ligature when encircling sutures are used for biopsy or partial hepatectomy. With hepatic trauma, anaerobic bacteria may proliferate in hypoxic portions of the liver and cause sepsis; therefore broad-spectrum antibiotics should be used in patients with severe hepatic trauma and in those undergoing hepatic surgery. Complications after major hepatic resection occurred in 13% of patients in one report.[6] Complications can include portal hypertension, ascites, fever, hemorrhage, hypophosphatemia, or persistent bile drainage. Dogs with ascites undergoing hepatic biopsy may have a shorter mean survival time than those without ascites. Icterus, hypoalbuminemia, a left shift on the leukogram, and enlarged portal lymph nodes may be associated with shorter survival in dogs with primary hepatitis. Dogs with lethargy, tachypnea, or anesthetic complications had increased mortality risk when undergoing liver lobectomy for neoplasia.[6]

SPECIAL AGE CONSIDERATIONS

PSS ligation (see p. 558) is often performed in young animals that are particularly prone to hypoglycemia; serum glucose concentrations should be carefully monitored. Hypothermia, also

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a particular problem in young patients, reduces the minimum alveolar concentration of inhalants used for anesthetic maintenance.

SPECIFIC DISEASES

PORTOSYSTEMIC VASCULAR ANOMALIES

DEFINITIONS

PSSs, also called **portosystemic vascular anomaly**, are anomalous vessels that allow normal portal blood draining from the stomach, intestines, pancreas, and spleen to pass directly into the systemic circulation without first passing through the liver. The term *portacaval shunt* is frequently used; however, this term technically refers to a specific type of vascular anomaly (i.e., portal vein to caudal vena cava). **Extrahepatic shunts** are vascular anomalies located outside the hepatic parenchyma; they may be **congenital extrahepatic portosystemic shunts** (CEPSSs) or acquired. **Congenital intrahepatic portosystemic shunts** (IHPSSs) are located in the liver. **Hepatic microvascular dysplasia** (HMD) is sometimes referred to as **portal vein hypoplasia**; unfortunately, there is nonuniformity in terminology. HMD is characterized by small or absent intrahepatic portal vessels and portal arteriolar hyperplasia associated with microscopic shunting of blood through the liver without a macroscopic PSS.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

When portal blood bypasses the liver, many substances that are normally metabolized or excreted in the liver enter the systemic circulation. In addition, important hepatotrophic substances from the pancreas (e.g., insulin) and intestines do not reach the liver, resulting in hepatic atrophy or failure of the liver to attain normal size. Hepatic insufficiency or hepatic encephalopathy frequently occurs. Hepatic encephalopathy is a clinical syndrome of altered CNS function resulting from hepatic insufficiency. A variety of substances (e.g., ammonia, methionine/mercaptans, short-chain fatty acids, alterations in the ratio between circulating levels of branched-chain and aromatic amino acids, γ-aminobutyric acid) are believed to cause false neurotransmitters.

PSSs are broadly categorized as congenital or acquired and as intrahepatic or extrahepatic. Congenital extrahepatic shunts are typically single anomalous vessels that allow abnormal blood flow from the portal vein directly to the systemic circulation; rarely two or more anomalous vessels may be present. CEPSS accounts for approximately 65% of single shunts in dogs and also occurs in cats. Many different types of CEPSS have been described in dogs, including (1) portal vein to caudal vena cava, (2) portal vein to azygos vein, (3) left gastric vein to caudal vena cava, (4) splenic vein to caudal vena cava, (5) left gastric, cranial mesenteric, caudal mesenteric, or gastroduodenal vein to caudal vena cava, and (6) combinations of these (Fig. 20.6). Cats most commonly have a large single vessel that empties directly into the prehepatic vena cava; however, they may have atypical CEPSS connections, and the shunt may flow into any systemic vessel, including renal, phrenicoabdominal, azygos, or internal thoracic veins. Intrahepatic shunts are usually congenital, singular shunts that occur because the ductus venosus fails to close after birth, or they may arise when other portal to hepatic vein or caudal vena cava anastomoses exist. Congenital IHPSS make up approximately 35% of single shunts in dogs and approximately 10% in cats.

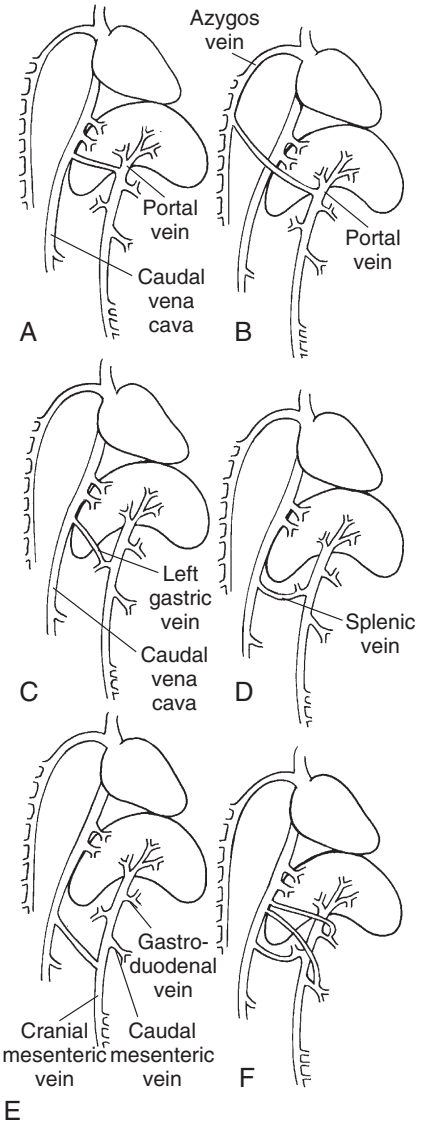


FIG. 20.6 Portosystemic shunts described in dogs and cats. (A) Portal vein to caudal vena cava. (B) Portal vein to azygos vein. (C) Left gastric vein to caudal vena cava. (D) Splenic vein to caudal vena cava. (E) Left gastric, cranial mesenteric, caudal mesenteric, or gastroduodenal vein to caudal vena cava. (F) Combinations of the above.

Acquired extrahepatic shunts are typically multiple and represent approximately 20% of all canine PSS. They arise in part because of increased resistance to portal blood flow, causing portal hypertension with a pressure gradient between portal and systemic circulation. This hypertension causes normal, nonfunctional microvascular connections, which are present at birth between portal and systemic veins, to become functional. Multiple shunts are most commonly associated with chronic, severe hepatic disease (i.e., cirrhosis) but have been reported secondary to hepatoportal fibrosis in young dogs. Veno-occlusive hepatic disease has been reported as a cause of multiple PSS in young cocker spaniels. Multiple shunts most commonly occur in the left renal area and the root of the mesentery (Fig. 20.7), and connections to the caudal vena cava or azygos veins are usually seen. Cats with acquired PSS typically develop connections between the

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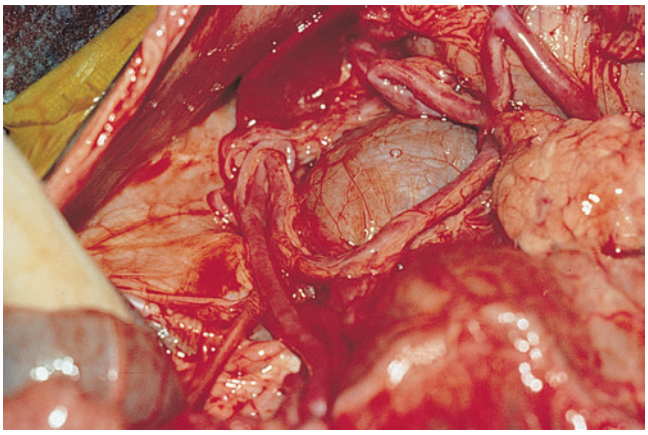


FIG. 20.7 Multiple shunts near the left kidney in a dog with hepatic disease and portal hypertension.

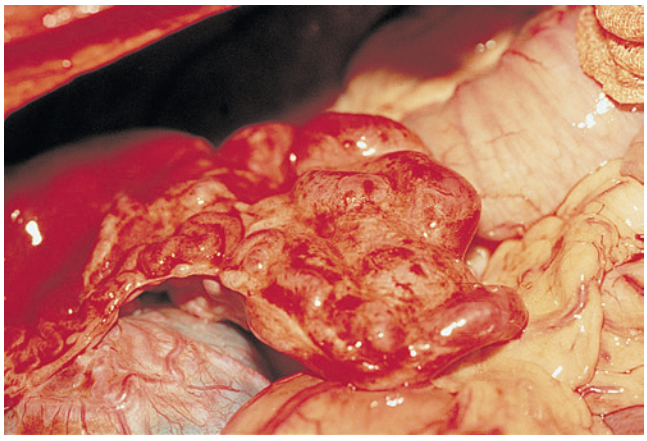


FIG. 20.8 Hepatic arteriovenous fistula in a 1-year-old Labrador. Note the dilated vessels in the hepatic parenchyma. The fistula was found during surgery for a gastric foreign body.

left gastric veins and the phrenicoabdominal veins, and from the left colic vein to the ipsilateral gonadal vein.

Our current understanding of HMD (i.e., sometimes referred to as *portal vein hypoplasia*) is that it is a congenital condition characterized by small or absent intrahepatic portal vessels and portal arteriolar hyperplasia that allows an abnormal communication between the portal and systemic circulations. This condition is sometimes difficult to differentiate from congenital PSS because both may have similarly increased serum bile acid concentrations. Furthermore, the histologic lesions of congenital PSS and HMD are identical, and HMD is common in some breeds that are at increased risk for congenital PSS. Most dogs with HMD are asymptomatic and do not have obvious microhepatica, but exceptions exist. In particular, there is concern that HMD can progress and result in noncirrhotic portal hypertension and/or hepatoportal fibrosis in some patients. Currently, HMD is diagnosed by hepatic histology plus eliminating macroscopic shunts. Mesenteric portograms, contrast computed tomography (CT), and nuclear scintigraphy in dogs with HMD should be normal.

AV fistulae account for approximately 2% of single shunts and may be congenital or acquired. Acquired AV fistulae occur secondary to trauma, tumors, surgical procedures, or degenerative processes that cause arteries to rupture into adjacent veins. The fistulae are typically macroscopic communications that form between branches of the hepatic artery and portal vein; however, microscopic hepatic AV fistulae have also been suspected. As congenital lesions, they are believed to develop as a result of failure of the common embryologic capillary plexus to differentiate into an artery or a vein. Affected animals usually develop portal hypertension and multiple collateral shunting vessels, resulting in acute onset of low-protein transudative ascites between the ages of 2 and 18 months (Figs. 20.8 and 20.9). In contrast, dogs with congenital PSS rarely have ascites.

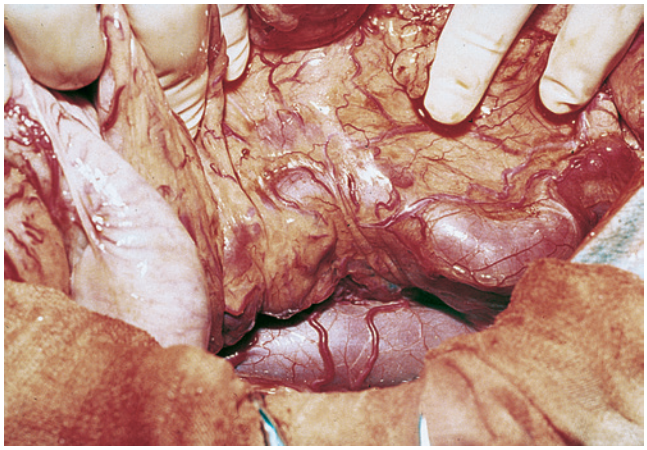


FIG. 20.9 Multiple collateral shunting vessels in the dog shown in Fig. 20.8.

DIAGNOSIS

Clinical Presentation

Signalment

Purebred dogs are at increased risk for CEPSS. Domestic shorthair cats are most commonly affected, although these aberrations also occur in purebred cats (e.g., Himalayan). Single PSS are usually congenital and are most commonly diagnosed in animals younger than 2 to 3 years of age, although dogs over 10 years old have been diagnosed. Extrahepatic shunts have been most frequently diagnosed in miniature and toy-breed dogs (e.g., Yorkshire terrier, Maltese, silky terrier, miniature schnauzer, poodle, lhasa apso, bichon frise, Jack Russell terrier, Shih Tzu, Pekingese). They are hereditary in Yorkshire terriers and Maltese and may be genetic in other breeds as well within the terrier, toy, and working groups of dogs.[7,8] Interestingly, there were more females with portoazygous than portocaval shunts in one study.[9] IHPSS are more commonly diagnosed in large-breed dogs (e.g., German shepherd, golden retriever, Doberman pinscher, Labrador retriever, Irish setter, Samoyed, Irish wolfhound). Small-breed dogs most likely to have IHPSS are toy and miniature poodles. A hereditary basis may exist for IHPSS in Irish wolfhounds. CEPSS and IHPSS have been reported in cats. No convincing gender predisposition has been noted for these anomalies in either species.

NOTE In general, small-breed dogs are more likely to have extrahepatic shunts and large-breed dogs are more likely to have IHPSS.

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Multiple acquired shunts are most commonly diagnosed in animals between 1 and 7 years of age; however, acquired PSS that occur secondary to hepatoportal fibrosis or infiltrative disease have been reported in dogs as young as 4 months. Breeds most commonly affected include German shepherds, Doberman pinschers, and cocker spaniels, but any breed may be affected. Multiple acquired shunts have been described in cats.

Most dogs with hepatic AV fistulae have been young (i.e., younger than 1.5 years of age) at the time of diagnosis. Congenital hepatic AV fistulae have rarely been reported in cats.

History

The presenting history for animals with congenital PSS varies considerably. Affected animals are usually evaluated because of failure to grow, small body stature, or weight loss. Other common abnormalities include intermittent anorexia, depression, vomiting, polydipsia or polyuria, ptyalism (especially in cats), pica, amaurosis, and behavioral changes. Some animals are presented for evaluation of urinary dysfunction (i.e., hematuria, dysuria, pollakiuria, stranguria, urethral obstruction) associated with urate urolithiasis (see later). Signs of hepatic encephalopathy can vary tremendously from those that are extremely mild and hard to identify as a significant abnormality (e.g., lethargy, being "tired," being "slow") to severe changes (e.g., ataxia, weakness, stupor, head pressing, circling, amaurosis, pacing, seizures, coma). These signs may be constant or intermittent and sometimes, but not invariably, worsen after eating (especially a high-protein diet composed of animal protein). Hepatic encephalopathy may also worsen after gastrointestinal hemorrhage (e.g., caused by parasites or ulceration). Neurologic signs are present in the majority of affected dogs, whereas approximately 40% have urinary signs.[10] In addition to ptyalism, affected cats typically show episodic central blindness. Some asymptomatic patients are diagnosed fortuitously when microhepatica or hypoalbuminemia is discovered while the patient is being evaluated for some other problem.

NOTE Consider congenital PSS in any young animal with a prolonged response to anesthetic agents or tranquilizers that require hepatic metabolism for clearance. These may be the first abnormalities noted in some affected animals.

The most common presenting sign in dogs with congenital hepatic AV fistulae is sudden onset of depression, ascites, and vomiting. Despite the chronic nature of this condition, the animal often has an acute onset of gastrointestinal or neurologic signs. The ascites is typically a pure low-protein transudate despite a serum albumin greater than 1.8 g/dL.

NOTE Animals with hepatic AV fistulae may be presented for evaluation of gastrointestinal foreign bodies. Presumably gastric irritation causes pica in these animals.

Physical Examination Findings

Most animals with congenital PSS have microhepatica, and the kidneys may feel prominent or plump. A golden or copper color to the iris has been observed in many cats with PSS. Neurologic abnormalities may be noted (see earlier). Ptyalism is a common finding in cats but is rare in dogs. Animals with hepatic AV fistulae may have a palpably enlarged liver (rare) or ascites. An audible bruit can sometimes be auscultated in the cranial abdomen of affected animals.

Diagnostic Imaging

Survey abdominal radiographs are an important part of screening for congenital PSS. Microhepatica is seen in most dogs with congenital PSS and may range from mild to marked. However, dogs with congenital PSS sometimes have normal-sized livers or even hepatomegaly if concurrent problems such as steroid hepatopathy are present. Microhepatica is not as common in cats with congenital PSS. Abdominal radiographs are more sensitive in detecting microhepatica than abdominal ultrasound.

Definitive diagnosis of congenital PSS requires identification of the shunt by ultrasound, contrast CT (CT angiography), nuclear scintigraphy, intraoperative positive contrast portography, or surgical localization. Various positive contrast techniques have been described, including splenoportography, cranial mesenteric arterial portography, celiac arteriography, transsplenic portal catheterization, jejunal vein portography, and CT angiography.[11] Jejunal vein portography is a simple and effective portographic technique that may be performed during shunt ligation (see later); however, with the increasing use of ameroid constrictors and cellophane banding procedures, it is less commonly done. Ultrasound-guided splenoportography can be performed in large-breed dogs.

NOTE CT angiography provides the best overall detail of shunt morphology and is useful in animals that have persistent clinical signs after surgery or important biochemical abnormalities (see later).

Ultrasound is a common diagnostic tool for imaging acquired and congenital PSS. Intrahepatic and extrahepatic shunts can be identified with this technique; however, an inconclusive ultrasound examination does not rule out congenital PSS. Ultrasound diagnosis of CEPSS depends on operator experience and the time allotted to examine the patient. Occasionally, a dilated intrahepatic vessel or communication of an intrahepatic shunt with the caudal vena cava is noted (Fig. 20.10). With extrahepatic shunts, overlying bowel may obscure the shunt, but a small liver with few detectable hepatic or portal veins may be

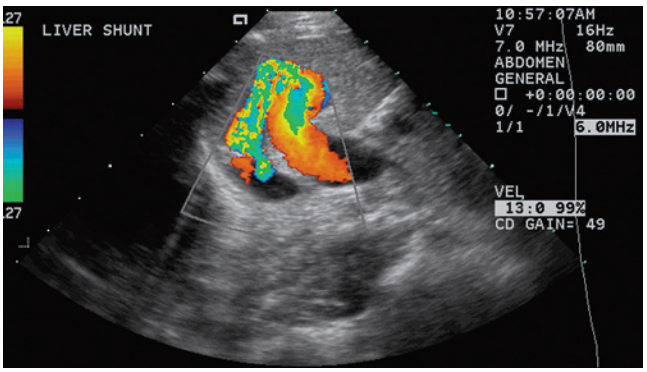


FIG. 20.10 Ultrasound image with color Doppler showing a large, tortuous intrahepatic shunt.

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noted. Increased hepatic arterial detection using Doppler is also a common finding. The bladder and renal pelves should be assessed for calculi because urate stones are usually radiolucent and difficult to see on survey abdominal radiographs. Ultrasound determination of hepatic volume may correlate with ability to completely ligate CEPSS. Ultrasound evaluation is also useful to identify the anechoic, tortuous vessels seen with hepatic AV fistulae. Pulsed wave Doppler ultrasound may assist in making a diagnosis of hepatic AV fistulae; visualization of retrograde (hepatofugal), pulsatile flow in the abnormal portal vein branch and the main portal vein may allow one to discern the involved liver lobe and resect it.

Nuclear scintigraphy is a rapid, noninvasive method of documenting abnormal hepatic blood flow. Sodium pertechnetate technetium-99m (^{99m}Tc) is typically used in scintigraphic studies to detect congenital PSS. After colonic administration of ^{99m}Tc, the time when activity in the region of the liver is first noted is compared with the time when activity appears in the region of the heart (Fig. 20.11). Animals with liver-to-heart intervals longer than 12 seconds are generally considered clinically normal. Sometimes studies in very small animals can be difficult to interpret because of the close proximity of the liver and heart, and occasional studies must be repeated if the ^{99m}Tc is not rapidly absorbed from the colon. If a study must be repeated, this must be done the following day to allow the body to eliminate the technetium from the invalid study. False-positive results have not been reported; however, false-negative results may occur if a small shunt involves only a peripheral portion of the portal system. HMD will have a normal scintigraphic study, which distinguishes it from congenital PSS. Transsplenic portal scintigraphy utilizes ultrasound guidance to inject a small amount of ^{99m}Tc into the parenchyma of the spleen. Dynamic phase imaging of the splenic vein drainage yields a nuclear angiogram of the portal system and is useful for detecting the presence of single or multiple extrahepatic shunts. An advantage of this technique is that a very small amount of radioactivity is used; thus the animal can be released from radiation isolation shortly after the procedure, depending on the release criteria of the state. Furthermore, the authors found that transsplenic portal scintigraphy yielded higher-quality images than were attained with transrectal scintigraphy.

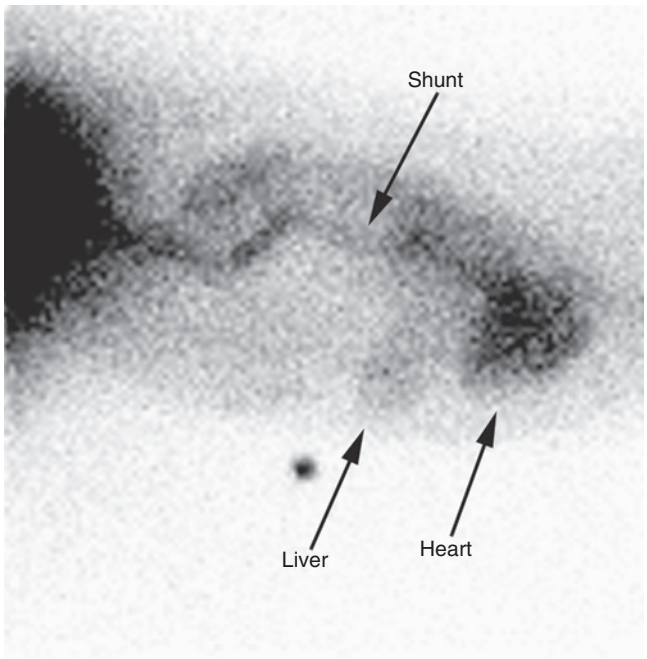


FIG. 20.11 Composite image of transrectal portal scintigraphy. The dog's head is to the right of the image. Note the severe lack of uptake by the liver; this supports the diagnosis of portosystemic shunting. The shunt vessel can be identified in this study.

Advanced imaging with CT angiography is the gold standard for the diagnosis of vascular disorders associated with the liver. Three-dimensional reconstructions enhance understanding of anatomy and surgical planning and can identify concurrent vascular anomalies. Calculation of hepatic perfusion using CT is also possible. Angiography has surpassed the ability of ultrasound to detect and define the anatomy of shunts in dogs.[12] It was also shown to be superior to intraoperative mesenteric portography.[13] CT angiography was better able to depict the portal supply to the liver; however, temporary shunt occlusion during mesenteric portography was helpful and should be considered if the presence of normal portal supply to the liver is questionable at surgery.[13] CT scans can also be used to document liver volume and have been used to demonstrate improvement in liver volume after shunt attenuation, whereas CT angiography may show return to normal hepatic arterial perfusion following shunt attenuation.[14]

NOTE Nuclear scintigraphy, a useful, noninvasive screening tool for diagnosing congenital or acquired shunts, distinguishes them from hepatic microvascular dysplasia.

Laboratory Findings

Hematologic, serum biochemical, and urine analysis of animals with congenital PSS may disclose various abnormalities, but dogs can have a congenital PSS with no abnormalities on complete blood count, serum biochemistry panel, or urinalysis. Hematologic abnormalities may include microcytosis with normochromic erythrocytes, mild nonregenerative anemia, target cells, or poikilocytosis. Low serum iron concentrations appear to cause microcytosis in dogs with congenital PSS. Biochemical tests often reveal a reduction in serum albumin, cholesterol, and/or blood urea nitrogen (BUN) concentrations. Low serum albumin is a common finding in affected dogs; however, some dogs (and most cats) with congenital PSS have normal albumin levels. Hypocholesterolemia is often seen concurrently with hypoalbuminemia. Low BUN results from reduced conversion of ammonia to urea in the hepatic urea cycle, but the polyuria-polydipsia seen in many affected patients may contribute. Other abnormalities occasionally include mild to marked increases in serum alanine aminotransferase. Manganese concentrations are typically elevated in dogs with congenital PSS compared with normal dogs and dogs with nonhepatic illness. Manganese is normally excreted via hepatic conjugation and may play a role in the development of hepatic encephalopathy; however, resolution of hepatic encephalopathy is not necessarily related to improved manganese levels.[15] The serum bilirubin concentration is usually normal. Fasting hypoglycemia rarely occurs. Prothrombin time, activated PTT, and activated coagulation time are usually normal, but antithrombin III levels typically mirror serum albumin concentrations. Routine urinalysis may disclose dilute urine or

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ammonium biurate crystals. Hyperuricemia and hyperammonemia lead to increased urinary excretion of urate and ammonia, promoting urinary precipitation of ammonium biurate crystals. Hematuria, pyuria, and proteinuria may occur if urate calculi form. The hematologic and biochemical profiles of canine hepatic AV fistulae can be similar to those of dogs with congenital or acquired PSS.

Hepatic function tests are important in screening for congenital PSS. Serum bile acids have been the standard hepatic function test in dogs and cats for years, but it is now recognized that they have some limitations. First, it is critical to measure both preprandial and postprandial serum bile acid concentrations; approximately 20% of dogs have a higher preprandial value. Second, some dogs with very high serum bile acid concentrations (>150 µmol/L) do not have clinically important hepatic disease, but some dogs with congenital PSS have serum bile acid concentrations that are only modestly increased (e.g., 50–60 µmol/L, normal <30 µmol/L). In very rare cases, dogs with congenital PSS have had normal fasting/postprandial serum bile acid concentrations. Third, unlike what is expected for most biochemical determinations, substantial variation in serum bile acid concentrations can occur from day to day (as much as or greater than 100%). Dogs with congenital PSS may have normal fasting bile acid concentrations, but a normal fasting bile acid concentration has been shown to have a negative predictive value of 96%.[16] Urinary bile acids appear to be as useful as serum bile acids but may have the advantage of being easier to collect (i.e., the owner can bring in a urine sample as opposed to bringing in the patient), especially in cats. Measurement of bile acids after stimulation of gallbladder contraction with a synthetic analog of cholecystokinin (ceruletide) may be more sensitive than postprandial measurements of serum bile acids in dogs with hepatic disease, including congenital PSS. Side effects of ceruletide may include pancreatitis, gastric distention, vomiting, and pain; they have yet to be reported in dogs, but further investigation is warranted.

Protein C is a sensitive test for hepatic insufficiency, and dogs with congenital PSS typically have lower protein C levels than dogs without macroscopic shunting. Dogs with hepatic failure have the lowest levels of protein C of all dogs with hepatobiliary disease. These differences may prove useful in differentiating cases of hepatic failure, congenital PSS, and HMD. Interleukin-6 (IL-6) has been found at higher levels in dogs with portosystemic shunting and may be involved in the pathophysiology of hepatic encephalopathy[17]; IL-6 has also been found to be elevated in dogs with liver disease.[18] IL-2, IL-8, and tumor necrosis factor alpha were not different between dogs with portosystemic shunting and normal dogs in the same study.

Hyperammonemia is very specific for hepatic insufficiency, but simply measuring resting blood ammonia is insensitive, even in patients experiencing hepatic encephalopathy; one study showed a negative predictive value of 52%.[16] The ammonia tolerance test is a very sensitive test (100% in one study),[16] but the test can have drawbacks (e.g., many animals vomit or defecate the administered ammonia). Measuring 6- to 8-hour postprandial blood ammonia concentrations appears to be more sensitive than measuring fasting blood ammonia, but is less sensitive than the ammonia tolerance test. Combining fasting bile acid concentration and fasting ammonia was associated with a 97% specificity and positive predictive value of 97%.[16] The biggest disadvantage of measuring blood ammonia is that it is easy to obtain artefactual values if instructions on collecting, storing, and preparing the blood are not followed exactly. This test must be run in-house; it cannot be sent to an outside laboratory. Transient hyperammonemia due to a presumed urea cycle enzymatic deficiency may be found in normal young Irish wolfhounds. Hyperammonemia similar to that found in cases of congenital PSS was identified in 6- to 8-week-old puppies; however, the levels were not accompanied by elevated bile acid concentrations and had normalized by the age of 3 to 4 months. The condition was theorized to be a congenital deficiency in urea metabolism. Serum bile acid concentrations were high in one puppy that had a congenital PSS. Therefore screening of the breed for congenital PSS should be done with the age of the patient in mind and should include serum bile acid testing if hyperammonemia is identified.

DIFFERENTIAL DIAGNOSIS

Congenital PSS must be differentiated from other diseases causing hepatic insufficiency (e.g., cirrhosis) and neurologic abnormalities (e.g., hydrocephalus, epilepsy). Survey abdominal radiographs (to look for microhepatica) and hepatic function testing (usually preprandial and postprandial serum bile acids) are the typical means of screening for congenital PSS. If either of these tests is normal, congenital PSS is still possible but is less likely, and other diseases must be seriously considered.

MEDICAL MANAGEMENT

Surgery appears to be the treatment of choice for animals symptomatic for congenital PSS. Medical management of patients with symptomatic, congenital PSS was associated with a mortality rate of 48%, with approximately 30% succumbing to shunt-related illness compared with 12% mortality and 10% of deaths related to PSS in cases treated surgically.[19] However, the best treatment for asymptomatic dogs with congenital PSS is currently uncertain, as is that for dogs older than 5 to 7 years of age that have minimal clinical signs. In these patients, one must weigh the reported 5% to 7% mortality rate associated with corrective surgery versus the likelihood of substantial deterioration if surgery is not done.[19] The best approach for asymptomatic patients is uncertain, but intuitively it appears that if these animals have only modest changes on serum biochemistry panel and only modest microhepatica on abdominal radiographs, they may be good candidates for long-term medical management (although they should be monitored annually for disease progression that may make corrective surgery appropriate). If histologic changes (e.g., bridging hepatic fibrosis, bridging biliary hyperplasia, massive lipogranuloma formation) would seem to make postsurgical portal hypertension more likely, medical management probably should be chosen over surgery. However, surgery is desirable because restoration of hepatotrophic factors to the liver postoperatively should promote hepatic regeneration. Medical management should be initiated before surgical intervention is begun in animals with substantial signs of hepatic encephalopathy (some believe that this should be routine; see later under Preoperative Management).

Goals of medical therapy are to identify and correct factors predisposing to hepatic encephalopathy (i.e., reduce absorption of toxins produced by intestinal bacteria, diminish the interaction between enteric bacteria and nitrogenous substances, and avoid drugs that predispose to encephalopathy) and possibly to decrease oxidative damage to hepatocytes. Precipitating factors for hepatic

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BOX 20.2 Drugs Used in the Management of Portosystemic Shunts

Neomycin (Biosol)
10–20 mg/kg PO q6–12h

Metronidazole (Flagyl)
10 mg/kg PO q12h

Amoxicillin
22 mg/kg PO, IV, IM, or SC q12h

Lactulose (Cephalac, Chronulac)
2.5–25 mL PO q8–12h, so that the animal has two or three soft but formed (not liquid) stools a day
Dogs: typically start at 0.5 mL/kg PO q8h
Cats: typically start at 2.5–5 mL/cat or 1 mL/4.5 kg PO q8h

IM, Intramuscular; *IV,* intravenous; *PO,* orally; *SC,* subcutaneous.

encephalopathy include high-protein meals (especially meat), bacterial infections (especially urinary tract with urease-producing bacteria), gastrointestinal bleeding, blood transfusions, inappropriate drug therapy, and electrolyte/acid-base abnormalities (especially alkalosis). General supportive care of the patient with hepatic encephalopathy should include fluid therapy (0.9% sodium chloride or 0.45% sodium chloride and 2.5% dextrose), normalization of acid-base disturbances, and supplementation of potassium as needed. A highly digestible diet in which the primary source of calories is carbohydrates should be fed. For long-term dietary management, feed the highest-protein diet (preferentially vegetable protein or cottage cheese) that the animal will tolerate. Moderately protein-restricted diets that contain high levels of branched-chain amino acids and arginine are often used, but no good evidence suggests that preferentially feeding branched-chain as opposed to aromatic amino acids benefits dogs with congenital PSS.

Antibiotics (Box 20.2) are used to reduce enteric flora that produce many of the toxins (i.e., ammonia) thought to cause hepatic encephalopathy. Oral neomycin is frequently used for this purpose, but it should be avoided because small amounts of neomycin are sometimes absorbed from the gut. Metronidazole or ampicillin (oral or parenteral) also reduces intestinal ammonia concentrations. Rifaximin is used in people and is beginning to be used in veterinary medicine; however, its advantage over metronidazole in dogs and cats, if any, is unknown. Lactulose is a synthetic disaccharide that acidifies colonic contents and traps ammonium ions in the lumen (see Box 20.2). It is also an osmotic cathartic that shortens intestinal transit time and reduces the production and absorption of ammonia. Lactulose may be given orally or as a retention enema. Side effects of lactulose administration may include diarrhea, vomiting, anorexia, and increased gastrointestinal loss of potassium and water. Treatment of animals presented in hepatic coma must be prompt and aggressive; cleansing enemas (warm water) and retention enemas with neomycin or lactulose (or both) should be given. Acid-base and electrolyte abnormalities and hypoglycemia must be identified and corrected. Diazepam is often ineffective in encephalopathic dogs having seizures; phenobarbital may be more effective, and some patients must be anesthetized with propofol. A medical text should be consulted for additional recommendations on the management of coma due to hepatic encephalopathy.

Dogs with congenital PSS may experience oxidative damage to the hepatocytes. Antioxidants may help protect the hepatocyte membrane. *S*-adenosyl-L-methionine and silybin are commonly used antioxidants, but their true clinical value in these patients is unknown. Consult a medicine text for further details on hepatosupportive therapy with such drugs.

A 2014 study evaluated long-term survival and quality of life in 97 dogs that underwent surgery and 27 dogs that were medically managed for congenital portosystemic shunting.[19] Fourteen medically treated dogs had intrahepatic portosystemic shunting; 78% of the medically treated dogs had extrahepatic portosytemic shunting. Mortality rate was 89% ($n = 24$) for medically managed and 22% ($n = 21$) for surgically treated dogs. Median survival time for medically managed dogs was 836 days. Because of the large number of surgically treated dogs that were alive at the time of the report, median survival time was not reported for this group. Acute mortality associated with surgery was 4%.[19] Quality-of-life scoring based on neurologic, gastrointestinal, and urinary signs was better at all follow-up times in the surgery group compared with the medically managed group. However, it is important to note that all patients were clearly symptomatic due to their shunts, and that there was no control over case allotment into surgery or nonsurgery categories.

SURGICAL TREATMENT

Only patients with congenital (as opposed to acquired) PSS are surgical candidates. The goal of surgery is to identify and occlude or attenuate the abnormal vessel. Ameroid constrictors (Fig. 20.12) or cellophane bands are now commonly used in animals with extrahepatic shunts to slowly occlude the shunt vessel. With an ameroid constrictor, initial shunt constriction is effected by swelling of the hygroscopic material that makes up the inner portion of the device (see Fig. 20.12); additional shunt occlusion occurs as fibrosis develops around the vessel. It is important to note that the rate of vascular occlusion may affect the animal's propensity to develop acquired shunts after occlusion. Plasma protein concentration may affect the rate of closure of ameroid constrictors, and applying silicone to the ameroid may slow its rate of closure. Complete occlusion of the vessel may not occur, and approximately half of cats may have persistent shunting 8 to 10 weeks after ameroid constrictor placement. Approximately one-fifth of dogs have persistent shunting 6 to

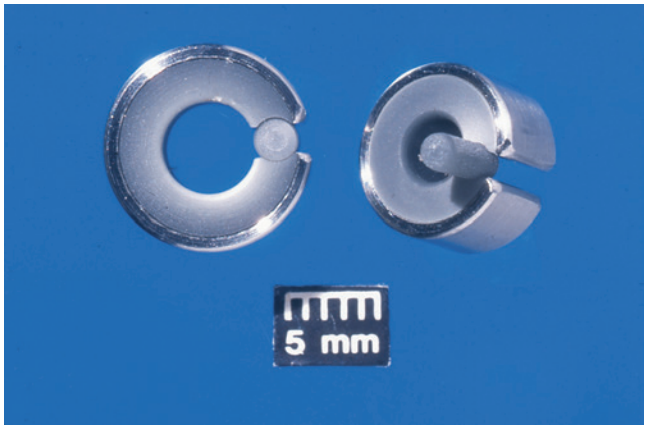


FIG. 20.12 Ameroid constrictor.

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10 weeks after placement of an ameroid. Vascular occlusion may occur owing to thrombus formation in some dogs.

Cellophane banding causes an initial acute inflammatory response followed by a chronic low-grade foreign body tissue reaction. Vascular attenuation may be slower and less complete with cellophane bands than with ameroid constrictors; however, clinical signs attributed to portosystemic shunting resolve or are substantially attenuated in most animals that survive the procedure. As with ameroid constrictors, multiple acquired shunts may occur after this procedure.

When the shunt or shunts are identified during abdominal exploration, positive contrast portography examination is generally unnecessary. However, if a shunt is not identified visually, mesenteric portography or retrograde portography may be used to help identify its location and nature. Sometimes, placing a catheter through the shunt during retrograde portography (see p. 559) and leaving it there will allow the surgeon to find some shunts that are especially difficult to find.

Preoperative Management

Encephalopathic patients should be stabilized before surgery. The value of pretreating surgical candidates with anticonvulsants is uncertain. Potassium bromide (dogs), phenobarbital (cats), or levetiracetam are typically used to attempt to lessen the incidence of postoperative seizures. Levetiracetam (20 mg/kg PO every 8 hours) administered to patients prior to surgery is believed to reduce or perhaps prevent seizures in animals with PSS. Because of the high incidence of postligation seizures in cats, pretreatment with this drug should be strongly considered. Fluid and electrolyte imbalances should be corrected preoperatively. Perioperative antibiotics (e.g., cephalosporins) are recommended for patients with PSS. See the earlier section on Medical Management for other recommendations.

Anesthesia

Care must be exercised when anesthetizing an animal with PSS. Because of reduced liver function and abnormal hepatic blood flow, drug absorption, metabolism, and clearance are markedly reduced (see anesthesia recommendations for hepatic disease on p. 540). The small size of these patients and their young age often makes anesthesia difficult because it puts them at increased risk for hypothermia, hypoglycemia, drug toxicity, hypervolemia, and hypovolemia. Even small amounts of blood loss can be significant, resulting in hypotension, hypovolemia, and anemia. Intraoperatively, patients often do well until the shunt is partially occluded (which is generally not an issue when an ameroid ring or cellophane band is used). At that time, preload may be markedly reduced, causing a reduction in cardiac output and systemic blood pressure. Because this is not a failure of the heart's ability to contract, the patient may not respond to inotropes (e.g., dobutamine, dopamine). Instead, return of blood flow to the heart needs to be increased by adjustment of the ligature. Central venous pressure measurements can aid in evaluating the consequences of ligation. It is recommended to have central venous pressures not decrease by more than 1 cm H_2O from the baseline measurement approximately 3 minutes after ligation.

Surgical Anatomy

The canine portal vein ranges from 3 to 8 cm long, depending on the size of the animal. On a radiographic contrast study of the normal portal system, the portal system usually originates at the level of the first lumbar vertebra (Fig. 20.13). Knowledge of the anatomy of the portal and hepatic venous systems is imperative in locating shunts, particularly intrahepatic ones (Fig. 20.14). The portal vein is formed by the confluence of the cranial and caudal mesenteric veins and the splenic vein. The splenic vein enters the portal vein at the level of the thoracolumbar junction. The phrenicoabdominal veins terminate in the caudal vena cava approximately 1 cm cranial to the renal veins. Any vein that enters the caudal vena cava cranial to the phrenicoabdominal veins (before the hepatic veins) may be considered an anomalous structure.

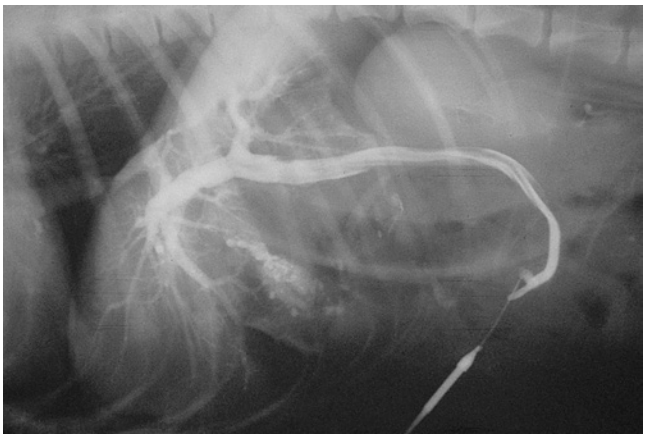


FIG. 20.13 Mesenteric portogram in a dog with a normal portal system. The portal system originates at the level of the first lumbar vertebra. Note the hepatic vasculature.

Abnormalities of the caudal vena cava, including segmental aplasia of the caudal vena cava, have been identified in dogs with congenital PSS. Careful evaluation of the entire abdominal venous system is important, because attenuation of the PSS is not possible if the portal system does not communicate with the liver.

> **NOTE** Examine the caudal vena cava carefully. The only vessels that should enter the caudal vena cava between the renal veins and the hepatic veins are the small phrenicoabdominal veins.

Positioning

A standard ventral midline celiotomy is performed from the xiphoid cartilage caudally, and the portal system is examined. For IHPSS and AV fistulae, the incision may need to extend cranially through the xiphoid process and the caudal sternebrae.

SURGICAL TECHNIQUES

The goals of surgery include the following: identify the abnormal shunting vessel(s), verify the presence of intact portal branching to the liver, ameliorate the flow of blood through the abnormal vessel, and sample the liver for histopathologic evaluation. Single CEPSS are generally treated by placing an ameroid constrictor or cellophane band on the vessel for gradual occlusion. Because the vessel slowly becomes occluded, portal hypertension is rare; most surgeons no longer evaluate portal pressures in conjunction with this surgery. When compared with surgical ligation, surgical time is shortened and intraoperative and

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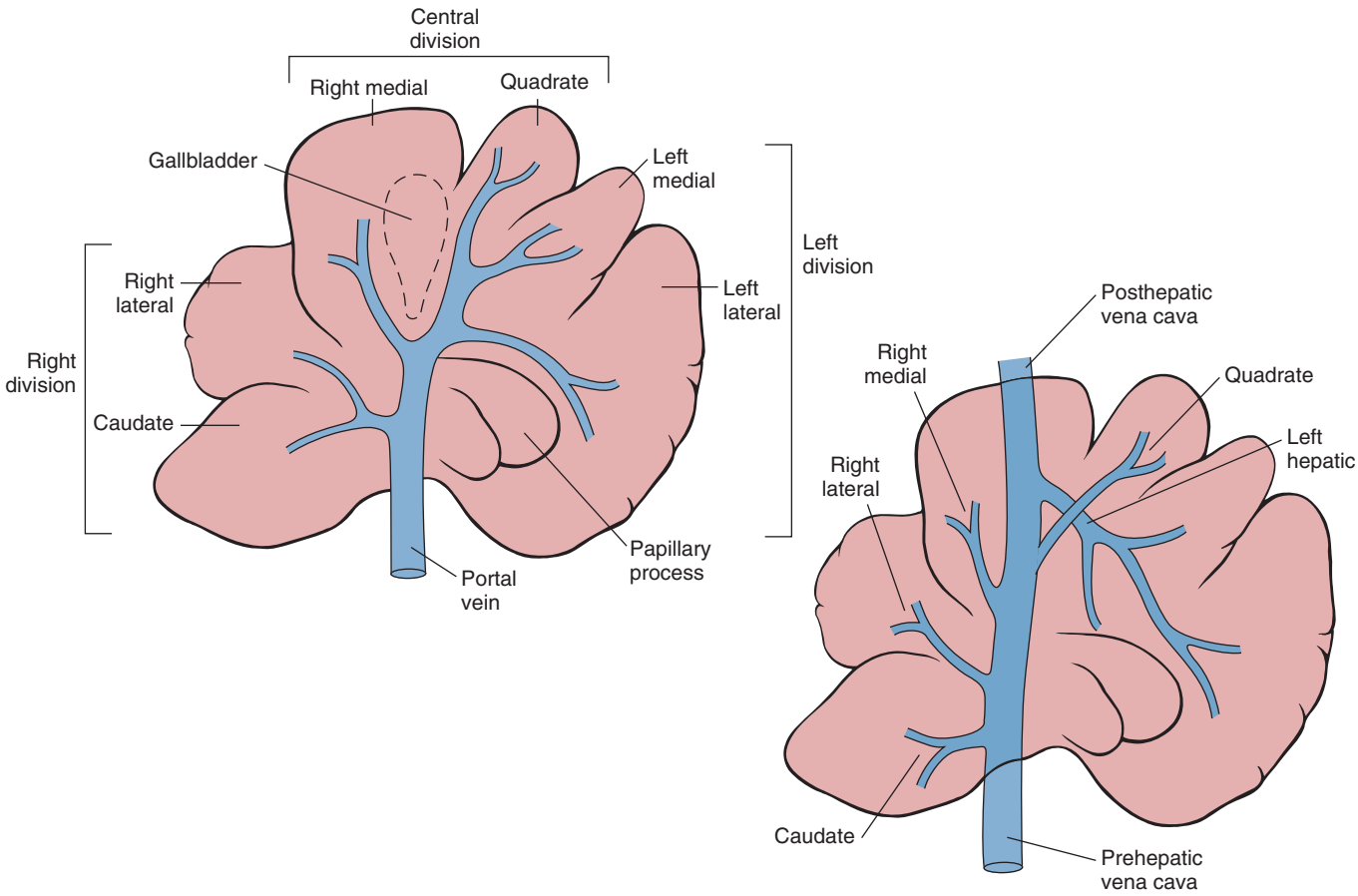


FIG. 20.14 Lobar and divisional anatomy of the portal vein and vena cava in the dog.

postoperative complications are decreased with ameroid constrictors. In rare cases, animals with IHPSS may have an ameroid constrictor or cellophane band placed on the vessel or the portal branch feeding the IHPSS; however, IHPSS generally requires ligation. Two additional techniques—intravascular coil occlusion and placement of a portacaval venograft using the jugular vein (with an ameroid constrictor placed on it) with complete ligation of the intrahepatic shunt—have been used for intrahepatic shunt occlusion. With the latter technique, an unacceptably high incidence of multiple, extrahepatic acquired shunts was noted at long-term follow-up. Transvenous coil embolization has been performed in dogs with CEPSS.

Generally, animals with congenital PSS should have a liver biopsy performed when the shunt is attenuated to rule out concurrent hepatopathy. However, if the gross appearance of the liver is clearly abnormal (e.g., rough, irregular), the liver should be biopsied and the histology report received before the shunt is attenuated. Maltese dogs seem to have a high incidence of hepatic parenchymal changes compared with other breeds; it may be prudent to biopsy them before shunt correction. Congenital PSS patients with chronic fibrotic or biliary hyperplastic changes may be at increased risk for portal hypertension following shunt attenuation.

Animals with multiple acquired PSS secondary to acquired hepatic disease may benefit from medical management directed at the cause of the hepatic disease (e.g., antiinflammatories, antifibrotic drugs, antioxidants), as well as subsequent ascites and encephalopathy (e.g., dietary restriction of protein and salt, diuretics), depending on the severity of the hepatic lesion. Good medical care may result in long-term survival and a good quality of life; noncirrhotic portal hypertension in particular may respond well. Caudal vena cava banding has been suggested to raise the systemic venous pressure in the abdomen to or slightly above that of the portal venous system, but this is uncertain and the technique is seldom performed. Multiple acquired PSS are usually evident upon exploratory laparotomy. Findings that may be noted include enlarged mesenteric veins, a larger than normal portal vein, and anomalous connections between the portal venous system and the systemic venous circulation. The most common location for multiple acquired PSS is the area of the left kidney; however, anomalous venous connections between the mesenteric circulation and the caudal vena cava or its tributaries may be noted throughout the abdomen (Fig. 20.15). Portoazygos connections have also been observed in approximately 25% of clinical cases.[9] Care should be taken when incising the abdominal wall in patients suspected of having acquired PSS because large, dilated vessels may be present in the falciform ligament, the greater omentum, or both. Trauma to these vessels upon abdominal entry may cause significant hemorrhage. Dissection of the falciform ligament usually requires ligation or cauterization of multiple vessels. Because many patients with multiple acquired PSS have ascites, suction should be available upon entry into the abdomen to evacuate ascitic fluid.

NOTE If you find multiple shunts in an animal, be sure to biopsy the liver.

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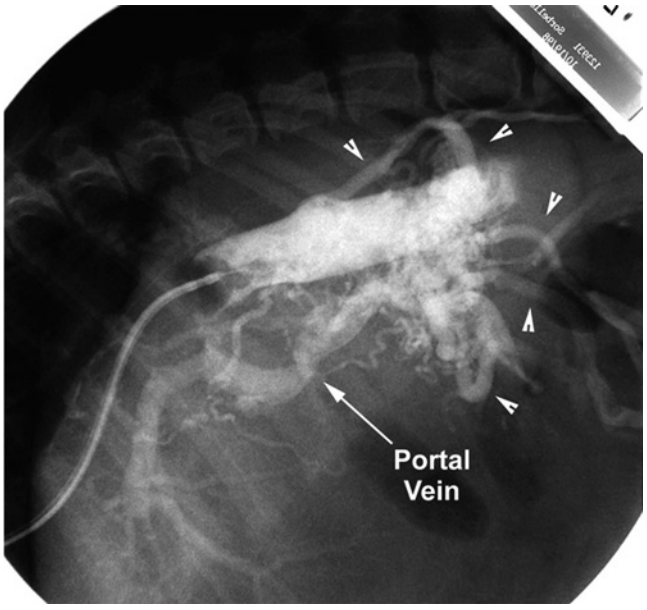


FIG. 20.15 Transvenous retrograde portogram in a dog with multiple acquired shunts *(arrowheads)*. (Courtesy Dr. Matt Miller, VETMED Consultants, Phoenix, AZ.)

BOX 20.3 Normal Pressures in Dogs

Portal Pressures
8–13 cm H_2O
6–10 mm Hg

Systemic Venous Pressures
0–6 cm H_2O
0–4 mm Hg

AV fistulae are treated by removing the affected liver lobe or in rare cases by ligating the fistulae directly. Portal pressures should be measured in conjunction with occlusion of IHPSS or vena cava banding. The normal portal pressure in dogs is 8 to 13 cm H_2O, which is 7 to 8 cm H_2O higher than the systemic venous pressure (Box 20.3). However, in animals with congenital PSS, the resting portal pressure is often closer to the systemic venous pressure. Excessive portal venous pressure can result in splanchnic congestion, portal hypertension, and death.

Ameroid Constrictor Placement on Extrahepatic Shunts

Perform a midline abdominal incision. Identify the portal vein by retracting the duodenum to the left and ventrally. Locate the caudal vena cava, renal veins, phrenicoabdominal veins, and portal vein (ventral to the caudal vena cava at the most dorsal aspect of the mesoduodenum). Note any veins entering the caudal vena cava proximal to the phrenicoabdominal veins. If the shunt has not been identified, open the omental bursa and retract the stomach cranially, the duodenum to the right and ventrally, and the left lobe of the pancreas caudally. Identify shunts that communicate with the caudal vena cava through the epiploic foramen by observing abnormal tributaries of the portal vein, left gastric vein, or splenic vein. Once the shunt has been identified, select the appropriately sized ameroid constrictor. Generally, use a 3.5- or 5-mm ameroid in most small dogs with extrahepatic shunts; use of ameroid constrictors larger than 5 mm may be associated with an increased rate of residual flow through the constrictor.[20] *Dissect around the shunt vessel to allow placement of the device, but avoid dissecting a large area next to the vessel.* Excessive dissection may allow movement of the ameroid on the vessel and may predispose to premature kinking of the vessel. *Place the vessel in the opening of the ameroid so that it sits in the inner circular space of the device. If necessary, place several loops of small (2-0) multifilament suture around the vessel and use these sutures to flatten the vessel and facilitate manipulation of the vessel into the opening of the ameroid. Once the vessel has been positioned in the ameroid, insert the key in the slit of the device. Evaluate the intestines for evidence of congestion (rare) and close the abdominal incision routinely. Dissect cautiously to avoid hemorrhage, which may be severe.*

In dogs with left divisional IHPSS, partial ligation may be superior to ameroid constrictor placement on the left hepatic vein because with the latter technique development of multiple shunts, persistent flow, or recanalization of the vessel encircled by the ameroid constrictor may be problematic.

NOTE The ameroid should fit on the vessel without compromising the lumen; however, avoid using an ameroid that is too large, because the weight of the device may cause the vessel to kink, obstructing flow prematurely.

Cellophane Banding of Extrahepatic Shunts

Fold a 10-cm long × 1.2-cm wide strip of cellophane (purchased from a store and sterilized, or use MS 350 grade cellophane) longitudinally to form a three-layer strip that is approximately 4-mm wide. The cellophane may be attached in either of two ways: (1) by causing partial obstruction of the shunt at the time of placement or (2) by attaching where the cellophane causes no shunt occlusion initially. The second technique is easier to perform, eliminates the need for monitoring portal pressures, and results in a more favorable outcome than the first technique.

For the first technique, pass the cellophane around the shunt and a pin of predetermined size, and place a titanium clip on the strip. To determine pin size in dogs weighing less than 10 kg (which will determine the diameter of the cellophane band), evaluate changes in heart rate, arterial pressure, intestinal color and motility, and pancreatic color when the shunt is totally occluded, or measure portal pressures (see later). If elevations in heart rare are minimal (<10 beats/min) and arterial systolic pressure does not decrease by more than 10 mm Hg, use a 2-mm pin to determine the final diameter of the band. If changes are moderate, use a 2.5-mm pin; if they appear to be severe, use a 3-mm pin. Secure the cellophane band with two titanium Ligaclips.

For the second technique, prepare the cellophane as described earlier and place it around the vessel without causing any occlusion of the shunt. Secure the cellophane with a hemoclip(s).

Ligation of Single Extrahepatic Shunts

If placement of an ameroid constrictor is not possible, the vessel may be ligated or attenuated; however, extreme care must be taken to avoid causing portal hypertension. *Identify the anomalous vessel, isolate it, and pass 2-0 silk suture around the vessel (Fig. 20.16). If jejunal portography was not performed (see later), exteriorize a segment of jejunum and insert a 20- to 22-gauge over-the-needle catheter (e.g., Angiocath) into a jejunal vein. Do*

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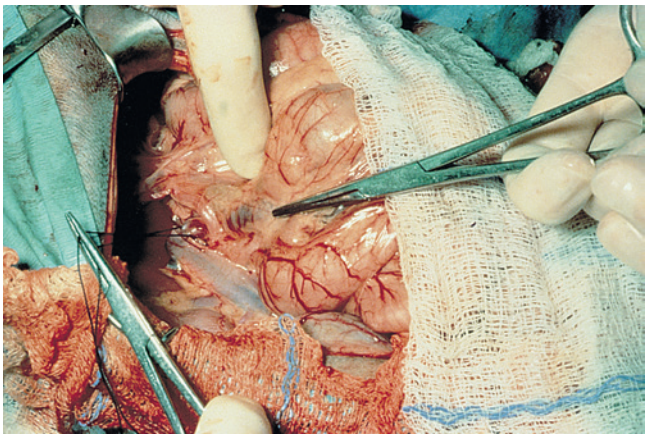


FIG. 20.16 To identify the portal vein, retract the duodenum to the left and ventrally. Note any vessels entering the caudal vena cava proximal to the phrenicoabdominal veins (hemostat). The suture is around a portocaval shunt.

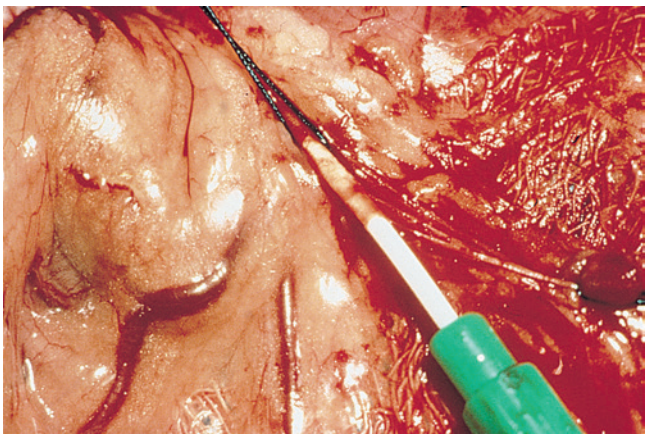


FIG. 20.17 To catheterize a jejunal vein, place one or two sutures around the selected vessel. Insert a 20- to 22-gauge over-the-needle catheter into the vein and use the preplaced sutures to secure it to the vessel.

not damage the corresponding jejunal artery. Obtain baseline portal pressures. Temporarily occlude the shunt and observe portal pressures during this manipulation. Occlusion of the shunt should result in a rapid increase in portal pressure, which aids in confirmation that it is an anomalous vessel. *Check portal pressures carefully before and during shunt ligation. If you are unsure whether complete ligation should be attempted, err on the conservative side and attenuate the shunt. If you are uncertain whether the vessel you have occluded is the shunt, perform jejunal portography.*

Once you have positively identified the shunt, slowly tighten the ligature while monitoring the portal pressure. If possible, completely occlude the shunting vessel, but do not allow postligation portal pressures to exceed 10 cm H_2O (8 mm Hg) above baseline pressures, or 20 to 23 cm H_2O (15–18 mm Hg). If intraoperative Doppler ultrasound is available, once hepatopetal flow is established in the cranial portal vein and shunt, do not occlude the shunt further. You may be able only to attenuate the vessel. *Observe the viscera for evidence of splanchnic congestion for 5 to 10 minutes. If excessive splanchnic congestion is noted, loosen the suture. If splanchnic congestion does not occur, remove the jejunal vein catheter and ligate the vein from which the catheter was removed. Examine the kidneys and bladder for calculi. If cystic calculi are present and the patient is in stable condition, remove the calculi during the shunt ligation surgery. If operative time has been lengthy or renal calculi are present, it may be best to schedule a second surgery. Obtain a liver biopsy (see p. 546) before closing the abdomen.*

Jejunal Portography

Positive contrast radiographs can determine whether the shunt is extrahepatic or intrahepatic. If the caudal extent of the congenital PSS is cranial to T13, the shunt is probably intrahepatic. If the caudal extent of the shunt is caudal to T13, it probably is extrahepatic. Sensitivity of this procedure may be less in dorsal and right lateral recumbency than in left lateral recumbency.

Exteriorize a loop of jejunum. Identify a jejunal vein near the mesenteric border of the intestine and place one or two sutures around the vessel. Insert a 20- to 22-gauge over-the-needle catheter into the vessel (Fig. 20.17), and use the preplaced sutures to secure it to the vessel. Attach a heparinized extension set and a three-way stopcock to the catheter. Inject a water-soluble contrast agent (e.g., Renovist) (2 mL/kg of body weight) as a bolus into the catheter,

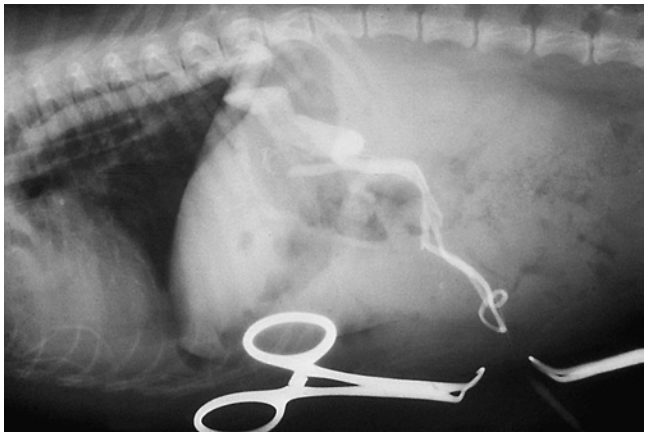


FIG. 20.18 Portogram in a dog with a portoazygos shunt. Note the lack of contrast filling in the hepatic portal system. Instead, it flows through a tortuous shunting vessel into the azygos vein.

and make an exposure while the last milliliter is injected. If necessary, make both lateral and ventrodorsal projections to help fully define the location of the shunt (Fig. 20.18). The catheter can also be used for pressure measurement.

With multiple acquired PSS, radiographic confirmation of the shunts is rarely necessary. The technique of intraoperative mesenteric portography in these patients is the same as for single PSS, except that exposures should be delayed approximately 3 or 4 seconds after the start of injection of the contrast material to allow filling of the shunting vessels.

Transvenous Retrograde Portography

Transvenous retrograde portography provides a method of identifying and characterizing shunts without the need for abdominal surgery; however, CT angiography is now more commonly done. This technique is most useful in dogs where occlusion of the shunt by intravascular means (e.g., coils) is being done; however, it may also be used to help identify IHPSS during surgery. The catheter, once placed, can be left in the shunt during surgery; palpation of it may help the surgeon identify the location of the shunt within the liver lobe.

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Place the patient in left lateral recumbency and aseptically prepare the right jugular furrow. In patients that weigh less than 10 kg, catheterize the jugular vein using a percutaneous technique. If the patient weighs more than 10 kg, perform a jugular venous cut-down to facilitate insertion of a large catheter into the vein. The large catheter is required in big dogs to totally occlude the caudal vena cava.

NOTE In patients weighing less than 10 kg, use a 7.5 French dual-lumen flow balloon (Swan-Ganz) catheter; in patients that weigh more than 10 kg, use variably sized balloon dilation catheters to facilitate occlusion of the caudal vena cava.

Following insertion into the introducer or venotomy site, direct the occluding catheter down the cranial vena cava and then in a dorsal direction into the azygos vein. Advance the catheter into the azygos vein as far as is possible without resistance. Inflate the balloon only enough to occlude the vein.

Occasionally, when larger balloon catheters are used, inflation of the balloon is unnecessary because the catheter itself may occlude the lumen sufficiently to allow for retrograde filling of the azygos vein. This seems to be especially true when the azygos vein is normal. *Make a vigorous hand injection of contrast (1–2 mL/kg) during fluoroscopic evaluation.* This injection normally results in retrograde filling of the intercostal and vertebral vessels. It is common to see the contrast flow in retrograde fashion into the abdominal cava. *Record the entire injection on videotape. If necessary, make an additional injection if hard copy of the study is required. Make initial images in the lateral projection; perform ventrodorsal or oblique views when indicated. After the initial injection into the azygos vein, withdraw the catheter into the right atrium; then advance it caudally through the right atrium and into the caudal vena cava. Advance the catheter to a position immediately cranial to the diaphragm. Once the catheter is in position, inflate the balloon enough to completely occlude the caudal vena cava. Once the caudal vena cava has been occluded, make a vigorous hand injection of contrast (1–2 mL/kg) during fluoroscopic evaluation (Figs. 20.19 and 20.20).* Occlusion of the caudal vena cava results in retrograde filling of the abdominal cava and the shunt.

In some cases, retrograde filling of the shunt is suboptimal. In those cases, positive-pressure ventilation (20 cm H_2O for 5–8 seconds) during the injection usually results in improved retrograde filling of the shunt.

NOTE It is imperative that the occlusion of the caudal vena cava and subsequent contrast injection be made immediately cranial to the diaphragm. If the occluding balloon is placed at the level of or caudal to the diaphragm, the ostia of shunts that arise in a cranial position may be occluded by the balloon, resulting in a false-negative study.

Once the shunt has been identified, attempt selective catheterization of the shunt. Selective catheterization with a flow-directed balloon allows for more specific opacification of the shunt, providing more detailed anatomic information. In addition, the configuration of the flow-directed balloon catheter allows for measurement of portal pressure both when the shunt is open and when it is occluded by the inflated balloon. Furthermore, selective catheterization of the shunt provides the opportunity to leave the catheter in the shunt lumen, facilitating intraoperative identification of the anomalous vessel. *Remove the catheter after the vessel has been identified and isolated. Remove the introducer and apply local pressure or sacrifice (ligate) the jugular vein and close the skin routinely.* The timing of removal of the introducer and catheters depends on whether the surgeon desires that the catheter be left in the lumen of the shunt during surgery.

Ligation or Attenuation of Intrahepatic Shunts

AP Both intravascular and extravascular methods have been described for ligation of IHPSS. Ligation of IHPSS can be extremely challenging because the vessel is often difficult to locate. Occasionally, the shunt can be identified as a palpable depression or a soft spot in a liver lobe, or it may be

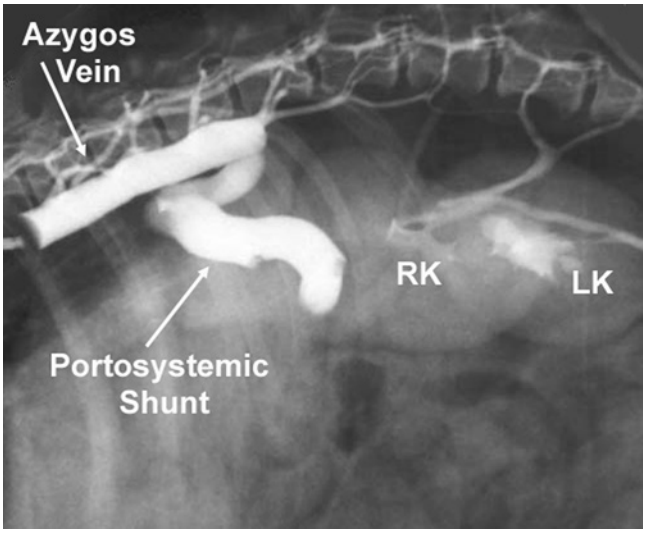


FIG. 20.19 Transvenous retrograde portogram (lateral view) in a dog with a portoazygos shunt. *LK,* Left kidney; *RK,* right kidney. (Courtesy Dr. Matt Miller, VETMED Consultants, Phoenix, AZ.)

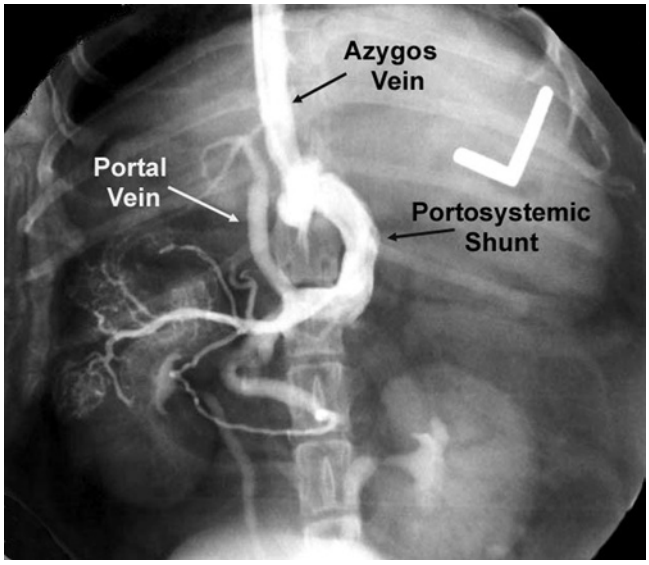


FIG. 20.20 Transvenous retrograde portogram (ventrodorsal view) in a dog with a portoazygos shunt. (Courtesy Dr. Matt Miller, VETMED Consultants, Phoenix, AZ.)

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seen entering the caudal vena cava if it is not completely encircled by hepatic parenchymal tissue. Intraoperative ultrasound scans have been used to help identify the shunt in hepatic tissue, but this technique is not always successful. Intrahepatic shunts may be classified as left, central, or right-sided. Left and central divisional shunts account for most shunts (see Fig. 20.14). Left-sided IHPSS (patent ductus venosus) is typically located in the left lateral or medial hepatic lobes. Ligation or attenuation of the left hepatic vein may be performed in these animals. Central shunts are generally found in the right medial lobe, and right shunts are typically located in the right lateral or caudate lobe. An intravascular technique involving temporary hepatic vascular occlusion in conjunction with caudal caval venotomy has been described for intrahepatic shunt occlusion; however, because this procedure is technically difficult and surgery time is prolonged, many surgeons prefer extravascular techniques. Isolation and obstruction of the specific branch of the portal vein supplying the IHPSS have been described. Indirect passage of suture for ligation of right-sided IHPSS was recently reported. The ligature should encircle the right portal branch approximately 4 mm lateral to its bifurcation from the parent vein (see later).

NOTE Warn owners that ligation of intrahepatic portosystemic shunts is difficult because the shunts are often hard to identify at surgery.

Isolation and Ligation of Intrahepatic Portosystemic Shunt Involving the Left Medial or Lateral Liver Lobes

AP Many shunts can be found cranial to the liver. *Extend the abdominal incision proximally into the caudal sternebrae. Incise the diaphragm if necessary. Incise the left triangular ligament and free the left lateral liver lobe so that it can be retracted to the right. Use a combination of sharp and blunt dissection to isolate the anomalous vessel at its junction with the hepatic vein. Place a single silk ligature around the vessel and attenuate flow while measuring portal pressures. Alternately, ligate or attenuate the left hepatic artery as it enters the liver while measuring portal pressures.*

Isolation and Ligation of Right-Sided Intrahepatic Portosystemic Shunt

AP *If necessary, ligate the right hepatic duct. Pass a Carmalt forceps from the dog's right to left over the dorsal surface of the main portal vein, just caudal to its bifurcation, but cranial to the termination of the gastroduodenal vein (Fig. 20.21). Grasp one end of a 2-0 silk suture and pull it back over the portal vein. Then, pass the forceps from the dog's right to left, dorsal to the left portal vein and within 5 mm of its bifurcation from the main portal vein. Grasp the opposite end of the suture and pull it back over the vein.*

Hydraulic Occlusion of Intrahepatic Portosystemic Shunt

Hydraulic occluders may be placed on the portal vein leading to the IHPSS. *Perform a ventral midline laparotomy with prehepatic dissection to identify the portal vein branch leading to the IHPSS. Ensure that appropriate portal, hepatic, and caudal vena caval vasculature is present. Measure the diameter of the portal vein branch of the vessel, while avoiding compression, and select the appropriate-sized hydraulic occluder (one that will not compress the vessel). Dissect the portal vein branch, thread 0 polypropylene suture through the occluder eyelets, and pass it around the branch. Appose the suture to close the occluder, and pass the actuating tubing through the body wall, tunneled subcutaneously through a separate stab incision to a site 5 cm lateral to the midline and 3 to 5 cm caudal to the ribs. Connect the tubing to the subcutaneous port, and secure it to the body wall. Inject a small amount of saline slowly (e.g., every other week) until complete shunt occlusion has been verified or side effects that make complete shunt occlusion not possible have been noted.*

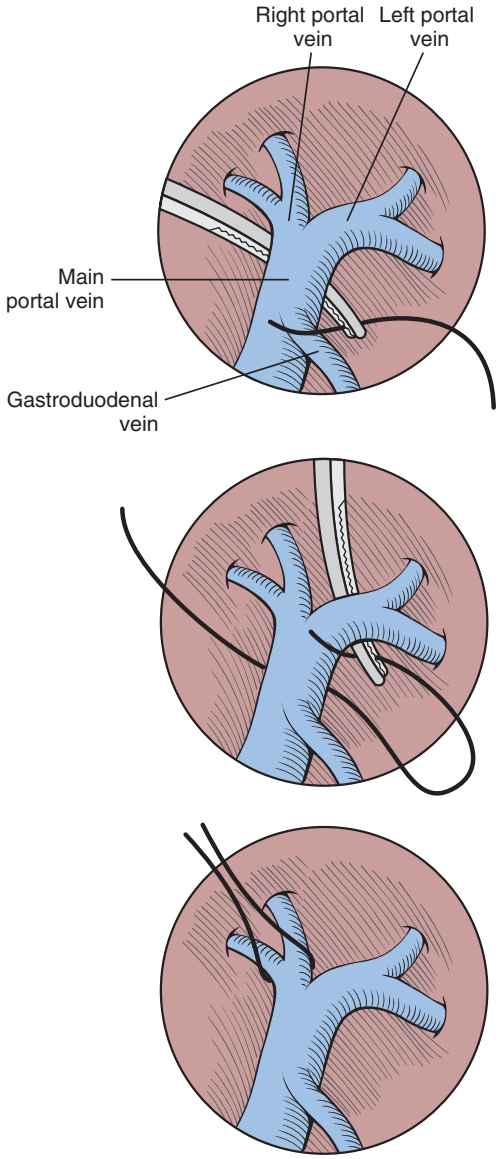


FIG. 20.21 Isolation and ligation of right-sided intrahepatic portosystemic shunt.

Complications of the procedure have led to redesign of the hydraulic occluders. Generally, occlusion is performed every 2 weeks in increments of 25% of the total occluding volume. Adding an additional 10% to 20% may be done to ensure complete occlusion.

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Intravascular Occlusion of Intrahepatic Portosystemic Shunt

Intravascular occlusion of IHPSS is performed by positioning a self-expanding Nitinol stent (Infiniti Medical) in the caudal vena cava over the orifice of the shunt. Once the stent is in place, the catheter is advanced through the interstices of the stent into the shunt as close to the stent as is feasible. Characterization of the shunt anatomy by abdominal ultrasonography, CT angiography, and/or retrograde portography (see p. 559) is important to determine the location of the shunt and the appropriate size of the stent. These diagnostic tests may also help ensure that the hepatic vasculature is not aplastic or hypoplastic, which may be a contraindication to the procedure.

With the patient in dorsal (left or right divisional shunts) or lateral (central divisional shunts) recumbency, make a stab incision over the jugular vein and position an intravascular introducer of appropriate size for the stent (typically 8–11 Fr) into the vein.[21] Advance a diagnostic catheter into the caudal vena cava and shunt, and perform a contrast injection in a left lateral or ventrodorsal projection using a diluted nonionic contrast agent (0.5–1 mL/kg) 1:1 with sterile saline to determine the size of the vena cava during 20 cm H_2O positive-pressure ventilation, which maximally distends the caudal vena cava. Deploy a stent of appropriate size (determined by caudal vena cava size and 10%–25% greater in diameter than the measured diameter) over the opening of the shunt into the cava. The stent should be at least 20 mm longer cranially and caudally to the shunt. Insert a catheter through the interstices of the stent into the shunt; use this catheter to deploy coils (e.g., stainless steel embolization coil) into the shunt and to measure portal pressures before and then after each coil is deployed. Do not allow portal pressures to increase by 6 to 7 mm Hg, by more than double, or if maximal pressure approaches 16 mm Hg.

Partial Hepatectomy for Removal of Hepatic Arteriovenous Fistula

Treatment of a hepatic AV fistula involves removal of the affected lobes and abnormal vascular structures; however, coil embolization of a large AV fistula in the abdomen has been described in the dog and may be possible for hepatic AV fistulae.[11] This has been done with or without temporary hepatic vascular occlusion. If temporary vascular occlusion is used, the vascular clamps and occlusive ligatures should be released within 15 minutes.

Extend the abdominal incision cranially through the caudal sternebrae and incise the diaphragm down to and partly around the hiatus of the caudal vena cava. Place moistened umbilical tapes around the thoracic portion of the caudal vena cava, the abdominal portion of the caudal vena cava (between the liver and renal veins), and the portal vein (just proximal to the first hepatic branch). Pass the umbilical tapes through a piece of rubber tubing (Rumel tourniquet). Identify, isolate, and ligate the phrenicoabdominal veins, and isolate the celiac and cranial mesenteric arteries. Place a purse-string suture in the portal vein or a splenic tributary and pass a 3.5 or 5 Fr catheter into the vessel to monitor portal pressures. Monitor blood pressure carefully during surgery; manipulation and ligation of the fistula may cause sudden, severe fluctuations. Isolate the affected lobes by dissection of the triangular, coronary, and hepatorenal ligaments and the ligaments of the lesser omentum. Identify the hepatic arterial branch supplying the affected lobe, and temporarily occlude it to see if pressure in the fistula diminishes. Double ligate the arterial supply of the fistula with nonabsorbable suture (e.g., 2-0 silk). Isolate the portal branch and biliary ducts to the affected lobe and double ligate them. Temporarily occlude the vasculature by tightening the preplaced umbilical tape ligatures and by placing vascular clamps on the celiac and cranial mesenteric arteries. Sharply dissect the liver parenchyma to resect the affected lobe. Ligate any vascular structures not already occluded, and control hemorrhage by packing the area for several minutes. Sometimes the affected portion of the liver can be removed by partial hepatectomy without performing vascular occlusion as described here.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Generally, 3.5 and 5 mm ameroid constrictors are used for occlusion of single extrahepatic shunts. Blunt-tipped, right-angled, or Mixter forceps are useful for dissecting around venous structures. Shunt ligation is usually performed with silk suture because of the relative knot security this suture affords. Delayed wound healing may be a problem if the patient is hypoproteinemic. To prevent dehiscence, a long-lasting absorbable suture material, such as polydioxanone, or a nonabsorbable suture material should be used to close the linea alba.

NOTE Ameroid constrictors are available through Research Instruments Northwest.

Right-angled forceps (e.g., Mixter, gallbladder, gall duct, or thoracic forceps) are widely available from many instrument manufacturers and suppliers.

POSTOPERATIVE CARE AND ASSESSMENT

In general, animals can be sent home the day after placement of an ameroid constrictor. Continuing medical management and feeding a low-protein diet may be necessary until the shunt vessel occludes and the hepatic parenchyma regenerates. These patients are often reevaluated 2 to 3 months after surgery and tested for evidence of improved hepatic function (e.g., normal serum albumin, normal serum cholesterol); however, whatever the outcome, if they are asymptomatic, further surgery is rarely warranted. Evaluating blood ammonia is probably more important in an animal that continues to have abnormal liver function because of the potential for development of ammonium urate calculi. The animal is weaned off medications and returned to a normal diet when hepatic function is determined to be reasonable.

Monitoring of the postoperative patient following PSS surgery should be intensive owing to the potential life-threatening complications. Close monitoring for portal hypertension and seizures is important regardless of the type of surgery performed. Dogs with PSS are also susceptible to hypoglycemia, despite IV dextrose infusion. Dextrose boluses may improve hypoglycemia in some cases; however, those that do not respond to dextrose may benefit from IV dexamethasone. The reason for the response to dexamethasone is unclear.

When shunt ligation or attenuation is performed, intensive care management and close observation of the patient are extremely important because portal hypertension may develop several hours after the procedure. Hypertension and splanchnic congestion may be evidenced as a painful abdomen, hemorrhagic diarrhea, endotoxic shock, and death. Many shunt patients have a painful abdomen during the early postoperative period; this makes it difficult to recognize life-threatening portal hypertension.

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However, should signs of endotoxic shock or hemorrhagic diarrhea or other signs of a deteriorating condition occur, emergency surgery to remove or loosen the ligature around the shunting vessel is advisable. Portal vein thrombosis may occur in congenital, single PSS cases in which the shunt has been partly ligated; it is a potentially life-threatening complication. If a shunt is only partly ligated, some authors recommend a single anticoagulant dose of regular heparin at the time of shunt attenuation. Ascites may occur after single shunt ligation; it is usually self-limiting, resolving in 1 to 3 weeks. Diuretics plus a low-salt diet may be used if drainage occurs from the incision site, or if the animal experiences dyspnea or discomfort from abdominal distention.

Seizures (typically cluster seizures) may occur after ligation of congenital PSS. These seizures are generally first noted 2 to 3 days after shunt ligation; their origin is unknown. This complication seems more common in cats, so much so that some recommend routine pretreatment of cats undergoing surgery for congenital PSS with phenobarbital (not potassium bromide, which can cause respiratory problems in cats). Diazepam is not recommended for these patients; constant-rate infusion propofol plus IV fluid support seems to be the most effective therapy. The patient is anesthetized for 24 hours and is then awakened. If seizures recur, then the patient is reanesthetized and awakened in another 24 hours. This cycle is repeated until control is achieved. Long-term anticonvulsant therapy may be required. Owners should be counseled that permanent neurologic abnormalities, such as blindness, may occur (especially in cats). Medical management of hepatic encephalopathy should be continued postoperatively until the hepatic parenchyma regenerates; this may take several months. If clinical signs have not improved within 2 to 3 months, repeating the contrast CT or other techniques (e.g., nuclear scintigraphy, jejunal portography) should be considered.

PROGNOSIS

Overall mortality is estimated to be between 2% and 25% for dogs following PSS surgery, with approximately one-fourth of these animals dying in the immediate postoperative period. Most dogs have an excellent or good outcome after placement of an ameroid constrictor. The main complication of ameroid constrictor placement is persistent flow through the ameroid constrictor; however, shunt fraction is insignificant in most of these dogs. Mortality associated with cellophane banding is less than 10%; potential complications include hemorrhage, ascites, seizures, and/or coagulopathies. Portal hypertension may occur secondary to kinking of the shunt; limiting dissection around the vessel may reduce this complication. The cause of seizures in these patients is not well understood. Some believe the cerebral edema plays a significant role. Animals that have grand mal seizures after surgery are more likely to die than those that have partial seizures characterized by disorientation, hyperesthesia, vocalization, salivation, and/or jaw clenching. Up to 12% of dogs develop neurologic signs within 6 days of surgical attenuation of congenital extrahepatic shunts. Prophylactic treatment with phenobarbital does not appear to significantly reduce the incidence of neurologic sequelae, but it may make the seizures less severe; however, levetiracetam may decrease the incidence of postoperative seizures (see earlier). Additionally, development of multiple acquired shunts may occur after occlusion of congenital PSS using any technique. With ligation, the surgical mortality is relatively high, a fact that reflects the many variables and unknown factors that exist in relation to portal physiology and dynamics.

Patients that only tolerate partial shunt occlusion and have persistence of clinical signs postoperatively require dietary and medical management. In such animals, reoperation and total shunt occlusion are recommended. Dogs that tolerate complete ligation of their shunt tend to have fewer clinical signs than those that tolerate only partial occlusion at surgery. Factors that appear to be significant predictors of continued portosystemic shunting include low preoperative albumin concentrations, high portal pressure during complete temporary occlusion of the PSS, and high portal pressure difference. Predictors of an unsuccessful long-term outcome include lower preoperative albumin concentration, high white blood cell count, high portal pressure measured during complete temporary occlusion of the PSS, postoperative seizures, and continued shunting. Outcome after placement of cellophane bands seems to be similar to that following use of ameroid constrictors.

Hemorrhage, hypotension, and acute hepatic congestion are possible complications during surgical correction of IHPSS in dogs. Packed cell volume and total protein may be positive prognostic indicators for long-term survival in dogs with IHPSS, whereas low body weight (<15 kg) and low total protein, albumin, and BUN may be negative prognostic factors. The long-term prognosis is good for dogs with hepatic AV fistulae that survive surgery.

Approximately 60% of cats develop postoperative neurologic signs after surgery for congenital PSS, with approximately 30% having seizures. Seizures may be treated successfully in the majority of these cats with the use of phenobarbital; however, some may require IV propofol therapy. Neurologic signs appear to be correlated with the grade of portal blood flow on intraoperative portograms, as well as the animal's age and duration of and response to medical management before surgery and with the level of bile acids after surgery. Animals that have less developed hepatic portal blood flow are more likely to have neurologic signs after surgery. Seizures are much less common in dogs than in cats after surgery.

Treatment of hepatic AV fistulae in dogs with traditional surgery, glue embolization, or interventional techniques is associated with a good prognosis for long-term survival. Immediate complications related to fistula ligation or removal include portal or mesenteric venous thrombosis, peritoneal effusion, icterus, hemorrhage, and hepatic encephalopathy. Some dogs may require reoperation if the fistula is not totally occluded or removed and serious complications (e.g., persistent ascites, neurologic signs, melena) may lead to euthanasia. Hepatic function tests may not normalize after surgery.

CAVITARY HEPATIC LESIONS

DEFINITIONS

Cavitary hepatic lesions are usually cysts or abscesses, although occasionally large neoplastic lesions, such as hemangiomas and adenomas, may cavitate. **Hepatic abscesses** are localized collections of pus in the hepatic parenchyma. **Hepatic cysts** are closed, fluid-filled sacs lined by secretory epithelium.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Hepatic abscesses are rare in dogs and cats and are usually associated with extrahepatic infection (e.g., ascending biliary

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tract infection, hematogenous infection via the portal vein or hepatic artery or via direct extension from areas adjacent to the liver), hepatic trauma (e.g., surgical biopsy, penetrating wounds, blunt trauma), or neoplasia. This is despite the normal presence of bacteria in the liver of dogs and may be the result of a well-developed local defense system provided by the rich blood supply of the liver and the phagocytic ability of reticuloendothelial cells. Diabetes mellitus has been associated with hepatic abscesses. Small abscesses may not cause clinical signs and may resorb without therapy.

Hepatic abscesses are most often recognized as a complication of omphalophlebitis in puppies and are usually diagnosed at necropsy.

Hepatic cysts are usually incidental findings, although in rare cases, they become large enough to interfere with the function of adjacent organs. A single hepatic cyst may be noted, or several cysts may be present in the same or different lobes. Concurrent polycystic renal disease has been reported in cats. If hepatic cysts are present in an animal with clinical evidence of hepatic dysfunction, liver biopsy is often warranted to determine the cause.

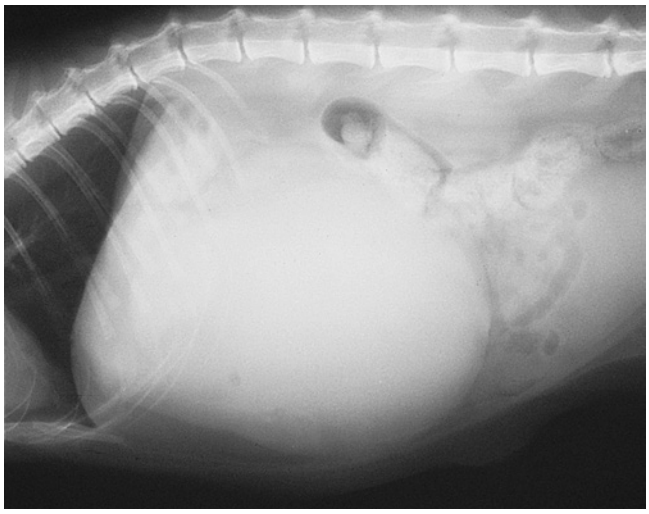


FIG. 20.22 Lateral abdominal radiograph of a 2-year-old cat with a large hepatic cyst. The cat was asymptomatic. Radiographically, a large, soft tissue mass arising from the liver can be seen. The diagnosis of a cyst would require ultrasound examination.

DIAGNOSIS

Clinical Presentation

Signalment

No gender or breed predisposition has been reported for hepatic abscesses or cysts.

History

Clinical signs of hepatic abscessation vary and may include anorexia, lethargy, weight loss, vomiting, and intermittent abdominal pain. Most animals with hepatic cysts are asymptomatic; however, some cysts cause abdominal distention. Secondary infections of hepatic cysts may cause clinical signs similar to those of hepatic abscesses.

Physical Examination Findings

Physical examination findings in patients with hepatic abscessation typically include persistent fever, hepatomegaly, and abdominal enlargement. Palpation of a firm abdominal mass and marked abdominal distention may be noted in some animals with hepatic cysts. Clinical signs are often vague (e.g., anorexia, lethargy, weight loss). Clinical signs of sepsis are common in cats with small multifocal abscesses and in those with microabscesses.

Diagnostic Imaging

Small hepatic cysts are often incidental radiographic and sonographic findings. Large hepatic cysts are usually well-defined, radiopaque structures in the cranial abdomen (Fig. 20.22). Abdominal radiographs may demonstrate hepatomegaly in animals with hepatic abscesses, but a well-defined hepatic mass is seldom evident. Occasionally, gas is noted in the hepatic parenchyma; this strongly suggests abscessation caused by gas-forming bacteria. Ultrasonography is the most useful diagnostic test for defining hepatic abscesses and cysts in dogs and cats. Hepatic abscesses appear as hypoechoic or anechoic structures that may contain mixed echogenicities, depending on the cellularity. The abscesses may appear solitary, multifocal, and small, or they may be microabscesses. Scintigraphy, CT, and magnetic resonance imaging (MRI) also are highly sensitive in diagnosing hepatic lesions, but these techniques are used less often than ultrasonography. Ultrasound-guided fine-needle aspirations of hepatic abscesses can be performed before surgery; however, there is a risk that the abscess will rupture or drain into the abdomen and cause diffuse peritonitis. Fluid removed from cysts during fine-needle aspiration is usually transudative.

NOTE Evaluate the kidneys for cystic disease in cats with hepatic cysts. Both conditions may be present.

Laboratory Findings

Laboratory abnormalities are seldom present with hepatic cysts. They are variable in animals with hepatic abscesses but may include an inflammatory leukogram and nonregenerative anemia. Serum biochemical abnormalities may include hypoalbuminemia, hypokalemia, hyperglycemia, and elevated hepatic enzymes; however, elevation of alanine transaminase activity is not a consistent finding. *E. coli* is the most commonly isolated organism from cats with hepatic abscesses. The organisms most often isolated from hepatic abscesses in dogs include *E. coli* and *Clostridium* spp.

DIFFERENTIAL DIAGNOSIS

Hepatic cysts, abscesses, neoplasms, cystic neoplasia, and parasitic lesions must be differentiated. Hepatic abscesses are often difficult to diagnose because they produce nonspecific signs that may be masked by associated disease processes. Large neoplastic hepatic lesions may necrose and become secondarily infected. Infection of hepatic cysts is also possible. Therefore histologic evaluation of surgically resected tissue is important.

MEDICAL MANAGEMENT

Medical management of hepatic abscesses entails administering fluid/electrolyte/acid-base therapy plus appropriate antibiotic therapy. Percutaneous ultrasound-assisted drainage and alcoholization using 95% ethanol may be considered.

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To perform this technique, position a spinal needle attached to extension tubing and a syringe into the abscess using ultrasound guidance; drain the fluid. Use a syringe that is twice the volume of the estimated amount of exudate in the lesion. Before removing the spinal needle, inject a volume of alcohol equal to half the volume of exudate removed. Leave the ethanol in the abscess cavity for 3 minutes and then gently remove it. Submit the exudate for culture and susceptibility testing. Continue appropriate antibiotic therapy for an additional 30 days.

If surgery is elected, resection of hepatic abscesses is indicated as soon as the animal is an acceptable anesthetic risk. Preoperative antibiotic therapy may be based on culture and sensitivity results if fine-needle aspiration has been performed, or antibiotics with bactericidal activity against anaerobes and gram-negative bacteria (e.g., ampicillin/sulbactam plus enrofloxacin, cefoxitin, clindamycin plus enrofloxacin, ampicillin/sulbactam plus amikacin; Box 20.4) may be given empirically. Parenteral antibiotics are indicated in the perioperative period. Combination therapy may be necessary, particularly if multiple organisms are isolated. Percutaneous drainage of hepatic cysts and sclerosis of the cyst lining have not been reported in dogs or cats.

SURGICAL TREATMENT

Whether hepatic cysts should be removed when diagnosed in asymptomatic animals is not clear. Although these cysts could enlarge or become infected and cause clinical signs, little information is available about the long-term follow-up of large hepatic cysts that are not surgically resected in dogs or cats. Hepatic cysts associated with clinical signs and hepatic abscesses should be promptly resected.

BOX 20.4 Antibiotics Used to Treat Hepatic Abscesses

Amoxicillin Plus Clavulanate (Clavamox)
Dogs: 12.5–25 mg/kg PO q12h
Cats: 62.5 mg PO q12h

Enrofloxacin (Baytril)[a]
Dogs: 7–20 mg/kg PO or IV[a] q24h slowly over 20–30 min
Cats: 5 mg/kg PO q24h; do not exceed 5 mg/kg q24h due to risk of blindness

Cefoxitin (Mefoxin)
30 mg/kg IV q6–8h

Cefazolin (Ancef, Kefzol)
22 mg/kg IV, IM or SC q8–12h

Clindamycin (Antirobe)
Dogs: 11–33 mg/kg q12h PO, or 11 mg/kg IV (dilute and give slowly when administering IV)
Cats: 11–33 mg/kg PO or SC q12–24h

Metronidazole (Flagyl)
10–12 mg/kg PO or IV (must be diluted and given slowly) q8h

Amikacin
Dogs: 15–22 mg/kg IV (diluted and given slowly) q24h
Cats: 10–14 mg/kg IV (diluted and given slowly) q24h

Ampicillin Sodium and Sulbactam Sodium (Unasyn)
10–50 mg/kg IV q12–24h off-label use

[a]Doses >5 mg/kg may be associated with blindness in cats.
IM, Intramuscular; *IV,* intravenous; *PO,* orally; *SC,* subcutaneous.

Preoperative Management

Symptomatic animals should be in stable condition before surgery. Antibiotics may be initiated before surgery, or in some animals, they may be administered after intraoperative cultures have been taken.

Anesthesia

See p. 540 for the anesthetic management of animals with hepatic disease.

Surgical Anatomy

See p. 543 for the surgical anatomy of the liver.

Positioning

The animal is positioned in dorsal recumbency for a midline abdominal incision. The prepped area should extend from midthorax to the pubis.

SURGICAL TECHNIQUE

Hepatic abscesses and cysts are generally treated by partial hepatectomy (see p. 546). If hepatectomy cannot be performed safely and the cyst completely removed, it may be omentalized. Although there is less concern about spillage of cystic contents into the abdomen, it is wise to try to remove the cyst without entering the lumen. Culturing hepatic cysts may be optional even if the fluid does not appear to be infected cytologically because some cysts can develop secondary bacterial infection.

Pack the area surrounding the liver with moistened laparotomy sponges to diminish intraoperative contamination if the lumen of the abscess or cyst is entered. If possible, resect the affected portion of the liver without entering the lesion. Culture the lesion and submit it for histologic examination. Palpate the remainder of the liver parenchyma for other nodules, and explore the abdominal cavity for associated infection or disease.

For omentalization, identify a segment of the omentum that will extend into the cyst cavity. Remove as much of the wall of the cyst as possible, and spread the omentum over the remaining cyst and adjacent liver. Tack it gently in place to the remaining cyst capsule.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

See p. 549 for recommendations regarding suture choices during partial hepatectomy.

POSTOPERATIVE CARE AND ASSESSMENT

Fluid therapy for animals with hepatic abscesses should be continued until the animal is drinking normally. Antibiotic therapy should be continued for 7 to 10 days. The animal should be monitored for peritonitis (e.g., leukocytosis, fever, abdominal fluid, abdominal pain) if abdominal contamination occurred. Minimal postoperative care is needed for most animals with hepatic cysts.

PROGNOSIS

The prognosis for animals with hepatic abscess depends on the rapidity with which the abscess is diagnosed, whether concurrent

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peritonitis is present, and the animal's overall health; however, the overall mortality rate is high and a guarded prognosis is warranted. The prognosis for animals with hepatic cysts (with or without surgery) is good unless concurrent hepatic or renal disease exists.

HEPATOBILIARY NEOPLASIA

DEFINITIONS

Hepatocellular tumors arise from hepatocytes; **cholangiocellular neoplasms** arise from intrahepatic or extrahepatic bile duct epithelium. The term *hepatoma* has historically referred to hepatocellular adenomas. However, current thought is that these hepatomas or adenomas are more accurately considered low-grade malignancies; therefore **hepatocellular carcinoma** is now considered the preferred term for such masses. Cholangiocellular carcinomas are also known as *bile duct carcinomas.*

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Primary hepatic neoplasms are uncommon in dogs and cats. They may be of epithelial or mesenchymal origin (Box 20.5). Hepatocellular carcinomas and cholangiocellular carcinomas are the most commonly diagnosed primary hepatic malignancies in dogs.[6] Hepatocellular carcinomas may involve a single liver lobe or may be nodular or diffuse, involving multiple lobes. In cats, cholangiocellular adenomas are the most common primary tumor. Hepatic carcinoids are rare tumors that arise from neuroectodermal cells in the liver. Biliary cystadenomas are uncommon, benign liver tumors of older cats that may occur as focal or multifocal cystic lesions. Benign hepatic masses (i.e., adenomas or cysts) are often incidental findings at necropsy. They may be more common than malignant tumors in both species but often go undiagnosed because they seldom cause clinical signs. Cholangiocellular carcinomas arise primarily from intrahepatic bile duct epithelium; neoplasms of the extrahepatic bile duct and gallbladder are rare.

Malignant primary hepatic tumors have typically been considered to be highly metastatic; however, long-term survival is common after lobectomy for hepatocellular carcinoma. The median survival time in cats undergoing surgery for hepatocellular carcinoma was 2.5 years in a 2016 study.[22] These tumors may metastasize by direct extension to other liver lobes or adjacent organs, or they may spread distantly via lymphatics or blood. Epithelial tumors most often metastasize to the regional lymph nodes and lungs. Mesenchymal tumors most often metastasize to the spleen.

Metastatic neoplasia is more common in the liver than are primary tumors. The liver is a common site for metastasis because it acts as a filter between abdominal organs and the systemic circulation. Lymphosarcoma is the most common secondary hepatic tumor. Other tumors that commonly metastasize to the liver are pancreatic adenocarcinomas, hemangiosarcomas, insulinomas, and tumors of the alimentary and urinary tracts.

BOX 20.5 Primary Hepatic Neoplasia in Dogs and Cats

Epithelial
- Hepatocellular carcinoma
- Hepatocellular adenoma
- Cholangiocellular carcinoma
- Cholangiocellular adenoma
- Hepatic carcinoids

Mesenchymal
- Hemangiosarcoma
- Fibrosarcoma
- Extraskeletal osteosarcoma
- Leiomyosarcoma

DIAGNOSIS

Clinical Presentation

Signalment

Primary hepatic neoplasia is usually a disease of aged dogs and cats. No breed predisposition is known. Hepatocellular carcinomas may be more common in male dogs, whereas cholangiocellular carcinomas may be more common in cats and female dogs. Dogs with metastatic liver cancer may be slightly younger (average, 7.8 years of age) than those with primary hepatic malignancy (average, 10 years of age).

History

Animals with primary hepatic neoplasia are often presented for treatment of signs associated with hepatic failure. The animal may be lethargic, weak, anorexic, losing weight, or vomiting, or may have polyuria or polydipsia. Clinical signs associated with metastatic hepatic neoplasia vary considerably. Primary hepatic tumors and metastatic hemangiosarcomas may rupture and bleed, causing signs of shock.

Physical Examination Findings

The most significant finding on physical examination of most primary hepatic tumors is an enlarged liver; however, hepatic carcinoids may not cause significant hepatomegaly. Additional findings may include jaundice and ascites. Hemangiosarcomas and hepatocellular adenomas may rupture, causing hemoperitoneum, shock, and pale oral mucous membranes. Marked hepatomegaly is less common with metastatic neoplasia; however, lymphosarcoma often causes diffuse hepatic enlargement.

Diagnostic Imaging

Survey radiographs help localize the mass to the liver (Fig. 20.23) and may reveal extrahepatic metastasis. Thoracic radiographs are indicated whenever hepatic neoplasia is suspected, because pulmonary metastasis is possible.[6] Ultrasonography often localizes and defines the extent of disease. Ultrasound-guided biopsies may allow presurgical diagnosis (see p. 545), but these tumors are often highly vascular and can bleed profusely. Although conventional ultrasound may detect lymph node enlargement, thereby helping to define metastasis in dogs with hepatic neoplasia, Doppler and contrast-enhanced ultrasound may be more sensitive diagnostic aids. Contrast-enhanced ultrasonography (e.g., perfluoropropane lipid emulsion contrast agent) may aid in distinguishing benign from malignant hepatic nodules and has the advantage of allowing real-time evaluation of tumor perfusion without anesthesia; however, further investigation of this technique is warranted.

NOTE Ultrasonography is particularly useful in animals with ascites. Radiographs often show little abdominal detail when ascites is present.

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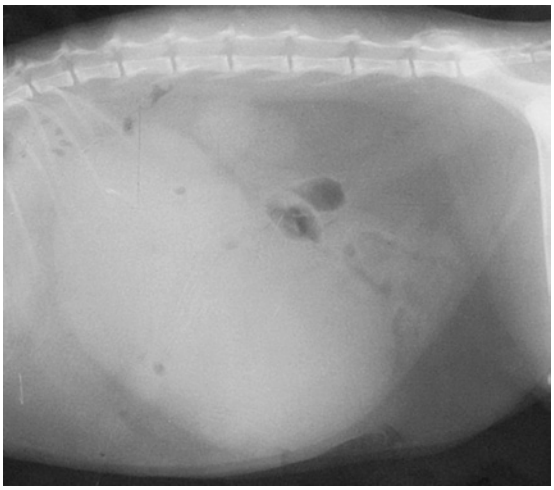


FIG. 20.23 Lateral abdominal radiograph of a dog with a large malignant hepatic tumor. Note the similarities between the radiographic appearance of this tumor and the benign hepatic cyst in Fig. 20.22. Benign and malignant hepatic masses cannot be differentiated radiographically.

Contrast CT has been studied to differentiate hepatic lesions in dogs. Dual-phase CT was not found to be of great value in one study.[23] General findings on CT included central and marginal enhancement during the arterial phase in hepatocellular carcinoma, hypoattenuation in the portal phase, and large cyst-like lesions.[24] Diffuse enhancement was present in hepatocellular adenomas and was also seen with nodular hyperplasia. However, even the use of triple-phase helical CT cannot definitively diagnose tumor type.[25] MRI has been evaluated in dogs as well. The ability to detect multiple lesions using gadoxetate disodium-enhanced MRI was variable in one study.[26]

Laboratory Findings

Neutrophilia and biochemical abnormalities compatible with hepatic disease (e.g., elevated serum alanine transaminase, aspartate transaminase, serum alkaline phosphate) are expected in most patients but may be absent in animals with hepatic neoplasia.[6] They are nonspecific, but recognition may prompt further evaluation of the hepatobiliary system. Mild to moderate anemia is less commonly associated with hepatic neoplasia. Serum bilirubin concentrations may be elevated, particularly if extrahepatic biliary obstruction occurs. Occasionally, hypoglycemia causes clinical signs. Albumin levels are usually normal in patients with primary hepatic neoplasia. Biochemical abnormalities seldom correlate with the extent of hepatic involvement with either primary or metastatic tumors.

NOTE Laboratory abnormalities often do not occur until the neoplasm is large enough to make surgical excision difficult.

DIFFERENTIAL DIAGNOSIS

Primary hepatobiliary tumors must be differentiated from regenerative nodules, abscesses, hematomas, and cysts. Histologic and/or cytologic evaluation of fine-needle aspirates or biopsy specimens is necessary to distinguish definitively between these lesions (see p. 544), but it can be difficult to distinguish well-differentiated, low-grade hepatocellular carcinomas from regenerative nodules. Percutaneous biopsies should not be performed in animals with clinical bleeding disorders or if the lesions appear cavitary or highly vascular. Cytologic evaluation of abdominal fluid is seldom helpful in differentiating between these lesions. A proposed classification of tumors in cats included nodular hyperplasia (single or multiple; 10%), cholangiocellular (41%), or hepatocellular (34%).[27] Hepatocellular tumors were subdivided into well-differentiated hepatocellular adenoma, poorly differentiated hepatocellular carcinomas, and small cell carcinoma, or carcinoids.

MEDICAL MANAGEMENT

Surgical excision of primary malignant hepatic tumors is the treatment of choice. Unfortunately, these tumors may not be diagnosed until they are large and metastasis has occurred. Because they are usually diagnosed in older animals, concurrent cardiac, renal, or other metabolic problems are common. Medical therapy should aim at correcting fluid and electrolyte imbalances and providing nutrition to improve the chances of surviving surgery (see Chapter 10).

INTERVENTIONAL ONCOLOGIC TREATMENT

Coil embolization has been reported in the treatment of a cat with hepatocellular carcinoma, and chemoembolization has been proposed as a means of therapy for large hepatic tumors in veterinary patients because it reduces systemic exposure to chemotherapeutic agents, subsequently decreasing morbidity associated with these agents.[28,29] Chemoembolization is used to treat diffuse hepatocellular carcinoma in people and has been performed in dogs in which surgery was declined or was unsuccessful.[30] However, the equipment required for super-selective intraarterial mapping and embolization is not readily available in veterinary medicine. Additionally, postembolization syndrome (malaise, fever, pain), hepatic infarction, hepatic abscessation, acute renal failure, and the potential for damage to normal parenchyma are possible side effects of this mode of therapy.

SURGICAL TREATMENT

If the tumor is localized to a single lobe or is confined to the gallbladder, surgical resection may be curative. Partial hepatectomy and cholecystectomy are described on pp. 547 and 574, respectively. Ultrasound is often used to screen for metastasis, but it is not a particularly sensitive way to detect such lesions, particularly if the metastases are on serosal surfaces. Although not as sensitive as an exploratory laparotomy, laparoscopy is more sensitive than ultrasound for finding metastasis, and one can first perform laparoscopy to look for gross evidence of metastasis before proceeding to surgery to resect a tumor. In addition, imaging with near-infrared fluorescence and indocyanine green administration may be of value in the future for evaluating the extent of mass lesions in the liver.[31]

Surgical biopsies should be performed on all animals with hepatomegaly or nodularity because differentiation of lesions requires histopathologic evaluation. Finding multiple hepatic masses is not pathognomonic for metastatic disease. Some benign lesions can present as multiple hepatic nodules (e.g., regenerative nodules associated with cirrhosis or lobular collapse), and primary hepatic tumors may spread to other portions of the liver. If

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neoplasia is suspected, the draining lymph nodes and surrounding organs should be carefully assessed for metastasis. Hepatocellular tumors are most commonly found in the left medial and left lateral liver lobes.

Preoperative Management

The animal's condition should be stabilized before surgery if possible. Fluid deficits and electrolyte imbalances should be corrected. Blood transfusions (see Table 4.5) should be given to severely anemic animals (i.e., packed cell volume less than 20%), especially if bleeding tendencies are present (i.e., petechiation, ecchymosis, or hemorrhage). If the animal has clinical evidence of a significant coagulopathy on the mucosal bleeding time (>5 minutes) or is severely thrombocytopenic (<20,000 platelets/μL), consider plasma or whole blood transfusions. Many patients with a prolonged one-stage prothrombin time and PTT do not have bleeding problems at surgery, but they should be monitored for such before, during, and after surgery. If the patient has substantial ascites, slow removal of some fluid before induction of anesthesia may help prevent hypoventilation associated with positioning the patient while it is prepared for surgery.

Anesthesia

Ventilation of patients with ascites requires support (i.e., intermittent positive-pressure ventilation). Compression of the caudal vena cava in patients with large hepatic masses or massive ascites may diminish venous return and reduce cardiac output. See p. 540 for additional comments about the anesthetic management of patients with hepatic disease.

Surgical Anatomy

See p. 543 for information on the surgical anatomy of the liver.

Positioning

Exploration of the liver is generally performed through a cranial ventral midline abdominal incision (see p. 516). Prepare the patient for caudal sternotomy and diaphragmatic incision, which may aid in the approach to large masses. Alternatively, the incision may be extended paracostally to allow enhanced visualization and manipulation of large tumors. The prepped area should extend from midthorax to the pubis.

SURGICAL TECHNIQUE

See pp. 547 and 574 for a description of surgical techniques for partial hepatectomy and cholecystectomy, respectively.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Absorbable suture material is used for hepatic biopsy (see p. 549). Ligation of the cystic duct for cholecystectomy is generally done with nonabsorbable suture material (see p. 574).

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative nutritional support of patients with hepatic neoplasia is often necessary (see Chapter 10). Nonresectable primary hepatic tumors seldom respond to chemotherapy or radiation therapy. Chemotherapy may palliate hepatic lymphosarcoma. For other considerations in animals undergoing partial hepatectomy, see p. 540.

PROGNOSIS

The prognosis for dogs and cats with primary hepatobiliary malignancies is often poor; however, some dogs may live a year or longer with aggressive therapy. In a 2015 study reporting lobectomy for liver masses, 23% of the animals died or were euthanized.[6] These animals were typically euthanized due to metastasis and invasiveness of the mass, development of liver failure, or the occurrence of hemoabdomen. In cats with malignant, nonlymphomatous hepatobiliary disease that undergo surgery, survival is generally days to a few months. The high rate of metastasis and degree of invasion make surgical resection unlikely to be curative in most patients. Benign tumors may be surgically resected, and long-term survival of patients with benign hepatic tumors has been reported. Survival times in cats with hepatobiliary cystadenomas have ranged from 12 to 44 months after surgery.

HEPATIC LOBE TORSION

DEFINITIONS

Hepatic lobe torsion occurs when a liver lobe twists around its axis.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Liver lobe torsions are rare in dogs and cats. Torsion of the left lateral or medial lobe appears to be most common, presumably because of greater mobility, size, and relative separation from adjacent lobes. In most animals the cause is unknown, but congenital absence or traumatic rupture of hepatic ligaments is generally suspected. When a liver lobe twists on its axis, it creates venous obstruction, causing increased hydrostatic pressure, ascites, and thrombosis. The liver lobe will eventually necrose.

Clinical Presentation

Signalment

No breed or sex predisposition has been identified for hepatic lobe torsion in dogs or cats. Most reported dogs have been middle-aged, large-breed dogs.

History

Clinical signs of hepatic lobe torsion are often nonspecific and may include depression, lethargy, vomiting, diarrhea, anorexia, collapse, and/or abdominal enlargement of one to several days' duration. Acute decompensation or death may occur, and chronic clinical courses have been documented.

Physical Examination Findings

Physical examination findings may include pain on abdominal palpation, tachycardia, dehydration, weak femoral pulses, cardiac arrhythmia, and ascites. The animal should be examined carefully for signs of trauma.

Diagnostic Imaging

Survey radiographs may show a mass effect, gas distention of the stomach and intestine, and ascites and should be reviewed for signs of associated trauma (e.g., diaphragmatic hernia). Ultrasonography is helpful to define the lesion, and Doppler ultrasonographic assessment usually identifies decreased blood flow to the affected segment of liver.

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Laboratory Findings

Bloodwork abnormalities are nonspecific and are not helpful in identifying hepatic lobe torsion. Mature neutrophilia and leukocytosis may be present, and biochemical abnormalities compatible with hepatic disease (elevated serum alanine transaminase, aspartate transaminase, and serum alkaline phosphate) are commonly seen. Coagulation testing may reveal prolongations, but coagulation difficulty was not reported commonly.

DIFFERENTIAL DIAGNOSIS

Liver lobe torsion must be differentiated from mass lesions and nonsurgical diseases of the liver, such as hepatitis.

MEDICAL MANAGEMENT

Surgical excision of torsed hepatic lobes is warranted. The animal should be stabilized before undergoing surgery.

SURGICAL TREATMENT

Surgical resection of the devitalized lobe is warranted. The technique for hepatic lobectomy is described on p. 547.

Preoperative Management

The animal's condition should be stabilized before surgery if possible. Fluid therapy should be initiated and electrolyte imbalances corrected. If the patient has massive ascites, slow removal of some fluid before induction of anesthesia may help prevent hypoventilation associated with positioning the patient while it is prepared for surgery.

Anesthesia

Ventilation of patients with ascites may require support (i.e., intermittent positive-pressure ventilation). See p. 540 for additional comments about anesthetic management of patients with hepatic disease.

Surgical Anatomy

The liver lobe is supported by a series of ligaments, including the left and right lateral triangular ligaments, which extend from the left and right lateral hepatic lobes to the muscular portion of the diaphragm; the left and right lateral coronary ligaments, which attach the right and left lobes to the central tendinous portion of the diaphragm; and the falciform ligament, which attaches to the liver, abdominal wall, and sternal portion of the diaphragm.

Positioning

Exploration of the liver is generally performed through a cranial ventral midline abdominal incision (see p. 516). The prepared area should extend from midthorax to the pubis.

SURGICAL TECHNIQUE

See p. 547 for a description of the surgical technique that should be used for removal of a liver lobe. Histologic evaluation of the excised liver lobe is warranted because the devitalized mass may appear similar in appearance to a hepatic tumor.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Absorbable suture material is used for liver lobectomy (see p. 549).

POSTOPERATIVE CARE AND ASSESSMENT

Information on postoperative care of the patient after liver lobe resection is provided on p. 549. Dogs with associated diaphragmatic hernias or other trauma should be carefully observed for evidence of respiratory distress after surgery. Fluid support should be continued until the animal is eating and drinking sufficiently on its own. Pain medication should be given postoperatively (see p. 549). Arrhythmias may require treatment and reportedly have responded to lidocaine therapy.

COMPLICATIONS

Potential complications after surgery include anemia, arrhythmia, hypoalbuminemia, and vomiting leading to aspiration pneumonia. Severe hemorrhage is uncommon, but animals should be monitored carefully after surgery for bleeding.

PROGNOSIS

The prognosis for dogs and cats with hepatic lobe torsion is good if an underlying disease process is identified and can be treated effectively. Liver lobectomy in dogs with liver lobe torsion without primary identifiable disease has an excellent prognosis.

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21

Surgery of the Extrahepatic Biliary System

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Cholecystotomy is the creation of an opening into the gallbladder for exploration, biopsy, and/or drainage; **cholecystectomy** is removal of the gallbladder. **Choledochotomy** is incision of the common bile duct for exploration or removal of a calculus. **Choledochoduodenostomy** is a rarely indicated procedure in dogs and cats that involves surgical anastomosis of the common bile duct to the duodenum. **Cholecystoenterostomy** is a term used to describe biliary diversion in which the gallbladder is opened and sutured to a portion of the intestine. **Cholecystoduodenostomy** and **cholecystojejunostomy** are surgical anastomoses of the gallbladder to the duodenum and jejunum, respectively. Calculi may form in the gallbladder (**cholelithiasis**) or in the bile duct (**choledocholithiasis**). **Cholangitis** refers to inflammation of the intrahepatic bile ducts; **cholangiohepatitis** refers to concurrent inflammation of the bile ducts and surrounding hepatic parenchyma.

PREOPERATIVE CONCERNS

Biliary disease may be caused by extrahepatic biliary tract obstruction (EHBO), neoplasia, stones, infection, or trauma. Lesions that cause EHBO may be extraluminal or intraluminal. Extraluminal obstruction may be caused by pancreatitis, pancreatic neoplasia, duodenal or pyloric neoplasia, hepatic or biliary neoplasia, diaphragmatic hernia, congenital abnormalities, or pancreatic abscessation. Intraluminal obstruction is less common but may occur in association with cholelithiasis, choledocholithiasis, inspissated bile, mucocele, tumor, or liver flukes (cats). Pancreatic disease is the most common cause of EHBO in dogs and may be important in cats as well. Scar formation may occur in or around the duct, or the duct may be compressed by fibrotic or inflamed pancreatic tissue. Pancreatic abscesses and cysts may rarely cause biliary obstruction.

Animals with EHBO should have electrolyte and fluid abnormalities corrected before surgery. Prolonged EHBO may cause vitamin K malabsorption, resulting in deficiencies of factors VII, IX, and X. Animals with clinical evidence of bleeding or increased mucosal bleeding time should receive vitamin K_1 (subcutaneous [SC], not intravenous [IV] or intramuscular [IM]) for 24 to 48 hours before surgery (Box 21.1) or fresh whole blood (see p. 33). However, some animals with EHBO are hypercoagulable, as demonstrated by thromboelastography.[1] Partial or complete biliary obstruction may allow ascending aerobic and anaerobic infection and subsequent bacteremia; therefore perioperative antibiotic therapy is indicated (see Chapter 9). The finding of *hemobilia* (upper gastrointestinal bleeding originating in the biliary tree) and *hemocholecyst* (accumulation of blood or blot clot within the gallbladder) associated with gallbladder neoplasia may not be accompanied by coagulopathy, but coagulation testing should be done.

Extrahepatic biliary injury may be caused by blunt or penetrating trauma. Common bile duct, gallbladder, cystic duct, or hepatic duct lacerations may cause bile peritonitis or, if the leakage is "walled off," a localized inflammatory process with adherence to surrounding organs. Necrotizing cholecystitis occurs when bacteria devitalize the gallbladder wall, often resulting in peritoneal spillage of bile (Fig. 21.1). This frequently leads to the development of severe, generalized septic peritonitis. Sometimes bile becomes inspissated before the gallbladder ruptures, and spillage of the relatively thick, gelatinous mass into the cranial abdomen causes a localized peritonitis. Adhesions or fistulous tracts occasionally occur around the gallbladder. See p. 583 for a discussion of the preoperative management of animals with bile peritonitis.

Animals with EHBO should be differentiated from those with intrahepatic cholestasis causing partial obstruction; the former rarely needs surgery, but the latter almost never require surgical correction. The indications for exploratory surgery in animals with suspected extrahepatic biliary obstruction are not well defined. Ultrasonographic evidence of EHBO with an increasing serum bilirubin level over 7 to 10 days in the absence of evidence of hepatic disease or pancreatitis can sometimes be an indication for surgery. When a cause cannot be found, pancreatitis is the most likely cause, even if there is no biochemical or ultrasonographic evidence of pancreatitis. In such a case, it is a good idea to repeat ultrasonography and feline pancreatic lipase immunoreactivity testing. Dogs with EHBO due to pancreatitis almost never need surgery, although very rare patients that do not respond to appropriate medical therapy may require a drainage procedure. Percutaneous ultrasound-guided decompression of the gallbladder has been done in dogs with acute pancreatitis, but the value of such is questionable. Aspiration of bile may be required more than once and does not obviate the need for surgery in all cases. A complication seen in a small number of dogs is mild, localized bile leakage from the aspiration site. Readers are referred to medicine texts for an in-depth discussion of the treatment of pancreatitis. Nuclear scintigraphy may aid in the diagnosis of complete extrahepatic biliary obstruction (see Diagnostic Imaging on p. 573).

ANESTHETIC CONSIDERATIONS

Anesthetic requirements and concerns for patients with biliary disease are similar to those for patients with hepatic disease (see

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ABSTRACT

Diseases of the extrahepatic biliary system may be life-threatening, particularly if the infection results in rupture of the gallbladder (e,g., with necrotizing cholecystitis). Prompt and aggressive management of affected animals is necessary. Biliary mucoceles are a common cause of surgically treatable biliary disease in which the prognosis is generally favorable.

KEYWORDS

biliary
gallbladder
surgery
cholelithiasis
peritonitis
cholecystis

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BOX 21.1 Dosage for Vitamin K_1 (AquaMephyton, Mephyton)

0.5–2.0 mg/kg SC[a] q24h

[a]Do not give intravenously or intramuscularly.
SC, Subcutaneous.



FIG. 21.1 Ruptured gallbladder in a dog with necrotizing cholecystitis.

p. 540), and similar complications have been documented in dogs requiring cholecystectomy compared with other surgeries.[2] An additional concern in patients with obstructive biliary disease relates to the effect of mu-agonists (e.g., hydromorphone, morphine) on smooth muscle tone. In human beings with biliary obstruction, these drugs increase both sphincter tone and pain. Mixed agonist-antagonists (e.g., butorphanol; see Tables 12.1 and 13.2) may be preferable as premedicants and analgesics in these patients. After corrective surgery of the biliary obstruction, mu-agonists can be utilized for postoperative analgesia.

ANTIBIOTICS

Prophylactic antibiotics are recommended in patients undergoing biliary surgery because of the detrimental effects of bacterial infection on healing. Antibiotic therapy for biliary infection should be based on the results of culture and susceptibility testing of liver parenchyma or bile or both. Organisms most often isolated from biliary infection are *Escherichia coli, Klebsiella* spp., *Enterobacter* spp., *Proteus* spp., and *Pseudomonas* spp. Antibiotics excreted in active form into the bile, which are commonly used to treat biliary disease, include amoxicillin, cefazolin, and enrofloxacin (Box 21.2).

NOTE Chloramphenicol is metabolized by the liver; do not use it in patients with severe hepatic dysfunction.

SURGICAL ANATOMY

The hepatic and cystic ducts, the bile duct (also called the *common bile duct*), and the gallbladder constitute the extrahepatic biliary

BOX 21.2 Antibiotics Commonly Used in the Treatment of Biliary Disease

Amoxicillin (Amoxi-Tabs, AmoxiDrops, Amoxi-inject)
22 mg/kg PO, IM, SC q8–12h

Cefazolin (Ancef, Kefzol)
22 mg/kg IV, IM, SC q8–12h

Clindamycin (Antirobe)
Dogs: 11–33 mg/kg q12h PO, or 11 mg/kg IV (dilute and give slowly when administering IV)
Cats: 11–33 mg/kg PO, SC q12–24h

Enrofloxacin (Baytril)[a]
Dogs: 7–20 mg/kg PO, IM, IV (diluted and given slowly over 30 min) q24h
Cats: 5 mg/kg PO, IM q24h

Amikacin (Amiglyde)
Dogs: 15–22 mg/kg IV q24h
Cats: 10–14 mg/kg IV q24h

[a]Doses >5 mg/kg may be associated with blindness in cats.
IM, Intramuscular; *IV,* intravenous; *PO,* oral; *SC,* subcutaneous.

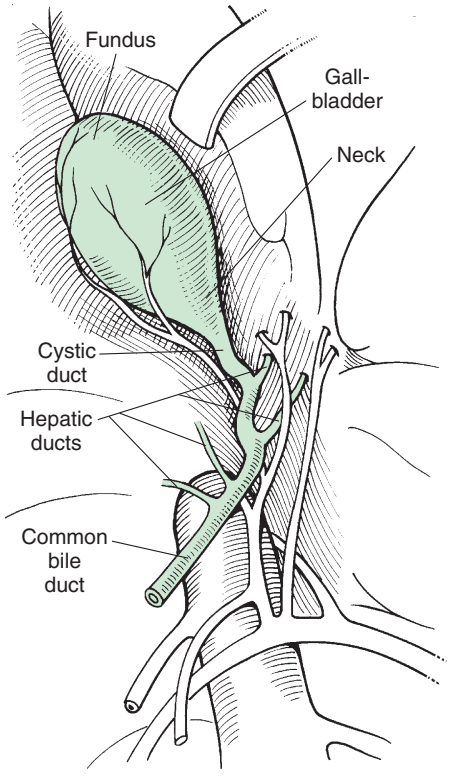


FIG. 21.2 Anatomy of the extrahepatic biliary system.

system (Fig. 21.2). Bile drains from the hepatic ducts into the bile duct and is stored and concentrated in the gallbladder. The gallbladder lies between the quadrate lobe of the liver medially and the right medial lobe laterally. It is a pear-shaped organ that, in medium-sized dogs, holds approximately 15 mL of bile. The rounded end is the fundus. Between the neck of the gallbladder (i.e., the tapering end leading into the cystic duct) and the fundus is the body, or middle portion, of the gallbladder.

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The cystic duct extends from the neck of the gallbladder to the junction with the first tributary from the liver. From this point to the opening of the biliary system into the duodenum, the duct is called the *bile duct.* The bile duct runs through the lesser omentum for approximately 5 cm and enters the mesenteric wall of the duodenum. The bile duct of dogs is generally approximately 3 mm in diameter, whereas that of cats is 2 to 2.5 mm. In contrast, the human bile duct is approximately 10 mm in diameter. The canine bile duct terminates in the duodenum near the opening of the minor pancreatic duct. This combined opening of the minor (accessory) pancreatic duct and the bile duct is the major duodenal papilla. The feline bile duct usually joins the major pancreatic duct before entering the duodenum. Thus cats with intestinal and hepatic disease may be at increased risk for pancreatitis caused by ascending infection.

Diagnostic Imaging

Radiographic findings in patients with extrahepatic biliary obstruction are generally nonspecific. Hepatomegaly and gallbladder distention may be noted. Mineral opacities in the right cranial abdominal quadrant may be indicative of cholecystic calculi (see p. 579); however, most animals with gallstones do not have radiopaque stones, and even when stones are present, they are often incidental findings. Poor serosal detail in the upper right quadrant or diffusely throughout the abdomen may be found in animals with or without gallbladder rupture, but many dogs without rupture have decreased abdominal detail locally or diffusely. Ultrasonography can be specific for ruptured gallbladder, but it is rather insensitive. Retrograde administration of contrast into the bile duct with duodenoscopy can be performed but is challenging.[3,4]

Ultrasound evaluation sometimes helps distinguish intrahepatic from extrahepatic causes of biliary obstruction. Identification of severe pancreatitis or a mass in the region of the pancreas may raise suspicion of EHBO. With experimental bile duct ligation, the gallbladder becomes distended within 24 hours, and the extrahepatic bile ducts distend within 48 to 72 hours. Intrahepatic ducts take longer to dilate (approximately a week). Remember that dilation from a previous obstructive episode may not resolve; thus it may be difficult to discern whether the present dilation is due to recent or past disease.

Hepatobiliary scintigraphy has been used to assess the patency of the bile duct. With ultrasound, it may be difficult to discern whether dilation of the common bile duct in the absence of an obvious obstruction (e.g., calculus, mass) is associated with complete or partial biliary obstruction. The usefulness of serum biochemical values and scintigraphic studies for differentiating animals with complete EHBO from those with partial obstruction is questionable. Those that do not have intestinal radioactivity within 3 to 4 hours should have additional imaging performed (up to 24 hours) to determine whether they have complete EHBO. Those that have partial obstructions have delayed arrival of radioactivity and less radioactivity in the intestine. Reevaluating these animals at 24 hours may lead to greater specificity. Hepatobiliary scintigraphy appears to be an insensitive test for structural hepatobiliary disease (e.g., cholangiohepatitis).

Computed tomography and magnetic resonance imaging have been evaluated in animals with cholangitis and pancreatitis.[5,6] The biliary tract is hyperintense on T2-weighted images and hypointense on T1-weighted images. Abnormalities include hyperintensity, gallbladder wall enhancement, and increased wall thickness. Further investigations are needed to fully elucidate the utility of advanced imaging of the biliary tract.

SURGICAL TECHNIQUES

Exploratory laparotomy should be performed when leakage of bile into the abdomen is suspected, and when biliary obstruction appears to be caused by neoplasia (biliary tract, intestinal, or pancreatic), biliary calculi, or parasitic disease. During exploration, patency of the common bile duct must be ensured by manually expressing the gallbladder or by catheterizing the duct, either retrograde (i.e., from the duodenum; see later) or in some cases normograde (i.e., from the gallbladder).

The treatment of animals with EHBO secondary to pancreatitis consists of aggressive medical management. If clinical or laboratory improvement (not necessarily resolution) is not seen within 10 to 14 days of initiation of appropriate therapy, or if clinical deterioration occurs despite excellent medical therapy, cholecystoduodenostomy or cholecystojejunostomy may be considered despite the high morbidity/mortality associated with the procedure. In extremely ill patients with biliary obstruction that cannot undergo surgical exploration, temporary decompression of the gallbladder may be warranted using ultrasound-guided aspiration, choledochal tube (bile duct) stenting, or a Foley or self-retaining accordion catheter.

Choledochal Tube Stenting

Stenting of the bile duct may be considered in cases of reversible disease processes (e.g., pancreatitis, inflammatory lesions of the biliary tract of duodenum) to allow stabilization before definitive surgical treatment, and for augmentation of bile duct repair (see later under Repair of Common Bile Duct Injuries). Chronic stenting may be associated with ascending infection.

After abdominal exploration, perform a longitudinal duodenotomy of the antimesenteric border of the duodenum over the major duodenal papilla. Flush the biliary tract as necessary, and treat primary disease (cholecystotomy, cholecystectomy, biliary duct repair). Pass a 5 to 10 cm fenestrated segment of red rubber catheter up the common bile duct via the duodenal papilla so that approximately one-half of the stent lies within the common bile duct and the other half resides within the duodenal lumen. Suture the stent to the submucosa of the duodenum aboral to the duodenal papilla using absorbable suture. Stents should be passed in the feces in 1 to 11 months or can be removed endoscopically if inflammation or infection is suspected.

Cholecystotomy

Cholecystotomy is rarely indicated to remove some choleliths (see p. 579), or when the contents of the gallbladder are inspissated and cannot be aspirated into a syringe. However, in almost all cases, cholecystectomy, which has a lower morbidity/mortality rate, is the preferred technique.

Pack the area surrounding the gallbladder with sterile, moistened laparotomy sponges. Place stay sutures in the gallbladder to facilitate manipulation and reduce spillage. Make an incision in the fundus of the gallbladder (Fig. 21.3). Remove the gallbladder contents and submit for culture. Lavage the gallbladder with warmed, sterile saline. Catheterize the common bile duct via the cystic duct with a 3.5- or 5-Fr soft catheter, and flush it to ensure patency. Close the incision with a one- or two-layer inverting suture pattern using absorbable suture (3-0 to 5-0).

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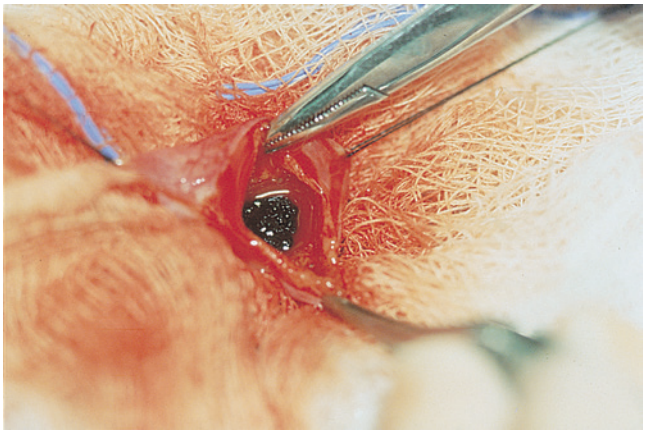


FIG. 21.3 Before cholecystotomy, place stay sutures in the gallbladder to facilitate manipulation and reduce spillage. Then make an incision in the fundus of the gallbladder.

Laparoscopic-Assisted Cholecystostomy

Laparoscopic-assisted short-term (temporary) biliary diversion has been described in dogs.[7] Definitive surgery was recommended when the catheter was removed to prevent biliary leakage, because maturation of the tract between the gallbladder and body wall did not occur in the report within the 25-day period of the study.

Position the dog in dorsal recumbency and place an endoscope port 1 cm cranial to the umbilicus. Place a second port 4 cm caudal and lateral to the xiphoid process. Grasp and stabilize the gallbladder with Babcock forceps, and pass a pigtail catheter via a separate stab incision to the right of the xiphoid process. Insert the catheter into the gallbladder; secure the locking mechanism, and suture the catheter to the skin. Positive contrast cholecystography may be performed to evaluate placement.

Cholecystectomy

Cholecystitis that is not responsive to or that relapses after antibiotics, biliary mucocele, spontaneous rupture, and cholelithiasis that is associated with disease (many cases are asymptomatic) are best treated by cholecystectomy Cholecystectomy may also be indicated for primary neoplasia or traumatic rupture of the gallbladder.

Expose the gallbladder and use Metzenbaum scissors to incise the visceral peritoneum along the junction of the gallbladder and the liver (Fig. 21.4A). Apply gentle traction to the gallbladder and use blunt dissection to free it from the liver. Free the cystic duct to its junction with the common bile duct. Be sure to identify the common bile duct and avoid damaging it during the procedure. If necessary, identify the common bile duct by placing a 3.5- or 5-Fr soft catheter into the duct via the duodenal papilla. Make a small enterotomy in the proximal duodenum, locate the duodenal papilla, and place a small red rubber tube into the common bile duct (see Fig. 21.4B). Flush the duct to ensure its patency. Clamp and double ligate the cystic duct and cystic artery (see Fig. 21.4C) with nonabsorbable suture (2-0 to 4-0). Sever the duct distal to the ligatures and remove the gallbladder. Submit a portion of the wall, plus bile, for culture if infection is suspected. Submit the remainder of the gallbladder for histologic analysis if indicated (for cholecystitis or neoplasia). Close the duodenal incision in a simple interrupted pattern with absorbable suture (2-0 to 4-0).

NOTE Be sure that the common bile duct is patent before performing a cholecystectomy. The animal must have a pathway for biliary drainage into the intestinal tract.

Laparoscopic Cholecystectomy

Laparoscopic cholecystectomy has been performed in dogs with uncomplicated gallbladder mucoceles, cholecystitis, or mucosal cystic hyperplasia.[8] Cases should be carefully chosen and should have no biochemical or diagnostic imaging evidence of biliary tract obstruction or rupture.

Establish an endoscope port approximately 1 cm caudal to the umbilicus and explore the abdomen. Triangulate three additional ports; place them under direct visualization. Place one port on the left, 3 to 5 cm cranial and 5 to 8 cm lateral to the umbilicus. Place two ports on the right, 3 to 5 cm and 5 to 8 cm lateral to the umbilicus. A single incision, multichannel laparoscopic port for passage of multiple instruments can be placed in the caudal abdomen, and articulated instruments used for the procedure. *Retract the gallbladder with a fan retractor to expose the cystic duct (Fig. 21.5). Dissect the cystic duct with curved and right-angle dissecting forceps. Ligate the cystic duct with three extracorporeally tied modified Roeder knots using 0 or 2-0 polydioxanone (PDS) and transect the duct with scissors, leaving two of the ligatures with the portion of the duct remaining in the patient. Alternatively, clip the cystic duct twice with endoscopic clip appliers, transect the cystic duct between clips with scissors, and ligate the portion of the duct remaining in the abdomen with a pretied loop ligature. Grasp the duct or suture tail of the portion of the cystic duct to be removed with the gallbladder. Retract the duct and gallbladder, and dissect them from the fossa using a harmonic scalpel. Place the gallbladder in a specimen retrieval bag and partially retract it through one of the operating ports. Aspirate bile until the bag and the gallbladder can be extracted. Evaluate the gallbladder fossa and ligation site for hemorrhage and leakage, respectively, and collect samples of liver for histopathologic examination, culture and susceptibility testing, and metal analysis as needed.*

Choledochotomy

Direct incision of the bile duct should be performed only in animals in which the duct is markedly dilated, such as those with chronic obstruction, and in which the obstruction can be removed (e.g., choledocholithiasis, biliary sludge). An attempt should be made first to remove the obstruction by flushing the common bile duct using a catheter placed by means of an enterotomy or by cholecystotomy. Extraluminal obstruction or stricture of the duct is best treated with biliary diversion techniques (see later).

Pack the area surrounding the common bile duct with sterile, moistened laparotomy sponges. Place traction sutures in the distended duct. Make a small incision in the duct and remove the obstruction (Fig. 21.6). Flush the duct with copious amounts of warmed, sterile saline, and pass a 3.5-to 5-Fr soft catheter into the gallbladder and duodenum to ensure patency. Close the incision in a simple continuous or simple interrupted pattern with absorbable suture (4-0 or 5-0). If leakage is a concern, pass a catheter into the duct through an incision in the proximal duodenum (see previously). Treat small leaks by stenting the incision with a 3.5-to 5-Fr soft catheter (see discussion on repairing common bile duct injuries).

Bile Flow Diversion

Bile flow diversion is indicated if medical therapy cannot resolve obstruction of the common bile duct in a timely fashion, or if

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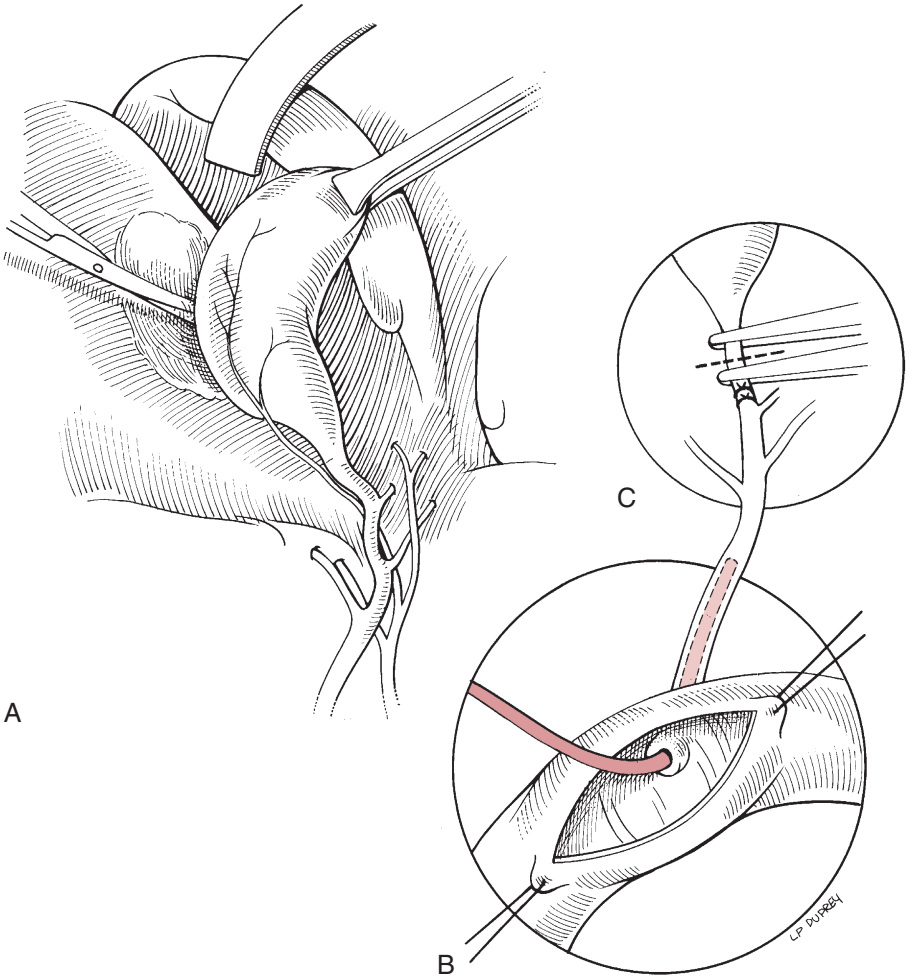


FIG. 21.4 Cholecystectomy. (A) Expose the gallbladder, then use Metzenbaum scissors to incise the visceral peritoneum along the junction of the gallbladder and the liver. (B) Identify the common bile duct; take care not to damage it during the procedure. If necessary, cannulate the duct via the duodenal papilla. (C) Clamp and double ligate the cystic duct and cystic artery.

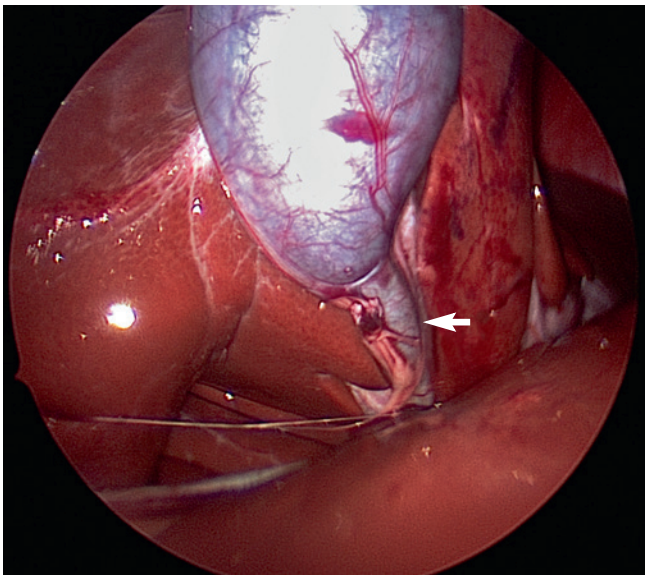


FIG. 21.5 Laparoscopic appearance of the gallbladder just before dissection, which begins at the cystic duct *(arrow)*.

the duct has been severely traumatized and the gallbladder is not directly involved in the disease process. Hepatic changes due to EHBO are typically reversible unless obstruction persists for 5 to 6 weeks, by which time irreversible changes such as biliary cirrhosis may develop. Cholecystoenterostomy (i.e., cholecystojejunostomy or cholecystoduodenostomy) is preferred over choledochoduodenostomy in dogs and cats because the small size of the common bile duct in these species often makes the latter procedure difficult to perform successfully. If cholecystojejunostomy is performed, the proximal jejunum should be used to reduce the incidence of postoperative maldigestion of lipids. In dogs, it has been recommended that the stoma between the bowel and the gallbladder should be at least 2.5 cm long to minimize the potential for obstruction of bile flow or retention of bowel contents in the gallbladder. Making the stoma too small is more apt to result in ascending or chronic cholecystitis than is making the stoma too large.

NOTE If possible, avoid biliary diversion in dogs with pancreatitis. Almost all affected animals improve with medical management, making this potentially dangerous technique unnecessary.

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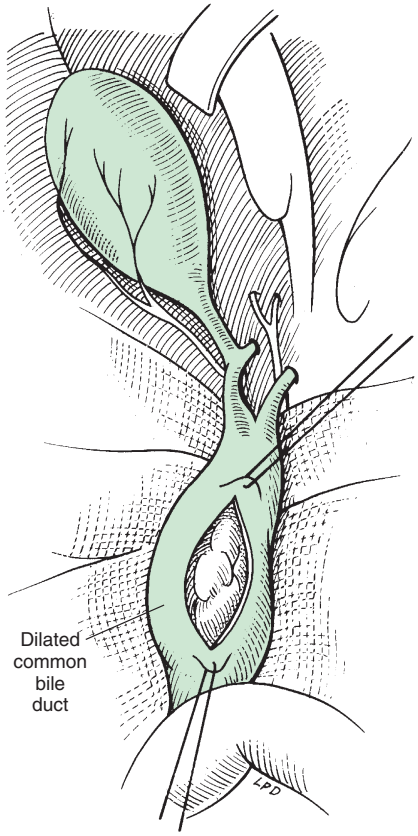


FIG. 21.6 Choledochotomy.

Mobilize the gallbladder from the liver as described for cholecystectomy. Place stay sutures approximately 3 cm apart in the gallbladder. Bring the gallbladder into apposition with the antimesenteric surface of the descending duodenum so that little or no tension is exerted on the gallbladder or intestine. Pack the area surrounding the gallbladder and duodenum with sterile, moistened laparotomy sponges. Place a continuous suture of absorbable suture material (2-0 to 4-0) between the serosa of the gallbladder and the serosa of the duodenum, near the mesentery (referred to as the original suture line; Fig. 21.7A). Make the suture line 3 to 4 cm long. Leave the ends of the suture long and use them to manipulate the intestine and the gallbladder. Drain the gallbladder and make a 2.5- to 3-cm incision in it, parallel to the preplaced suture line (Fig. 21.7B). Have an assistant occlude the duodenum proximal and distal to the proposed incision site. Make a similar parallel incision in the antimesenteric surface of the duodenum (Fig. 21.7C). Place a continuous suture line of absorbable suture material (2-0 to 4-0) from the mucosa of the gallbladder to the mucosa of the duodenum, beginning with the edges closest to the original suture line (Fig. 21.7D). Then use the same suture material to suture the mucosal edges of the stoma farthest from the original suture line (Fig. 21.7E). Complete the stoma by suturing the serosal edges of the gallbladder and intestine over the near side of the stoma (i.e., the side farthest from the original suture line; Fig. 21.7F).

NOTE If you cannot identify the mucosa of the gallbladder and suture it as a separate layer, perform a two-layer anastomosis of the far side, followed by a two-layer anastomosis of the near side.

Repair of Common Bile Duct Injuries

The surgical technique used to repair lacerations of the common bile duct depends on the location and severity of the lesion. Severely damaged ducts are difficult to repair primarily, particularly with bile leakage or adhesion formation. Incisional dehiscence, leakage, and stricture formation are common. If the injury is distal to the entrance of the hepatic ducts, the bile duct should be ligated proximal and distal to the injury, and biliary diversion (i.e., cholecystoduodenostomy or cholecystojejunostomy; see earlier) performed. If the duct has been cleanly severed and the luminal diameter is greater than 4 to 5 mm (which is seldom), primary suturing and anastomosis are possible. Similarly, proximal lacerations or perforations may be treated with primary suturing. The mucosa of the bile duct should be accurately reapposed. Small sutures should be used and tension on the suture line avoided. The use of stenting catheters in the bile duct is controversial, but temporary bile diversion may allow healing of bile duct injuries that would otherwise dehisce, leak, or form a stricture (see earlier). The tube acts to decompress the biliary tree and minimizes bile leakage from the site during healing. Disadvantages of tubes placed in the bile duct include greater risk of stricture formation because of the presence of a foreign body at the injury site, obstruction of the tube, and ascending infection. If the bile duct is stented, a soft tube that is smaller than the diameter of the duct should be used to minimize irritation to the duct wall. The use of rubber tubes or catheters that enter the duodenum and T-tubes that exit the duct and are exteriorized through the abdominal wall has been described in the veterinary literature. The use of a straight catheter (e.g., Sovereign feeding tube, Dover red rubber Robinson catheter) has been described here.

Identification of the common bile duct may be facilitated by passing a catheter into the duct from the duodenum (see previous discussion of cholecystectomy). *Be careful not to interfere with the blood supply to the duct during manipulation. Carefully debride the transected ends of the duct, but be sure to leave adequate duct length to avoid tension on the suture line when the ends are reapposed. Reappose the ends of the duct in a simple interrupted pattern using absorbable suture (4-0 to 6-0). Place a 3.5- to 5-Fr soft catheter in the duct from the duodenum to stent the suture line (Fig. 21.8). Suture the distal end of the catheter to the duodenal lumen with small chromic gut suture (3-0 or 4-0). As the suture dissolves, peristalsis will cause the catheter to enter the intestinal lumen, where it will pass in the feces.*

HEALING OF THE BILIARY TRACT

Studies have shown that if just a small strip of the common bile duct wall remains intact, the duct regenerates. However, longitudinal tension on the suture line of a repaired biliary duct causes severe stenosis. In addition to promoting stricture of the duct, there is some suggestion that intraluminal tubes may interfere with normal biliary drainage, thus promoting cholangitis. Because of uncertainties regarding healing of the duct in the presence of infection, leakage, or tension, drainage procedures such as cholecystojejunostomy, rather than direct repair of the common bile duct (see earlier), are commonly performed.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Absorbable suture material should be used when suturing the gallbladder or bile duct because nonabsorbable suture may act

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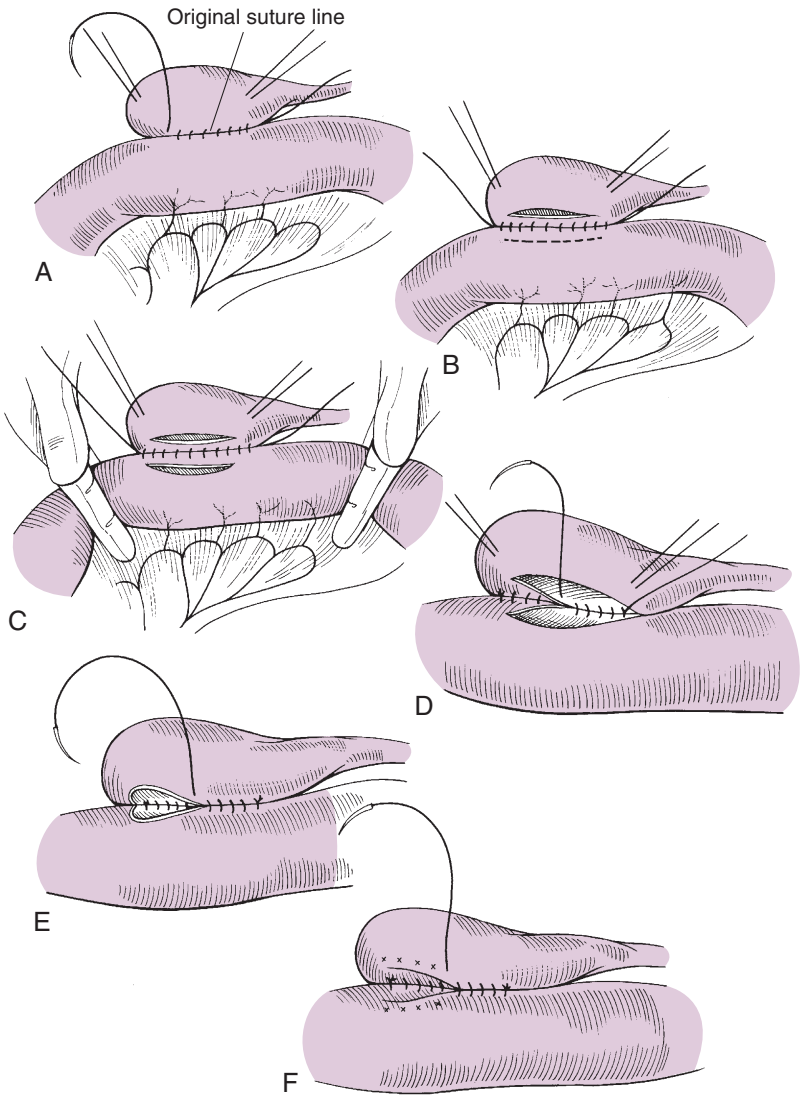


FIG. 21.7 For cholecystoduodenostomy (or cholecystojejunostomy), bring the gallbladder into apposition with the antimesenteric surface of the descending duodenum. (A) Place a 3- to 4-cm continuous suture line between the serosa of the gallbladder and the serosa of the duodenum (original suture line). (B) Drain the gallbladder and make a 2.5- to 3-cm incision in it, parallel to the preplaced suture line. (C) Have an assistant occlude the duodenum proximal and distal to the proposed incision site, and make a parallel incision in the antimesenteric surface of the duodenum. (D) Place a continuous suture line from the mucosa of the gallbladder to the mucosa of the duodenum, beginning with the edges closest to the original suture line. (E) Suture the mucosal edges of the stoma farthest from the original suture line. (F) Complete the stoma by suturing the serosal edges of the gallbladder and intestine over the near side of the stoma.

as a nidus for stone formation. Biliary duct surgery is aided by the use of small instruments such as those used for ophthalmic surgery. With biliary diversion surgery, the gallbladder should be emptied with a syringe and needle, or a needle attached to suction before surgical manipulation, to reduce spillage of bile during the procedure.

POSTOPERATIVE CARE AND ASSESSMENT

Fluid therapy should be continued until the animal is able to maintain hydration with oral fluids. Electrolytes and acid-base status should be assessed and corrected during the postoperative period. Many patients with bile peritonitis are debilitated before surgery, and nutritional supplementation and temporary biliary diversion may be beneficial (see Chapter 10). Antibiotic therapy should be continued for 7 to 10 days if cholecystitis was present, or if bile leakage occurred before or during surgery. Open abdominal drainage may be considered in patients with generalized bile peritonitis. For postoperative analgesia of patients with bile peritonitis see Table 13.2. Biliary diversion surgery in cats is commonly associated with intraoperative and postoperative hypotension and anemia, often significant enough to warrant therapy. Careful monitoring of these cases during surgery is strongly recommended.

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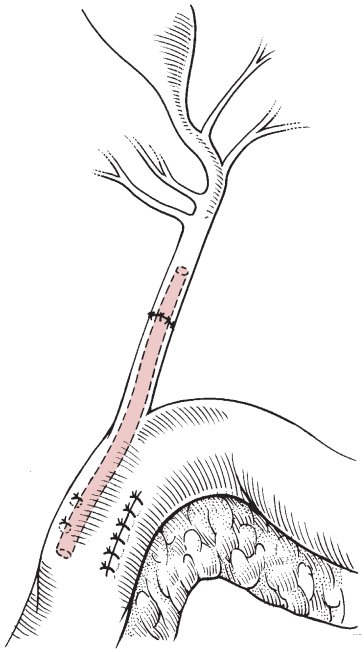


FIG. 21.8 Lacerations in the common bile duct may be sutured over a stent placed by means of a duodenotomy.

PROGNOSIS

Biliary surgery in dogs is associated with a mortality of approximately 15% to 25%; those surviving the immediate postoperative period often have good long-term outcomes, although infection is common when cholecystoenterostomy is performed. Biliary diversion techniques have a worse prognosis than cholecystectomy. Development of postoperative dyspnea and hypotension may be associated with decreased survival.

The prognosis for cats with EHBO is guarded, regardless of whether neoplasia (pancreatic or biliary) or nonneoplastic disease is present; however, median survival time of cats with inflammatory disease is significantly longer than that of cats with neoplasia. Biliary diversion in cats may be associated with high early mortality, and surviving cats may have chronic vomiting and anorexia. Vomiting is typically transient in nature and is responsive to antibiotics. Thus biliary diversion should be avoided unless no other viable alternatives exist. The prognosis after biliary diversion in dogs is also guarded, and bleeding at the stoma site and ascending infections are known to occur.

COMPLICATIONS

Surgery of the extrahepatic biliary tree requires technical competence, manual dexterity, and sound surgical judgment to prevent serious complications. Potential complications after cholecystectomy (particularly if perforation was present) include generalized peritonitis, shock, sepsis, hypoglycemia, hypoproteinemia, and hypokalemia. Close monitoring of clinical signs, blood pressure, complete blood count, biochemical profile, and coagulation tests during the postoperative period should be done to allow diagnosis and treatment at the earliest sign of sepsis or systemic inflammatory response syndrome. Stricture, bile leakage, and dehiscence may occur after surgery of the common bile duct. Ascending cholangiohepatitis may occur in some animals after biliary diversion, particularly if the stoma of the enteric-biliary anastomosis is too small and intestinal contents remain in the gallbladder lumen for prolonged periods. Intermittent antibiotic therapy may be necessary in such animals. Long-term complications after biliary decompression include cholangiohepatitis, recurrence of obstruction, and chronic weight loss.

In one study, conversion to open surgery during laparoscopic cholecystectomy occurred in nearly one-third of cases due to identification of gallbladder rupture, large or abnormal cystic duct anatomy, leakage during dissection, or difficulty with visualization.[7]

SPECIAL AGE CONSIDERATIONS

Trauma should be suspected in young animals with bile peritonitis. Obstruction that occurs secondary to pancreatitis or neoplasia is more common in middle-aged or older animals.

SPECIFIC DISEASES

CHOLELITHIASIS

DEFINITIONS

Calculi found in the gallbladder are **choleliths;** those found in the common bile duct are **choledocholiths.** Calculi are often called *gallstones.*

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Choleliths are often fortuitous findings at necropsy or during imaging with radiographs or ultrasound. They are frequently clinically silent; however, they may be associated with cholecystitis and/or cholangiohepatitis, vomiting, anorexia, icterus, fever, or abdominal pain. Whereas human beings usually develop dietary-induced cholesterol gallstones, cholesterol, bilirubin, and mixed stones have been reported in dogs and cats. The relative rarity of canine cholelithiasis may be due to (1) decreased concentrations of cholesterol in canine bile, (2) absorption of ionized calcium from the gallbladder, limiting the amount of free ionized calcium in bile, and (3) failure to recognize choleliths. Calcium salts are the major components of pigment gallstones; therefore the availability of ionized calcium may be important in gallstone formation in dogs. Pigment gallstones can be experimentally produced in dogs after 6 weeks of a methionine-deficient diet or with a high-cholesterol diet that is deficient in taurine. Pigment gallstones may also form secondary to bilirubin overproduction during hemolysis. Calcium carbonate and mixed choleliths (calcium carbonate, calcium bilirubinate, and cholesterol) are the most common types found in cats.

NOTE Most choleliths are asymptomatic; treatment is indicated only when the calculi are associated with clinical signs.

DIAGNOSIS

Clinical Presentation

Signalment

Aged female small-breed dogs appear to be at increased risk for the development of choleliths. Middle-aged to older male cats may be more commonly affected.

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History

Most animals with choleliths are asymptomatic; however, they may be presented for treatment of fever, vomiting, icterus, or abdominal pain if cholecystitis or biliary obstruction occurs. Clinical signs may be mild and intermittent in some animals. The most common clinical signs in cats ill because of cholelithiasis are progressive vomiting, dehydration, anorexia, icterus, and lethargy.

Physical Examination Findings

Icterus may be noted if the calculus causes biliary obstruction or ascending cholangitis. Abdominal pain and vomiting may also occur. In rare cases, choleliths have been associated with perforation of the gallbladder or common bile duct (see p. 582).

Diagnostic Imaging

Gallstones are seldom radio-opaque (Fig. 21.9), but they are readily identified by ultrasound. A hyperechoic focus with acoustic shadowing originating from the lumen of the gallbladder may be noted (Fig. 21.10). If obstruction is present, dilation of the common bile duct or intrahepatic ducts may also be detected. Contrast radiographs of the biliary tree are rarely useful in icteric patients. Endoscopic retrograde pancreatography is technically difficult and is rarely performed in dogs or cats. Direct injection of contrast into dilated bile ducts via transabdominal placement of a "slim" needle has been accomplished in people but is seldom done in dogs.

Laboratory Findings

Abnormalities are uncommon; however, clinically ill animals typically have abnormalities compatible with EHBO. Dogs typically have a marked increase in serum alkaline phosphatase and cholesterol, along with some increase in alanine aminotransferase (ALT). Hyperbilirubinemia is typical in partial or complete obstruction and when ascending cholangitis occurs. Coagulation profiles are typically normal. Cats are similar but have lesser elevations of serum alkaline phosphataste than dogs and may have marked increases in ALT. Urinalysis may be especially helpful in cats because bilirubinuria is always abnormal in that species and often presages hyperbilirubinemia.

DIFFERENTIAL DIAGNOSIS

Evidence of concurrent cholecystitis should be sought in symptomatic animals with choleliths. Sludge and true concretions may be difficult to differentiate in some animals before surgery. Ultrasonographically guided, percutaneous cholecystocentesis with cytology and culture of bile appears to be the most sensitive test for cholecystitis.

MEDICAL MANAGEMENT

Medical dissolution of gallstones in dogs and cats has not been reported and is probably not reliable because of the expected content of most gallstones. Medical management of animals with biliary obstruction is discussed on p. 571. Concurrent cholecystitis should be treated with appropriate antibiotics.

SURGICAL TREATMENT

Because choleliths may be associated with cholecystitis and may cause vomiting, anorexia, icterus, fever, or abdominal pain, they should be removed if they are found in a patient with biliary tract disease.

Preoperative Management

See p. 571 for the preoperative management of patients with biliary obstruction. The bacteria isolated from animals with choleliths are typically sensitive to aminoglycosides and fluoroquinolones.

Anesthesia

See pp. 540 and 571 for discussions of the anesthetic management of patients with hepatic and obstructive biliary disease, respectively.

Surgical Anatomy

Surgical anatomy of the biliary tract is described on p. 572.

Positioning

Choleliths are generally removed via a cranial midline abdominal incision.



FIG. 21.9 Lateral abdominal radiograph in a dog with radiopaque choleliths *(arrows)*.

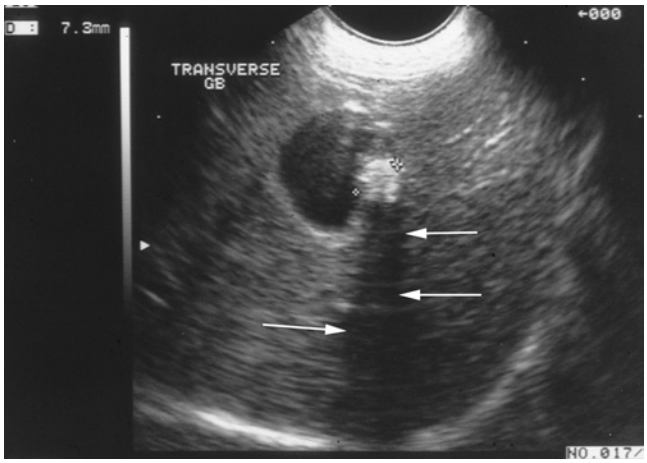


FIG. 21.10 Abdominal ultrasound showing a calculus in the lumen of the gallbladder. Note that the calculus is hyperechoic and casts an acoustic shadow *(arrows)*.

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SURGICAL TECHNIQUE

Cholecystectomy (see p. 574) is the surgical treatment of choice in dogs with clinical signs that occur secondary to cholelithiasis. If stones are also present in the common bile duct, the duct can be catheterized via the duodenum and the stones flushed into the gallbladder (see p. 574). As an alternative, if the bile duct is enlarged, the duct can be incised (choledochotomy) and the stones removed directly; however, care must be taken when suturing the common bile duct to avoid stricture formation (see p. 574). Clinical studies have found a greatly increased mortality in human patients with cholelithiasis when choledochotomy is performed rather than cholecystectomy. Cholecystoenterostomy (see p. 574) should be performed when ductal obstruction cannot be relieved. Bile should be cultured.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

The gallbladder and the common bile duct should be sutured with absorbable suture material to reduce the likelihood of suture serving as a nidus for calculi formation.

POSTOPERATIVE CARE AND ASSESSMENT

See p. 577 for the postoperative management of patients with obstructive biliary disorders. For cholangitis or cholangiohepatitis, aggressive antibiotic therapy plus antioxidant therapy (e.g., *S*-adenosyl-L-methionine, Silybin) is indicated. Ursodeoxycholic acid (15 mg/kg given orally once a day) will also help protect hepatocyte membranes but should be used only if there is no obstruction.

PROGNOSIS

The prognosis is excellent when ductal obstruction can be relieved and a cholecystectomy is performed.

GALLBLADDER MUCOCELES

DEFINITIONS

A **gallbladder mucocele** is a mucus-filled dilation or distention of the gallbladder that is associated with dysfunction of mucus-secreting cells within the gallbladder mucosa. The gallbladder contents are so thick that they cannot be excreted out the bile duct (Video 21.1).

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Gallbladder mucoceles are second only to pancreatitis as the most common cause of extrahepatic biliary obstruction in dogs. They are probably the most common diagnosis resulting in the need for biliary surgery. They are rarely reported in cats. Mucoceles are characterized histologically by hyperplasia of mucus-secreting glands within the gallbladder mucosa, resulting in an abnormal accumulation of thick mucus within its lumen. They may be caused by obstruction of the common bile duct, or they may be associated with a functional obstruction due to altered gallbladder wall contractility. Hyperadrenocorticism, hypothyroidism, excessive androgens, and abnormal lipid metabolism appear to be risk factors for mucoceles. The pathophysiology of the association is unknown, but some theorize that hypertriglyceridemia or hypercholesterolemia may be associated with their formation in these dogs owing to augmentation of dyslipidemia. EHBO may occur secondarily if mucus extends into the common bile ducts and/or the cystic and hepatic ducts. If intraluminal pressure from the thickened contents compromises perfusion of the gallbladder wall, rupture may occur, with subsequent bile peritonitis (see p. 581) or a modified transudative effusion. Cholecystitis or necrotizing cholecystitis has been suspected as a predisposing cause; however, histopathology does not typically indicate an inflammatory or bacterial origin in most animals.

DIAGNOSIS

Clinical Presentation

Signalment

Aged female and male dogs are most commonly affected. Shetland sheepdogs, cocker spaniels, Scottish terriers, and miniature schnauzers may be predisposed to gallbladder disorders and biliary mucoceles.

History

Some animals with gallbladder mucoceles are asymptomatic; however, they may be presented for signs of systemic illness of several days' to several weeks' duration. Clinical signs in animals that are not obstructed are nonspecific and may include vomiting, anorexia, lethargy, polyuria and polydipsia, fever, diarrhea, and abdominal distention.

Physical Examination Findings

Physical examination findings include signs of abdominal discomfort on palpation, depression, icterus, tachypnea, fever, and/or tachycardia. Abdominal distention may also be noted if gallbladder rupture has occurred.

Diagnostic Imaging

Abdominal radiographs are not helpful in diagnosing gallbladder mucoceles; however, they may indicate the presence of abdominal fluid. Mature gallbladder mucoceles have a classic appearance on ultrasound. Typically, ultrasound reveals echogenic material within the lumen of the gallbladder, which is not gravity dependent and has a classic stellate or "kiwi fruit" appearance (Fig. 21.11). Classification of mucoceles based on ultrasound has been reported as follows: type 1, echogenic immobile bile occupying the gallbladder; type 2, incomplete stellate pattern; type 3, typical stellate pattern; type 4, kiwi fruit–like pattern and stellate combination; type 5, kiwi fruit–like pattern with residual central echogenic bile; type 6,

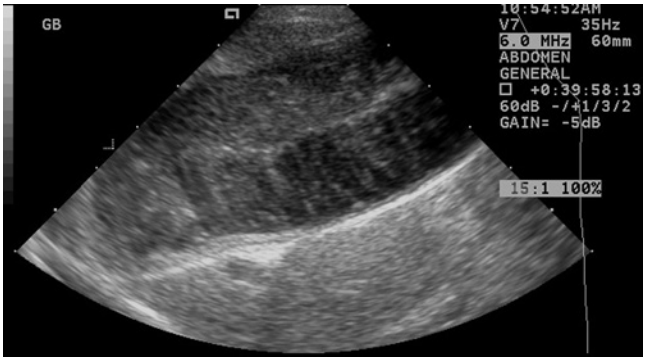


FIG. 21.11 Ultrasound image of a gallbladder mucocele. Note the typical hyperechoic striations within the lumen of the gallbladder ("kiwi fruit" appearance).

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kiwi fruit–like pattern without central echogenic bile.[9] The gallbladder may or may not appear distended, and the wall may or may not appear thickened. Gallbladder rupture may be suspected on ultrasonography based on the finding of free abdominal fluid, lack of continuity of the gallbladder wall, and the presence of hyperechoic cranial abdominal fat. A trilaminated appearance is also suggested as indicating rupture.[9] However, ultrasonography may not be sensitive for detecting the severity of disease in affected animals, and focal peritonitis and adhesions may go undetected. One study showed that contrast-enhanced ultrasound evaluation was 100% sensitive and specific for necrosis and rupture compared with 75% sensitivity and 81% specificity of traditional ultrasound.[10] Sulfurhexafluoride echo-signal enhancer has been given intravenously followed by saline and resulted in a thick, double-rim enhancement in the intact gallbladder and absence of contrast enhancement in cases of necrosis/rupture. Gallbladder motility has been assessed ultrasonographically, and hypomotility has been suggested to be a risk factor for mucocele formation.

Laboratory Findings

Common abnormalities include elevated activities of ALT, alkaline phosphatase (ALP), aspartate aminotransferase, and γ-glutamyltransferase. Serum total bilirubin is inconsistently elevated in affected animals. Leukocytosis, neutrophilia, and monocytosis may be seen on hematologic examination. Elevations of ALP, aspartate aminotransferase, γ-glutamyltransferase, and serum bilirubin are typically higher in dogs with gallbladder rupture than in those without. High venous lactate levels may also indicate biliary rupture. If warranted by concurrent clinical signs, testing for hypothyroidism and hyperadrenocorticism is recommended.

DIFFERENTIAL DIAGNOSIS

Definitive documentation of a gallbladder mucocele may require surgery. Other conditions causing partial or complete EHBO should be considered.

MEDICAL MANAGEMENT

Medical management (e.g., ursodeoxycholic acid, *S*-adenosyl-L-methionine) should be considered in dogs that are asymptomatic. Exploratory celiotomy and cholecystectomy are generally recommended in symptomatic animals. Because gallbladder rupture is common in affected animals, serial examinations should be performed in animals managed medically, and surgical intervention should be considered if biliary obstruction is evident. Asymptomatic dogs treated medically and reevaluated remained static (4 of 7), improved (2 of 7), or had asymptomatic bile duct dilation (1 of 7) in one study.[9]

SURGICAL TREATMENT

Cholecystectomy or cholecystoenterostomy should be performed and cultures of the gallbladder and/or peritoneal fluid submitted for susceptibility testing (Video 21.2).

Preoperative Management

See p. 571 for the preoperative management of patients with biliary obstruction. If gallbladder rupture is present, the animal may be debilitated; prompt and aggressive surgery is indicated. Perioperative antibiotics are indicated, although they may be delayed in some cases until after cultures have been obtained. The bacteria most commonly isolated from the abdomen of dogs with gallbladder mucoceles are *Streptococcus* and *E. coli;* however, negative cultures are common.

Anesthesia

See pp. 540 and 571 for discussions of the anesthetic management of patients with hepatic and obstructive biliary disease, respectively.

Surgical Anatomy

Surgical anatomy of the biliary tract is described on p. 572.

Positioning

Cholecystectomy and cholecystoenterostomy are performed via a cranial midline abdominal incision.

SURGICAL TECHNIQUE

Cholecystectomy (see p. 574) is the surgical treatment of choice in animals with ruptured gallbladders, particularly when the gallbladder wall is nonviable. In animals with a viable gallbladder, cholecystoenterostomy may be considered. Surgery is indicated on an emergency basis in dogs with gallbladder rupture, and those with an intact gallbladder can usually have surgery performed on an elective basis. Hepatic biopsies (see p. 546) are indicated.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Nonabsorbable monofilament suture should be used on the cystic duct during cholecystectomy (see p. 576).

POSTOPERATIVE CARE AND ASSESSMENT

See p. 577 for the postoperative management of patients with obstructive biliary disorders. For information on bile peritonitis, see p. 583.

PROGNOSIS

The prognosis is excellent for animals that survive the postoperative period (73%) with one study showing a mean survival time of 19 months.[11] These dogs typically have complete resolution of their clinical signs and no recurrence. Prompt surgical intervention in dogs with gallbladder rupture typically results in a good or excellent outcome; however, if bile peritonitis is severe, a high mortality can be expected (see p. 584). In the aforementioned study, 3 of 43 (7%) dogs died within 2 weeks of cholecystectomy. Two of these dogs died during hospitalization: one related to multiple organ failure and pulmonary thromboembolism and one with hypotension and anemia. A third dog was euthanized after release from the hospital due to recurrence of signs.[11] Seven dogs lived more than 2 weeks after surgery but less than 2 months; four of these dogs were euthanized because of presumptive liver disease. Postoperative elevation of lactate and hypotension negatively affected survival; however, presence of gallbladder rupture did not affect outcome.

BILE PERITONITIS

DEFINITION

Bile peritonitis is an inflammation of the peritoneum caused by leakage of bile into the abdomen. This condition has also been called *bilious ascites*.

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GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Acute abdomen (i.e., shock or pain or both caused by severe abdominal disease) may be caused by leakage of bile into the abdominal cavity, particularly with concurrent septic peritonitis. Leakage of bile into the abdominal cavity may occur with traumatic rupture of any portion of the extrahepatic biliary tree or may occur secondary to necrotizing cholecystitis or chronic obstruction (rare).

Untreated bile peritonitis is typically lethal; therefore early diagnosis is imperative. If rupture is associated with biliary tract infection, clinical signs of bile peritonitis can develop quickly. Dogs with sterile bile peritonitis (i.e., rupture caused by trauma) may have only ascites and icterus for weeks. Bile in the abdominal cavity causes chemical peritonitis, which may not be associated with overt clinical signs initially; however, changes in intestinal mucosal permeability may lead to secondary bacterial infection of the effusion. If diagnosis of a ruptured biliary tract is delayed, repair of the biliary tract is complicated by necrotic tissues and adhesions. Diagnostic peritoneal lavage (see p. 534) may assist in the early diagnosis of bile peritonitis (before the onset of clinical signs) in animals that have suffered abdominal trauma.

NOTE You need to repair ruptured gallbladders or common bile ducts as soon as possible. Delaying surgery means that the animal will be more debilitated, and fibrosis and adhesions in the areas will make surgery more difficult.

Rupture of the extrahepatic biliary ducts or gallbladder may be due to blunt abdominal trauma, necrotizing cholecystitis, or obstruction that occurs secondary to calculi, neoplasia, or parasites. Trauma usually causes rupture of the common bile duct rather than the gallbladder. Ductal rupture probably occurs when a force is applied adjacent to the gallbladder sufficient to cause rapid emptying, combined with a shearing force on the duct. Among human beings, biliary duct rupture has been reported in individuals who had previously undergone cholecystectomy, suggesting that shearing of the duct alone is sometimes sufficient. The most common site of ductal rupture appears to be the common bile duct just distal to the entrance of the last hepatic duct; however, rupture may occur in the distal common bile duct, the cystic duct (rare), or the hepatic ducts. Gallbladder rupture is principally caused by necrotizing cholecystitis (either from infection or mucocele) or cholelithiasis, but it has also been reported secondary to gunshot wounds. Many dogs with necrotizing cholecystitis have obstruction of the common bile duct; however, rupture can be caused by necrosis and perforation of only the gallbladder wall.

DIAGNOSIS

Clinical Presentation

Signalment

Traumatic rupture of the common bile duct or gallbladder may occur in animals of any age. Necrotizing cholecystitis is more common in middle-aged or older animals.

History

The animal may have sustained trauma several weeks before presentation. Clinical signs may be slowly progressive or acute if the bile becomes infected (see later).

Physical Examination Findings

Clinical signs of bile peritonitis depend on the presence of bacteria and on whether the peritonitis is diffuse or localized. Animals with infected bile peritonitis are generally in shock and have acute abdominal pain, fever, vomiting, and anorexia. Animals that develop localized peritonitis secondary to inspissated bile tend not to be as sick as those with diffuse peritonitis. Pain can sometimes be localized to the anterior abdomen. Some animals are diagnosed before a diseased gallbladder ruptures, in which case signs are similar to those seen with localized peritonitis.

NOTE Perform diagnostic peritoneal lavage to help identify bile peritonitis in dogs that have suffered trauma, before the onset of clinical signs.

Diagnostic Imaging

Radiographs of animals with bile peritonitis may show generalized loss of visceral detail if the peritonitis is diffuse, or a soft tissue ill-defined opacity in the cranial abdomen if the infection is localized. Survey radiographs infrequently reveal radiopaque gallstones or very rarely air in the gallbladder wall or lumen. Ultrasonography may also delineate the location of mass lesions and may evaluate the gallbladder and biliary ducts. Abnormal findings on ultrasound include echogenic peritoneal fluid, thickening of the gallbladder wall, and echogenicity of the gallbladder fossa. The absence of contrast enhancement on ultrasound can be used to help in the diagnosis of rupture/necrosis.[10] Reaction in the gallbladder fossa and localized or diffuse fluid on ultrasound examination did not indicate rupture; 55% of dogs with intact gallbladders had one or more of these ultrasound findings. Exploratory laparotomy is indicated in any patient with bile peritonitis and negates the need for extensive diagnostic workups.

Laboratory Findings

Comparing bilirubin concentrations in serum and abdominal fluid is 100% effective in diagnosing bile leakage. Bilious effusions have bilirubin concentrations greater (typically two times) than those found in serum. Neutrophilia is often noted if the peritonitis is generalized; however, the white blood cell count may be normal with localized infection. A normal or nearly normal peripheral white blood cell count, plus low numbers of immature neutrophils and no evidence of toxicity, may be associated with improved survival. Gallbladder rupture is positively correlated with increased white blood cell and neutrophil counts. The presence of rupture may be negatively correlated with normal coagulation profiles. Serum biochemical abnormalities commonly found in dogs with bile peritonitis include hyperbilirubinemia, increased ALP, increased ALT, hypoalbuminemia, and hyponatremia. Other findings are inconsistent and depend on the severity of the peritonitis. With septic biliary effusion, multiple types of gram-negative bacteria are typically found on bacterial culture and susceptibility.

DIFFERENTIAL DIAGNOSIS

A bilious effusion is obvious because the fluid looks like bile; it is usually easy to distinguish from effusions of other causes that have been stained by bilirubin. However, simultaneous bilirubin

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concentrations in the serum and effusion should be compared (see earlier).

NOTE To diagnose bile peritonitis, compare bilirubin concentrations in the fluid with those in serum. With bilious effusions, the bilirubin concentration is generally at least twice the bilirubin concentration of serum.

MEDICAL MANAGEMENT

Animals with bile peritonitis may be anemic, hypoproteinemic, or dehydrated or may have electrolyte imbalances. The irritating effects of bile on the peritoneum cause inflammation and fluid transudation into the abdominal cavity, and the animal may be presented for treatment in hypovolemic or systemic inflammatory response syndrome (or both). Aggressive fluid therapy may be needed, and electrolyte imbalances should be corrected. Broad-spectrum antibiotics should be administered before, during, and after surgery. Whole blood transfusions (see Table 4.5 and Box 4.1) may be indicated (i.e., hematocrit <20%). Administration of vitamin K_1 (see Box 21.1) or fresh-frozen plasma should be considered if coagulation abnormalities are detected (vitamin K malabsorption and disseminated intravascular coagulation are potential complications).

SURGICAL TREATMENT

Surgical treatment options for common bile duct rupture include ductal repair and biliary diversion (see p. 576). Repair is possible if the rupture is diagnosed early but becomes difficult once adhesions develop. Cholecystoduodenostomy or cholecystojejunostomy is usually easier and safer. Rupture of a hepatic duct can be treated by ligation of the leaking duct. Gallbladder rupture that occurs secondary to infective processes should be treated by cholecystectomy (see p. 574).

Treatment of necrotizing cholecystitis includes early surgical exploration once the animal's condition has been stabilized. Treatment consists of cholecystectomy, antibiotics, and appropriate therapy for peritonitis. Generally, attempts to salvage the gallbladder by closing the defect are inappropriate because the wall is usually necrotic. Be sure that the common bile duct is not ligated when the gallbladder is removed. Delayed diagnosis probably contributes to the high mortality associated with necrotizing cholecystitis.

NOTE Animals with necrotizing cholecystitis have infected bile; therefore clinical signs often begin soon after the gallbladder ruptures. Unless diagnosis and surgical intervention are prompt, mortality is high.

Preoperative Management

Surgery should be performed as soon as the animal's condition has been stabilized. Electrolyte and fluid abnormalities should be corrected before surgery. See also the earlier section on Medical Management of patients with bile peritonitis.

Anesthesia

Hypovolemic, septic, or shocky dogs may be induced with hydromorphone plus diazepam or midazolam IV (Box 21.3), given to effect. If intubation is not possible, etomidate, propofol, or alfaxalone may be given, or mask induction with isoflurane or sevoflurane is used if the patient is not vomiting. For patients in stable condition, see p. 540 for the anesthetic management of patients with hepatobiliary disease.

BOX 21.3 Selected Anesthetic Protocol for Dogs With Biliary Disease

Induction

- Hydromorphone (0.1 mg/kg IV) *or*
- Oxymorphone (0.1 mg/kg IV) *or*
- Fentanyl (2–5 µg/kg IV) *plus*
  - Diazepam (0.2 mg/kg IV) or
  - Midazolam (0.2 mg/kg IV)

Give in Incremental Dosages; Intubate if Possible

If necessary, give (to effect):

- Etomidate (0.5–1.5 mg/kg IV) (give a benzodiazepine prior to the etomidate) *or*
- Propofol (4–6 mg/kg IV) *or*
- Alfaxalone (2–5 mg/kg IV)

Maintenance

Isoflurane or sevoflurane
Give anticholinergics as needed.

IV, Intravenous.

Surgical Anatomy

See p. 572 for information on the surgical anatomy of the extrahepatic biliary system.

Positioning

The gallbladder is generally exposed via a cranial midline abdominal incision (see p. 516). The caudal thorax and the entire abdomen should be prepared for aseptic surgery.

SURGICAL TECHNIQUE

Cholecystectomy is discussed on p. 574. Laceration or transection of the bile ducts may be treated by primary repair (see p. 576) or biliary diversion (p. 574). A damaged hepatic duct may be ligated because alternative routes for biliary drainage from a single liver lobe will develop. The abdominal fluid and the site of rupture or perforation should be cultured during surgery. Once the site of leakage has been identified and corrected, the abdomen should be flushed with copious amounts of warm, sterile fluids. Open abdominal drainage (see p. 534) may be considered if generalized peritonitis is present.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

A Poole suction tip is useful for removing abdominal fluid and can help identify the site of leakage. It is also used to remove fluid instilled in the abdomen during lavage.

POSTOPERATIVE CARE AND ASSESSMENT

Fluid therapy should be continued until the animal is able to maintain hydration on its own. Electrolytes and acid-base status should be monitored. Many patients with bile peritonitis are extremely debilitated before surgery. Animals with bile peritonitis are in extreme pain. Postoperative analgesia may be provided with

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hydromorphone (see Table 13.2) or a fentanyl-lidocaine-ketamine constant-rate infusion (see Table 13.2 and Box 13.4). Butorphanol is also effective, but the analgesia is of shorter duration than that achieved with hydromorphone. Nutritional supplementation via a needle-catheter jejunostomy or parenterally is beneficial in these patients (see Chapter 10). Antibiotic therapy based on culture of bile should be continued for at least 7 to 14 days after surgery.

PROGNOSIS

The prognosis for patients with diffuse, septic bile peritonitis is guarded. Without aggressive surgical management, many of these patients die. The prognosis is better if the condition is diagnosed and treated early, and in patients with smaller elevations in white blood cell counts. Survival in dogs and cats with nonseptic biliary peritonitis is high. Hypotension (preoperative, intraoperative, or postoperative) and disseminated intravascular coagulation may be negative prognostic indicators.

CHOLECYSTITIS

DEFINITION

Cholecystitis is an inflammation of the gallbladder that may be due to obstruction of the cystic duct by stones or secondary to bacterial infection. It may be accompanied by cholangitis or infection of the biliary tree.

GENERAL CONSIDERATIONS AND RELEVANT PATHOPHYSIOLOGY

Bacteria may enter the biliary tree via ascending infection of the bile duct or via a hematogenous route from the portal vasculature. Normally, ascending infection is prevented by pressures at the major duodenal papilla, flushing of the ducts during bile flow, and the bacteriostatic action of bile salts. Kupffer cells, secretory immunoglobulin A, and mucus in the liver provide immunologic protection from portal blood as a source of infection. An increased risk of bacterial infection may occur where there is biliary stasis, increased portal inoculation, or when the animal has been administered immunosuppressant agents. Biliary mucoceles may become infected, and other conditions (e.g., cholelithiasis, gallbladder calcification) may become secondarily infected. Gastrointestinal conditions that alter anatomy or function of the proximal duodenum can predispose to ascending infection (e.g., inflammatory bowel disease, pancreatitis).

DIAGNOSIS

Clinical Presentation

Signalment, clinical signs, and treatment of animals with cholecystitis were reported in a 2016 study.[12] Dogs of any age and various sizes (2.3–38 kg) were affected. Presenting complaints included vomiting (89%), anorexia (70%), lethargy (67%), and diarrhea (44%). Less common, historical findings were polyuria and polydipsia. Clinical signs were evident for 1 day to months prior to presentation. Acute signs were present in approximately one-third of the cases; the remainder had waxing and waning signs.

Physical Examination

In the aforementioned study, the most common findings on physical examination included jaundice (56%), abdominal discomfort (56%), and fever (33%). Ascites was present on physical examination in 18%.

Diagnostic Imaging

The most common findings on abdominal ultrasound are dilation of the bile duct (38%), thickening of the gallbladder wall (35%), gallbladder distention (35%), and gallbladder sludge (35%).[12] Abdominal effusion was present in 31% of dogs in this study and was bilious or exudative in nature. A mucocele was diagnosed in 24%, and the gallbladder was ruptured in a small number of animals. Rarely, gas was noted in the gallbladder and hepatic enlargement was seen. Choleliths were present in 15% of the dogs.

Laboratory Findings

Hyperbilirubinemia is a common finding and was present in the majority of cases (83%) in the aforementioned study; other common findings are neutrophilia and hyperglobulinemia.

Bile can be cultured by percutaneous ultrasound guidance. When performing this procedure, complete evacuation of the gallbladder should be done to minimize the risk of leakage. Bile culture was positive in 96% of affected dogs in a 2016 multicenter, retrospective cases series of 27 dogs with bacterial cholangitis, cholecystitis, or both.[12] In this study, 100% and 33% of gallbladder and liver cultures, respectively, were positive. *E. coli* was most often cultured, but other isolates included *Enterococcus* spp., *Clostridium perfringens,* and occasionally *Enterobacter cloacae, Klebsiella, Proteus,* and *Bacteroides* spp. Multibacterial infection was present in 26% of the dogs. In another study of 140 bile samples from dogs and cats who had undergone cholecystocentesis, cultures were positive in 30% of dogs and 22% of cats, and concurrent inflammation was present in 5% and 19%, respectively.[13] Similar to the previous study, *E. coli* and *Enterococcus* species were most commonly isolated. An increase in canine pancreatic lipase immunoreactivity was associated with the presence of bacteria and inflammation of the bile.

DIFFERENTIAL DIAGNOSIS

The clinical signs of abdominal pain and jaundice seen with cholecystitis are similar to those seen with many hepatic disease, including chronic hepatopathy, biliary mucocele, and cholelithiasis. Lack of obvious gastrointestinal abnormalities on radiographs, combined with abdominal ultrasound, usually aid in the diagnosis of cholecystitis; however, chronic cases may require specific diagnostic testing.

MEDICAL MANAGEMENT

Supportive care may include IV fluid support, antimicrobial therapy, and analgesics. *E. coli* may be resistant to antibiotics often used in animals with hepatic disease, such as amoxicillin clavulanate and potentially fluoroquinolones; therefore culture and susceptibility testing is recommended in these animals. Concurrent use of hepatic support (s-adenosyl-L-methionine, Silybin) and ursodeoxycholic acid is recommended in many animals.

SURGICAL TREATMENT

Cholecystectomy may be considered in affected animals, particularly if the condition is chronic or has not responded

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to medical management; it is a necessity when a mucocele (see p. 580), choleliths, or gallbladder rupture are present.

Preoperative Management

Where feasible, the animal should be stabilized before surgery. Some animals with chronic disease have been treated with antibiotics and hepatic support prior to presentation.

Anesthesia

See pp. 540 and 571 for the anesthetic management of animals with hepatic and biliary diseases.

Surgical Anatomy

See p. 572 for the anatomy of the biliary tract.

Positioning

The patient is placed in dorsal recumbency for abdominal exploration, cholecystectomy, liver biopsy, and evaluation of the biliary tree.

SURGICAL TECHNIQUE

In most animals, perform a cholecystectomy (see p. 574) and liver biopsy (see p. 546). Evaluate the biliary tree and lavage and stent it, as necessary. Submit samples of bile, gallbladder wall, and liver parenchyma for aerobic and anaerobic culture and susceptibility testing. This is important because of the resistant nature of the bacteria involved and also because many of these patients have previously been or are currently being administered antibiotics.

POSTOPERATIVE CARE AND ASSESSMENT

See p. 577 for the postoperative care following cholecystectomy. Antibiotics (based on culture and sensitivity testing), liver support, and ursodeoxycholic acid (Ursodiol) should be continued after surgery.

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22

Surgery of the Endocrine System



Surgery of the Adrenal and Pituitary Glands

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Adrenalectomy is the removal of one or both adrenal glands. **Hypophysectomy** is the removal of the pituitary gland (hypophysis). **Hyperadrenocorticism** (HAC) is a multisystemic disorder caused by an excess of glucocorticoids. **Cushing disease** refers to HAC caused by a pituitary adenoma. **Addison disease** is caused by a deficiency of glucocorticoids or mineralocorticoids or both.

PREOPERATIVE MANAGEMENT

Adrenocortical insufficiency may be primary, secondary to other diseases, or iatrogenic (i.e., due to administration of glucocorticoids, progestins, or drugs that suppress the adrenal glands [e.g., trilostane, mitotane, etomidate]). The history should include the dosages of glucocorticoids or other drugs given, type of glucocorticoids, duration of administration, and time since the last dose. It is easier to inhibit secretion of glucocorticoids than mineralocorticoids (see the discussion of anatomy on p. 587). When glucocorticoid secretion is severely suppressed, the patient may experience depression, inappetence, lethargy, collapse, and/or weakness without electrolyte abnormalities. If mineralocorticoid secretion is suppressed, hyponatremia, hyperkalemia, acidosis, and/or azotemia may occur. Diminished ability to retain sodium results in volume depletion, diminished cardiac output, and reduced vascular tone, which may cause acute vascular collapse. Gastrointestinal disturbances and prolonged vomiting may contribute to electrolyte abnormalities and volume depletion. Electrolyte concentrations should be corrected before surgery. Some dogs with hypoadrenocorticism are hypoalbuminemic. A protective steroid release normally occurs during surgery that prevents circulatory collapse; however, animals with hypoadrenocorticism may be unable to respond appropriately and often require glucocorticoid supplementation before and during surgery. When minor elective surgery is performed in animals with adrenocortical insufficiency, glucocorticoid therapy may be given intravenously before induction of anesthesia (Box 22.1). The same dose can be given intravenously or intramuscularly after recovery from anesthesia, and the animal is returned to its oral maintenance glucocorticoid therapy the day after surgery. A similar protocol is used for major surgery, except that glucocorticoid therapy is continued at approximately five times the maintenance dose for 2 to 3 days (Box 22.2). Normal maintenance doses are then reinstituted. Once the animal is eating, medications can be given orally (Box 22.3).

HAC is found primarily in dogs; it is rare in cats. Iatrogenic HAC is the most common type. Spontaneous HAC is usually caused by excessive pituitary secretion of adrenocorticotropic hormone (ACTH), resulting in bilateral adrenocortical hyperplasia or pituitary-dependent hyperadrenocorticism (PDH) (80%–90% of noniatrogenic cases). Functional adrenocortical tumors (adrenal-dependent hyperadrenocorticism [ADH]) are less common (10%–20% of noniatrogenic cases). Differentiation of PDH from ADH is potentially complex, and the reader is referred to a medical text for further information.

Patients with HAC are catabolic and often protein depleted; this may adversely affect wound healing. They may have connective tissue abnormalities and muscle wasting, resulting in a pot-bellied appearance, redistribution of fat, and thin, fragile skin. Pyodermas are common in affected dogs, which may cause postoperative suture line healing to be compromised. Affected dogs may pant because of their catabolic state, but intraabdominal fat deposition plus abdominal muscular weakness sometimes causes ventilatory abnormalities. Hypernatremia, hypokalemia, and alkalosis may be present; substantial abnormalities should be corrected before surgery. Concurrent abnormalities (e.g., congestive heart failure, diabetes mellitus [DM]) increase the patient's anesthetic risk. Cardiovascular abnormalities may occur secondary to hypervolemia and hypertension; a thorough preoperative cardiac examination including blood pressure measurement is appropriate.

Animals with HAC are at increased risk for postoperative pulmonary thromboembolism (PTE). If hypercoagulability is suspected, preventive measures may be indicated before surgery. For prevention of PTE, animals may be started on heparin during surgery and continued on it postoperatively (see Box 29.1) or they may be administered clopidogrel (2–3 mg/kg per day in dogs); however, prospective studies are needed to determine the relative benefit of these therapies. Many animals with HAC have clinically silent urinary tract infection; therefore urine culture is indicated in all patients, regardless of urinalysis findings.

ANESTHESIA

A variety of anesthetic protocols may be used in adrenocortical-insufficient or hyperadrenal animals. Etomidate causes transient adrenal suppression and should be avoided in patients with hypoadrenocorticism and those in which postoperative hypoadrenocorticism is anticipated. Steroid replacement should be provided in animals showing signs of adrenal insufficiency. Maintenance of electrolyte and glucose concentrations is important. Glucocorticoid supplementation is often necessary in animals with adrenocortical insufficiency undergoing surgery (see previous

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ABSTRACT

Organs of the endocrine system that may require surgical intervention are the pituitary gland, adrenal glands, thyroid and parathyroid glands, and the pancreas. Conditions affecting the endocrine system often result in abnormal production and release of hormones into the circulation. Excessive production of hormones, most often due to a neoplastic process, may require removal of the affected gland (e.g., adrenalectomy, thyroidectomy). Diseases of the pancreas can be challenging as they may be diffuse, or a neoplastic lesion requiring resection may be in an anatomically challenging area. Thorough diagnostic evaluation for suspected endocrinopathies is critical to determine the best mode of treatment.

KEYWORDS

adrenal gland
adrenalectomy
pituitary gland
hypophysectomy
hyperadrenocorticism
pheochromocytoma
pancreas
insulinoma
partial pancreatectomy
neoplasia
abscess
pseudocyst
gastrinoma
thyroid gland
parathyroid gland
hyperthyroidism
hyperparathyroidism

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BOX 22.1 Protocol for Glucocorticoid Administration in Animals With Adrenocortical Insufficiency Undergoing Minor Elective Procedures

1. Prior to surgery give double the maintenance dose of oral glucocorticoids.
2. If oral supplementation is not reliable or feasible, then give:
   - Prednisolone sodium succinate 1 mg/kg IV *or*
   - Hydrocortisone sodium succinate 2 mg/kg IV
3. As soon as the patient can reliably take oral medications, repeat oral prednisolone.

Dexamathasone is more ulcerogenic than other steroids and should be used with caution.

BOX 22.2 Protocol for Glucocorticoid Administration in Animals With Adrenocortical Insufficiency Undergoing Major Elective Procedures

1. Administer preoperative steroids as described in Box 22.1.
2. Administer dexamethasone (0.05–0.1 mg/kg IV) during surgery.
3. Taper the dexamethasone dose by 0.02 mg/kg (IV bolus) every 12 hours (but do not give <0.02 mg/kg).
4. As soon as the dog can reliably be given oral medications, switch to oral prednisolone (see Box 22.3).

BOX 22.3 Postoperative Drug Therapy After Adrenalectomy in Dogs

Fludrocortisone Acetate (Florinef)[a]
0.2 mg/kg PO q12h for 1–2 wk postoperatively

Dexamethasone
See Box 22.2

Prednisolone
0.5 mg/kg q12h for 2–3 days, then slowly decrease the dose every 3 wk to the lowest tolerated dose

[a]Fludrocortisone is much less reliable than desoxycorticosterone pivalate for normalizing serum electrolyte concentrations; however, the effects of fludrocortisone last only 1 day, whereas one injection of desoxycorticosterone acetate lasts 28 days.
IV, Intravenous; *PO,* orally; *SC,* subcutaneous.

discussion). Glucocorticoid therapy should be instituted before surgery in patients with HAC that are undergoing adrenalectomy. Because of the close association of the adrenals and the caudal vena cava, retraction of the caudal vena cava is often necessary for adrenalectomy. Vascular pressures should be closely monitored during surgery, and retraction should be done carefully to avoid obstructing venous return. Special anesthetic considerations are required for animals with pheochromocytomas to prevent complications associated with excessive secretion of catecholamines (see p. 594).

ANTIBIOTICS

Animals with HAC are at increased risk of developing postoperative infection owing to high levels of circulating glucocorticoids.

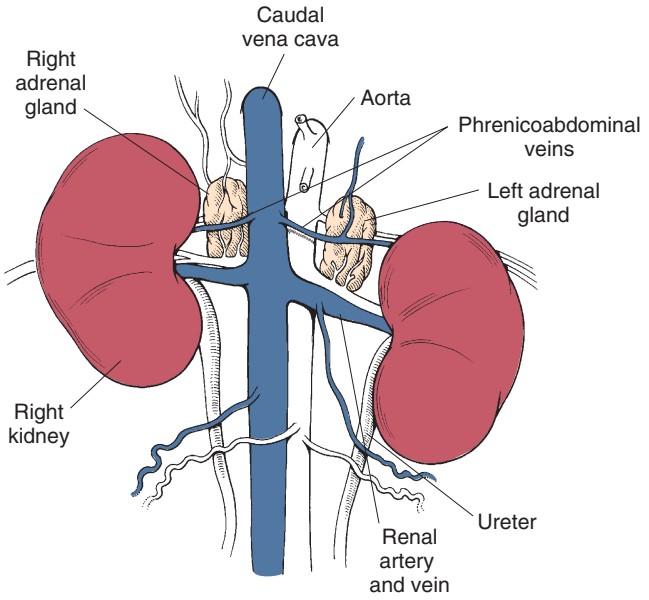


FIG. 22.1 Location of the adrenal glands.

Perioperative prophylactic antibiotics are recommended for these patients.

SURGICAL ANATOMY

The adrenal glands are near the craniomedial pole of the kidneys (Fig. 22.1). The left adrenal is slightly larger than the right. The left gland lies ventral to the lateral process of the second lumbar vertebra; the right adrenal is more cranial, lying ventral to the lateral process of the last thoracic vertebra. Because of the proximity of the right adrenal to the caudal vena cava, surgical removal of neoplastic glands can be difficult. The phrenicoabdominal (cranial abdominal) vessels cross the ventral surface of the adrenal. The adrenal glands are composed of two functionally and structurally different regions. The *outer cortex* produces mineralocorticoids (e.g., aldosterone), glucocorticoids, and small quantities of androgenic hormones. Mineralocorticoids regulate sodium and potassium concentrations. Aldosterone causes transport of sodium and potassium through the renal tubular walls and also causes hydrogen ion transport.

The adrenal *medulla* is functionally related to the sympathetic nervous system and secretes epinephrine and norepinephrine in response to sympathetic stimulation. Epinephrine and norepinephrine have almost the same effects as direct sympathetic stimulation (e.g., vascular constriction, resulting in increased arterial pressure; inhibition of the gastrointestinal tract; pupillary dilatation; increased rates of cellular metabolism throughout the body), except that their effects last significantly longer because they are removed from the circulation slowly.

SURGICAL TECHNIQUE

Adrenalectomy is usually performed for adrenal tumors. Bilateral adrenalectomy for canine PDH is controversial and uncommonly performed, but it has been effective for feline PDH. One of two open approaches can be used, or alternatively a laparoscopic approach can be performed. A ventral midline approach allows the entire abdomen to be explored for metastasis and bilateral adrenalectomy to be performed with a single surgical incision

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if necessary. However, exposure and dissection of the adrenal may be difficult with this approach, particularly in large dogs. A paracostal incision provides better access to the adrenal gland but does not allow evaluation of the liver or other organs for metastasis. It may be considered in animals with unilateral lesions that have no evidence of metastasis on ultrasound, computed tomography (CT), or magnetic resonance imaging (MRI). Concurrent DM might be a contraindication to bilateral adrenalectomy because lack of endogenous catecholamines may make it difficult to regulate the diabetes. For laparoscopic adrenalectomy, dogs are positioned in lateral recumbency, with the adrenal gland to be removed on the up side. Dogs may also be positioned in sternal recumbency with the thorax and pelvis elevated, such that the abdomen is not in contact with the surgical table.[1] This allows for gravitational displacement of the abdominal viscera and better visualization of the affected adrenal gland.

Adrenalectomy via a Midline Abdominal Approach

Prepare the entire ventral abdomen and caudal thorax for aseptic surgery. Make a ventral midline abdominal incision that extends from the xiphoid cartilage to near the pubis. Identify the affected adrenal gland and carefully inspect the entire abdomen, including the other adrenal gland, for abnormalities or evidence of metastasis. Palpate the liver for evidence of nodularity and biopsy if indicated. Palpate the caudal vena cava near the adrenal glands for evidence of tumor invasion or thrombosis. If additional exposure is necessary for adrenalectomy, extend the incision paracostally on the side of the affected gland by incising the fascia of the rectus abdominis muscle and the fibers of the external abdominal oblique, internal abdominal oblique, and transversus abdominis muscles, respectively. Use self-retaining retractors to improve visualization of the abdominal cavity. Retract the liver, spleen, and stomach cranially, the kidney caudally, and the caudal vena cava medially to expose the entire adrenal gland. Identify the blood supply and ureter to the ipsilateral kidney, and avoid these structures during dissection. Ligate the phrenicoabdominal vein and divide it between sutures. Using a combination of sharp and blunt dissection, carefully dissect the adrenal gland from surrounding tissue (Fig. 22.2). Numerous vessels may be encountered. *Obtain hemostasis with electrocautery, a vessel-sealing device, or with hemoclips. If possible, do not invade the adrenal capsule. Remove the adrenal in one piece, if possible, to reduce the chances of leaving small pieces of neoplastic tissue in the abdominal cavity. If tumor thrombosis is present in the caudal vena cava but extensive metastasis is not apparent, temporarily occlude the vena cava using Rumel tourniquets (see pp. 562 and 795). Make a longitudinal incision in the vein and remove the thrombus. Close the vena cava in a continuous pattern with 5-0 or 6-0 vascular suture, and close the abdomen routinely (see discussion of suture material on p. 589). If a paracostal incision was made, begin the closure by approximating the abdominal wall at the junction of the combined ventral and paracostal incisions. After closing the linea alba, suture each muscle layer of the paracostal incision with a continuous pattern of synthetic absorbable sutures. Close the skin and subcutaneous tissue routinely.*

Adrenalectomy via a Paralumbar Approach

Place the patient in lateral recumbency with a rolled towel or a sandbag between the abdomen and the operating table. Prepare the caudal hemithorax and lateral abdomen for aseptic surgery. Make an incision just caudal to the 13th rib, extending it from the lateral vertebral processes to within 3 to 4 cm of the ventral midline (the incision will be approximately 10–14 cm long, depending on the animal's size; Fig. 22.3). Incise the abdominal muscles individually and identify the adrenal gland cranial to the kidney. Retract the kidney ventrally and ligate any vascular structures that cross its surface. Dissect the gland free from surrounding tissue (Fig. 22.4). Suture each muscle layer of the paracostal incision in a continuous suture pattern of synthetic absorbable (i.e., 2-0 or 3-0) material. Close the skin and subcutaneous tissues routinely.

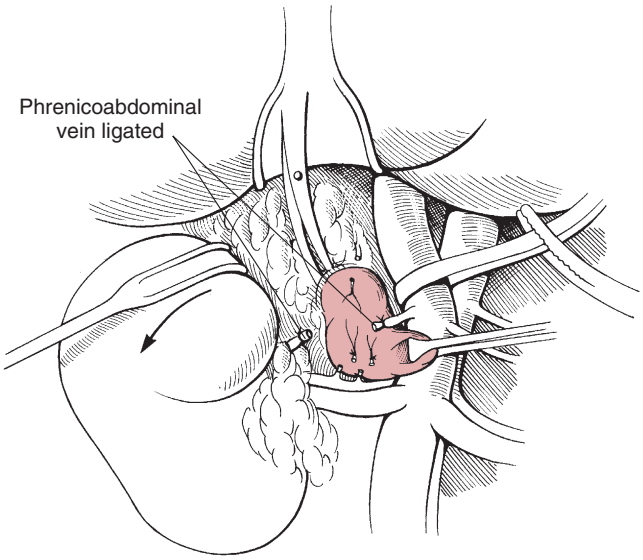


FIG. 22.2 To resect the right adrenal gland, retract the vena cava medially. Ligate the phrenicoabdominal vein and divide it between sutures. Carefully dissect the adrenal gland from surrounding tissue.

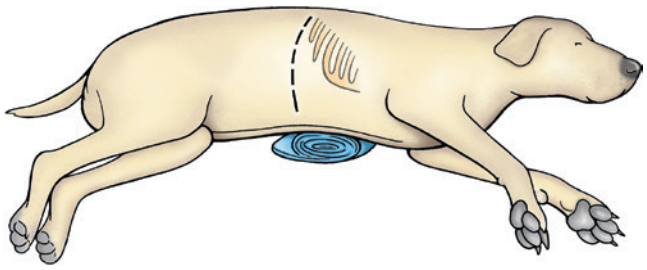


FIG. 22.3 Adrenalectomy may be performed via a paralumbar approach. Place the animal in lateral recumbency with a rolled towel or sandbag between the abdomen and the operating table. Make an incision just caudal to the 13th rib, extending it from the lateral vertebral processes to within 3 to 4 cm of the ventral midline.

Laparoscopic Adrenalectomy

When no evidence of caudal vena cava involvement is found, laparoscopic adrenalectomy may be an option. Laparoscopic adrenalectomy has been described in veterinary patients; however, the procedure is challenging and requires careful patient selection. Removal of the right adrenal gland in dogs is especially challenging as the adrenal gland capsule can be continuous with the tunica externa of the caudal vena cava. Preoperative diagnostic imaging is important in the workup for adrenal masses and is the basis for deciding whether a laparoscopic approach might be feasible. The dimensions of the mass are vital, as are relationships to surrounding organs and vascular structures. Approximately 25% of adrenal neoplasms invade the caudal vena cava, phrenicoabdominal

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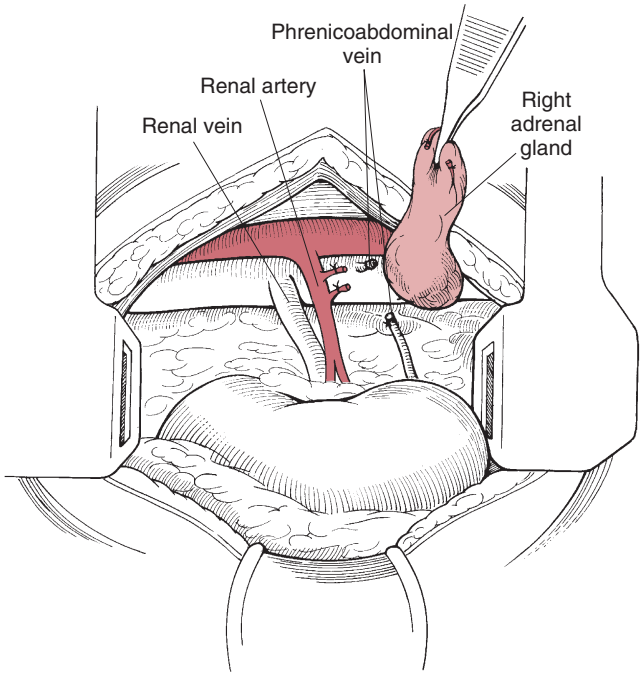


FIG. 22.4 To expose the adrenal gland via a paralumbar approach, retract the kidney ventrally and ligate any vascular structures that cross its surface.

veins, or renal vasculature; pheochromocytomas are more likely to invade than are adrenocortical tumors. Vascular invasion is an indication for an open surgical approach. Ultrasonography and CT are used most often for preoperative imaging. Ultrasonography has a sensitivity and a specificity of approximately 80% and 90%, respectively, for detection of tumor thrombus. A 2015 study found excellent agreement between signs of vascular invasion in CT images and finding vascular invasion at surgery or necropsy with sensitivity, specificity, and agreement measuring 92%, 89%, and 94%, respectively.[2] Animals with functional tumors causing clinical signs, and those with tumors measuring more than 2 cm that do not exhibit vascular invasion may be candidates for laparoscopic adrenalectomy. Animals that are systemically unstable and those that have uncontrolled metabolic or acid-base disturbance, uncontrolled coagulopathies, untreated severe arrhythmias, or hypertension should undergo an open approach. Animals that may be poorly tolerant of pneumoperitoneum (e.g., severe cardiorespiratory disease, diaphragmatic herniation) are also poor candidates for a laparoscopic approach. Vascular invasion of the mass into surrounding vessels and large masses (>7 cm) are indications for open adrenalectomy. An upper limit of 5 to 7 cm maximal diameter of the adrenal mass has been recommended for laparoscopic removal of adrenocortical tumors.[3]

Place the animal in lateral recumbency with elevation of the vertebral column or sternal recumbency with the thorax and pelvic elevated. Use a three-port technique in the paralumbar fossa, caudal to the last rib, triangulating the approximate position of the adrenal gland. Place an additional port dorsally if additional retraction is needed. Isolate the adrenal gland by careful dissection of the peritoneum and periadrenal tissue dorsolateral to the gland. Use large hemoclips or a vessel-sealing device to ligate the phrenicoabdominal vein. Carefully dissect the adrenal gland out of the retroperitoneal space using dissecting forceps, electrocautery, or a vessel-sealing device, keeping the adrenal capsule intact. Following completion of the dissection, place the adrenal gland in a specimen retrieval bag and remove it from the body. Close the port sites routinely following evacuation of the pneumoperitoneum.

HEALING OF THE ADRENAL AND PITUITARY GLANDS

Because adrenal or pituitary biopsies are rarely performed, little information is available about the healing of these glands after surgery.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

HAC may cause delayed wound healing; therefore abdominal closure should be performed with strong, slowly absorbed, or nonabsorbable suture material (e.g., polydioxanone, polyglyconate, polypropylene, nylon). Self-retaining retractors, such as Balfour abdominal retractors, are recommended to improve abdominal visualization. Malleable retractors covered with moistened sponges are used to retract viscera from the adrenal glands. Hemostasis is easier to achieve with vessel-sealing devices, electrocautery, and hemoclips than with suture ligation of vessels (see Chapter 7).

POSTOPERATIVE CARE AND ASSESSMENT

After adrenalectomy, the patient's hydration status and electrolyte balance should be monitored carefully and corrected as necessary. Bilateral adrenalectomy causes permanent adrenal insufficiency, and these animals require lifelong glucocorticoid (e.g., prednisolone) and/or mineralocorticoid (e.g., deoxycorticosterone) replacement (see Box 22.3). Animals should be closely monitored for hypoadrenocortical collapse. They are most likely to have an Addisonian crisis after they have been released to the owner's care. Owners must be advised to watch for malaise, inappetence, vomiting, weakness, and other clinical signs suggesting decompensation. Temporary adrenal insufficiency occurs after unilateral removal of functional adrenal tumors because the tumor has suppressed the function of the contralateral adrenal. Glucocorticoids should be supplemented postoperatively (see Box 22.3) but may be discontinued when the remaining adrenal begins to function normally, as determined by the results of an ACTH stimulation test.

PTE is a potentially life-threatening complication of adrenal surgery, particularly in dogs with adrenal neoplasia. Sudden, severe postoperative respiratory distress may indicate PTE. Lung perfusion scans may help identify lung regions that are underperfused. To help prevent this have the dog walk every 2 to 3 hours to promote circulation. Sufficient analgesics should be provided so that the dog can be coaxed to walk 4 hours after it wakes up. Treatment with strict cage rest, oxygen, anticoagulants (e.g., clopidogrel, aspirin, heparin), and thrombolytic agents (e.g., streptokinase, tissue plasminogen activator) has been proposed in the past, but the value of these pharmacologic agents is uncertain (see Box 29.1). Animals treated for PTE with thrombolytics should be assessed frequently for evidence of hemorrhage, and the hematocrit should be checked every 2 hours. If the packed cell volume (PCV) drops or hemorrhage is noted, the thrombolytic infusion should be discontinued.

COMPLICATIONS

The main complications of adrenalectomy are hemorrhage, fluid and electrolyte imbalance, pancreatitis, wound infection, delayed

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wound healing, and thromboembolism. Postoperative hemorrhage is usually associated with incomplete occlusion of small vessels surrounding an enlarged, highly vascular tumor. Judicious use of vessel-sealing devices, electrocautery, and hemoclips helps prevent this complication. Delayed wound healing is often encountered in animals with HAC because of the adverse effect of steroids on wound healing; therefore care should be taken with abdominal closure in these animals (see previous discussion). Strong monofilament absorbable (e.g., polydioxanone or polyglyconate) or nonabsorbable (e.g., polypropylene) suture should be used.

SPECIAL AGE CONSIDERATIONS

Animals with adrenal neoplasia are usually older and often have concurrent abnormalities, such as hypertension or cardiovascular abnormalities; therefore extreme care should be exercised during anesthesia. These animals also require extensive postoperative monitoring. If the animal is debilitated, anorexic, or vomiting, placement of an enteral feeding tube during surgery (see p. 101) or parenteral nutrition (see p. 94) is advised.

SPECIFIC DISEASES

ADRENAL NEOPLASIA

DEFINITIONS

Adrenal carcinomas are autonomously functioning, malignant tumors of the adrenal cortex; **adrenal adenomas** are benign adrenocortical tumors. **Pheochromocytomas** are catecholamine-secreting tumors of the chromaffin tissue, which usually arise in adrenal medullary tissue. Pheochromocytomas are also known as *paragangliomas*. "Incidentalomas" (incidental adrenal masses) are adrenal masses that are fortuitously found during imaging in animals that are not suspected of having adrenal disease. They may be carcinomas or pheochromocytomas, or they may be undefined, nonfunctioning masses.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Adrenal gland tumors include adrenal adenomas, carcinomas, and pheochromocytomas. Most adrenal tumors are nonfunctional, and clinical signs are caused by local invasion of the tumor into surrounding tissue, distant metastases, or both. Functional cortical tumors secrete excessive amounts of cortisol or aldosterone. In dogs, functional tumors typically secrete cortisol, which inhibits pituitary ACTH secretion and causes atrophy of the contralateral adrenal. Adrenocortical adenomas and carcinomas appear to occur with equal frequency. They are usually unilateral, although bilateral adrenocortical neoplasia rarely occurs. History, physical examination, and laboratory findings do not differentiate between bilateral and unilateral adrenal neoplasia. Ultrasonographic evaluation of the adrenals often identifies adrenomegaly on one side and adrenal atrophy on the other, which localizes the tumor. Colonic perforation is a rare sequela of excessive glucocorticoid secretion. Corticosteroids may inhibit collagen synthesis and increase collagen breakdown. They may also cause breakdown of the mucosal barrier and inhibit normal immune responses.

NOTE Most animals with hyperadrenocorticism have pituitary (rather than adrenal) tumors.

Unilateral or bilateral adrenal gland tumors may also occur in cats. In a 2016 study of 33 feline adrenal tumors, 17 were carcinomas, 13 were adenomas, and 3 were pheochromocytomas.[4] Nineteen of these cats were found to have functional tumors, with 16 of 19 causing hypersecretion of aldosterone. Common clinical signs include weakness (most often due to significant hypokalemia), blindness, respiratory issues, and gastrointestinal signs.

Pheochromocytomas are tumors of the adrenal medulla that secrete excessive amounts of catecholamines (primarily norepinephrine, but also epinephrine and dopamine) and other vasoactive peptides (e.g., vasoactive intestinal polypeptide, somatostatin, enkephalin, corticotropin). Excessive catecholamine and vasoactive peptide levels may manifest as cardiovascular, respiratory, or central nervous system (CNS) disease. Although these tumors have classically been reported as benign, recent reports suggest that regional invasion and distant metastases (liver, regional lymph nodes, lungs, spleen, ovaries, diaphragm, and vertebrae) occur in as many as 50% of affected dogs. Invasion of the caudal vena cava, phrenicoabdominal (cranial abdominal) artery or vein, renal artery or vein, or hepatic vein may cause signs of ascites, edema, or venous distention. Pheochromocytomas are usually unilateral, although bilateral tumors occur. These masses are usually reddish-tan, multilobulated, firm, or friable, and may be completely or partly encapsulated. Occasionally, pheochromocytomas may be associated with neoplastic transformation of multiple endocrine tissues of neuroectoderm origin (e.g., pituitary, adrenocortical, or thyroid adenomas; or pancreatic islet cell tumors). Extra-adrenal pheochromocytomas have been reported in dogs and cats. Other tumors rarely arising from the adrenal medulla include neuroblastoma and ganglioneuroma.

DIAGNOSIS

Clinical Presentation

Signalment

Adrenocortical tumors usually occur in older, large-breed dogs and appear to be diagnosed more commonly in females. A definitive breed predisposition has not been identified. Pheochromocytomas usually occur in older dogs but have been reported in dogs as young as 1 year; both genders appear to be equally affected.

History

It is critical to understand that HAC can be diagnosed only in animals with clinical signs consistent with HAC. To diagnose HAC, there must be abnormalities (e.g., polyuria-polydipsia, polyphagia, pendulous abdomen [i.e., "pot belly"], endocrine alopecia, hyperpigmentation, and/or calcinosis cutis) that are typical of HAC. One might also see muscle wasting, weakness, lethargy, and/or panting in some patients, but these are not strongly suggestive of HAC. No imaging or laboratory findings (including adrenal gland function tests) allow diagnosis of HAC in the absence of obvious clinical signs. Pheochromocytoma and nonfunctional tumors will not cause these signs.

Vomiting has been associated with intestinal perforation in a dog with adrenocortical adenoma, but this is rare. High circulating levels of glucocorticoids may make diagnosis of intestinal perforation difficult because signs of peritonitis (abdominal discomfort, restlessness, panting, weakness, and/or dyspnea) are initially obscured. Cats reported with primary hyperaldosteronism present with a history of weakness, vomiting, inappetence, dehydration, diarrhea, and cervical ventroflexion.

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Pheochromocytomas may cause vague, intermittent signs of weakness or panting due to episodic hypertension and tachycardia. Many times they are incidental findings on CT, ultrasonography, or necropsy. Signs of nonfunctional adrenocortical tumors may include anorexia, abdominal enlargement, abdominal pain, diarrhea, vomiting, and lethargy; however, they too may be incidental findings on CT, ultrasonography, or necropsy. In a study of 270 dogs undergoing abdominal CT for reasons other than adrenal gland disease, incidental adrenal gland masses were identified in 9.3%.[5]

Physical Examination Findings

Clinical findings in animals with adrenocortical tumors depend on whether the tumors are functional. Dogs with ADH should have obvious signs of HAC (see previous discussion). Ascites, abdominal pain, edema, diarrhea, and vomiting are more common with nonfunctional tumors, although many are asymptomatic. Cats with hyperaldosteronism may have pelvic limb weakness, cervical ventroflexion, or a plantigrade stance. For cats presenting with blindness, there may be hyphema, retinal detachment, or intraocular hemorrhage. A palpable abdominal mass may also be noted in cats.

Clinical findings in animals with pheochromocytomas may include tachycardia or cardiac arrhythmia, acute collapse, polypnea, panting, cough, lethargy, anorexia, dyspnea, weakness, abdominal distention, congestive heart failure, ataxia, incoordination, polyuria-polydipsia, and alopecia. Hypertension (paroxysmal or sustained) is also frequently present. However, pheochromocytomas can be incidental findings in dogs that have no clinical signs associated with the tumor.

Diagnostic Imaging

Adrenal tumors are difficult to detect radiographically unless they are associated with significant adrenal enlargement (≥20 mm) or calcification. Food should be withheld for 24 hours before radiography to allow the gastrointestinal tract to empty. In some dogs, mineralization of tissue cranial to the kidney may be seen on survey radiographs and may or may not be associated with obvious adrenal enlargement. This finding is suggestive of adrenocortical neoplasia (adenoma or carcinoma). Nonneoplastic mineralization of adrenal glands is rare in dogs; however, bilateral adrenal calcification may occur with PDH. Conversely, adrenal gland mineralization is considered an incidental finding in cats (Fig. 22.5). Hepatomegaly, calcinosis cutis, or osteoporosis may be seen with PDH and ADH. Enhanced abdominal contrast caused by increased abdominal fat may occur. Dogs with HAC are more likely to have calcium-containing uroliths than dogs without clinical evidence of HAC. Although pheochromocytomas may be detected radiographically if sufficiently large, ultrasonography and CT are more sensitive.

NOTE If you note mineralization of an adrenal gland radiographically in a dog, consider the possibility of neoplasia.

Ultrasonography is useful for assessing the adrenal size, echogenicity, and shape of the gland and whether invasion into adjacent structures has occurred. The normal canine adrenal gland size in mature dogs is dependent on body size. Several methods can be used to ultrasonographically assess the size of the adrenal gland. One method is to compare the maximal width with the length of the gland; this should be less than approximately

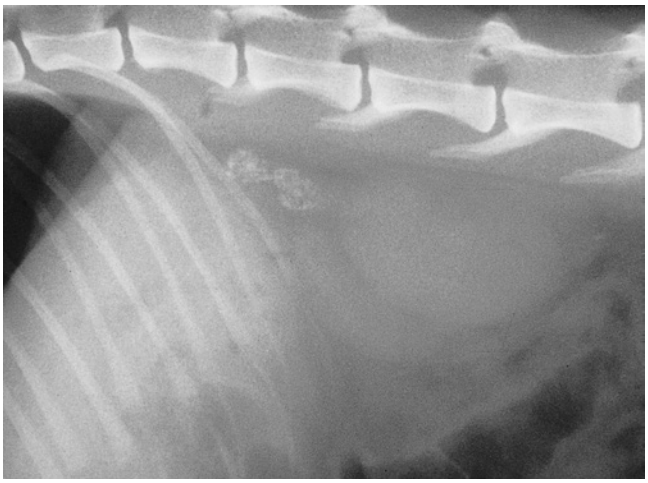


FIG. 22.5 Lateral abdominal radiograph of a cat with a mineralized adrenal gland. (Courtesy L. Homco, Ithaca, NY.)

30%. Alternatively, the width of the gland should not be larger in diameter than the diameter of the adjacent aorta. Absolute measurements have also been reported (e.g., the canine adrenal should be <7.4 mm; the feline adrenal should be <4.3 mm), but recently the adrenal thickness at the caudal pole was evaluated in context with body weight, sex, and age of the dog.[6] For nonadrenal gland illness, adrenal thickness is significantly lower in dogs weighing less than 12 kg and measurement should not exceed 0.62 cm, whereas adrenal thickness in dogs more than 12 kg should not exceed 0.72 cm.

Both adrenals should be routinely imaged in dogs with HAC because one normal adrenal gland does not exclude the existence of a contralateral functional adrenocortical tumor. Bilateral adrenocortical tumors are rare. Pheochromocytomas have been reported to have mixed echo patterns and cannot be definitively differentiated from adrenocortical tumors. Although bilateral adrenal enlargement is suggestive of PDH, atrophy of the contralateral adrenal gland in dogs with functional adrenocortical tumors may not be apparent ultrasonographically. Adrenal metastasis may be diagnosed ultrasonographically. In cats, unilateral or bilateral adrenal masses may be identified by abdominal ultrasound.

NOTE It is not possible to definitively differentiate between benign and malignant adrenal lesions using ultrasonographic criteria alone unless invasion of the vena cava has occurred.

CT and MRI allow accurate localization of adrenal neoplasia, but they do not allow differentiation of tumor type. Adrenal carcinomas may appear as well-demarcated, homogeneous masses, or they may be poorly demarcated, with irregular texture and contrast enhancement. Masses that are poorly demarcated, irregularly shaped, and nonhomogeneous with mineralization are usually carcinomas. Ultrasound can detect caudal vena cava invasion, but contrast-enhanced CT is more sensitive. Administration of contrast agents in patients with pheochromocytoma may produce severe hypertension; thus it should be done cautiously. If contrast-enhanced CT and ultrasound are not performed, caudal vena caval angiography (note precautions in previous discussion) may be considered before surgery if caudal vena caval thrombosis is suspected (Fig. 22.6). Excretory urography

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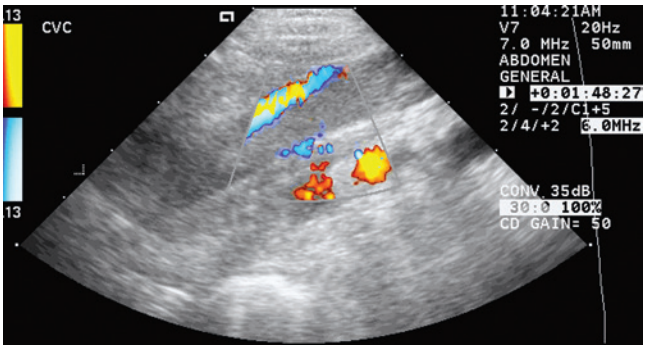


FIG. 22.6 Color Doppler evaluation of the caudal vena cava. A hyperechoic mass is seen within the lumen of the cava from invasion of an adrenal tumor. Note the disruption of blood flow around the mass *(blue)*.

may help identify tumor invasion requiring nephrectomy (e.g., ureteral obstruction or renal invasion).

NOTE CT and MRI do not differentiate adrenal adenomas, carcinomas, or pheochromocytomas. Clinical signs, biochemical testing, and a tissue sample are necessary for a definitive diagnosis.

Laboratory Findings

No laboratory changes are reliably seen in all animals with HAC; however, common laboratory abnormalities include substantially increased serum alkaline phosphatase, neutrophilic leukocytosis, lymphopenia, eosinopenia, mild polycythemia, modestly increased alanine aminotransferase, hypophosphatemia, and hypercholesterolemia. Mild hypernatremia and mild hypokalemia are rarely seen. Urinary tract abnormalities may include hyposthenuria (urine specific gravity <1.007) or isosthenuria (1.008–1.012). Urinary tract infections are common, even when bacteriuria and pyuria are absent.

Diagnosis of HAC requires clinical signs consistent with the disease. Abnormal adrenal function test results in the absence of clinical signs are not diagnostic of HAC. The ACTH stimulation test (Box 22.4) and the low-dose dexamethasone suppression (LDDS) test are the primary adrenal function tests used. The ACTH stimulation test is easy and quick to perform, and is the best test to look for iatrogenic HAC (Box 22.5). However, the ACTH stimulation test has disadvantages. Dogs with ADH can have almost any response (i.e., normal, exaggerated, or diminished) to ACTH, and many clinically ill dogs without HAC have exaggerated test results that mimic HAC. Classically, one measures cortisol concentrations before and after administration of ACTH, but one may also measure sex steroids (e.g., 17-hydroxyprogesterone). The sensitivity and specificity of sex steroid concentrations for diagnosing HAC are uncertain. The LDDS test requires 8 hours to perform, but it is a better test to look for spontaneous HAC. Furthermore, the LDDS test often permits differentiation of PDH and ADH.

Functional adrenocortical carcinoma may cause decreased aldosterone concentrations and increased deoxycorticosterone concentrations in the presence of hypokalemia. These metabolic abnormalities have been shown to resolve with resection of the carcinoma. Elevated serum aldosterone levels have been found in all cats with hyperaldosteronism secondary to an adrenal tumor. Other laboratory findings include hypokalemia, increased creatine kinase, alkalemia, hyperglycemia, hypophosphatemia, and hyponatremia, as well as evidence of chronic kidney disease.

It is critically important to note that HAC cannot be diagnosed simply by performing an adrenal function test; these tests may be substantially altered by nonadrenal disease or drugs. Exaggerated test results often occur in chronically stressed or ill dogs. In particular, animals with abnormal adrenal function tests but normal-sized adrenal glands should be carefully scrutinized before beginning therapy. PDH and ADH very rarely coexist. Because of the complexity of diagnosing HAC and differentiating PDH from ADH, readers are referred to a medical text for further information.

Laboratory abnormalities are inconsistent and nonspecific in animals with pheochromocytomas. However, measurement of urine and plasma catecholamines and metanephrines have been used to help differentiate between dogs with HAC and dogs with pheochromocytomas. Dogs with pheochromocytomas classically have significantly higher urinary normetanephrine- and metanephrine-to-creatinine ratios, and significantly higher plasma-total and -free normetanephrine and plasma-free metanephrine concentrations compared with dogs with HAC and dogs with nonadrenal disease.[7]

BOX 22.4 Adrenocorticotropic Hormone Stimulation Test in Dogs

1. Obtain serum to determine pretest cortisol concentration.
2. Administer 2.2 IU/kg of ACTH gel or 5 µg/kg of synthetic ACTH (Cortrosyn) IM or IV
3. Obtain serum for testing 1 hour after ACTH administration.

ACTH, Adrenocorticotropic hormone; *IM*, intramuscular; *IV*, intravenous.

BOX 22.5 Patterns of Adrenocorticotropic Hormone Stimulation Tests (Post–Adrenocorticotropic Hormone Cortisol)[a]

<24 µg/dL: suggestive of hyperadrenocorticism[b]
19–24 µg/dL: equivocal for hyperadrenocorticism
8–18 µg/dL: normal
<4 µg/dL: potentially consistent with iatrogenic Cushing disease
<1 µg/dL: consistent with hypoadrenocorticism, spontaneous or iatrogenic

[a]Substantial variation between laboratories may occur. To convert µg/dL to nmol/L, multiply µg/dL × 27.59.
[b]Severe nonadrenal disease can be associated with stress, causing values this high or higher; hyperadrenocorticism is not diagnosed simply by performing an adrenal function test.

DIFFERENTIAL DIAGNOSIS

Pheochromocytomas and adrenocortical tumors must be differentiated because operative management is different. Generally, clinical signs and laboratory analysis allow preoperative differentiation (see previous discussion). At surgery, pheochromocytomas may be identified grossly by application of Zenker's solution (potassium dichromate or iodate), which oxidizes catecholamines, forming a dark brown pigment within 10 to 20 minutes after application to the surface of a freshly sectioned tumor. Although adrenal carcinomas are apt to be large and invasive, differentiation of adenomas and carcinomas is impossible without histopathology. Apparent metastatic lesions in the liver or draining lymph nodes may suggest malignancy, but care should be taken to differentiate benign hepatic nodules from neoplastic disease.

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NOTE Be sure to submit the adrenal gland for histologic examination. Definitive diagnosis of adrenal tumors requires histopathology.

MEDICAL MANAGEMENT

Adrenergic blockage (e.g., phenoxybenzamine, phentolamine, prazosin) is used to control blood pressure in patients with pheochromocytoma. These drugs are also used preoperatively and intraoperatively (see discussions of Preoperative Management and Anesthesia on p. 586). If tachycardia or cardiac arrhythmias are present, β-adrenergic blockade may be used; however, unopposed β-blockade may cause severe, life-threatening hypertension. β-Blockade should not be started until α-adrenergic blockade has been determined to be adequate. In one study, phenoxybenzamine-treated dogs undergoing adrenalectomy for pheochromocytoma had a significantly decreased mortality rate compared with untreated dogs (13% vs. 48%, respectively).[8] In general, it is recommended to pretreat dogs for 2 weeks with phenoxybenzamine (see p. 594) prior to surgery for a pheochromocytoma.

Medical therapy for HAC is potentially complex and can have significant side effects. Therefore the reader is referred to a current medical text for a more complete discussion. Trilostane inhibits one of the synthetic enzymes, thus blocking production of cortisol and other adrenal steroids; it usually does not cause adrenal necrosis. Trilostane appears to be as safe or perhaps safer than other drugs used to treat HAC and is effective in most patients (Box 22.6). The major disadvantages are that it must be given daily and it has occasional side effects (e.g., hypoadrenocorticism, adrenal necrosis).

Mitotane (o,p′-DDD) destroys the adrenal cortex in a dose-dependent fashion. It was the major drug used to treat canine HAC before the advent of trilostane. Mitotane can often control clinical signs in animals with ADH; however, tumors are more resistant to the adrenocorticolytic effects of mitotane than are normal or hyperplastic adrenal cortices. Larger doses (see Box 22.6) are often required to attain and maintain control in ADH dogs than in PDH dogs, and stronger side effects (e.g., gastric irritation, vomiting) can be expected. Major advantages of mitotane include the following: (1) it is effective, and (2) after an induction therapy (usually 4–14 days), maintenance therapy consists of one to two treatments per week. Major disadvantages are: (1) it can easily destroy the entire adrenal gland, resulting in temporary or permanent hypoadrenocorticism (or death), (2) some dogs are resistant to its effects, and (3) some dogs are very sensitive to its effects. The last two disadvantages mean that there is substantial variation in how patients respond. Some patients respond well and are easy to treat, whereas others are extremely difficult to control using mitotane. For this reason, trilostane has become the more popular treatment for HAC.

Ketoconazole causes reversible inhibition of adrenal steroid production and has little effect on mineralocorticoid production. Therefore ketoconazole may be used (1) in dogs with ADH who are not surgical candidates, (2) before surgery to reduce anesthetic and surgical risks in animals with uncontrolled HAC, and (3) as a diagnostic trial in dogs in which equivocal test results make the diagnosis of HAC difficult. If used for diagnostic purposes, the drug should be given for a minimum of 4 to 8 weeks. Major advantages of ketoconazole include the following (1) it is relatively safe, and (2) it is very effective. Major disadvantages are: (1) it works only while there are blood levels of drugs, (2) it is relatively expensive, and (3) it is so effective at decreasing cortisol concentrations that it is easy to make patients feel sick when first starting the drug. Furthermore, adverse reactions to ketoconazole (e.g., anorexia, depression, vomiting, diarrhea, icterus) may occur, necessitating that the drug be dropped or the dosage reduced. If an overdose is suspected of causing acute illness or collapse, one should administer glucocorticoids and stop the ketoconazole. As previously noted, it is seldom used for HAC since the introduction of trilostane.

Amlodipine, spironolactone, and potassium gluconate are used in the medical management of primary hyperaldosteronism associated with adrenal tumors in cats. Aldosterone is released from adrenal glands via the action of the renin-angiotensin-aldosterone system. Aldosterone stimulates renal sodium reabsorption, resulting in volume expansion. Signs of hyperaldosteronism typically result from systemic hypertension. Aldosterone also stimulates potassium excretion and may result in a hypokalemic myopathy.

BOX 22.6 Medical Treatment for Adrenal Tumors

Trilostane (Primary Drug Used for Adrenal Tumors)

1. Start with 1 mg/kg bid or 2 mg/kg once daily (twice daily is probably more effective). If that dose is insufficient, then gradually increase the dose orally with food. Observe for signs of lethargy, vomiting, diarrhea, or decreased appetite.
2. Perform a clinical examination, serum biochemistry profile, and ACTH stimulation 4–6 hours after the morning capsule on days 10–14.
3. Increase the dose until the owner reports a good clinical response and the post-ACTH serum cortisol is <9 µg/dL.

Mitotane[a]

1. Administer 50–75 mg/kg once daily with food plus 0.2 mg/kg prednisolone/day. Observe for signs of lethargy, vomiting, diarrhea, or decreased appetite. Recheck dog in 10–14 days. If no response, then increase dose by 50 mg/kg/day.
2. Perform ACTH stimulation test. Appropriate response is ACTH-stimulated cortisol <1 µg/dL. If patient is responding, decrease mitotane to 75–100 mg/kg/wk and continue prednisolone. If no response at 14 days, then increase mitotane by 50 mg/kg/day (continue prednisolone) for another 14 days and recheck.

ACTH, Adrenocorticotropic hormone.
[a]A total cumulative dose of up to 3000 to 5000 mg/kg may be needed. Treatment for ablation generally is 10 days to 11 weeks (mean, 24 days). Two-thirds of dogs eventually relapse.

SURGICAL TREATMENT

The overall health of the animal, the presence of nonresectable metastases, and the apparent invasiveness of the tumor (i.e., evidence of caudal vena cava thrombosis on CT or ultrasound) should be considered when the appropriateness of surgery for adrenal tumors is determined. Long-term survival (>1 year) may be possible, even in dogs with widespread metastatic lesions. If the tumor appears invasive, a midline abdominal approach is preferred to allow evaluation of the caudal vena cava and other abdominal structures. Thrombus removal may require that the midline incision be extended into the caudal thorax through a caudal median sternotomy approach (see p. 891). Thrombi more commonly occur because of intraluminal extension via the adrenal or renal vein, and less commonly by direct invasion. Caval thrombi occur in approximately one-fourth of dogs with adrenal gland tumors. They are more common with pheochromocytoma than with adrenocortical tumor, but they may occur with either. Venotomy may be used to remove tumors extending into the caudal vena cava. If venotomy cannot be performed, gradual

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occlusion of the caudal vena cava may allow removal of adrenal gland tumors with vascular invasion that would otherwise be difficult or impossible to resect; en bloc resection of the caudal vena cava during removal of a pheochromocytoma may also be performed. Small tumors and those that do not appear invasive may be removed through a paralumbar approach (see p. 588). Laparoscopic adrenalectomy has also been performed in dogs and cats (see p. 588).

Preoperative Management

Preoperative management for a function adrenal tumor causing hyperadrenocorticism should include 3 to 4 weeks of trilostane therapy (see Box 22.6). Renal function should be determined before surgery in case ipsilateral nephrectomy is necessary. Substantial electrolyte or acid-base abnormalities, blood glucose concentrations greater than 200 mg/dL, and hypertension should be corrected before surgery, if possible. Fluid therapy should be initiated before induction of anesthesia. Animals with HAC are at increased risk of postoperative PTE (see p. 586). Fresh-frozen plasma transfusions, aspirin, and/or plasma incubated with heparin may be indicated in patients with disseminated intravascular coagulation (DIC) (Box 22.7). Aspirin, heparin, and clopidogrel have been used preoperatively in patients with adrenocortical tumor to prevent thromboembolism (see Box 22.7), but the value of these preventative measures is controversial. Heparin (75–100 U/kg) may be added to plasma during surgery, with the animal continuing to take heparin for 3 to 4 days. Low-molecular-weight heparin (150 U/kg) may be more effective than unfractionated heparin but requires different monitoring than unfractionated heparin (see Box 22.7). Perioperative antibiotics should be administered and continued in the immediate postoperative period in hyperadrenal animals. Do not give steroids to animals with functional adrenal tumors before surgery as they may increase blood pressure and promote PTE. Wait until surgery to give steroids in these animals.

Particular emphasis should be placed on preoperative examination of the cardiovascular system for evidence of arrhythmias or congestive heart failure in animals with pheochromocytomas. If cardiac arrhythmias are present, a β-blocker may be added, but only after the phenoxybenzamine dosage (see under Anesthesia) has been determined to be adequate and blood pressure has returned to normal. Both α- and β-blockade will allow the return of a normal fluid volume; however, they may unmask renal insufficiency and anemia. α-Adrenergic blockade has been shown to drastically reduce the incidence of severe perioperative hypertension, thereby reducing mortality (Table 22.1).

Anesthesia

Anesthetic complications are common during adrenalectomy for pheochromocytoma, and wide fluctuations in heart rate (HR) and blood pressure are typical in unblocked animals. Even if hypertension is well controlled, patients can be extremely difficult to manage under anesthesia. Appropriate monitoring is critical and includes cardiac rhythm, arterial blood pressure, end-tidal carbon dioxide ($EtCO_2$), and pulse oximetry. Treatment for several weeks before surgery with an α-adrenergic blocker (e.g., phenoxybenzamine; see Table 22.1) is recommended. An initial dose of phenoxybenzamine, 0.25 mg/kg given orally every 12 hours is gradually increased every 2 to 3 days until blood pressure is within the normal range. This process may take 1 to 2 weeks before the patient is adequately blocked. Maximum dose is 2 mg/kg. HR can be controlled with a β-blocker (e.g., metoprolol, esmolol); however, this treatment should not be initiated until adequate α-blockade has been established (i.e., normal blood pressure). Intraoperative β-blockade with esmolol (see Table 22.1) is preferred because of its short half-life and can be given as boluses or constant-rate infusion (CRI). Cardiac arrhythmias may be treated with lidocaine (Box 22.8) or esmolol (see Table 22.1). Hypertension may result from tumor manipulation and can be minimized by isolating the blood supply of the tumor before manipulating the tumor. Hypertension may be treated with phentolamine given as an intravenous (IV) bolus (see Table 22.1). Sodium nitroprusside or nitroglycerin may also be infused if hypertension is present. Hypotension frequently occurs after tumor removal; large doses of crystalloids should be given to replace estimated blood loss, as well as third-space fluid loss. If hypotension persists, dobutamine could be administered (2–10 μg/kg per minute IV). In human pheochromocytoma patients, the vasopressors of choice are phenylephrine and norepinephrine. Because removal of the tumor initiates cessation of norepinephrine release into the bloodstream, infusion of an alpha-1 agonist (phenylephrine or norepinephrine) provides

BOX 22.7 Therapeutics for Disseminated Intravascular Coagulation

Plasma (Fresh Frozen)[a]

10–20 mL/kg, then reassess plasma ATIII activity. Repeat as needed to increase ATIII to near-normal concentrations.

NOTE: Large amounts of FFP may be necessary to increase ATIII, but it is thought that adequate levels of ATIII are critical in these patients.

Aspirin[b]

0.5 mg/kg PO q24h (efficacy not proven in dogs or cats)

Clopidogrel

Loading dose of 2–3 mg/kg (some suggest as high as 10 mg/kg), then 1–2 mg/kg q24h (efficacy not proven in dogs or cats)

Heparin-Activated Plasma[c]

Place the first heparin dose (50–100 U/kg) into the plasma and incubate for 30 minutes before administration. Once ATIII levels are above 60%, continue the heparin subcutaneously. If additional plasma is needed, incubation with heparin is not necessary (efficacy not proven in dogs or cats).

Heparin (Unfractionated)[c,d]

50–300 U/kg SC q8–12h; adjust dose based on monitoring

Low-Molecular-Weight Heparin (Dalteparin)[c]

100–150 U/kg SC q8–24h (dogs)
180 U/kg SC q6h (cats)

[a]Replacing clotting factors and ATIII is probably the best therapy for DIC; however, large doses of FFP are required to raise ATIII. Thus the efficacy of this treatment has been questioned.
[b]Is believed effective in DIC associated with immune-mediated hemolytic anemia; efficacy in DIC caused by other diseases unproven.
[c]Heparin is controversial in the treatment of DIC. Low-molecular-weight heparin is probably more effective than unfractionated heparin, but its effectiveness for treating DIC is questionable. Substantial differences have been noted between the two forms, and the reader is referred to a medical text for a more complete discussion, including differences in monitoring the effectiveness of therapy. As more becomes known regarding low-molecular-weight heparin, this dosage recommendation may change.
[d]Heparin is no longer considered to be an optimal treatment for DIC. There are no adequately designed publications in veterinary medicine critically evaluating heparin therapy in patients with DIC. Rightly or wrongly, current thinking at the time of this writing is that heparin should not be used for patients with DIC that have concurrent inflammatory conditions, those that are actively bleeding, or those that appear hypocoagulable. Although heparin seems beneficial in patients that are at risk for thromboembolism, there is no study that confirms this hypothesis. Readers are referred to a medical text for advances in the treatment of DIC.
ATIII, Antithrombin III; *DIC,* disseminated intravascular coagulation; *FFP,* fresh-frozen plasma; *SC,* subcutaneous.

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TABLE 22.1 Anesthetic Considerations in the Canine Pheochromocytoma Patient

Preoperative Considerations

Associated conditions	• Anemia (may be occult) • Hypovolemia • Hypertension • Tachydysrhythmias • Ventricular ectopy • Cardiac dysfunction • Renal insufficiency (may be occult) • Cardiogenic pulmonary edema
Bloodwork	• HCT • Electrolytes • BUN • Cr • TP • Urinalysis
Physical examination	May be hypovolemic, tachycardic, and hypertensive if untreated with phenoxybenzamine. May have orthostatic hypotension if treated.
Other diagnostics	• Blood pressure is essential • ECG • Radiographs (thoracic, abdominal) • Ultrasound
Premedications	Give: • Phenoxybenzamine until the morning of surgery • Diazepam (0.2 mg/kg IV), *or* • Midazolam (0.2 mg/kg IV, IM), *plus* • Hydromorphone (0.05–0.2 mg/kg IV, IM), *or* • Oxymorphone (0.1–0.2 mg/kg IV, IM), *or* • Morphine[a] (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM) • Avoid ketamine, xylazine, medetomidine, dexmedetomidine, atropine, glycopyrrolate, and acepromazine

Intraoperative Considerations

Induction	• Titrate propofol (2–4 mg/kg IV if sedated or 4–8 mg/kg IV if unsedated), *or* • Give alfaxalone (2–3 mg/kg IV if sedated or 3–5 mg/kg IV if unsedated) • If CHF, titrate etomidate (0.5–1.5 mg/kg)

Maintenance	• Isoflurane or sevoflurane *plus*
	  • Fentanyl (2–10 µg/kg IV PRN) for short-term pain relief, *plus*
	  • Fentanyl CRI (1–5 µg/kg IV loading dose, then 2–30 µg/kg/h IV), *or*
	  • Hydromorphone (0.05–0.2 mg/kg IV PRN), *or*
	  • Oxymorphone (0.1–0.2 mg/kg IV PRN), *or*
	  • Morphine[a] (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM) if minimal hypotension, *plus*
	• For hypertension (to keep MAP 70–90 mm Hg)
	  • Phentolamine (0.02–0.1 mg/kg IV) bolus and/or CRI (0.5–3 µg/kg/min IV) *and*
	  • Nitroprusside (0.5–5 µg/kg/min IV) *or*
	  • Nitroglycerin (1–5 µg/kg/min IV) *and*
	  • Esmolol (0.05–0.25 mg/kg IV) boluses every 2–5 min to effect and/or CRI (50–200 µg/kg/min IV) to maintain normal heart rate
	• For hypotension (to keep MAP 70–90 mm Hg)
	  • Phenylephrine (20–200 µg IV boluses and/or CRI 0.1–1 µg/kg/min IV), *or*
	  • Norepinephrine CRI (0.05–2 µg/kg/min IV), *or*
	  • Dopamine (5–15 µg/kg/min IV)
	• For hypotension with CHF
	  • Epinephrine (0.1–1 µg/kg/min IV) *or*
	  • Dobutamine (2–15 µg/kg/min IV)

Fluid needs	• 5–10 mL/kg/h to replace evaporative losses plus 3 × EBL If CHF, then 5–10 mL/kg and slower replacement of fluid losses over 3–4 h if necessary
Monitoring	• BP: essential • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature • Arterial line • U/O
Blocks	Epidural: • Morphine (0.1 mg/kg preservative free) *or* • Buprenorphine (0.003–0.005 mg/kg diluted in saline) Incisional: • Lidocaine (<5 mg/kg), *or* • Bupivicaine (<2 mg/kg)

Continued

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TABLE 22.1 Anesthetic Considerations in the Canine Pheochromocytoma Patient—cont'd

Postoperative Considerations

Analgesia	• Fentanyl CRI (1–10 µg/kg loading dose, then 2–20 µg/kg/h IV), *or* • Morphine[a] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h) if minimal hypotension *or* • Hydromorphone (0.05–0.2 mg/kg IV, IM q3–4h), *or* • Hydromorphone CRI (0.025–0.1 mg/kg/h IV), *or* • Oxymorphone (0.1–0.2 mg/kg IV q2–4h)
Monitoring	• SpO_2 • Blood pressure is essential • ECG • HR • Respiratory rate • Temperature • U/O
Bloodwork	• HCT • TP • Serial BUN/Cr for the next 2–4 weeks
Estimated pain score	Moderate to severe

[a]Give slowly to prevent histamine release.
BP, Blood pressure; *BUN,* blood urea nitrogen; *CHF,* congestive heart failure; *Cr,* creatinine; *CRI,* constant-rate infusion; *EBL,* estimated blood loss; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *MAP,* mean arterial pressure; *PRN,* as needed; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein; *U/O,* urine output.

BOX 22.8 Lidocaine Administration for Ventricular Arrhythmias

Dogs

Give IV (2 mg/kg bolus, up to 8 mg/kg total dose) to determine responsiveness to this drug. If the arrhythmias decrease or stop, lidocaine should be given by IV CRI of 50–75 µg/kg/min (for 50 µg/kg/min, place 500 mg lidocaine in 500 mL of fluids and administer at maintenance rate [66 mL/kg/d]).

Cats

Give IV (1 mg/kg bolus, up to 4 mg/kg total dose); if necessary administer 25–50 µg/kg/min by CRI.

CRI, Constant-rate infusion; *IV,* intravenous.

reliable vasoconstriction. These tumors tend to be highly vascular, and significant intraoperative hemorrhage may require blood transfusions, particularly if caudal vena cava venotomy is performed to remove a thrombus.

Atropine, glycopyrrolate, xylazine, medetomidine, dexmedetomidine, and ketamine should not be used in patients suspected of having a pheochromocytoma. As anticholinergics, atropine and glycopyrrolate block the parasympathetic pathways, allowing the sympathetic nervous system to be unopposed. Tachyarrhythmias and severe hypertension are potential side effects, especially in patients with pheochromocytoma. Xylazine, medetomidine, and dexmedetomidine are primarily alpha-2 agonists. They typically cause transient hypertension followed by prolonged hypotension. Although they may increase myocardial sensitivity to catecholamines, changes in blood pressure make alpha-2 agonists an undesirable addition to the anesthetic protocol. Ketamine should be avoided because it increases HR, blood pressure, and circulating levels of catecholamines. Because an increase in arterial CO_2 causes an increase in catecholamine release, $EtCO_2$ monitoring and prevention of hypoventilation decrease the chance of an additional catecholamine response. If etomidate is used in patients with arrhythmias, the need for perioperative steroid replacement should be anticipated. When etomidate is used, the advantages of cardiovascular stability should be weighed against the possibility of transient adrenal suppression, which may occur in patients undergoing unilateral adrenalectomy. Isoflurane and sevoflurane are the inhalation agents of choice because they do not sensitize the myocardium to epinephrine-induced arrhythmias; halothane should be avoided. In addition, drugs that release histamine such as morphine and meperidine should be avoided. Other opioids such as hydromorphone and/or fentanyl provide good pain control without histamine release.

Surgical Anatomy

See p. 587 for the discussion of the surgical anatomy of the adrenal gland.

Positioning

The animal is positioned in dorsal recumbency or in lateral recumbency with the affected side up, depending on the operative approach chosen. With large or invasive tumors, a generous area should be clipped and prepared for surgery to allow a caudal thoracotomy (median sternotomy) to be performed if necessary.

SURGICAL TECHNIQUE

Adrenalectomy via a midline abdominal or a paralumbar approach is described on pp. 587 to 588. Laparoscopic adrenalectomy is discussed on p. 588. Concurrent nephrectomy may be necessary in some patients with invasive tumors. Surgical resection of adrenal tumors should be aggressive to ensure complete tumor removal. En bloc resection should be performed if possible to prevent leaving behind small fragments of neoplastic tissue. The vascular supply to pheochromocytomas should be isolated before tumor manipulation to reduce catecholamine release and help prevent shedding of tumor cells. Venotomy may be required to remove tumor thrombi. The entire abdomen should be explored, with special attention paid to the bladder, pelvic canal, kidneys, and aorta near the junction of the caudal mesenteric artery, where extra-adrenal neoplasia is reported to occur.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Care should be taken when selecting the appropriate suture because delayed wound healing may occur in any debilitated

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animal (see p. 589). Self-retaining retractors (e.g., Balfour abdominal retractors) and malleable retractors are useful for improving visualization of the adrenal glands. With vascular tumors, hemostasis is attained more easily with electrocautery, Ligasure, and hemoclips than with suture ligation of vessels.

POSTOPERATIVE CARE AND ASSESSMENT

Animals with HAC caused by ADH often develop hypoadrenocorticism postoperatively as a result of atrophy of the contralateral gland. These animals require glucocorticoid therapy postoperatively (see discussion of postoperative care on p. 589). If HAC continues postoperatively, medical therapy should be considered. Fluid therapy should be continued until the animal is able to maintain hydration. Blood pressure, HR, and heart rhythm should be carefully monitored after surgery. Blood transfusions may be required intraoperatively or postoperatively in some patients. Dogs should be reevaluated periodically for tumor recurrence. The most common complications after adrenal tumor removal include dyspnea, hemoperitoneum, ventricular arrhythmias, anuric acute renal failure, and coagulopathies. Dogs with adrenocortical tumors are at increased risk of developing thromboembolism (see p. 586).

PROGNOSIS

Although there can be significant perioperative complications, median survival for dogs that survive to discharge exceeds 10 months. In a study of 52 dogs undergoing adrenalectomy, survival time was significantly shorter in dogs with carcinoma, tumors with a major axis length greater than 5 cm, thrombosis, metastasis, and when adrenalectomy was combined with another abdominal surgical procedure.[9] Dogs in this study had a median survival of 953 days, with more than 65% living for more than 1 year.

Perioperative mortality ranges from 10% to 20%. Risk factors that have been identified for poor short-term survival (death <14 days after surgery) include caval invasion, extent of invasion (extension beyond the hepatic hilus), receipt of intraoperative transfusion, dogs with pheochromocytoma, and postoperative complications such as DIC, pancreatitis, hypotension, hypoxemia, and renal failure.[10] In a study of nine dogs undergoing bilateral adrenalectomy, median survival of the eight dogs that survived to hospital discharge was 525 days; no dogs died of metastatic disease or from complications of hypoadrenocorticism.[11]

Outcomes following laparoscopic adrenalectomy have been reported in several studies. When comparing perioperative morbidity and mortality rates for laparoscopic versus open adrenalectomy in dogs with noninvasive adrenocortical tumors, laparoscopic adrenalectomy was associated with shorter surgical and hospitalization times, with no perioperative deaths.[3] In a study of 10 dogs that had laparoscopic removal of noninvasive pheochromocytomas, only one dog required conversion to an open approach.[12] Prognostic factors for improved survival for pheochromocytoma include preoperative use of phenoxybenzamine, younger age, lack of intraoperative arrhythmias, and decreased surgical time.[13]

Surgical treatment of adrenal gland tumors in cats has good long-term survival regardless of tumor type. Perioperative mortality (death <14 days after surgery) was 23% in a 2016 study; median survival time was 50 weeks.[4]

NOTE Warn owners that animals with pheochromocytoma may die suddenly as a result of arrhythmias and hypertension.

PITUITARY NEOPLASIA

DEFINITIONS

Pituitary tumors arise from the hypophysis in the **sella turcica. Hypophysectomy** is the surgical removal of the pituitary gland. The pituitary gland is also called the *hypophysis.*

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Functional pituitary tumors are the most common cause of canine HAC. However, 40% of pituitary tumors are nonfunctional. Clinical signs are usually caused by hypersecretion of ACTH from tumors in the pars distalis (adenohypophysis) or pars intermedia. Large pituitary tumors often grow dorsally into the brain because the diaphragm of the sella is incomplete. Such tumors may cause clinical signs by impinging on adjacent brain tissue (e.g., optic chiasm, hypothalamus, thalamus, infundibular recess, and third ventricle). Size of the tumor and development of neurologic signs do not always correlate. Adenomas and carcinomas may arise from pituitary tissue; however, carcinomas represent less than 3% of all pituitary neoplasms. Adenomas are usually classified as microadenomas (<1 cm in diameter) or macroadenomas (>1 cm in diameter). Microadenomas are most common, accounting for nearly 70% of all pituitary tumors.

DIAGNOSIS

Clinical Presentation

Signalment

Poodles, Malteses, dachshunds, and boxers may be predisposed to PDH. Middle-aged and older dogs are most commonly affected; however, young dogs may occasionally develop pituitary tumors.

History

Most dogs are presented for evaluation of typical signs of HAC (polyuria, polydipsia, polyphagia, abdominal enlargement, endocrine alopecia, muscle wasting, weakness, lethargy, panting, and/or hyperpigmentation). Concurrent neurologic signs (e.g., seizures, visual deficits, ataxia, incoordination, facial hemiplegia, head tilt, somnolence, compulsive walking, depression) may be noted. The diversity of neurologic signs in dogs with pituitary tumors is probably a result of impingement on various parts of the brain responsible for differing functions. Mental depression and stupor were reported as the most common abnormalities in two studies describing dogs with large pituitary tumors. In animals with nonfunctional macroadenomas or carcinomas, neurologic signs may be the only presenting abnormality, and some owners report only anorexia with no obvious CNS abnormalities.

Cats with insulin-resistant DM secondary to pituitary adenoma present with a ravenous appetite, increased body weight, polyuria/polydipsia, and a dull hair coat.

NOTE Large, nonfunctional pituitary tumors may cause neurologic abnormalities. Functional tumors usually cause hyperadrenocorticism but may also cause neurologic signs.

Physical Examination Findings

Typical signs of HAC (see previous discussion) are expected in animals with functional pituitary tumors. Neurologic

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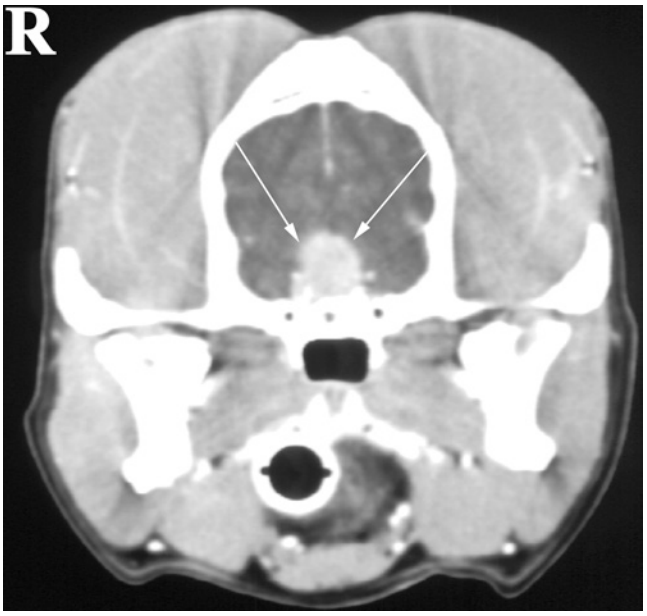


FIG. 22.7 Contrast-enhanced computed tomography image of a dog with a macroadenoma of the pituitary gland *(arrows)*.

abnormalities (e.g., papillary edema, ataxia, incoordination) and poor appetite occasionally occur as the only signs.

Diagnostic Imaging

Diagnosis of pituitary neoplasia is best made with CT or MRI (Fig. 22.7). Pituitary adenomas and carcinomas cannot be differentiated with CT; however, animals with microadenomas, which might benefit from hypophysectomy, can be differentiated from animals with macroadenomas. The latter seldom benefit from surgery. Bilateral adrenal enlargement is usually indicative of PDH.

Laboratory Findings

Laboratory abnormalities are generally consistent with HAC. Small, nonfunctional pituitary tumors seldom cause laboratory abnormalities. Large tumors may cause increased intracranial pressure. See p. 592 for differentiation of PDH from ADH. In a diabetic cat with insulin resistance secondary to somatotroph pituitary adenoma, plasma concentrations of growth hormone (51 µg/L, reference range 0.8–7.2 µg/L) and insulin-like growth factor 1 (3871 µg/L, reference range 39–590 µg/L) were highly elevated.

DIFFERENTIAL DIAGNOSIS

Animals with PDH must be differentiated from those with iatrogenic HAC or ADH (see p. 592). Once a diagnosis of pituitary dysfunction has been made, pituitary neoplasms must be differentiated from other lesions that may arise in the pituitary (e.g., cysts, abscesses, and craniopharyngiomas); however, such lesions are rare.

MEDICAL MANAGEMENT

Clinical signs of HAC may be treated medically (see p. 593). External-beam radiation therapy (RT) appears to be an effective treatment for large pituitary tumors when combined with concurrent adrenal-suppressive treatment (e.g., trilostane, mitotane, ketoconazole). Long-term survival may be possible with RT. Single-fraction modified radiosurgery is a safe and effective approach to the treatment of pituitary tumors in cats. In one study, 11 client-owned cats referred for treatment of pituitary tumor causing neurologic signs or poorly controlled DM secondary to acromegaly or pituitary-dependent HAC underwent MRI of the brain to manually plan RT.[14] Modified radiosurgery was performed by delivering a single large dose (15 or 20 Gy) of radiation while arcing a linear accelerator–generated radiation beam around the cat's head with the pituitary mass at the center of the beam. Eight cats were treated once, two cats were treated twice, and one cat received three treatments. Five of nine cats with poorly regulated DM had improved insulin responses, and both cats with neurologic signs had clinical improvement. No confirmed acute or late adverse radiation effects were reported. Overall median survival was 25 months (range, 1–60 months), and three cats were still alive at the time of the report.

RT in dogs with pituitary masses may enhance survival and control neurologic signs. In a retrospective study, 19 dogs with pituitary masses identified on CT or MRI were irradiated with 48 Gy given in daily-dose fractions of 3 Gy.[15] Twenty-seven dogs with pituitary masses that received no RT were used for comparison. Mean survival time in the treated group was 1405 days (range, 1053–1757 days) with 1-, 2-, and 3-year estimated survival of 93%, 87%, and 55%, respectively. Median survival in the nonirradiated group was 359 days (range, 48–916 days), with a mean of 551 days (range, 271–829 days). The 1-, 2-, and 3-year estimated survival was 45%, 32%, and 25%, respectively. Dogs that received RT for their pituitary tumors had significantly longer survival times than untreated dogs. Treated dogs with smaller tumors (based on maximal pituitary-to-brain height ratio or area of tumor to area of brain) had longer survival than those with larger tumors ($P < .001$). Stereotactic radiosurgery and stereotactic radiotherapy have been shown to be effective treatments for reducing tumor volume, particularly in dogs with pituitary tumors.[16]

SURGICAL TREATMENT

Hypophysectomy can be performed in animals with pituitary microadenomas and functional adenohypophyseal hyperplasia (rare); however, it is seldom performed by veterinary surgeons. Advocates of this procedure suggest that most dogs with PDH are surgical candidates for hypophysectomy, and that this technique is preferable to long-term medical management. The transoral paramedian approach to the pituitary gland has been advocated to remove pituitary tumors, even though some normal tissue may be left behind. Dogs with normal-sized pituitary glands may have fewer postoperative complications than dogs with enlarged pituitary glands. However, if concurrent neurologic signs are present, or if the tumor has extended intracranially or transsphenoidally, hypophysectomy is not indicated. Hypophysectomy should not be considered in animals intended for breeding purposes because it renders them infertile. Transphenoid cryohypophysectomy has been performed safely and effectively in cats. Cats may present with acromegaly caused by elevated growth hormone and insulin-resistant DM.

NOTE Hypophysectomy should be performed only by surgeons familiar with the regional anatomy and experienced in the technique.

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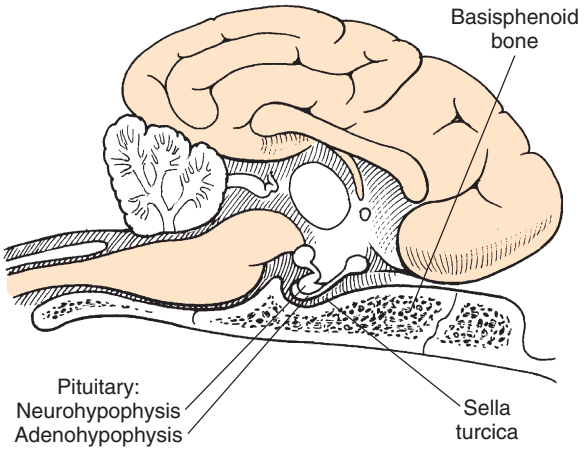


FIG. 22.8 Location of the pituitary gland.

Preoperative Management

If surgery is considered for a pituitary neoplasm, extensive preoperative workup is indicated to confirm and localize the lesion. CT or MRI of the pituitary fossa (including a contrast-enhanced image) is used to determine the height and width of the pituitary gland and to localize pertinent landmarks. Animals with HAC are at increased risk of developing postoperative infection because of high levels of circulating glucocorticoids. Perioperative prophylactic antibiotics are recommended. See p. 594 for additional comments on the preoperative management of animals with HAC.

Anesthesia

Most animals with pituitary tumors do not require special anesthetic consideration; however, patients with large masses that increase intracranial pressure need special precautions. Fluid therapy should be restricted to the volume required to maintain adequate circulation. Isoflurane and sevoflurane are the inhalants of choice because they interfere less with autoregulation of cerebral blood flow than does halothane. The faster recovery that sevoflurane offers provides some advantage over isoflurane. Patients with increased intracranial pressure should be modestly hyperventilated ($EtCO_2$ approximately 30 mm Hg) during surgery. For further discussion of increased intracranial pressures and preoperative management and anesthetic considerations, see Chapter 39, pp. 1338–1342, and Table 39.2.

Surgical Anatomy

The pituitary is a small appendage of the diencephalon (Fig. 22.8). It occupies a shallow, oval recess in the basisphenoid bone called the *sella turcica.* The gland varies greatly in size among breeds of dogs and within the same breed, but it is usually approximately 1 cm long. The pituitary is composed of the adenohypophysis and the neurohypophysis, and the adenohypophysis is further subdivided into the pars proximalis, the pars intermedia, and the pars distalis. The arterial supply of the pituitary arises from the internal carotid arteries and caudal communicating arteries.

SURGICAL TECHNIQUE

AP Transsphenoidal, intracranial, and peripharyngeal approaches have been described for hypophysectomy.

BOX 22.9 Postoperative Drug Therapy After Hypophysectomy

Desmopressin Acetate[a]

Nasal preparation: give 1–4 drops of 100 µg/mL intranasally or in conjunctiva q12–24h

Parenteral form: give 0.5–2 µg/dog SC or IV q12–24h

Tablets: 0.05–0.1 mg/kg q12h PO; can increase to 0.2 mg/kg if needed

Hydrocortisone (Solu-Cortef) or Prednisolone

Hydrocortisone: 1 mg/kg IV q6h

Prednisolone: 0.2 mg/kg q24h

Levothyroxine (Soloxine, Thyro-Tabs, Synthroid)

18–22 µg/kg PO q12h

[a]Can often be discontinued 2 weeks after surgery.
IV, Intravenous; *PO,* orally; *SC,* subcutaneous.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Delayed wound healing may occur in animals with HAC; therefore incisions should be closed with strong, slowly absorbed or nonabsorbable suture material (e.g., polydioxanone, polyglyconate, polypropylene, nylon).

POSTOPERATIVE CARE AND ASSESSMENT

See p. 597 for the postoperative management of animals with HAC. See also Box 22.9.

COMPLICATIONS

Diabetes insipidus–like symptoms have been reported after hypophysectomy in dogs, although these symptoms tend to resolve in approximately 2 weeks. It is unclear whether return of arginine vasopressin secretion is involved. The presence of increased ACTH concentration after hypophysectomy is a risk factor for recurrence of HAC.

PROGNOSIS

Long-term survival is possible after hypophysectomy, RT plus chemotherapy, or chemotherapy alone in dogs with PDH caused by microadenomas. Long-term survival has also been reported in dogs with large, functional tumors after RT. Successful radiation treatment for pituitary tumors has been reported in cats. In a study of 306 dogs with PDH undergoing transsphenoidal hypophysectomy, median survival was 781 days and median disease-free interval was 951 days.[17]

Surgery of the Pancreas

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Pancreatectomy is surgical removal of all or part of the pancreas. **Insulinoma** is a functional tumor of pancreatic β-islet cells; excessive insulin production commonly causes hypoglycemia in affected animals (see p. 609). **Zollinger-Ellison** syndrome is a

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condition caused by non–β-islet cell tumors, in which excess gastrin is secreted.

PREOPERATIVE MANAGEMENT

Although acute onset of anorexia, vomiting, and anterior abdominal pain have been considered the hallmarks of canine pancreatitis, affected dogs may have a wide range of clinical signs, ranging from peracute to chronic and including ascites, shock, dyspnea, and melena. Clinical signs in cats are even more variable, with the primary signs being lethargy, poor appetite, and dehydration; vomiting is much less common or much less noticeable in this species. It is important to try to diagnose pancreatitis before surgery because (1) these animals often neither require nor benefit from surgery if pancreatitis is the major problem (dogs with abscesses may be different; see later discussion) and (2) poor visceral perfusion due to anesthesia and/or unnecessary manipulation of the pancreas during surgery can exacerbate the disease.

Abdominal ultrasonography has been an important tool in the diagnosis of pancreatitis. Ultrasonography is useful in diagnosing pancreatic disease (especially pancreatitis and pancreatic abscess) and in guiding aspirates and biopsies. Sensitivity has been suggested to be in the 40% to 60% bracket. However, it is worth noting that the ultrasonographic appearance of the pancreas in a dog with pancreatitis can change remarkably in hours; therefore repeating ultrasonography a day later might increase its sensitivity. Pancreatitis, pseudocysts, abscesses, neoplastic lesions, nodular hyperplasia, exocrine pancreatic insufficiency (EPI), pancreatolithiasis, congenital anomalies, and pancreatic edema cannot always be reliably differentiated.

CT is the technique of choice for diagnosing pancreatitis in humans. In a 2015 pilot study, abdominal CT angiography was performed in 10 dogs under sedation to confirm clinically suspected pancreatitis.[18]

Serum pancreatic lipase immunoreactivity as measured by Spec cPL (canine) or Spec fPL (feline) is widely used for the diagnosis of pancreatitis. Sensitivity of Spec cPL is reported to range from 64% to 94%, whereas sensitivity of Spec fPL ranges from 54% to 100%. The reader is referred to a medical text for a more detailed discussion of the diagnosis of pancreatitis.

NOTE Spec cPL and Spec fPL tests are sensitive enough to detect histologic pancreatitis that is not clinically important; therefore one should not blindly use these tests as a "litmus" test.

Vomiting animals often require correction of fluid, electrolyte, and acid-base abnormalities before surgery. Diabetic animals may be prone to pancreatitis and are often anesthetized for elective and nonelective procedures. Diabetics should be carefully evaluated before surgery, including complete blood cell count, serum biochemical panel (including fasting blood glucose, blood urea nitrogen, and creatinine), urinalysis, and urine culture. Severe hyperglycemia (>300 mg/dL), ketoacidosis, major electrolyte abnormalities (e.g., hypokalemia, hypophosphatemia), and urinary tract infection should be corrected before surgery. Animals with pancreatic tumors may have a wide variety of metabolic disorders.

ANESTHESIA

Many protocols have been described for anesthetic management of diabetic animals. Blood glucose concentrations should ideally be maintained between 100 and 300 mg/dL during surgery. Hypoglycemia may occur if animals are given their regular insulin dose and if food is withheld before surgery; however, the stress of surgery usually results in hyperglycemia. Animals should be fed their normal diet the day before surgery, and their regular dose of insulin should be administered. Food should be withheld 6 to 8 hours before surgery or a small meal given after the morning insulin. Surgery should be performed in the morning. Blood glucose concentrations should be measured the morning of surgery. One to 2 hours before surgery, if the blood glucose concentration is between 150 and 300 mg/dL, the animal should receive one-half of its usual morning dose of insulin subcutaneously. Blood glucose should be checked at induction and hourly thereafter. If the blood glucose level is low, 0.45% saline and 2.5% dextrose (10–15 mL/kg for the first hour, then 5 mL/kg thereafter if blood and evaporative fluid losses are small) should be administered. If the blood glucose level is normal, administer lactated Ringer's solution (at the same rate). Fluids should be changed to 5% dextrose and an additional small dose of regular insulin given if the blood glucose concentration is greater than 300 mg/dL. The key to management of the diabetic patient is frequent blood glucose checks and an appreciation of the variability of patient responses to insulin therapy.

NOTE Be sure to maintain excellent perfusion during surgery to help prevent postoperative pancreatitis.

Selected anesthetic protocols for animals with pancreatic disease that are in stable condition are provided in Table 22.2. These animals may be premedicated with an anticholinergic and opioid, induced with thiopental or propofol, and maintained on isoflurane or sevoflurane inhalants. If the animal is shocky, dehydrated, or hypovolemic, anesthesia must be induced and maintained with greater care. Suggested anesthetic protocols are provided in Table 22.3. As an alternative, animals that are not vomiting may be induced with a mask or placed in a chamber, or they may be given thiopental or propofol at reduced dosages. If ketamine is not contraindicated, reduced dosages of diazepam and ketamine may be used.

NOTE It is important to realize that dehydration is often not appreciated or is underestimated, especially in obese animals.

ANTIBIOTICS

Antibiotics have not been shown to benefit dogs with pancreatitis, which is almost exclusively a nonseptic disease. They are often administered in an effort to prevent secondary infection in necrotic pancreatic and peripancreatic tissue, but no evidence suggests that they benefit the patient. Nonetheless, prophylactic antibiotic therapy (e.g., cefazolin 22 mg/kg IV) is often administered in animals undergoing pancreatic biopsy or partial pancreatectomy to prevent pancreatic abscessation. Treatment with imipenem or ciprofloxacin has been shown to reduce early and late septic pancreatic complications and to improve survival in experimental pancreatitis in rats; however, no evidence indicates that these drugs benefit dogs or cats with pancreatitis. Antibiotic therapy should be based on the results of culture and sensitivity testing of infected tissue in animals with septic pancreatic abscessation.

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TABLE 22.2 Anesthetic Considerations in the Stable Patient With Pancreatic Disease

Preoperative Considerations

Associated conditions	• +/– Diabetes • Often healthy patients before pancreatic disease
Bloodwork	• HCT • Electrolytes • BUN • Cr • TP • Blood glucose, often serial glucose checks • Urinalysis
Physical examination	• Often middle-aged or elderly patients • Painful abdomen
Other diagnostics	• Blood pressure • ECG
Premedications	• Midazolam (0.2 mg/kg IV, IM), *or* • Diazepam (0.2 mg/kg IV), *plus* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM in dogs; 0.05–0.1 mg/kg IV, IM in cats), *or* • Oxymorphone (0.1–0.2 mg/kg IV, IM), *or* • Morphine[b] (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM), *or* • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM)

Intraoperative Considerations

Induction	• If premedicated, give:
	  • Propofol (2–4 mg/kg IV), *or*
	  • Alfaxalone (2–3 mg/kg IV), *or*
	  • Etomidate (0.5–1.5 mg/kg IV)
	• If no premedication given, then:
	  • Propofol (4–8 mg/kg IV), *or*
	  • Alfaxalone (3–5 mg/kg IV), *or*
	  • Ketamine (5.5 mg/kg IV) with diazepam (0.28 mg/kg IV)

Maintenance	• Isoflurane or sevoflurane *plus*
	  • Fentanyl (2–10 µg/kg IV PRN in dogs; 1–4 µg/kg IV PRN in cats) for short-term pain relief, *plus*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Morphine[b] (0.1–1 mg/kg IV PRN in dogs; 0.05–0.2 mg/kg IV PRN in cats), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV PRN), *plus*
	  • Ketamine (low dose) (0.5–1 mg/kg IV once), *or*
	  • Ketamine CRI (0.5 mg/kg IV loading dose, then 10 µg/kg/min IV)
	• For hypotension (to keep MAP 60–80 mm Hg), give phenylephrine, ephedrine, or dopamine as needed

Fluid needs	• 5–10 mL/kg/h if minimal EBL and minimal evaporative losses, or 10–20 mL/kg/h if open abdomen with higher evaporative losses, plus 3 × EBL
Monitoring	• Blood pressure • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature • U/O
Blocks	Epidural: • Morphine (0.1 mg/kg preservative free) *or* • Buprenorphine (0.003–0.005 mg/kg diluted in saline) Incisional: • Lidocaine (<5 mg/kg in dogs; 2–4 mg/kg in cats), *or* • Bupivicaine (<2 mg/kg)

Postoperative Considerations

Analgesia	• Fentanyl CRI (1–10 µg/kg IV loading dose, then 2–20 µg/kg/h IV), *or* • Morphine[b] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.05–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h in cats), *or* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats), *or* • Hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs), *or* • Oxymorphone (0.1–0.2 mg/kg IV, IM), *or* • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM q4–8h; 0.01–0.02 mg/kg OTM q6–12h in cats), *plus* • +/– Ketamine CRI (2 µg/kg/min IV). If no previous loading dose, give 0.5 mg/kg IV prior to CRI)

Continued

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TABLE 22.2 Anesthetic Considerations in the Stable Patient With Pancreatic Disease—cont'd

Monitoring	• SpO_2 • Blood pressure • HR • Respiratory rate • Temperature • U/O • ECG if electrolyte abnormalities
Bloodwork	• HCT if significant blood loss • Repeat abnormal preoperative bloodwork • Serial blood glucose checks if necessary
Estimated pain score	Moderate to severe if open abdominal surgery or if underlying pancreatitis

[a]Monitor for hyperthermia in cats.
[b]Give slowly to prevent histamine release.
[c]Buprenorphine is a better analgesic than morphine in cats.
BUN, Blood urea nitrogen; *Cr,* creatinine; *CRI,* constant-rate infusion; *EBL,* estimated blood loss; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *MAP,* mean arterial pressure; *OTM,* oral transmucosal; *PRN,* as needed; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein; *U/O,* urine output.

TABLE 22.3 Anesthetic Considerations in the Compromised Patient With Pancreatic Disease

Preoperative Considerations

Associated conditions	• Dehydration • Electrolyte abnormalities • Hypotension • Abnormal blood glucose • Adrenal suppression may be present in the critically ill patient
Bloodwork	• HCT • Electrolytes • BUN • Cr • TP • Blood glucose, often serial glucose checks • Urinalysis
Physical examination	• Often elderly patients • May be dehydrated, tachycardic or bradycardic, hypotensive, and/or hypothermic • Painful abdomen if pancreatitis
Other diagnostics	• Blood pressure • ECG

Premedications	• Rehydrate over 4–6 hours if possible; if emergent, may have to give more rapid boluses to expedite time to surgery
	• Correct electrolyte abnormalities
	• Avoid sedatives in sick patients
	• Avoid alpha-2 agonists and acepromazine
	• If patient is anxious, give:
	  • Midazolam (0.1–0.2 mg/kg IV, IM) *or*
	  • Diazepam (0.1–0.2 mg/kg IV)
	• If patient is not depressed, then give:
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM in dogs; 0.05–0.1 mg/kg IV, IM in cats), *or*
	  • Morphine[b] (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM), *or*
	  • Oxymorphone (0.1–0.2 mg/kg IV, IM), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM)

Intraoperative Considerations

Induction	• If dehydrated, give the following:
	  • Etomidate (0.5–1.5 mg/kg IV); if possible, avoid its use in critically ill patients, *or*
	  • Propofol (1–4 mg/kg IV) slowly, *or*
	  • Alfaxalone (2–3 mg/kg IV)
	• If hydrated, give the following:
	  • Propofol (2–6 mg/kg), *or*
	  • Alfaxalone (3–5 mg/kg IV)

Maintenance	• Isoflurane or sevoflurane *plus*
	  • Fentanyl (2–10 µg/kg IV PRN in dogs; 1–4 µg/kg IV PRN in cats) for short-term pain relief, plus PRN
	  • Fentanyl CRI (1–5 µg/kg IV loading dose, then 2–30 µg/kg/h IV), *or*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV PRN), *plus*
	  • Ketamine (low dose) (0.5–1 mg/kg IV), *or*
	  • Ketamine CRI (0.5 mg/kg IV loading dose, then 10 µg/kg/min IV)
	• For hypotension (to keep MAP 60–80 mm Hg), give phenylephrine, ephedrine, or dopamine as needed

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TABLE 22.3 Anesthetic Considerations in the Compromised Patient With Pancreatic Disease—cont'd

Fluid needs	• 5–10 mL/kg/h if minimal EBL and minimal evaporative losses, or 10–20 mL/kg/h if open abdomen with higher evaporative losses, plus 3 × EBL • Consider colloids if persistent hypotension
Monitoring	• Blood pressure • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature • U/O
Blocks	Epidural: • Morphine (0.1 mg/kg preservative free) *or* • Buprenorphine (0.003–0.005 mg/kg diluted in saline) • Avoid local anesthetics for spinals and epidurals in hypotensive patients Incisional: • Lidocaine (<5 mg/kg in dogs; 2–4 mg/kg in cats), *or* • Bupivicaine (<2 mg/kg)
Postoperative Considerations	
Analgesia	• Fentanyl CRI (1–10 µg/kg IV loading dose, then 2–20 µg/kg/h IV), *or* • Morphine[b] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.05–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h in cats) if no hypotension, *or* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats), *or* hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs), *or* • Oxymorphone (0.1–0.2 mg/kg IV, IM), *or* • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM q4–8h; 0.01–0.02 mg/kg OTM q6–12h in cats), *plus* • +/– Ketamine CRI (2 µg/kg/min IV). If no previous loading dose, give 0.5 mg/kg IV prior to CRI • Avoid NSAIDs in patients with hypotension
Monitoring	• SpO_2 • Blood pressure • HR • Respiratory rate • Temperature • U/O • ECG if electrolyte abnormalities
Bloodwork	• HCT if significant blood loss • Repeat abnormal preoperative bloodwork • Serial blood glucose checks if necessary
Estimated pain score	Moderate to severe if open abdominal surgery or if underlying pancreatitis

[a]Monitor for hyperthermia in cats.
[b]Give slowly to prevent histamine release.
[c]Buprenorphine is a better analgesic than morphine in cats.
BUN, Blood urea nitrogen; *Cr,* creatinine; *CRI,* constant-rate infusion; *EBL,* estimated blood loss; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *MAP,* mean arterial pressure; *NSAID,* nonsteroidal antiinflammatory drug; *OTM,* oral transmucosal; *PRN,* as needed; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein; *U/O,* urine output.

SURGICAL ANATOMY

The pancreas of dogs and cats is composed of a right and a left limb and a small central body (Fig. 22.9). The right limb of the pancreas lies within the mesoduodenum and is closely associated with the duodenum, particularly at its cranial aspect. The dorsal aspect of the right pancreatic lobe is visualized by retracting the duodenum ventrally and toward the midline; the ventral aspect of the right pancreatic lobe is examined by retracting the duodenum laterally. The pancreatic body (angle) lies in the bend formed by the pylorus and the duodenum. The left pancreatic lobe is viewed within the deep leaf of the greater omentum by retracting the stomach cranially and the transverse colon caudally.

NOTE Visualize the left lobe of the pancreas by looking in the deep leaf of the greater omentum while retracting the stomach cranially.

The main blood supply to the left pancreatic lobe is provided via branches of the splenic artery; however, branches from the common hepatic and gastroduodenal arteries also supply portions of it. The main vessels of the right lobe of the pancreas are the pancreatic branches of the cranial and caudal pancreaticoduodenal arteries that anastomose in the gland. The cranial pancreaticoduodenal artery is a terminal branch of the hepatic artery; the caudal pancreaticoduodenal arises from the cranial mesenteric vessel. These vessels also provide branches that supply the duodenum. Because they are closely associated with the proximal portion of the right lobe of the pancreas, care must be taken not to damage these vessels during pancreatic surgery, or devitalization of the duodenum may occur.

NOTE The proximity and shared blood supply of the pancreas and duodenum make duodenal resection difficult if pancreatic function is to be maintained.

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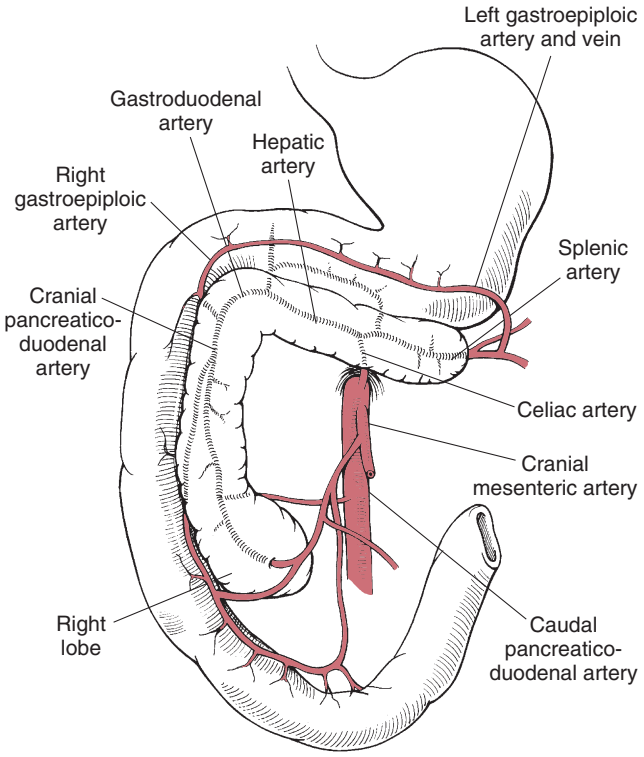


FIG. 22.9 Vascular supply to the pancreas.

The pancreas has both endocrine (insulin) and exocrine (digestive secretions) functions. Digestive secretions enter the duodenum via one of two ducts. These ducts may communicate within the gland or may cross each other. When the two ducts do not communicate, the *pancreatic duct* drains the right lobe and the *accessory pancreatic duct* drains the left lobe. The accessory pancreatic duct is the largest excretory pancreatic duct in dogs. It opens into the duodenum at the *minor duodenal papilla.* The smaller pancreatic duct is occasionally absent. The latter usually enters the duodenum on the *major duodenal papilla,* adjacent to the common bile duct. The pancreatic duct is the principal and oftentimes the only duct in cats.

NOTE Extrahepatic biliary obstruction may occur secondary to pancreatic swelling or masses because of impingement of the common bile duct as it enters the major duodenal papilla. Attempting to resect such masses is typically contraindicated because it can easily result in laceration and/or rupture of the bile duct.

SURGICAL TECHNIQUE

A ventral midline abdominal incision is made, extending from the xiphoid cartilage caudal to the umbilicus, and the pancreas is examined using a combination of gentle palpation and visual inspection. The free portion of the greater omentum is retracted cranially and is covered with moist sponges. The omental leaf overlying the pancreas can be bluntly separated to allow direct visualization of the left pancreas. When neoplasia is suspected, lymph nodes that lie along the splenic vessels and portal vein and those at the hilus of the liver and head of the pancreas should be examined for evidence of metastasis.

NOTE Handle the pancreas gently to avoid causing pancreatitis.

Pancreatic biopsy and partial pancreatectomy are performed in dogs and cats. Because of the difficulty in diagnosing feline pancreatitis, biopsy may be indicated more often than currently performed. Laparoscopic biopsy of the canine and feline pancreas is generally well tolerated. Pancreatic biopsy is occasionally performed in dogs to differentiate benign pancreatic conditions (e.g., pancreatitis, pancreatic fibrosis) from neoplastic disease. Although ultrasound-guided biopsies of large pancreatic lesions may be possible, exploratory laparotomy and direct visualization of pancreatic tissue are usually indicated. Partial pancreatectomy is indicated in animals with insulin-secreting or gastrin-secreting tumors and in those with pancreatic adenocarcinoma (see p. 613). Total pancreatectomy is infrequently performed in veterinary patients. Removal of the pancreas without duodenectomy requires that pancreatic tissue be bluntly dissected from the pancreaticoduodenal vessels without damage to branches supplying the duodenum. This is difficult in animals with pancreatic disease because of adhesions, fibrosis, and edema. Therefore total pancreatectomy is usually performed in conjunction with resection and anastomosis of the proximal duodenum (i.e., Billroth II procedure), ligation of the common bile duct, and cholecystojejunostomy (see p. 574) and is associated with high rates of morbidity and mortality. Pancreatic drainage or omentalization is indicated in conditions (e.g., large abscesses or cysts) in which pancreatectomy is not feasible.

NOTE Total pancreatectomy is difficult because it usually necessitates cholecystoenterostomy and removal of the duodenum.

Laparoscopic Pancreatic Biopsy

This is generally performed during two-port laparoscopy. As previously noted, diagnosing pancreatitis in cats can be more difficult than in dogs; therefore pancreatic biopsy is more commonly done in cats. *Use a punch type of biopsy forceps (as opposed to "double spoon" forceps). Obtain biopsies from the edge of the pancreas and take care to examine both sides (a large vein typically runs under the pancreas, near the edge). If you see obviously diseased pancreatic tissue, take a biopsy; however, some cats with pancreatitis do not have grossly diseased pancreatic tissue. Because canine pancreatitis can be a focal or multifocal lesion, take multiple biopsies from such animals.*

Surgical Pancreatic Biopsy

If obvious, diffuse pancreatic disease is present, a biopsy is best obtained by removing a small portion of the caudal aspect of the right pancreatic limb (see the discussion of Partial Pancreatectomy in the next section). Focal lesions near the extremity of the pancreas may be removed in a similar fashion. If no obviously diseased tissue is noted, then multiple biopsies should be taken, as pancreatitis can be a localized or a multifocal disease. *For focal lesions within the pancreatic parenchyma, use a TruCut (Cardinal Health) or Vim-Silverman needle (see p. 544), or take a portion of the lesion using a guillotine ligature of absorbable suture to obtain a small sample of pancreatic tissue. Take care not to damage adjacent blood vessels or pancreatic ducts.*

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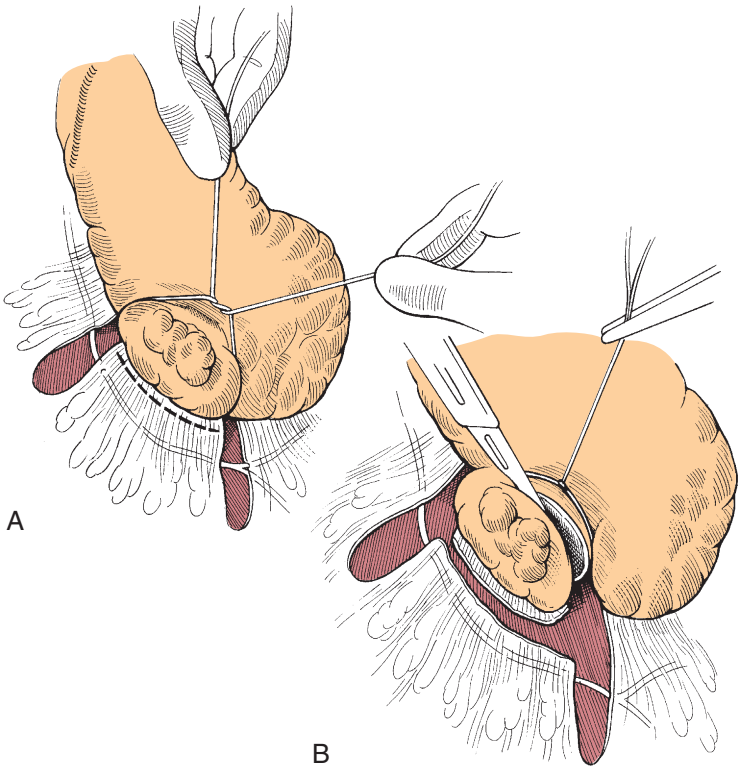


FIG. 22.10 Focal lesions near the extremity of the pancreas can be removed by the suture fracture technique. (A) Incise the mesoduodenum or omentum *(dotted line)* and pass nonabsorbable suture from one side of the pancreas to the other through the incisions. (B) Tighten the suture and allow it to crush through the parenchyma.

Partial Pancreatectomy

Focal lesions near the extremity of the pancreas can be removed by the suture fracture technique. *Incise the mesoduodenum or omentum on each side of the pancreas (Fig. 22.10A). Pass absorbable (3-0 to 4-0) suture material from one side of the pancreas to the other, through the incisions, so that the suture is just proximal to the lesion to be excised. Tighten the suture, and allow it to crush through the parenchyma, which ligates vessels and ducts (see Fig. 22.10B). Excise the specimen distal to the ligature. Close any holes in the mesoduodenum with absorbable suture material.*

Partial pancreatectomy was performed using a vessel-sealing device (Ligasure) in eight dogs and compared with a historical control group where partial pancreatectomy was performed using a traditional suture-fracture technique. Dogs in the Ligasure group had shorter surgical and hospitalization times and none developed clinical signs in the postoperative period consistent with pancreatitis.[19]

Blunt separation of pancreatic lobules and ligation of ducts can be performed for lesions anywhere in the pancreas. With small lesions, it may be possible to identify and preserve the pancreatic ducts. *Identify the lesion to be removed, and gently incise the mesoduodenum or omentum overlying it (Fig. 22.11A). For lesions involving the pancreatic body or the proximal aspect of the right lobe, use gauze sponges to bluntly dissect pancreatic tissue from the pancreaticoduodenal vessels. Ligate or cauterize small pancreatic vessels, but take care not to damage the pancreaticoduodenal vessels. Using sterile Q-tips or Halsted Mosquito hemostats, separate the affected lobules from adjoining tissue by blunt dissection (see Fig. 22.11B). Identify the blood vessels and ducts supplying the portion of the pancreas to be removed, and ligate them (see Fig. 22.11C). Excise the affected pancreatic tissue and close any holes in the mesoduodenum.*

NOTE Ligate the pancreatic ducts with absorbable suture. If there is or may be substantial pancreatitis, it is often a wise idea to put in an enteral feeding tube (see p. 94) during the procedure.

HEALING OF THE PANCREAS

The fibrous stroma of the pancreas allows healing to occur by protein synthesis, epithelialization, and fibrin polymerization. Obstruction of the pancreatic duct is seldom caused by wound contraction; rather, parenchymal edema or obstruction at the duodenal papilla is usually the cause. The main concern associated with pancreatic healing after surgery is the effect of healing on the flow and drainage of pancreatic secretions. If the duct to the remaining portion is left intact, as much as 80% of the pancreas can be removed without causing deleterious decreases in exocrine or endocrine function.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Duct ligation is performed with absorbable suture material (e.g., polydioxanone, polyglyconate, glycomer 631, or poliglecaprone 25) in animals with inflammatory, aseptic, or neoplastic conditions; braided suture material should be avoided.

POSTOPERATIVE CARE AND ASSESSMENT

Medical treatment of canine and feline pancreatitis is replete with opinions, but as of this writing, no well-designed, prospective, controlled studies have been performed. The following recommendations are generally agreed upon, but the reader is referred to a current medical text for a more complete discussion.

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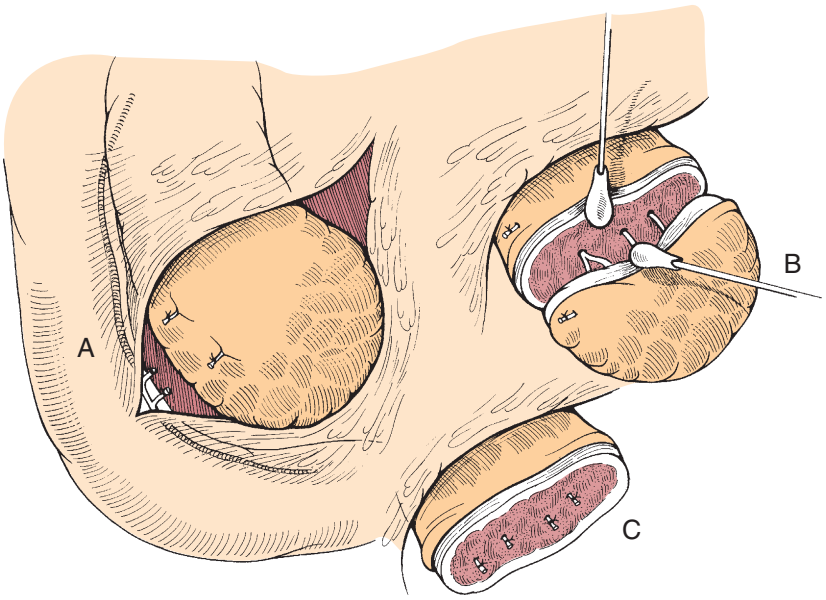


FIG. 22.11 Blunt separation of pancreatic lobules and ligation of ducts can be performed for lesions anywhere in the pancreas. (A) Identify the lesion to be removed and gently incise the mesoduodenum or omentum overlying it. (B) Separate the affected lobules from adjoining tissue by blunt dissection using sterile cotton-tipped swabs or Halsted Mosquito hemostats. (C) Ligate the blood vessels and ducts supplying the portion of the pancreas to be removed.

Classically, enteral feeding has been delayed for 2 to 5 days after extensive pancreatic surgery is performed, or if pancreatitis is present. It is now generally agreed that keeping dogs with pancreatitis off food and putting them into a negative nitrogen balance is deleterious. Patients with pancreatitis should be fed as soon as they can tolerate enteral alimentation (per os or via esophagostomy tube). Feeding should be initiated with small amounts of low-fat (probably less than 2–3 g fat/100 kcal), bland food (e.g., rice, defatted white chicken, or white turkey without the skin). Animals with severe or prolonged pancreatitis that cannot accept enteral nutrition without obviously becoming worse may benefit from parenteral nutrition (see Chapter 10). Hydration and electrolytes (especially potassium) should be maintained with IV fluid therapy because pancreatic perfusion is probably paramount in healing the diseased pancreas. Visceral perfusion may be inadequate in animals that subjectively appear normally hydrated; dehydration is often not obvious clinically. Plasma oncotic pressure should be maintained by administering colloids (e.g., hetastarch or pentastarch) if hypoalbuminemia (i.e., serum albumin <2 g/dL) occurs (see Chapter 4). Plasma is much less effective in raising plasma oncotic pressure. Treating patients with DIC consists of maintaining an adequate blood pressure and thus perfusion pressure, as well as replacing clotting factors with fresh-frozen plasma transfusions; heparin therapy may be given in addition. There is general consensus that early intervention in animals with DIC increases their chances of survival. Antiemetics and analgesics may be administered as needed. If sepsis is identified, appropriate antibiotic therapy should generally be continued for 10 to 14 days after surgery, but this is exceedingly rare in dogs.

COMPLICATIONS

The most common complication of pancreatic surgery is pancreatitis; this can be minimized by maintaining good visceral perfusion and gentle tissue handling. In a study of postoperative complications following pancreatic biopsy in dogs and cats, postoperative pancreatitis occurred in 11.6% of patients.[20] Anecdotally, octreotide (1–2 µg/kg given subcutaneously before surgery) has been used to try to prevent postoperative pancreatitis.

BOX 22.10 Pancreatic Enzyme Treatment of Exocrine Pancreatic Insufficiency

Pancrezyme

Up to 2 tsp in each meal

Viokase

1 to 2 tsp in each meal

If oral lesions (stomatitis, glossitis) occur, stop the preparation for 3–5 days, then start back at half the dose.

Severe, acute pancreatitis puts the patient at risk for mortality from multiorgan failure. EPI may occur if pancreatic drainage is completely obstructed. EPI is treated with pancreatic supplements of commercial pancreatic extract (e.g., Viokase; Box 22.10) and feeding of low-fat, highly digestible meals. Endocrine pancreatic insufficiency (DM) may result when more than 80% to 90% of the pancreatic tissue is removed. Supplementation with insulin may be necessary.

SPECIAL AGE CONSIDERATIONS

Pancreatic disease is usually found in middle-aged or older animals. Special care must be taken to meet the nutritional and metabolic needs of geriatric patients, particularly when disease may have caused inappetence or chronic vomiting. Parenteral hyperalimentation may be necessary before and after surgery in these patients.

SPECIFIC DISEASES

PANCREATIC ABSCESSES AND PSEUDOCYSTS

DEFINITIONS

Pancreatic abscesses are a collection of purulent material and necrotic tissue within and extending from the pancreatic parenchyma. **Pancreatic pseudocysts** are collections of pancreatic secretions and cellular debris enclosed within a wall of granulation

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tissue or fibrous sac that lacks an epithelial wall. Pancreatic pseudocysts have also been called *pancreatic cysts.*

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Pancreatic abscesses (Fig. 22.12) are pancreatic or peripancreatic collections of purulent, necrotic, and hemorrhagic tissue that probably occur as a consequence of acute pancreatitis. Pancreatic abscesses may also occur in human beings as a consequence of chronic ductal obstruction.

Pancreatic pseudocysts (Fig. 22.13) are a common complication of acute pancreatitis in human beings but are rarely diagnosed in small animals. They may be associated with recurrent bouts of pancreatitis or trauma. Fluid in the cysts is a combination of blood, pancreatic fluids, and enzymes. These are not true cysts because the fluid is thought to leak from damaged pancreatic ducts and vessels rather than being secreted by the lining of the cyst. Pancreatic pseudocysts may be incidental findings or may be associated with nonspecific abdominal signs, such as pain and vomiting. Complications such as infection, rupture, or acute hemorrhage may occur in people and are associated with a high mortality rate.

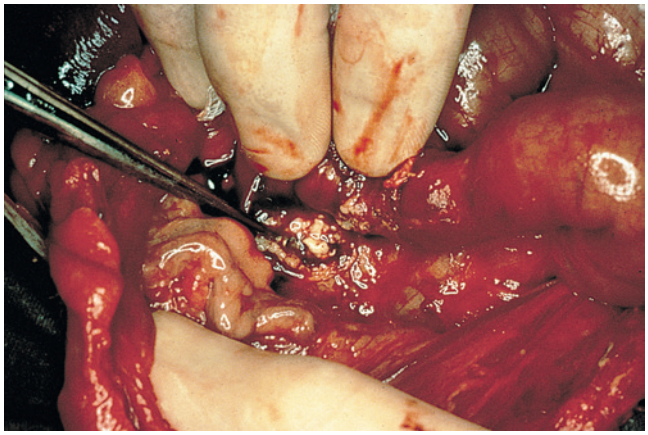


FIG. 22.12 Pancreatic abscess in a dog.

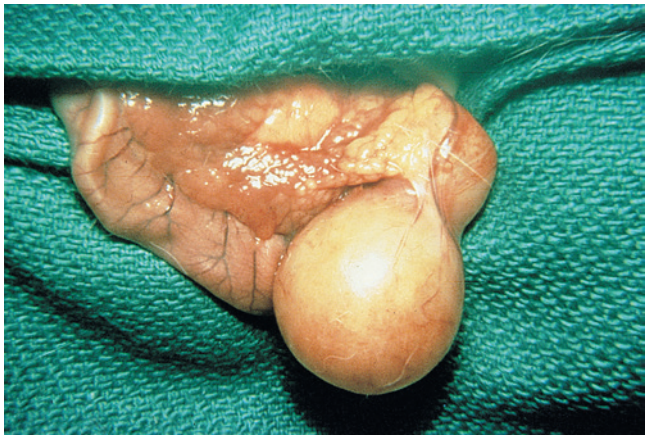


FIG. 22.13 Pancreatic pseudocyst. (Courtesy H.P. Hobson, Texas A&M University.)

NOTE Large pancreatic masses in symptomatic or asymptomatic animals may be pseudocysts or sterile abscesses; cancer is a much less common event. Do not recommend euthanasia solely on the basis of a radiographic finding of an abdominal mass.

DIAGNOSIS

Clinical Presentation

Signalment

Pancreatic abscesses/pseudocysts probably arise after acute pancreatitis; therefore the signalment of these animals closely parallels that of animals diagnosed with acute pancreatitis (see p. 600). Most animals are middle-aged or older, and dogs are more commonly affected than cats.

History

Animals with pancreatic abscesses/pseudocysts may have a previous history of acute onset of anorexia, depression, diarrhea, or vomiting; some have previously been treated for gastroenteritis that was probably pancreatitis. Other clinical findings may include ataxia, anorexia, abdominal pain, or pyrexia. These patients may have dramatic acute signs, or vague, smoldering, chronic signs, or they may be asymptomatic.

Physical Examination Findings

Typical findings with pancreatic abscesses/pseudocysts may include pain during abdominal palpation, depression, icterus, pyrexia, palpable cranial abdominal mass, or abdominal distention. Some animals may be weak and reluctant to stand. Pyrexia is an uncommon finding. However, some animals are asymptomatic.

Diagnostic Imaging

The most consistent finding with pancreatic abscess/pseudocyst on survey abdominal radiographs is an ill-defined increase in soft tissue density in the right cranial abdominal quadrant. If peritonitis is present, a generalized increase in soft tissue opacity and loss of visceral detail in the right quadrant or throughout the abdomen may be observed. Abdominal ultrasonography is more sensitive and usually reveals a mass in the area of the pancreas. Gallbladder and bile duct distention may also be noted. Ultrasonography may also identify pancreatitis (Fig. 22.14). Gastric outflow obstruction is rarely observed on contrast studies

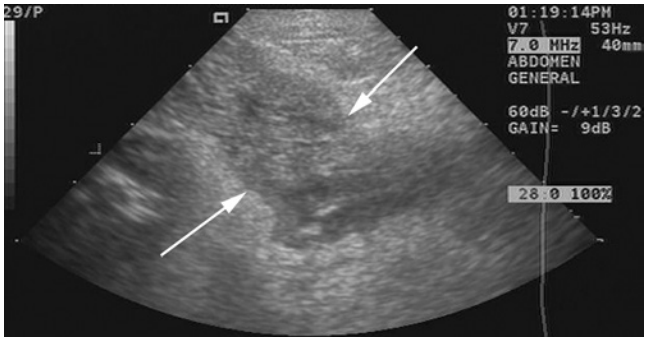


FIG. 22.14 Ultrasound parasagittal image of the pancreas. It is hypoechoic *(arrows)*, enlarged, and surrounded by hyperechoic fat consistent with pancreatitis.

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of the upper gastrointestinal tract. Ultrasonography is the best tool for identifying pancreatic abscess/pseudocyst; however, differentiation of pseudocysts from other fluid-filled masses is not possible without evaluation of the fluid. Percutaneous fine-needle aspiration of masses is reasonable in dogs because of the extremely low incidence of septic pancreatitis in this species. Risk must be weighed against the advantage of a preoperative diagnosis. Resolution of pancreatic abscess/pseudocysts after percutaneous, ultrasound-guided drainage is possible.

NOTE Fine-needle aspiration of cavitary pancreatic masses may involve some risk. Perform with caution and observe these animals carefully after the procedure.

Laboratory Findings

Hematologic and serum biochemical findings with pancreatic abscess/pseudocyst are inconsistent but may include leukocytosis, neutrophilia with or without a left shift, lymphopenia, or monocytosis. Serum biochemical abnormalities may include hyperbilirubinemia and high serum alkaline phosphatase caused by extrahepatic cholestasis, high alanine aminotransferase, hypocholesterolemia, hyponatremia, hypochloremia, and hypokalemia. Serum lipase and serum amylase are insensitive and nonspecific; they should not be requested. Bilirubinuria is often present.

NOTE The absence of neutrophilia does not exclude pancreatic abscessation.

DIFFERENTIAL DIAGNOSIS

Pancreatic abscess/pseudocyst must be differentiated from other causes of vomiting and cranial abdominal pain (e.g., pancreatitis, gastric foreign bodies, intestinal foreign bodies, gastritis, cholecystitis, pancreatic neoplasia, gastrointestinal neoplasia). Ultrasound evaluation of the pancreas is the most useful test for differentiating these abnormalities preoperatively; however, exploratory surgery may be required in some animals to make a definitive diagnosis.

MEDICAL MANAGEMENT

Pancreatic abscesses/pseudocysts have classically been considered surgical diseases. However, we now realize that in some patients they can be resolved with percutaneous drainage, and others (typically those fortuitously diagnosed on abdominal ultrasound for some other reason) may need no treatment. These lesions are almost invariably sterile in dogs, but septic abscesses can occur in cats. The mortality rate in human beings with pancreatic abscesses is nearly 100% when medical therapy without drainage is used; with surgical treatment, mortality has been reduced. Similar studies have not been reported in dogs or cats. Some pancreatic abscesses/pseudocysts may resolve spontaneously without therapy.

SURGICAL TREATMENT

Symptomatic dogs with abscesses or pseudocysts probably benefit from drainage procedures; however, many dogs with pancreatitis have hypoechoic areas in the pancreas that do not represent fluid accumulations that can be drained. Generalized, sterile peritonitis is present in some dogs with pancreatic abscess/pseudocyst; if septic peritonitis or pancreatitis is present, a thorough search should be made for a primary cause such as a perforated duodenum. On opening the abdomen, a mass is observed originating from the pancreas in the cranial portion of the abdomen. The mass may be firm and fibrotic or friable. Multiple adhesions to omentum and adjacent loops of small or large intestine are often present. These lesions can look malignant; however, a vast majority of pancreatic lesions and masses are inflammatory without malignancy, regardless of how bad they appear. Adhesions may be present if the lesion has ruptured and reformed. If the abscess or pseudocyst is surgically resolved, omentalization is preferred over external drainage (see later discussion).

Preoperative Management

If clinically apparent pancreatitis is present, medical management should be initiated before surgery (percutaneous drainage may proceed if deemed important). See the discussion on p. 606 for information on the treatment of pancreatitis. A broad-spectrum antibiotic can be administered intravenously before surgery if sepsis is believed to be present, but this is seldom necessary. If infection is found by cytology or culture, then antibiotics should be used for at least 10 to 14 days postoperatively.

Anesthesia

See the discussion of the anesthetic management of animals with pancreatic disease on p. 600.

Surgical Anatomy

See the discussion of the surgical anatomy of the pancreas on p. 603.

Positioning

The animal is positioned in dorsal recumbency, and the caudal thorax and the entire abdomen are prepared for aseptic surgery.

SURGICAL TECHNIQUE

Pancreatic Abscesses/Pseudocysts

Perform a midline abdominal laparotomy that extends from the xiphoid cartilage caudally to distal to the umbilicus. Gently explore the abdomen. Locate the pancreatic mass and obtain cultures of infected tissue. Gently break down adhesions in the intestine and omentum as needed to visualize the lesion. Preserve the pancreatic ducts, common bile ducts, and adjacent vascular structures during dissection. Debride necrotic or purulent areas of the pancreas using a combination of sharp and blunt dissection. Resect as much of the necrotic pancreas as possible without damaging adjacent blood vessels or tissue. Once the lesion has been debrided, place a piece of omentum in it and secure it with sutures. If possible, loop the omentum through a tunnel in the pancreatic tissue and suture it back to itself. Determine common bile duct patency by gently expressing the gallbladder. If the common bile duct is not patent, catheterize the duct and try to obtain flow or perform a cholecystoenterostomy (see p. 574). Make sure you do not ligate the common bile duct. If generalized peritonitis is present, lavage the abdomen thoroughly with warm, sterile saline or lactated Ringer's solution. If peritonitis is present close the abdomen, insert a drain, or leave it open for drainage (see p. 534).

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SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Absorbable suture material should be used for partial pancreatectomy in animals with pancreatic abscesses/pseudocysts. Aerobic and anaerobic culture swabs should be available. Copious amounts of warmed fluids should be available for abdominal flushing; suction allows complete removal of instilled fluid and facilitates dilution of infected fluids in the abdominal cavity.

POSTOPERATIVE CARE AND ASSESSMENT

The patient should be treated for pancreatitis, as described previously. Antibiotic therapy should be continued if infection is found. Animals should be monitored postoperatively for signs of worsening inflammation. Clinical assessment of these patients (e.g., presence or absence of fever, abdominal pain, anorexia, vomiting, and icterus) is more important than the complete blood cell count or ultrasonographic appearance. Repeat operations may be required occasionally. Blood cultures are warranted if bacteremia is suspected. Pancreatitis is a potential complication of any surgery involving the pancreas (see p. 606).

NOTE Warn owners that repeat surgery may be necessary in some animals with pancreatic abscesses/pseudocysts.

PROGNOSIS

The prognosis in animals with inflammatory pancreatic lesions is guarded. Mortality rates appear to be higher with necrotic mass lesions of the pancreas and pancreatic abscesses than with pancreatic pseudocysts. Cats undergoing surgery because of extrahepatic biliary obstruction secondary to severe acute pancreatitis that does not respond to medical management may have a good prognosis. Postoperative complications that have been described include progression of DM, septic peritonitis, local gastrostomy tube stoma inflammation, local gastrostomy tube stoma infection, and mild dermal suture reaction.

INSULINOMAS

DEFINITION

Insulinomas are functional tumors of the β-cells of the islets of Langerhans. These tumors secrete insulin despite the presence of hypoglycemia. They have also been called *pancreatic β-cell tumors, adenomas,* or *adenocarcinomas of the pancreatic islets.*

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Insulinomas are pancreatic islet cell tumors that secrete excessive amounts of insulin, causing hypoglycemia. They are more commonly recognized in dogs than in cats. Unlike human beings, in whom up to 90% of insulinomas are benign, malignant tumors predominate in dogs. They typically metastasize to the regional lymph nodes, liver, and omentum. Occasionally nodules may be found in the lung. They are slow-growing tumors that compress adjacent pancreatic parenchyma. Because they are typically sharply delineated and encapsulated, palliative surgical excision often prolongs survival. Tumor state may correlate with survival time following surgery and medical management (see Prognosis section).

NOTE More than 90% of canine insulinomas are malignant. They nearly always metastasize even though they may lack histologic criteria of malignancy.

DIAGNOSIS

Clinical Presentation

Signalment

Insulinomas generally occur in middle-aged or older dogs; no gender predisposition has been noted. Medium- to large-breed dogs (e.g., Irish setters, German shepherds, Labrador retrievers, standard poodles, boxers) appear to be more commonly affected.

History

The clinical signs are attributable to hypoglycemia and include muscle tremors, muscle weakness, ataxia, mental dullness, disorientation, collapse, and/or convulsions. Dogs may be easily agitated and may have intermittent periods of excitability and restlessness. These clinical signs suggest hypoglycemia from any cause, not just insulinoma. Owners may notice clinical signs for months before presenting the animals for evaluation. The clinical signs are often intermittent initially but occur more frequently as the disease progresses. Owners often report that clinical signs diminish or resolve with feeding. Animals are sometimes treated for seizures with anticonvulsant agents before the diagnosis is made.

NOTE Warn owners that chronic, severe hypoglycemia may cause permanent neurologic abnormalities.

Physical Examination Findings

Physical examination findings may reveal a normal or ataxic animal, muscle weakness (usually seen as shaking or collapse in the rear), mental dullness, or disorientation. Affected dogs are usually normal between hypoglycemic episodes, a fact that may help differentiate insulinoma from other causes of hypoglycemia. Withholding food before and during the evaluation may precipitate seizures in affected animals. Neuronal demyelination and axonal degeneration may result from chronic hypoglycemia. Although the cause is not known for certain, direct toxic effects of hypoglycemia on peripheral nerves or a paraneoplastic neuropathy has been postulated. Signs of peripheral polyneuropathy, such as ataxia and weakness, may continue despite appropriate therapy.

Diagnostic Imaging

Thoracic and abdominal radiographs do not contribute to the diagnosis; however, the location of the tumor in the pancreas can sometimes be determined using ultrasound. Unfortunately, tumor masses are often so small that they are difficult to identify. Ultrasonography may reveal metastasis to the liver and regional lymph nodes in some affected animals. Thoracic radiographs are indicated to look for metastasis, although pulmonary metastasis is rare. Abdominal and thoracic CT can be used to identify thoracic and lymph node metastasis, but identification of false-positive lesions is possible. Therefore intraoperative inspection and palpation of the pancreas are superior to CT. Positron emission tomography/CT may be more effective than ultrasound or CT in identifying canine insulinomas, but this has yet to be fully evaluated.

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BOX 22.11 Whipple's Triad

- Clinical signs associated with hypoglycemia (usually neurologic abnormalities)
- Fasting blood glucose concentrations of ≤40 mg/dL
- Relief of neurologic signs with feeding or glucose administration

Laboratory Findings

A tentative diagnosis of insulinoma is based on demonstration of Whipple's triad (Box 22.11). Fasting or nonfasting blood glucose concentrations are often below 70 mg/dL. If blood glucose concentrations initially are within the normal range, most affected dogs can be made hypoglycemic by fasting for 12 to 24 hours. Blood glucose measurements should be determined every 2 to 3 hours in these animals until hypoglycemia is detected. Serum fructosamine may be useful for diagnosing chronic, occult hypoglycemia.

Once hypoglycemia has been confirmed, blood for serum insulin measurement should be obtained immediately. If food has been withheld from the animal to induce hypoglycemia, serum insulin concentrations should be measured on the first hypoglycemic sample (i.e., <55 mg/dL). Normal fasting serum immunoreactive insulin concentrations range from 5 to 26 μIU/mL, whereas insulin levels in affected animals often exceed 70 μIU/mL. Evaluating the absolute insulin concentration when the patient is hypoglycemic while considering the history, physical examination, and other clinical pathology data is the best approach. In some cases, definitive diagnosis of insulinoma may require exploratory surgery.

DIFFERENTIAL DIAGNOSIS

Insulinomas should be considered a differential diagnosis in any dog with persistent and progressive seizures. Once hypoglycemia has been verified, these tumors must be differentiated from other causes of hypoglycemia, including extrapancreatic neoplasms, hunting dog hypoglycemia, sepsis, hepatic failure, hypoadrenocorticism, glycogen storage disorders (very rare), and hypopituitarism.

MEDICAL MANAGEMENT

Dogs with insulinomas should be fed frequent, small meals. Three to six meals a day of a diet high in protein and complex carbohydrates but low in refined sugar reduces clinical signs. Exercise restriction may help alleviate clinical signs. Glucocorticoid therapy (Box 22.12) may help prevent hypoglycemia caused by islet cell tumors by increasing hepatic glucose production and decreasing cellular glucose uptake. The lowest possible dose that controls hypoglycemia should be used to prevent iatrogenic HAC (e.g., polyphagia, polydipsia, bilateral symmetric alopecia, thin epidermis). If clinical signs of HAC occur, glucocorticoid therapy may be reduced and alternate drugs used; however, HAC may be preferable to hypoglycemia. Diazoxide (see Box 22.12) is an oral hyperglycemic agent that inhibits pancreatic insulin secretion and glucose uptake by tissue. It raises blood glucose concentrations in some dogs with insulinomas; however, side effects, such as anorexia, vomiting, aplastic anemia, cataracts, bone marrow suppression, thrombocytopenia, anorexia, diarrhea, tachycardia, and fluid retention, may occur. Diazoxide should be used cautiously in animals with hepatic dysfunction. If hypoglycemia is severe and unresponsive, IV administration of 5% or 10% dextrose may be necessary to maintain blood glucose concentrations in the normal range until surgery can be performed. Alloxan and a somatostatin analog (octreotide) have been used in a few dogs with insulinomas; however, too little information is available at this time to recommend their use.

BOX 22.12 Oral Hyperglycemic Agents

Prednisolone
0.25–2 mg/kg q12h

Diazoxide (Proglycem)
Start with 5 mg/kg q12h with meals; may gradually increase to 60 mg/kg divided q12h; concurrent administration of hydrochlorothiazide may enhance effects of diazoxide

Streptozotocin may be efficacious in dogs with insulinomas that have metastatic disease. It has been suggested that it can be given safely to dogs at a dosage of 500 mg/m^2 IV every 3 weeks when combined with a protocol for induction of diuresis. Give 0.9% NaCl administered at a rate of 18.3 mL/kg per hour IV for 3 hours before streptozotocin administration. Dilute the streptozotocin to an appropriate volume and give over 2 hours at the same rate as the fluid administration. Then give 0.9% NaCl for an additional 2 hours. To reduce vomiting, butorphanol or antiemetics may be given intramuscularly immediately after streptozotocin administration; however, these drugs are usually ineffective. Repeat the treatment every 3 weeks until tumor progression is evident (i.e., the tumor increases significantly in size), recurrence of hypoglycemia is observed, or toxicity (e.g., renal, hepatic) is noted that is unresponsive to supportive care. Other abnormalities that may occur in association with streptozotocin administration include neutropenia, thrombocytopenia, anorexia, diarrhea, and DM.

SURGICAL TREATMENT

Preoperative Management

Fluid therapy with 5% glucose should be initiated 12 to 24 hours before surgery. Food is withheld 6 to 8 hours before surgery. Blood glucose concentrations should be measured immediately before surgery and additional glucose given if the concentration is below 75 to 100 mg/dL.

Anesthesia

The goal of surgery is to maintain blood glucose concentrations above 75 to 200 mg/dL. Thiopental, propofol, or alfaxalone may be used for induction of anesthesia because they reduce cerebral glucose metabolism. Etomidate should be avoided because it may cause adrenal suppression. After intubation, anesthesia should be maintained with isoflurane or sevoflurane. Isoflurane and sevoflurane reduce the cerebral metabolic rate more than does halothane. Blood glucose concentrations should be monitored regularly during surgery (i.e., every 20–40 minutes) to prevent intraoperative hypoglycemia.

Surgical Anatomy

See the discussion of the surgical anatomy of the pancreas on p. 603.

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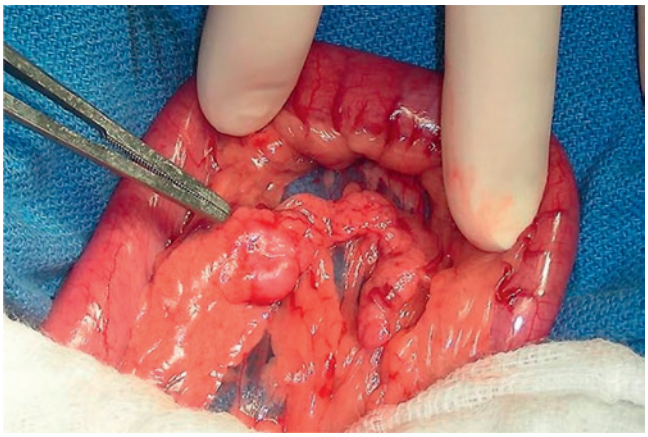


FIG. 22.15 Functional islet cell adenocarcinoma in a dog.

BOX 22.13 Methylene Blue Administration

Dilute 3 mg/kg of 1% methylene blue in 250 mL of 0.9% sterile saline and give intravenously over 30–40 min.

Positioning

The animal is positioned in dorsal recumbency, and the caudoventral thorax and the entire abdomen are prepared for aseptic surgery.

SURGICAL TECHNIQUE

Explore the cranial abdominal cavity thoroughly for evidence of neoplasia. Carefully and gently palpate the entire pancreas for evidence of tumor nodules. Most dogs have solitary nodules (Fig. 22.15). Tumors are found with equal frequency in the left and right lobes of the pancreas and in the body. Metastasis is noted in approximately 50% of cases at the time of surgery. Metastasis usually occurs to the regional lymph nodes and liver; however, duodenal, mesenteric, and omental metastasis may also be noted. *Perform a partial pancreatectomy (see p. 605), removing tumor nodules with as wide a margin of normal tissue as possible. Submit excised lesions for histopathologic examination. Excise metastatic nodules if possible.*

If the tumor cannot be identified, methylene blue may be administered intravenously (Box 22.13). Methylene blue may stain neoplastic islet cells, helping to differentiate them from surrounding normal tissue. Maximum staining occurs within 30 minutes. A common side effect of methylene blue administration is hemolytic anemia resulting from the formation of Heinz bodies.

NOTE Fatal Heinz body anemia in dogs has been reported with the use of methylene blue.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Balfour abdominal retractors are useful for abdominal exploration. Sterile Q-tips or fine hemostats are useful for separating pancreatic tissue during partial pancreatectomy. Duct ligation is performed using 3-0 or 4-0 nonabsorbable suture material (see p. 605).

BOX 22.14 Postoperative Recommendations for Maintaining Glucose Concentrations in Patients With Insulinomas

- Initially monitor blood glucose every 1–2 h.
- Continue providing glucose-containing fluids until the blood glucose concentration is >75 mg/dL, then measure every 2–4 h, depending on how high it is.
- If hypoglycemia persists, administer steroids or diazoxide.

Methylene blue may be given intravenously to help identify primary and metastatic nodules (see previous discussion).

POSTOPERATIVE CARE AND ASSESSMENT

Blood glucose concentrations should be measured frequently during the first 24 hours after surgery. Pancreatitis may result from surgical manipulation of the pancreas and should be treated aggressively, as described on p. 605. Small amounts of water may be administered the day after surgery, and if vomiting does not occur, feeding of small, frequent meals may be initiated. Once the blood glucose concentration stabilizes at 75 to 100 mg/dL or higher, the glucose infusion can be discontinued (Box 22.14). If persistent hypoglycemia continues, medical therapy (glucocorticoids, diazoxide; see Box 22.12) should be initiated. Prolonged hypoglycemia may cause cerebral laminar necrosis. Neurologic signs (e.g., ataxia, bizarre behavior, coma, seizures) may persist in such animals despite normoglycemia. Transient hyperglycemia occasionally occurs and may persist for years after surgery. Insulin therapy may be indicated if blood glucose concentrations above 180 mg/dL persist for longer than 3 to 5 days.

COMPLICATIONS

Complications of surgery in animals with insulinomas include persistent hypoglycemia, pancreatitis, DM, epilepsy, and diffuse polyneuropathy. The most common causes of postoperative hypoglycemia are unrecognized or nonresectable metastases and multiple or incompletely resected primary tumors. Persistent hyperglycemia occurs in up to one-third of dogs undergoing surgical removal of insulinomas and is thought to be a result of suppression of normal β-cells by tumor insulin, resulting in loss of insulin production.

PROGNOSIS

The median survival times for dogs with insulinoma has ranged from 12.3 to 18.2 months, but survival times between studies are difficult to compare because of differences in treatment and data reported (Table 22.4). In a study of 28 dogs with insulinoma, median survival time was 547 days.[21] The median survival of 19 dogs undergoing partial pancreatectomy was 785 days, and for those subsequently receiving prednisone therapy on relapse, median survival was 1316 days. Median survival of dogs with insulinoma treated with medical therapy alone was 196 days.

Prognostic biomarkers have been studied to facilitate optimal patient management. The Ki67 index has proved prognostically significant for both the disease-free interval and overall survival time. In addition to known factors such as tumor size and stage,

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TABLE 22.4 Reported Survival Times for Dogs With Insulinomas

Reference	Number of Cases	Approximate Median Survival Time (Months[a])	Comments
Kruth et al., 1982[34]	25	12.3	Mean survival time; medically and surgically treated cases; peri-mortality rate associated with owners euthanizing due to presence of inoperable lesions or presence of presumed metastatic lesions
Mehlhaff et al., 1985[35]	35	14.2	Mean survival time; medically and surgically treated cases; peri-mortality rate associated with owners euthanizing due to presence of inoperable lesions or presence of presumed metastatic lesions
Leifer et al., 1986[36]	18	14.5	Cases surviving surgery, no relapse
Caywood et al., 1988[37]	47	18	Stage I[a]; surgically and medically treated cases
Tobin et al., 1999[38]	26	12.7	Surgically treated cases only
Polton, 2006[21]	28 19	18.2 16.5	Surgically and medically treated cases Median postoperative time to remission
Northrup et al., 2013[39]	19	10.3	Medical management with biweekly streptozotocin; serious adverse events reported

[a]Statistically significant.

Ki67 can act as a biomarker of insulinoma that may be used to predict clinical outcome.

NOTE If metastasis is not apparent at surgery, survival of longer than 1 year may occur, even though cures are unlikely.

GASTRINOMAS

DEFINITIONS

Gastrinomas are tumors that secrete excessive gastrin. **Zollinger-Ellison syndrome** is the term used to describe a syndrome of gastric acid hypersecretion, gastrointestinal ulceration, and non–β-cell pancreatic tumors. Gastrinomas are also called *non–β-cell tumors* and *gastrin-secreting tumors*. The terms *gastrinoma* and *Zollinger-Ellison syndrome* are often used interchangeably; however, gastrinomas can arise in other parts of the alimentary tract. Zollinger-Ellison syndrome refers specifically to gastrinomas arising in the pancreas.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Gastrinomas are rare tumors in dogs and cats. They are derived from ectopic amine precursor uptake decarboxylase cells in the pancreas and produce an excess of the hormone gastrin. Gastrin is normally secreted by cells of the antral and duodenal mucosa in response to antral distention and stimulation by amino acids. Excess gastrin causes hyperacidity, which produces multiple duodenal ulcerations and/or erosions. Pancreatic gastrin-secreting tumors are usually locally invasive into adjacent parenchyma and frequently metastasize to regional lymph nodes or the liver or both.

DIAGNOSIS

Clinical Presentation

Signalment

Dogs and cats may be affected. Too few cases have been reported to determine breed or gender predisposition. Affected animals are usually middle-aged to older.

History

Most animals have clinical signs of anorexia, vomiting (which is occasionally blood tinged), regurgitation, intermittent diarrhea, weight loss, and/or dehydration. Clinical signs may be present for several days or months before diagnosis. Animals may have been treated for gastric ulcers for months with poor response.

Physical Examination Findings

Clinical findings are nonspecific and may include dehydration, diarrhea, melena, hematemesis ("coffee grounds" appearance), steatorrhea, and/or weight loss. Abdominal pain is inconsistent. Gastric ulcer perforation may cause generalized peritonitis (see p. 527).

Diagnostic Imaging

Radiographs and ultrasonography are nondiagnostic for gastrinomas because pancreatic masses are generally too small to be visualized. Endoscopy is the most useful technique for diagnosing esophagitis, gastric mucosal hypertrophy, or duodenal ulceration in dogs with suggestive clinical signs. Ulcers are most commonly located in the proximal duodenum.

Endoscopy

On endoscopy, patients typically have esophagitis (because of profuse vomiting of acid) and duodenal ulcers or erosions. Duodenal biopsies typically have minimal inflammation. Gastric ulceration is much less common, but erosions and/or mucosal hypertrophy may be seen.

Laboratory Findings

Nonspecific laboratory abnormalities noted in animals with gastrinoma include anemia, hypoproteinemia, elevated serum alkaline phosphatase activity, and/or leukocytosis. Electrolyte and acid-base abnormalities (e.g., hypochloremia, hypokalemia, metabolic alkalosis, metabolic acidosis) may occur if vomiting has been severe. Preoperative diagnosis of gastrinoma is based on demonstration of hypergastrinemia. Blood samples for serum gastrin analysis should be obtained after a 12-hour fast and before treatment with any antacid drug. Serum gastrin levels of animals with Zollinger-Ellison syndrome may exceed 1000 pg/mL.

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DIFFERENTIAL DIAGNOSIS

Gastrinomas must be differentiated from other causes of gastrointestinal tract ulceration, including nonsteroidal antiinflammatory drugs, dexamethasone, local neoplasia, infiltrative disease, mast cell tumors, DIC, hepatic failure, circulatory shock, and septic shock (see also p. 427). Other causes of hypergastrinemia include renal failure, gastric outflow obstruction, chronic gastritis, and recent proton pump inhibitor therapy.

MEDICAL MANAGEMENT

Because of the aggressive biological behavior of this malignant neoplasm, the prognosis for long-term cure is poor; however, aggressive medical management includes the use of proton pump inhibitors (Box 22.15). Proton pump inhibitors (e.g., omeprazole) are the most potent known inhibitors of gastric acid secretion. Other agents that may be used to help treat ulcers in dogs with gastrinomas include those that protect the gastric mucosa from damage (see Box 22.15). The effectiveness of these drugs in animals with gastrinoma can be limited, and beneficial effects vary from short to long periods of time.

Sucralfate forms a protective coating over the ulcer or erosion (see Box 22.15). Cimetidine, ranitidine, and famotidine are H_2-receptor blockers that reduce acid secretion; they are much less effective than proton pump inhibitors.

SURGICAL TREATMENT

Exploratory laparotomy is often required to confirm the diagnosis. Surgical resection of the pancreatic mass may provide a cure if metastasis is not present. If metastasis is present, surgical debulking of the mass and removal of operable metastatic lesions may improve the efficacy of medical therapy and prolong survival. The gastrointestinal tract should be closely inspected during surgery for evidence of ulcerations that may perforate. Any such lesions should be removed or should have a serosal patch (see p. 447). Total gastrectomy has been recommended for animals in which the condition is unresponsive to medical therapy; however, because of long-term complications (e.g., malnutrition, dysphagia, bile reflux), this procedure is seldom performed.

Preoperative Management

If possible, the animal's condition should be stabilized before surgery. Whole blood should be given if the animal is severely anemic (i.e., PCV <20% [see Box 4.1 and Table 4.5]), and anemic animals should be oxygenated before induction of anesthesia. Electrolyte and acid-base abnormalities should be corrected and fluid therapy initiated before surgery.

Anesthesia

See p. 600 for anesthetic recommendations for animals undergoing pancreatic surgery. Preoperative volume resuscitation may be necessary.

Surgical Anatomy

See p. 603 for discussion of the surgical anatomy of the pancreas and p. 399 for discussion of the surgical anatomy of the stomach.

Positioning

The animal is placed in dorsal recumbency, and the abdomen is prepared for a ventral midline incision. The caudal thorax and the entire ventral abdomen should be prepared for aseptic surgery.

BOX 22.15 Medical Therapy for Animals With Gastrinoma

Omeprazole (Prilosec)[a]
Dogs: 1–2 mg/kg PO q12h

Pantoprazole (Protonix)
Dogs: 1 mg/kg (anecdotal dose) IV q24h

Sucralfate (Carafate) (Suspension, Not Tablets)
Dogs: 0.5–1 g/dog PO q6–8h
Cats: 0.25 g/cat PO q8–12h

[a]Takes 2–5 days to reach maximal effect.
PO, Orally; *IV,* intravenous.

SURGICAL TECHNIQUE

Perform a thorough abdominal exploration. Inspect the draining lymph nodes, liver, duodenum, and mesentery for evidence of metastasis. Inspect the entire pancreas for a mass lesion. Perform a partial pancreatectomy (see p. 605) and resect metastatic lesions that are accessible. Submit excised tissues for histopathologic examination.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

If the animal is severely hypoproteinemic or anemic, wound healing may be delayed. In such cases, polydioxanone or polyglyconate suture is preferred to close gastrotomy and abdominal incisions (see p. 415). These sutures may also be used to perform a serosal patch.

POSTOPERATIVE CARE AND ASSESSMENT

Anemic animals benefit from nasal oxygen given postoperatively. The patient must be monitored, and if signs suggestive of pancreatitis are seen, aggressive therapy is indicated (see p. 605 for treatment of pancreatitis). Small amounts of water should be given the day after surgery, and the patient observed for vomiting. If vomiting does not occur, small amounts of food can be given 24 hours postoperatively. The diet should be low in fat and fiber and should contain moderate amounts of protein and carbohydrates to aid gastric emptying. Fluid therapy should be continued until the animal is eating and drinking. Medical therapy for ulcers should be continued until clinical signs resolve. Long-term medical therapy may be necessary to control gastric hypersecretion caused by hypergastrinemia and to reduce the incidence and severity of ulcers.

PROGNOSIS

Because of the malignant nature of this tumor and its high propensity for metastasis, the long-term prognosis is generally grave.

EXOCRINE PANCREATIC NEOPLASIA

DEFINITION

Exocrine pancreatic carcinomas are malignant tumors that arise from acinar or ductular epithelial cells. They are also called *pancreatic adenocarcinomas.*

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GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Exocrine pancreatic tumors are slightly more common than tumors of the pancreatic islet cells in dogs and cats. Pancreatic tumors are more common in human beings than in dogs and are associated with an extremely high mortality rate (approximately 90% within 1 year of diagnosis). Most pancreatic tumors are malignant (adenocarcinoma); they are aggressive tumors that invade locally and metastasize readily. The most common sites for metastasis are the liver, lungs, peritoneum, and regional lymph nodes. Metastatic pancreatic carcinoma has been diagnosed in a dog with diabetes insipidus. Benign pancreatic tumors (i.e., adenomas) are rare.

NOTE Most pancreatic masses are caused by pancreatitis, not neoplasia. Never euthanize a patient with a pancreatic mass without a histologic diagnosis, no matter how "bad" the mass looks grossly.

DIAGNOSIS

Clinical Presentation

Signalment

Pancreatic adenocarcinomas occur more commonly in older animals; Airedale terriers and boxers have been reported to be at higher risk for this tumor. A gender predisposition has not been proved in dogs, although pancreatic carcinoma seems to be more common in males.

NOTE Clinical signs or gross appearance does not help differentiate pancreatic adenocarcinoma from benign pancreatic disease.

History

Animals with pancreatic adenocarcinoma may have vomiting, abdominal pain, anorexia, weight loss, lethargy, abdominal distention, and/or diarrhea. The history may be acute or chronic. Adenomas are usually incidental findings at surgery or at necropsy and are not associated with clinical signs.

Physical Examination Findings

Physical examination findings for exocrine pancreatic carcinoma may include abdominal pain on palpation and/or ascites occurring secondary to compression of the portal vein or other vessels or as the result of widespread abdominal metastasis. Some animals may have a palpable abdominal mass; others may have icterus secondary to common bile duct obstruction.

Diagnostic Imaging

An ill-defined increase in soft tissue opacity in the right cranial abdominal quadrant may be noted on survey abdominal radiographs. If ascites is present, loss of visceral detail throughout the abdomen may be observed. Abdominal ultrasonography often reveals a mass in the area of the pancreas, but it is not necessarily easy to distinguish from pancreatitis. Distention of the gallbladder and bile ducts may be noted with obstruction of the extrahepatic biliary tract. Obstruction of gastric outflow may be seen on contrast studies of the upper gastrointestinal tract.

Laboratory Findings

Laboratory abnormalities have not been well defined in animals with exocrine pancreatic neoplasia. Abnormalities consistent with extrahepatic cholestasis (i.e., elevated alkaline phosphatase and hyperbilirubinemia) are often present. Some animals may show mild leukocytosis, dehydration, and hemoconcentration. Extremely high serum lipase values might suggest pancreatic carcinoma, but the test is not generally recommended.

DIFFERENTIAL DIAGNOSIS

Exocrine pancreatic carcinoma must be differentiated from benign and metastatic pancreatic disease. Nodular pancreatic hyperplasia, a condition seen in older animals, is characterized by multiple small, white lesions that protrude minimally from the pancreatic surface. Adenomas are usually small masses that may contain cysts. These conditions are not associated with clinical signs. Pancreatic carcinomas are usually well advanced at the time of diagnosis, and it may be difficult to determine the site of origin for neoplastic masses.

MEDICAL MANAGEMENT

Although numerous treatments have been used in human beings in an attempt to improve the survival of patients with pancreatic adenocarcinoma, only those with resectable lesions at the time of laparotomy have a fair prognosis. Chemotherapeutic agents have not prolonged the life of people or animals with this tumor.

SURGICAL TREATMENT

Surgical resection is the treatment of choice; however, many animals are presented with advanced disease and surgical resection is not possible.

Preoperative Management

The animal's condition should be stabilized before surgery with administration of IV fluids and correction of acid-base and electrolyte abnormalities.

Anesthesia

See the discussion of anesthetic management of animals with pancreatic disease on p. 600.

Surgical Anatomy

The surgical anatomy of the pancreas is described on p. 603.

Positioning

The animal is prepared for a ventral midline exploratory procedure. The entire abdomen and the caudal thorax should be prepared for aseptic surgery.

SURGICAL TECHNIQUE

Make an abdominal incision that extends from the xiphoid cartilage as far caudally as necessary to allow complete exploration of the abdominal cavity. After identifying the pancreatic mass (Fig. 22.16), explore the abdominal organs, peritoneum, and regional nodes for evidence of metastasis. Euthanasia should be considered in animals with widespread metastasis. *Perform a partial pancreatectomy, if possible. Confirm the patency of the common bile duct before closing the abdomen.*

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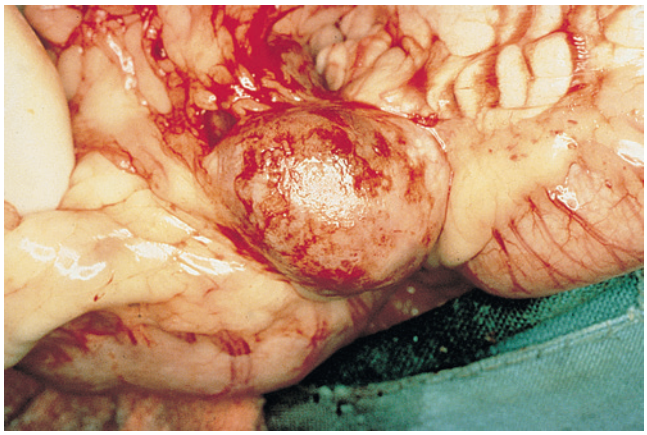


FIG. 22.16 Pancreatic carcinoma in a dog.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

A standard soft tissue pack or a general surgery pack is usually all that is required. See p. 605 for requirements for partial pancreatectomy.

POSTOPERATIVE CARE AND ASSESSMENT

These animals may have pancreatitis secondary to tumor at the time of diagnosis, and this may require therapy (see p. 605). Animals presented for treatment that have pancreatic carcinomas are often debilitated and require special attention to ensure that their nutritional needs are met postoperatively. Enteral or parenteral hyperalimentation should be considered. See also p. 605 for postoperative care of patients with pancreatic disease.

PROGNOSIS

The prognosis is extremely poor for animals with pancreatic carcinomas. Most have widespread disease at the time of diagnosis, and many are euthanized at surgery. Survival of less than 3 months should be expected for most of the remaining animals.

Surgery of the Thyroid and Parathyroid Glands

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Thyroidectomy is removal of a thyroid gland. **Hypothyroidism** is deficient secretion of thyroxine. **Goitrous hypothyroidism** is caused by an abnormal iodine uptake or by defects in iodine uptake, organification, or thyroglobulin formation. **Nongoitrous hypothyroidism** is spontaneous hypothyroidism that may be immune mediated (i.e., lymphocytic thyroiditis) or may result from idiopathic atrophy. **Hyperthyroidism** is excessive secretion of thyroxine. **Primary hyperparathyroidism** is excessive secretion of parathyroid hormone (PTH) by one or more abnormal parathyroid glands.

BOX 22.16 Treatment of Canine Hypothyroidism

Maintenance

Levothyroxine (Soloxine) 18–22 µg/kg PO q12h

Before Surgery (If Not on Maintenance Therapy, Which Is Preferred)

1. Oral: liothyronine (T_3; Cytobin or Cytomel) 4.4 µg/kg PO q6–8h, *or*
2. Intravenous: L-thyroxine 4–5 µg/kg (1 dose) (use with caution)

PO, Orally.

PREOPERATIVE MANAGEMENT

Hypothyroidism is a common endocrinopathy in dogs. It is usually due to thyroid dysfunction (primary hypothyroidism), although pituitary and hypothalamic causes are occasionally diagnosed. Three weeks of administration of trimethoprim-sulfamethoxazole (14.1–16 mg/kg orally, twice daily) will depress total thyroxine and free thyroxine and will elevate canine thyroid-stimulating hormone (TSH) concentrations, mimicking hypothyroidism. Thyroid function tests should be interpreted carefully in dogs that are receiving glucocorticoids, phenobarbital, and carprofen. Secretion of thyroid hormones triiodothyronine (T_3) and thyroxine (T_4) from the thyroid is controlled by a feedback mechanism between the hypothalamus, pituitary, and thyroid glands. Thyrotropin (TSH) is produced in the pars distalis of the pituitary gland. It stimulates the synthesis and release of thyroglobulin, a precursor of T_3 and T_4, as well as T_3 and T_4. Release of thyrotropin is controlled by a neuropeptide produced in the hypothalamus called *thyrotropin-releasing hormone* (TRH). TRH secretion is inhibited by high circulating levels of glucocorticoids (e.g., HAC) or thyroid hormone. Primary hypothyroidism is usually caused by idiopathic follicular atrophy or lymphocytic thyroiditis. Dogs with lymphocytic thyroiditis often have circulating thyroglobulin antibodies that form antigen-antibody complexes in the gland, causing functional glandular tissue to be replaced by fibrous tissue. Feline hypothyroidism is usually caused by thyroidectomy, damage to the blood supply during parathyroidectomy, or destruction by iodine-131 (I^{131}) therapy. Congenital hypothyroidism may occur in Abyssinian cats. The disease is inherited as an autosomal recessive trait and appears to be the result of a defect of iodide organification. Congenital hypothyroidism has also been reported in dogs.

Hypothyroidism may be manifested as lethargy, exercise intolerance, weight gain, constipation, nonpruritic symmetric alopecia, peripheral neuropathies (e.g., laryngeal paralysis, vestibular deficits), reproductive problems, cardiovascular changes (i.e., bradycardia and weak apex beat), and/or coagulopathies. Hypothyroidism may also result in diminished activity of factor VIII or of factor VIII–related antigen, which may predispose animals with von Willebrand disease (vWD) to spontaneous bleeding or serious hemorrhage during surgery. The mean von Willebrand factor/antigen (vWF) concentration in hypothyroid dogs has been found to be significantly reduced compared with that in euthyroid dogs. It appears that reduced concentrations of plasma vWF can be found in dogs in association with congenital vWD or with vWD acquired through hypothyroidism. Animals with untreated severe hypothyroidism and bleeding tendencies undergoing emergency procedures should be given oral L-triiodothyronine (Box 22.16) three or four times a day, or a

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single IV dose of L-thyroxine. Elective procedures should be postponed until replacement therapy has been maintained for a minimum of 2 weeks. If excessive bleeding is noted despite thyroid supplementation, whole blood, plasma, or cryoprecipitate should be given (see Box 4.1 and Table 4.5).

NOTE Hypothyroid animals may bleed excessively during surgery. Monitor hemostasis carefully.

Animals with hyperparathyroidism are often brought in because of signs caused by hypercalcemia. PTH is synthesized by chief cells of the parathyroid glands. PTH stimulates renal reabsorption of calcium, mobilizes calcium from bone, and promotes intestinal calcium reabsorption. PTH also controls hydroxylation of 25-hydroxyvitamin D_3 to 1,25-dihydroxyvitamin D_3 in the proximal renal tubules. 1,25-Dihydroxyvitamin D_3 regulates PTH secretion through a negative feedback mechanism. PTH is synthesized and secreted in response to decreases in circulating calcium levels. Functional parathyroid neoplasms (primary hyperparathyroidism; see p. 625) cause hypercalcemia through excessive secretion of PTH; this causes increased renal reabsorption of calcium and increased renal excretion of phosphorus, increased release of calcium and phosphorus from bone, and increased intestinal absorption of calcium and phosphorus. The preoperative management of animals with functional parathyroid tumors is described on p. 616. Primary hypoparathyroidism is rare in dogs and cats. It primarily affects middle-aged female dogs, occurring secondary to lymphocytic parathyroiditis. Most affected animals have a history of neurologic abnormalities (particularly seizures) or neuromuscular disease.

Cystic thyroid and parathyroid lesions have been reported in older cats but are uncommon. These lesions may be benign (thyroid cysts or cystadenomas) or malignant (parathyroid adenocarcinomas). Surgical resection may be curative, and long-term survival is excellent. Differentials for a cystic ventral cervical mass should include branchial cyst, thyroglossal cyst, thyroid cyst, thyroid cystadenoma, parathyroid cyst, parathyroid cystadenoma, thyroid carcinoma, salivary mucocele, and abscess.

ANESTHESIA

Hypothyroidism may prolong recovery from anesthesia. Dosages of premedications and anesthetics should be reduced and titrated to effect in moderately or severely affected animals. Blood pressure and hematocrit should be closely monitored during anesthesia and in the early postoperative period. Blood should be available in case excessive bleeding occurs intraoperatively. Hypothermia may be of greater concern in these patients because of their inability to regulate body temperature normally; care should be taken to maintain body temperature during surgery and to rewarm these patients after surgery. See pp. 621 and 628 for anesthetic recommendations for animals undergoing thyroidectomy.

NOTE Anesthetize hypothyroid animals with care; these patients may require reduced dosages of anesthetics.

ANTIBIOTICS

Guidelines for appropriate use of perioperative antibiotics should be followed in hypothyroid patients (see Chapter 9). Prophylactic antibiotic therapy should be considered in animals that are debilitated or obese, have pyoderma, or that have concurrent HAC.

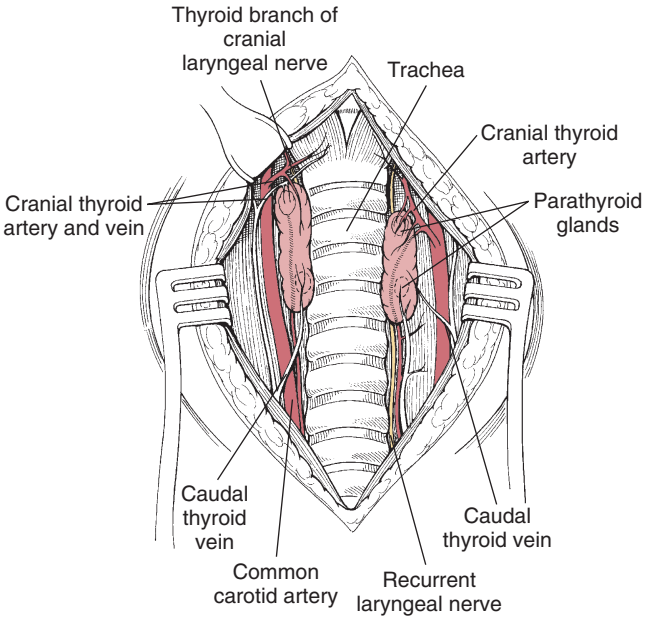


FIG. 22.17 The thyroid gland is lateral and slightly ventral to the fifth to eighth cartilage rings.

SURGICAL ANATOMY

The thyroid gland (with two lobes) is a dark red, elongated structure attached to the outer surface of the proximal portion of the trachea (Fig. 22.17). The lobes are usually positioned laterally and slightly ventral to the fifth to eighth cartilage rings. The left lobe is usually located one to three tracheal rings caudal to the right lobe. In adult dogs, they are approximately 5 cm long and 1.5 cm wide; in cats, they are 2 cm long and 0.3 cm wide. Occasionally the right and left lobes are connected by a ventral isthmus. Unlike most glandular organs, they can often be palpated when enlarged. Thyroid secretions (T_4, T_3, and calcitonin) exert a major effect on metabolism. Thyroid hormone is synthesized by follicular cells, stored intercellularly, and released into the circulation. In adults, it causes an increase in the overall metabolic rate; in juveniles, it stimulates growth. Calcitonin (formed by parafollicular C cells) lowers blood calcium by stimulating calcium uptake. Functional accessory thyroid tissue is common along the trachea, thoracic inlet, mediastinum, and thoracic portion of the descending aorta. Thyroid follicular cells arise from a midline outpouching known as the *thyroid diverticulum* on the ventral pharyngeal floor. The pharyngeal connections of the diverticulum usually separate completely; however, a persistent connection that has functional glandular epithelium and cysts along its course may remain *(thyroglossal duct)*.

The cranial and caudal thyroid arteries are the principal blood supply of the thyroid. The cranial thyroid artery arises from the common carotid artery; the caudal thyroid artery typically arises from the brachiocephalic artery. The cranial and caudal thyroid arteries anastomose on the dorsal surface of the gland, where they send numerous vessels that supply the gland. The cranial thyroid artery in dogs usually sends a branch that supplies the

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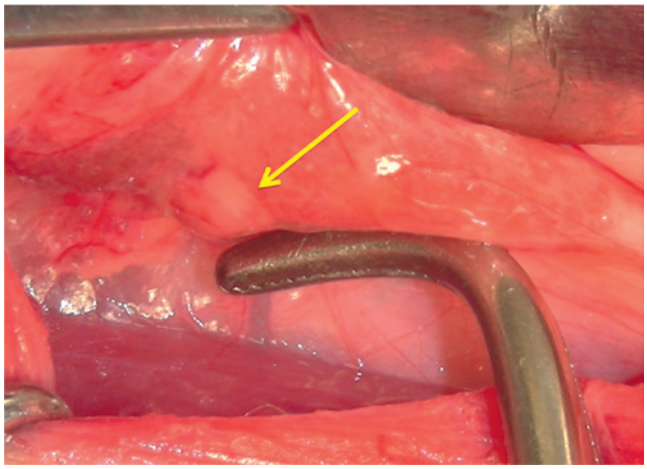


FIG. 22.18 Enlarged and nodular external parathyroid gland *(arrow)* in a dog with hypercalcemia.

external parathyroid gland before entering the thyroid parenchyma. In cats, the branch that supplies the external parathyroid gland may arise from the cranial thyroid artery after it has perforated the capsule. Caudal thyroid arteries may not be present in cats. Innervation to the thyroid is provided via the thyroid nerve, which is formed from the cranial ganglion and the cranial laryngeal nerve.

The parathyroid glands are small, ellipsoid discs that usually occur as four structurally independent glands in close association with the thyroid glands. The external parathyroid glands (so named because they lie outside the thyroid capsule) are normally found on the cranial dorsolateral surface of the respective thyroid (Fig. 22.18). The internal parathyroid glands are embedded within the thyroid parenchyma, usually at the caudomedial pole.

SURGICAL TECHNIQUE

Thyroidectomy may be performed via an intracapsular or extracapsular approach. The extracapsular approach is used in dogs with malignant thyroid tumors (e.g., carcinomas; see p. 628), and no attempt is made to spare the ipsilateral parathyroid glands. Intracapsular and modified extracapsular approaches have been described for thyroidectomy in cats (see pp. 621–623). These techniques spare the external parathyroid glands in an attempt to prevent complications associated with hypoparathyroidism. A modification of the original intracapsular approach, developed to reduce the incidence of postthyroidectomy hyperthyroidism, involves excising most of the thyroid capsule once the thyroid tissue has been removed. Recurrence of hyperthyroidism in cats after thyroidectomy is thought to be the result of hypertrophy of small nests of functional thyroid tissue attached to the capsule and not removed.

HEALING OF THE THYROID AND PARATHYROID GLANDS

Abnormal thyroid tissue (i.e., adenomatous tissue) appears to regenerate and hypertrophy after incomplete feline thyroidectomy. Parathyroid tissue may be able to revascularize and regain function even if it has been totally separated from its blood supply. Therefore most surgeons recommend implanting an inadvertently excised parathyroid gland into surrounding muscle rather than discarding it. Ectopic parathyroid tissue may hypertrophy after removal of the parathyroid glands, resulting in normal parathyroid function.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Delayed wound healing may occur in animals with hypothyroidism; care should be used in closing surgical wounds in these patients. See pp. 623, 626 and 628for a discussion of the instruments for thyroidectomy and parathyroidectomy, respectively.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative care and assessment of animals undergoing thyroidectomy for hyperthyroidism or neoplasia are provided on pp. 627 and 629, respectively.

SPECIFIC DISEASES

FELINE HYPERTHYROIDISM

DEFINITIONS

Hyperthyroidism is a multisystemic disease that results from excessive production and secretion of T_4. **Goiter** is an enlargement of the thyroid gland. **Graves disease** describes an autoimmune disorder of human beings in which circulating autoantibodies stimulate thyroid tissue. It is the most common cause of human hyperthyroidism.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Hyperthyroidism may occur in dogs or cats; however, it is much more common in cats, in which it is generally associated with adenomatous hyperplasia of one or both thyroid glands. Approximately 90% of affected cats have bilateral thyroid lobe involvement, although the enlargement is usually asymmetric. In 5% to 10% of cats, the thyroid mass is ectopic (i.e., at the thoracic inlet or in the cranial mediastinum). Feline hyperthyroidism secondary to malignant thyroid carcinoma is rare. The cause of feline hyperthyroidism is unknown. Suggested causes have included circulating thyroid-stimulating immunoglobulins, serum thyroid growth-stimulating immunoglobulins, dietary goitrogens, and viral causes.[40,41] Studies suggest that cats that (1) consume food packaged in cans, (2) have diets containing fish, (3) eat more than 50% wet cat food, or (4) use a litter box have increased risk of hyperthyroidism. It has been suggested that iodine recommendations for commercial cat foods in the past may have inadvertently contributed to the recent epidemic of feline hyperthyroidism, because changes in the recommended iodine concentration of foods may have resulted in a reduction in iodine supplementation since the late 1970s. Canned food may predispose to hyperthyroidism because toxic chemicals such as bisphenol-A are released into the food from the plasticizer linings placed in cans during the canning process. Exposure to fertilizers, herbicides, plant pesticides, flea products, and smoke did not seem to be associated with increased risk of this disease. Advanced age is a risk factor, and purebred cats have a decreased risk of developing hyperthyroidism.

Excessive circulating T_4 causes multisystemic organ dysfunction. Thyrotoxicosis increases the metabolic rate and sensitivity

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BOX 22.17 Neurologic Abnormalities in Hypokalemic, Hyperthyroid Cats

- Generalized weakness
- Neck ventroflexion
- Fatigue
- Muscle tremors
- Ataxia
- Incoordination
- Inability to jump
- Muscle atrophy
- Breathlessness (due to weakness of intercostal muscles)
- Collapse

to catecholamines and causes significant cardiovascular and metabolic abnormalities. Up to 80% of affected cats may have thyrotoxic heart disease; approximately 20% of these may have congestive heart failure. Hypertension is sometimes identified but does not appear to be as common as cardiac disease. Multifactorial mechanisms may cause neuromuscular and CNS dysfunction in some hyperthyroid cats. Neurologic signs associated with feline hyperthyroidism are listed in Box 22.17. T_4 and T_3 bind to receptor sites in the sarcoplasm that increase skeletal muscle heat production and mitochondrial oxygen consumption. The hyperthyroid state may reduce muscle contraction by uncoupling oxidative phosphorylation. Thyroid hormones may lower the threshold for cerebral tissue activation, may alter the activity of some brain enzymes, and may interact with catecholamines to alter the mental state of some affected animals. Abnormalities of the CNS may include hyperexcitability, irritability, aggression, seizures, confusion, and stupor.

DIAGNOSIS

Clinical Presentation

Signalment

Hyperthyroidism generally affects cats older than 8 years of age (mean age, 13 years); however, it may rarely occur in young cats. No gender predisposition has been noted. Siamese and Himalayan cats may be at decreased risk of developing hyperthyroidism. Cats fed primarily canned food and those using cat litter may be at increased risk. Cats that prefer to eat canned cat food of fish or liver and giblet flavor may have an increased risk of developing this condition.

History

Most affected cats are presented for treatment because of weight loss despite a normal or voracious appetite, restlessness, and/or hyperactivity. Occasionally, a small mass is noted in the ventral cervical region. Vomiting, diarrhea, polyuria, polydipsia, aggression, and/or a rough hair coat may occur. Frequency of defecation is sometimes increased. Body temperature may be slightly elevated. Approximately 10% of hyperthyroid cats are depressed, lethargic, inappetent, and/or weak (i.e., "apathetic" hyperthyroidism).

Physical Examination Findings

A palpable cervical mass is present in most affected cats. The weight of the enlarged gland often causes it to gravitate ventrally because the thyroid is loosely attached to tracheal fascia. Occasionally the gland may descend into the thoracic inlet, where it can no longer be palpated. Additional physical examination findings may include emaciation, a thin and/or roughened hair coat, and cardiac abnormalities (e.g., tachycardia, gallop rhythms, murmurs, left anterior fascicular block, and/or atrial and ventricular tachyarrhythmias). Electrocardiographic abnormalities may include tachycardia, prolonged QRS duration, increased R-wave amplitudes in lead II, and ventricular preexcitation.

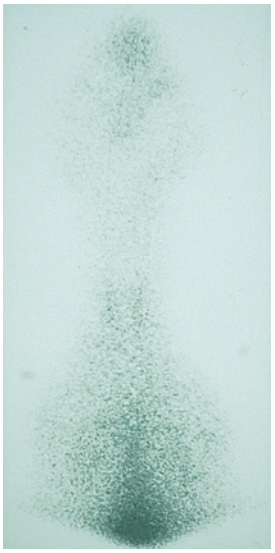


FIG. 22.19 Thyroid scintigraphy may be used to identify functional thyroid tissue. Compare this normal ventral view of the cervical region in this cat with that of the hyperthyroid cat in Fig. 22.20.

Diagnostic Imaging

An enlarged heart consistent with hypertrophic cardiomyopathy is often found on thoracic radiography and echocardiography. If the cat is in congestive heart failure, pleural effusion or pulmonary edema (or both) may occur. Ectopic thyroid tissue is rarely visible radiographically. Thyroid scintigraphy is the optimal diagnostic test because it can both definitively identify hyperthyroidism and locate functional ectopic thyroid tissue. With this procedure, technetium-99m is administered intravenously or intramuscularly. The radionuclide is trapped in functional thyroid tissue but is not organified. Delayed phase imaging of the body using a gamma camera provides functional and rudimentary anatomic locations of the hyperfunctioning thyroid tissue (Figs. 22.19 and 22.20). A ratio of uptake in thyroid tissue compared with that in salivary glands of greater than 2 is diagnostic of hyperthyroidism.

NOTE Approximately 20% of hyperthyroid cats have multiple areas of hyperfunctional thyroid tissue and/or intrathoracic hyperfunctional thyroid tissue where surgical thyroidectomy would not be curative. Warn owners of the possibility of ectopic thyroid tissue when performing thyroidectomy.

NOTE Before anesthetizing animals for a thyroidectomy, perform echocardiography and obtain thoracic radiographs to identify thyrotoxic heart disease.

Laboratory Findings

Most affected cats have high serum total T_4 (TT4) and free T_4 (fT4) by equilibrium dialysis concentrations. However, the

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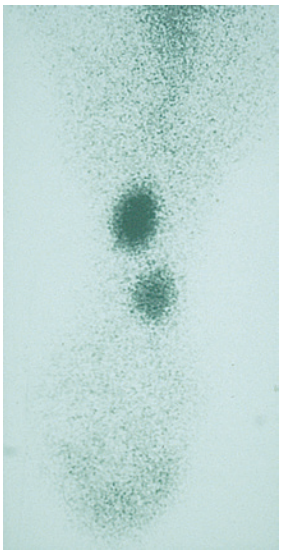


FIG. 22.20 Thyroid scintigraphy of a cat with bilateral thyroid adenomas.

BOX 22.18 T_3 Suppression Test[a]

Day 1
Obtain morning baseline serum T_4 and T_3 concentrations.

Days 1 and 2
Give sodium liothyronine (Cytobin), 25 µg/cat PO q8h for 2 days.

Morning of Day 3
Administer last dose of sodium liothyronine in morning, wait 2–4 h, then measure serum T_4 and T_3.

[a]Also see text.
PO, Orally; *T_3,* triiodothyronine; *T_4,* thyroxine.

diagnosis of hyperthyroidism cannot be excluded on the basis of a normal TT4 concentration, and cats without hyperthyroidism may have increased fT4 concentrations. In general, fT4 is measured only if a cat that is suspicious for hyperthyroidism has a normal TT4. Hyperthyroidism increases glomerular filtration rate (GFR), which may lessen azotemia and thereby mask clinical signs of chronic kidney disease (CKD). This is important because successful therapy of hyperthyroidism may result in clinical manifestation of CKD. At the same time, CKD may lower serum TT4 concentrations (i.e., euthyroid sick syndrome) into the reference range, making diagnosis more difficult. The combined measurement of fT4 and TT4 may be necessary when trying to determine if cats with moderate to severe CKD have hyperthyroidism.

Other abnormalities may include mild elevations in red blood cell numbers, increased PCV, neutrophilic leukocytosis, eosinopenia, lymphopenia, and elevated alanine aminotransferase and alkaline phosphatase. Serum creatinine and blood ionized calcium concentrations are often decreased, and serum phosphorous concentrations are often increased.

If baseline serum thyroid hormone concentrations are normal in a cat with appropriate clinical signs, or if a ventral cervical mass is palpable, serum TT4 and fT4 concentrations should be remeasured in 3 to 4 weeks, or nuclear scintigraphy should be performed. As an alternative, a T_3 suppression test (Box 22.18) or a TRH stimulation test may be performed. In normal cats, the serum T_4 concentration should decline by more than 50% after administration of sodium liothyronine (i.e., <1.5 mg/dL), whereas in hyperthyroid cats, a minimal decrease in the serum T_4 concentration is seen. The T_3 concentration should increase in both hyperthyroid and euthyroid cats if the medication was given appropriately. For the TRH test, serum T_3 and T_4 concentrations are measured before and 4 hours after IV administration of TRH (0.1 mg/kg). Hyperthyroid cats usually have a relative increase in T_4 of less than 50%; normal cats have a relative increase of greater than 50%. As an alternative, the response to oral antithyroid drugs may help confirm the diagnosis.

NOTE If total T_4 fails to diagnose hyperthyroidism, measurement of free T_4 should be requested.

Cats with hyperthyroidism usually have an increased GFR compared with normal cats; resolution of hyperthyroidism may reduce the GFR, resulting in signs of renal failure. Significant changes in kidney function typically occur within 4 weeks posttreatment, but are normal afterwards. Measurement of GFR and/or urine specific gravity and serum TT4 may help identify those animals that will develop evidence of renal azotemia after treatment. If in doubt as to the safety of thyroidectomy or ^{131}I therapy, one may first administer a course of methimazole and monitor serum creatinine and blood urea nitrogen concentrations to see how the patient will tolerate euthyroidism. For cats that develop overt renal failure after establishment of euthyroidism, withdrawal of methimazole should result in improved renal function. Hypokalemia and concurrent muscle weakness may occur in cats with hyperthyroidism.

DIFFERENTIAL DIAGNOSIS

Cats presented for treatment with weight loss or vomiting caused by hyperthyroidism must be differentiated from those with intestinal lymphoma or inflammatory bowel disease. Those with neurologic signs must be differentiated from cats with primary CNS abnormalities. Cardiac dysfunction that occurs secondary to hyperthyroidism should be differentiated from that resulting from other acquired or congenital causes.

MEDICAL MANAGEMENT

Treatment of feline hyperthyroidism may include long-term administration of antithyroid drugs (see Preoperative Management later) or ^{131}I, or surgical removal of the affected glands. The choice of treatment for the individual cat depends on the age and condition of the animal (i.e., presence of cardiovascular or renal disease) and the therapies available to the practitioner.

Propylthiouracil is an effective oral antithyroid drug in cats; however, it is not recommended because of severe side effects (i.e., autoimmune hemolytic anemia and immune-mediated thrombocytopenia). Long-term administration of methimazole or carbimazole (not available in the United States) can cause remission; however, clinical signs return once the drug is discontinued. Methimazole inhibits several steps in thyroid hormone synthesis and is effective in restoring a euthyroid status to most cats; however, up to 20% of cats experience gastrointestinal upset

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BOX 22.19 Possible Side Effects of Methimazole Therapy[a]

- Anorexia
- Vomiting
- Pruritus
- Lethargy

Polyarthritis

- Development of serum antinuclear antibodies
- Hepatopathy
- Thrombocytopenia with or without bleeding

Anemia

- Agranulocytosis
- Leukopenia
- Positive Coombs' test result

[a]Fewer adverse effects are seen when using transdermal gel as opposed to oral administration. Also see text.

during treatment (Box 22.19). In rare cases, drug-induced hepatopathy, thrombocytopenia, and agranulocytosis occur with long-term therapy. Although in general the drug is well tolerated and many side effects resolve with continued therapy, administration of an oral compound may be difficult in fractious cats and in those with impaired or elderly owners. A pluronic organogel formulation for transdermal application has recently been offered. The incidence of adverse gastrointestinal signs is less with the transdermal application; however, the efficacy of this route of administration does not appear to be as high as that of the oral route. Timing of blood sampling after oral methimazole administration does not appear to be a significant factor when response to methimazole treatment is evaluated. If thyroid carcinoma is suspected, medical therapy with antithyroid drugs may palliate clinical signs while allowing tumor growth. The safety and efficacy of a novel controlled-release formulation of carbimazole (a pro-drug to methimazole) has been evaluated. Treatment was started at 15 mg once daily; response was assessed after 10 days and 3, 5, 8, 26, and 53 weeks thereafter; and the dosage was adjusted. The median dose used in these cats was 10 and 15 mg once daily after 3 and 53 weeks, respectively.[22]

^{131}I is a safe and effective method of treating hyperthyroidism; however, facilities are required to safely handle the isotope. The cat must be confined for days to weeks (depending on specific state laws), during which time it is a human health hazard. It is important to detect other diseases before treating with ^{131}I, so that minimal contact with the cat is required during treatment. Radioactive iodine is trapped in the thyroid gland and causes tissue destruction. However, normal thyroid tissue is spared because it is suppressed and thus does not uptake the radioactive iodine. In the past, recent administration of antithyroid medications has been proposed to decrease the efficacy of radioactive iodine treatment because of decreased uptake. In normal cats methimazole may cause an increase in uptake as the result of upregulation of TSH. A definitive conclusion will require evaluation in hyperthyroid cats. If carcinoma is present, larger doses of ^{131}I may be necessary, requiring longer isolation periods.

NOTE Percutaneous ethanol ablation of bilateral thyroid nodules is not recommended as a treatment for hyperthyroidism in cats.

BOX 22.20 Preoperative Drug Therapy for Cats With Hyperthyroidism

Methimazole (Tapazole)

1.25–2.5 mg/cat PO q12h for 7–14 days. If needed, increase dose by 2.5 mg/d until control is achieved up to 5–10 mg/cat PO q12h.[a]
Transdermal application (to the hairless skin of the pinna) is also available.[b]

Carbimazole

5 mg/cat PO q8–12h, then adjust to 5 mg/cat q12h as needed

Carbimazole (Controlled-Release Tablet Formulation)[c]

15 mg/cat PO q24h, then adjust as necessary (10–25 mg/cat)

Propranolol (Inderal)[d]

2.5–5 mg/cat (0.4–1.2 mg/kg) PO q8–12h

[a]Use lower dose in small or debilitated animals. If long-term administration is considered, this dose should be adjusted to maintain the T_4 concentration within the normal range.
[b]The overall efficacy of transdermal methimazole may not be as high as the orally administered drug; however, it is associated with fewer gastrointestinal adverse effects.
[c]Vidalta, Merck Animal Health, Intervet International, The Netherlands. Carbimazole is not available in the United States but is available through compounding pharmacies (e.g., Diamondback Drugs; www.diamondbackdrugs.com, 1-866-578–4420).
[d]Propranolol should be used with care in hyperthyroid cats. Administration of propranolol to hypokalemic cats may cause sudden death.
PO, Orally; *T_4,* thyroxine.

SURGICAL TREATMENT

Surgical treatment of hyperthyroidism involves thyroidectomy. Complications of thyroid surgery include intraoperative hemorrhage and clinical signs associated with damage to the recurrent laryngeal nerves, the parathyroid blood supply, or parathyroidectomy.

The major complication of bilateral thyroidectomy is hypoparathyroidism, which occurs secondary to removal or damage of the parathyroid glands. The procedure must be performed carefully to prevent this complication. If the parathyroid gland is inadvertently removed, it should be transferred to a nearby muscle belly (e.g., sternohyoideus muscle) so that the gland may revascularize and become functional again (parathyroid gland autotransplantation). To prevent the complication of hypocalcemia, some surgeons recommend a two-stage procedure in which one thyroid lobe is removed and its associated parathyroid is reimplanted into adjacent musculature during the first surgery. Two to three weeks later, the other thyroid lobe is removed and its associated parathyroid is similarly reimplanted. Although this may reduce the risk of postoperative hypocalcemia, the added risk of a second anesthetic event is introduced.

Preoperative Management

Metabolic and cardiovascular abnormalities associated with hyperthyroidism make anesthesia risky; therefore cats should be made euthyroid preoperatively by administering methimazole (Tapazole) (Box 22.20). Generally, administration for 1 to 3 weeks before surgery is sufficient; however, measurement of the TT4 concentration should be repeated to ensure that it is within the normal range before surgery is performed (see comment on side effects earlier). If preoperative therapy with methimazole is not tolerated, propranolol, a β1- and β2-blocker, may be given for 1 to 2 weeks before surgery (see Box 22.20) to reduce the HR. Propranolol may be discontinued 24 to 48 hours before surgery

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because of its β-blocking effects, which may interfere with treatment of hypotension; however, this increases the risk of tachycardia and hypertension, especially at induction. Another preanesthetic choice would be to use metoprolol, a specific β1-blocker, and continue antihypertensive therapy until the morning of surgery. Although atenolol (β-blocker) effectively reduces HR in most cats with hyperthyroidism, elevated systemic blood pressure is poorly controlled, and the addition of another vasodilator such as amlodipine or an angiotensin-converting enzyme inhibitor is often necessary to treat associated hypertension. Hypotension secondary to vasodilation induced by the anesthetic gases can be treated with small boluses of phenylephrine.

Because cardiac abnormalities are common, an electrocardiogram, blood pressure readings, and chest radiographs should be obtained before surgery. An echogram needs to be obtained if a cardiac murmur has been auscultated. Many hyperthyroid cats have concurrent renal disease, hypokalemia, and/or azotemia. If clinical signs of hyperthyroidism have not been resolved with the preoperative methimazole, hypovolemia may persist secondary to increased catecholamine production. These cats should be given fluids before, during, and after surgery, and care should be taken to ensure that uremia does not occur during surgery (see the discussion of anesthetic management of animals with renal disease on p. 650) or after surgery, when cardiac output drops because the cat becomes euthyroid. Fluid therapy should be adjusted if the cat is in congestive heart failure.

Anesthesia

Cats with cardiomyopathy may be premedicated with butorphanol or buprenorphine (Table 22.5). As soon as an IV catheter is placed, diazepam or midazolam can be given to minimize the patient's stress. Because mask induction may stress the animal and cause increased catecholamine release, its use is generally not recommended; the animal can be reoxygenated by holding the end of the circuit near the animal's face. These patients have a high oxygen requirement, and adequate oxygenation should be maintained throughout the perioperative period. Anesthesia can be induced with propofol or etomidate. Ketamine and thiopental can cause tachycardia and should be avoided during induction. A couple of drops of lidocaine given via a tuberculin syringe may aid intubation and reduce the elevations in HR and blood pressure associated with intubation. Maintenance on isoflurane or sevoflurane with oxygen should be used; halothane should be avoided. Tachyarrhythmias usually can be controlled with esmolol intravenously. If elevated HR and/or blood pressure continues, low doses of metoprolol can be titrated to effect. If the arrhythmias are ventricular in nature, lidocaine can be given as an IV bolus (see Box 22.8).

It bears repeating that these patients often have high catecholamine production with underlying hypovolemia and renal insufficiency, masked anemia, abnormal cardiac output, tachyarrhythmias, and increased metabolic rates, drug metabolism, and oxygen consumption. Therefore they need to be sedated with benzodiazepines and must be adequately hydrated before surgery. Elevated blood pressure and HR need to be controlled with β-blockers. If hypotension arises during surgery and blood losses are minimal, small boluses of phenylephrine are usually sufficient to maintain mean arterial pressures between 60 and 80 mm Hg. Intraoperative fluid rates may need to be 5 mL/kg per hour in patients that have decreased cardiac function, and 10 mL/kg per hour in patients with normal cardiac function. *Thyroid storm* is a condition caused by excessive thyroid hormone production due to excessive catecholamine release. Clinical signs of thyroid storm may include marked elevations in HR, blood pressure, and temperature, as well as cardiac arrhythmia and shock.

Surgical Anatomy

See p. 616 for a description of the surgical anatomy of the thyroid gland.

Positioning

The animal is placed in dorsal recumbency with the neck slightly hyperextended and the forelimbs pulled caudally. The entire ventral neck and the cranioventral thorax should be prepared for aseptic surgery.

SURGICAL TECHNIQUE

Intracapsular Thyroidectomy

Make a skin incision from the larynx to a point cranial to the manubrium. Bluntly separate the sternohyoid and sternothyroid muscles. Use a self-retaining (e.g., Gelpi) retractor to maintain exposure. Identify the enlarged thyroid gland and the external parathyroid gland (Fig. 22.21). Make an incision on the caudoventral surface of the gland in an avascular area (Fig. 22.22), and extend it cranially with small scissors (e.g., iris scissors). Using a combination of blunt and sharp dissection, carefully remove the thyroid tissue from the capsule. Perform the dissection carefully to prevent damage to the parathyroid gland or its blood supply. Use bipolar cautery to achieve hemostasis, but avoid damaging the gland's blood supply. After the thyroid parenchyma has been removed, excise most of the thyroid capsule; however, do not excise the capsule that is intimately associated with the external parathyroid gland. If the parathyroid gland is inadvertently excised, or if its blood supply is damaged, transplant the gland to a nearby muscle belly (see p. 613). Close subcutaneous tissue in a simple continuous suture pattern (e.g., 3-0 or 4-0 absorbable). Close the skin in a simple continuous or simple interrupted suture pattern (e.g., 3-0 nonabsorbable).

NOTE Sterile cotton-tipped swabs are useful to help separate the gland from the capsule.

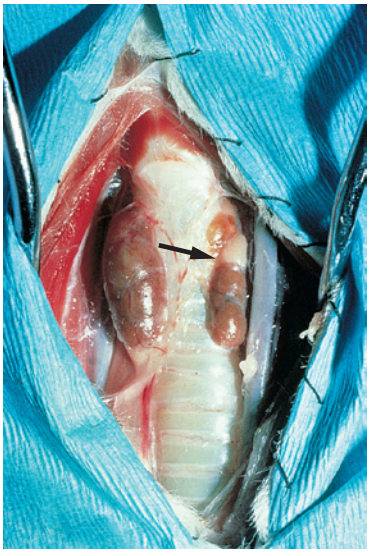


FIG. 22.21 Thyroid enlargement in a cat. Note the parathyroid gland at the cranial pole of the left thyroid gland *(arrow)*.

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TABLE 22.5 Anesthetic Considerations in the Feline Hyperthyroid Patient

Preoperative Considerations

Associated conditions	• Anemia (may be occult) • Hypovolemia • Hypertension • Tachydysrhythmias • Ventricular ectopy • Cardiac dysfunction • Renal insufficiency (may be occult) • Cardiomyopathy
Bloodwork	• HCT • Electrolytes • BUN • Cr • TP • Urinalysis
Physical examination	May be hypovolemic, tachycardic, and hypertensive if untreated with methimazole
Other diagnostics	• Blood pressure is essential • ECG • Radiographs (thoracic) • Echocardiography
Premedications	Give: • Diazepam (0.2 mg/kg IV), *or* • Midazolam (0.2 mg/kg IV, IM), *plus* • Buprenorphine[a] (0.005–0.02 mg/kg IV, IM) *or* • Butorphanol (0.2–0.4 mg/kg IV, IM) *or* • Morphine[b] (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM) • Avoid ketamine, xylazine, medetomidine, dexmedetomidine, atropine, glycopyrrolate, and acepromazine

Intraoperative Considerations

Induction	• Titrate propofol (2–6 mg/kg IV), *or* • Give alfaxalone (2–3 mg/kg IV) • If CHF, titrate etomidate (0.5–1.5 mg/kg IV)

Maintenance	• Isoflurane or sevoflurane *plus*
	  • Fentanyl (1–4 µg/kg IV PRN) for short-term pain relief, *plus*
	  • Buprenorphine[a] (0.005–0.02 mg/kg IV PRN), *or*
	  • Hydromorphone[c] (0.05–0.1 mg/kg IV PRN), *or*
	  • Morphine[b] (0.05–0.2 mg/kg IV PRN) if minimal hypotension
	• For hypertension (to keep MAP 70–90 mm Hg)
	  • Esmolol (0.05–0.25 mg/kg IV) boluses every 2–5 min to effect and/or CRI (50–200 µg/kg/min IV) to maintain normal heart rate, *plus*
	  • Nitroprusside (0.5–5 µg/kg/min IV) *or*
	  • Nitroglycerin (1–5 µg/kg/min IV)
	• For hypotension (to keep MAP 60–80 mm Hg), give phenylephrine or dopamine as needed

Fluid needs	• 5–10 mL/kg/h if minimal blood loss
Monitoring	• BP: essential • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature • +/– Arterial line

Postoperative Considerations

Analgesia	• Buprenorphine[a] (0.005–0.02 mg/kg IV, IM q4–8h or 0.01–0.02 mg/kg OTM q6–12h)
Monitoring	• SpO_2 • Blood pressure • ECG • HR • Respiratory rate • Temperature • U/O
Bloodwork	• Serial calcium for 48–72 h • Serial BUN/Cr for 2–4 wk
Estimated pain score	Mild; however, these patients need to have minimal stress and to be kept pain free for approximately 24 h to assist in avoiding thyroid storm (see text)

[a]Buprenorphine is a better analgesic than morphine in cats.
[b]Give slowly to prevent histamine release.
[c]Monitor for hyperthermia.
BP, Blood pressure; *BUN,* blood urea nitrogen; *CHF,* congestive heart failure; *Cr,* creatinine; *CRI,* constant-rate infusion; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *MAP,* mean arterial pressure; *OTM,* oral transmucosal; *PRN,* as needed; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein; *U/O,* urine output.

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Modified Extracapsular Approach for Thyroidectomy

Position the animal as previously described. Locate the thyroid gland as described before, and ligate or cauterize the caudal thyroid vein. Using fine-tipped, bipolar cautery forceps (Fig. 22.23A), cauterize the thyroid capsule approximately 2 mm from the external parathyroid gland. With small, fine scissors, cut the gland at the cauterized area, and use sharp and blunt dissection to remove the gland from the parathyroid gland (see Fig. 22.23B). Carefully dissect all thyroid gland from the surrounding tissue and parathyroid gland (see Fig. 22.23C). Do not damage the cranial thyroid artery or its branches to the external parathyroid gland. If the parathyroid gland is inadvertently excised, or if its blood supply is damaged, transplant the gland to a nearby muscle belly (see p. 613). Close as previously described.

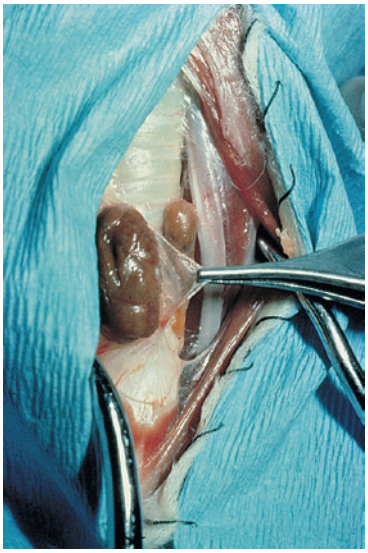


FIG. 22.22 For intracapsular thyroidectomy, make an incision on the caudoventral surface of the gland in an avascular area and extend it cranially with small scissors (e.g., iris scissors). Using a combination of blunt and sharp dissection, carefully remove the thyroid tissue from the capsule.

NOTE To prevent hypocalcemia, take special care to avoid damaging the cranial thyroid artery.

Modified Intracapsular Approach for Thyroidectomy

Position the animal as described previously. Locate the thyroid gland and remove the gland as described previously in the intracapsular thyroidectomy section. Using a No. 15 scalpel blade or fine scissors, create a peninsula of capsular tissue containing only the parathyroid gland and its blood vessel. Excise the remainder of the capsule. If the parathyroid gland is inadvertently excised, or if its blood supply is damaged, transplant the gland to a nearby muscle belly (see p. 613). Close as previously described.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Small, fine instruments, such as iris scissors and Bishop-Harmon thumb forceps, facilitate removal of the thyroid glands. Bipolar cautery forceps are advantageous for providing hemostasis because they allow finer control of coagulation than do unipolar forceps.

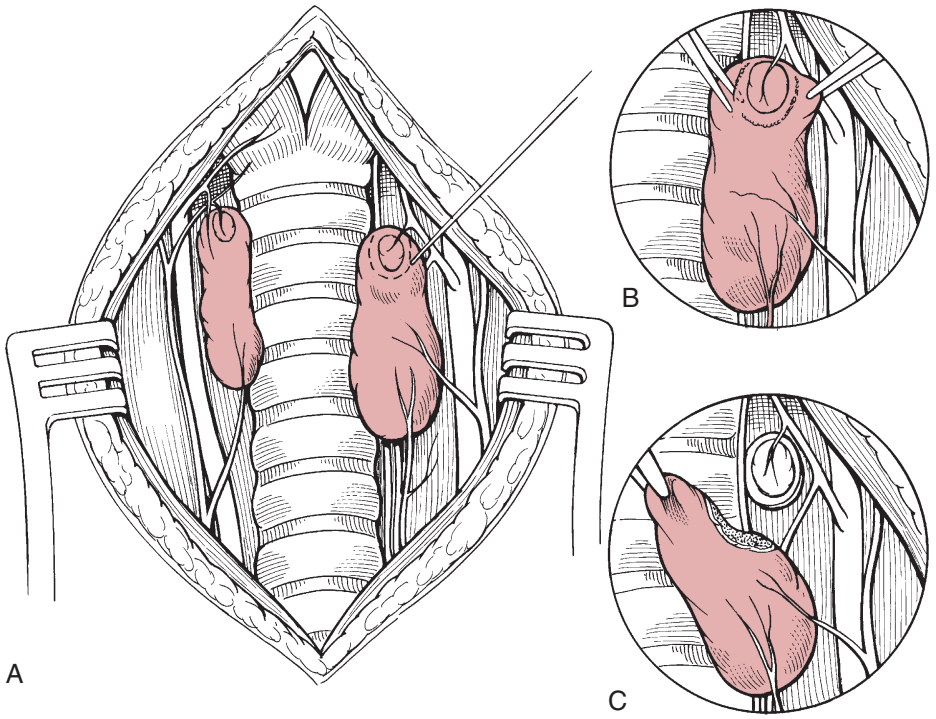


FIG. 22.23 Modified extracapsular thyroidectomy. (A) Using fine-tipped bipolar cautery forceps, cauterize the thyroid capsule approximately 2 mm from the external parathyroid gland. (B) With small, fine scissors, cut the gland at the cauterized area and remove from the parathyroid gland. (C) Carefully dissect all of the thyroid gland from the surrounding tissue and parathyroid gland.

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Sterile Q-tips are useful for dissecting the thyroid glands from the parathyroid glands.

POSTOPERATIVE CARE AND ASSESSMENT

Complications may include hypocalcemia, hypothyroidism, recurrence of hyperthyroidism, worsening of renal disease, Horner syndrome, and/or laryngeal paralysis. Hypocalcemia (serum calcium level <9 mg/dL in adult dogs and <8.5 mg/dL in adult cats) is the most important, acute, life-threatening complication of thyroidectomy. Hypocalcemia may occur owing to removal of or trauma to the parathyroid glands or their blood supply. Transient hypocalcemia may be caused by local edema of the parathyroid gland associated with trauma (including use of electrocautery) to the gland's vascular supply. Hypocalcemia appears to occur in 6% of cats undergoing bilateral thyroidectomy; surgeon experience may be an important factor in determining whether hypocalcemia occurs. Most animals do not develop clinical signs until the serum calcium level is below 7.5 mg/dL, depending on acid-base status. Animals should be closely observed for 2 to 4 days for signs of hypocalcemia (e.g., panting, nervousness, facial rubbing, muscle twitching, ataxia, seizures). In cats, early signs may include lethargy, anorexia, panting, and facial rubbing. Clinical signs are usually noted within 24 to 96 hours, although delayed signs have been reported up to 5 or 6 days later. Acute signs of hypocalcemia may be treated with IV 10% calcium gluconate; calcium chloride should never be administered. The 10% calcium gluconate should be given slowly intravenously (Box 22.21), and the cardiac rate and rhythm should be monitored during administration. Calcium administration should be discontinued if bradycardia develops. Calcium gluconate can also be added to the fluids, or the IV dose can be diluted in an equal volume of saline (or for greater safety, dilute 1:3 or 1:4) and given subcutaneously every 6 to 8 hours (see Box 22.21) until the animal is eating and can be given oral medications. Administration of undiluted calcium gluconate may be associated with abscess formation (Fig. 22.24). Subcutaneous or IV administration of calcium should be discontinued when the serum calcium level is above 8 mg/dL.

Maintenance therapy consists of oral calcium and vitamin D administration (see Box 22.21). The form of vitamin D most commonly used is dihydrotachysterol. It does not accumulate in fat and has a more rapid onset of action than vitamin D_3. Serum calcium levels should be monitored weekly and the dosage of calcium changed accordingly. Vitamin D supplementation can often be discontinued once the parathyroid gland revascularizes. Some animals must be maintained on vitamin D for months before the dose can be reduced; others require lifelong therapy.

Recurrence of hyperthyroidism is likely associated with the presence of ectopic hyperplastic thyroid tissue (EHTT). In a study of 2096 cats undergoing thyroid scintigraphy, ectopic thyroid tissue was found in 81 (3.9%).[23] This reinforces the importance of performing preoperative thyroid scintigraphy in affected cats.

NOTE Do not administer calcium chloride (especially intravenously); you can overdose the animal too easily.

PROGNOSIS

Hypocalcemia due to iatrogenic hypoparathyroidism may be permanent or temporary. Persistent hypocalcemia may occur if all four parathyroid glands are removed, or if their blood supply is irreversibly damaged. Temporary or transient hypocalcemia is usually caused by disruption of the parathyroid blood supply, which may be associated with edema and swelling of the gland or its blood supply. Hypocalcemia should not occur after unilateral thyroidectomy. Recurrent hyperthyroidism may result from hypertrophy of adenomatous tissue not removed during thyroidectomy or from adenomatous changes in EHTT (see earlier). Hyperthyroidism may occur within months or years in a small portion of cats undergoing bilateral thyroidectomy. If possible, a thyroid scan should be performed to localize hyperfunctioning tissue in these animals before surgery is repeated.

NOTE Hypocalcemia is extremely rare after unilateral thyroidectomy for feline hyperthyroidism.

BOX 22.21 Treatment of Hypocalcemia Following Thyroidectomy

Management of Acute Signs

Give 0.5–1.5 mL/kg of 10% calcium gluconate slowly IV (over 10–20 min) and monitor the heart; then add 10 mL of 10% calcium gluconate to 250 mL of lactated Ringer's solution, and drip at a maintenance rate or give IV dose diluted in saline[a] (1:3–1:4) SC (at multiple sites). Monitor serum calcium frequently (q8–12h if necessary).

Maintenance Therapy

1. Oral calcium supplementation (cats require 0.5–1.0 g of calcium/cat/day): different oral preparations contain different amounts of calcium. Calcium lactate is 13% calcium with 1 g = 6.5 mEq of calcium (325 mg tablet has 42 mg of calcium); calcium gluconate is 9% calcium with 1 g = 4.5 mEq of calcium (325 mg tablet has 30 mg calcium). Give calcium lactate 0.2–3.0 g/cat/day in divided doses PO and reassess.
2. Give vitamin D, either[b]:
   - Dihydrotachysterol at 0.02–0.03 mg/kg/d PO for 3–5 d; then 0.02 mg/kg/d for 4 d; then 0.005 mg/kg/d for 1–4 mo; or
   - Calcitriol at 0.02–0.03 µg/kg/d for 2–4 d, then maintenance at 0.005–0.015 µg/kg/d.

[a]It is important to dilute calcium gluconate when it is administered SC.
[b]It is important to give EITHER dihydrotachysterol OR calcitriol, but not both.
IV, Intravenous; *PO,* orally; *SC,* subcutaneous.



FIG. 22.24 A dog that received undiluted calcium gluconate subcutaneously and developed skin necrosis and tissue sloughing.

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In a study of 101 hyperthyroid cats undergoing thyroidectomy, two cats died within 72 hours of surgery, one from laryngospasm and the other from unknown causes.[23] In cats treated with ^{131}I, methimazole, or both, cats with preexisting renal disease were shown to have significantly shorter survival times than cats without preexisting renal disease.[24] When cats with preexisting renal disease were excluded, median survival time for cats treated with methimazole alone was 2 years, and median survival time for cats treated with ^{131}I alone was 4 years. Treatment with both resulted in a median survival of 5.3 years. Age, preexisting renal disease, and treatment type affected survival times.

In another study, it was found that iatrogenic hypothyroidism appeared to contribute to the development of azotemia after treatment of hyperthyroidism.[25] In the aforementioned study, cats with azotemia had reduced survival time compared with cats with normal renal function. The authors suggested that restoration of hypothyroid cats to euthyroidism may be indicated to normalize renal function and improve survival.

HYPERPARATHYROIDISM

DEFINITION

Primary hyperparathyroidism is a disorder resulting from excessive secretion of PTH by the parathyroid gland or glands.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Primary hyperparathyroidism is uncommon in dogs and rare in cats. It is usually caused by parathyroid adenomas, although parathyroid carcinomas and parathyroid hyperplasia have also been reported. Parathyroid adenomas are typically small, well-encapsulated tumors that appear brown or red and are located near the thyroid glands; however, ectopic adenomas may be located near the thoracic inlet or in the cranial mediastinum. Clinical signs are caused by PTH increasing calcium absorption and phosphorus excretion in the kidneys and enhancing bone resorption. The net result is an increase in serum calcium levels and a decrease in serum phosphorus levels. Clinical abnormalities caused by hypercalcemia may include dystrophic calcification, impaired renal tubular concentrating ability, renal failure, and calcium oxalate nephrolithiasis and urolithiasis.

DIAGNOSIS

Clinical Presentation

Signalment

Parathyroid tumors usually occur in older dogs. No gender predisposition has been noted. Keeshonden (and possibly German shepherds and Norwegian elkhounds) may be predisposed to the disorder. Primary gland hyperplasia has been reported in young dogs.

History

Dogs may be asymptomatic or may be presented because of polyuria-polydipsia or nonspecific signs (e.g., vomiting, weakness, constipation, lethargy, inappetence). Clinical signs may be insidious at onset. The most common clinical signs in cats with primary hyperparathyroidism are anorexia, lethargy, vomiting, weakness, and weight loss; polyuria and polydipsia are less common in cats than in dogs. Occasionally, bone and joint pain and pathologic fractures may occur secondary to skeletal demineralization. Cystic calculi may develop secondary to hypercalcemia.

NOTE Consider primary hyperparathyroidism as a differential diagnosis in animals with hypercalcemia, dystrophic calcification, calcium oxalate urolithiasis, and/or nephrolithiasis.

Physical Examination Findings

The physical examination findings usually are nonspecific. The enlarged parathyroid gland can seldom be palpated in dogs; however, a cervical mass may be palpated in some cats.

Diagnostic Imaging

Cervical radiographs rarely identify the neoplasm. Notable demineralization of the skeleton, nephrolithiasis, and/or nephrocalcinosis may be seen radiographically. Ultrasonography is an excellent modality to evaluate the parathyroid glands. Ultrasonographic detection of a parathyroid gland exceeding 4 mm in diameter is highly suspicious for parathyroid adenoma or carcinoma. Parathyroid scintigraphy does not appear to be a sensitive or specific indicator for definitive identification of abnormal parathyroid glands in dogs with hypercalcemia.

Laboratory Findings

Serum biochemical abnormalities in dogs with primary hyperparathyroidism include hypercalcemia and hypophosphatemia. Hypercalcemia is the most consistent finding in affected cats. Measurement of PTH in animals with normal renal function is a sensitive test. High-normal or increased serum concentrations of PTH in hypercalcemic animals with normal renal function are strongly suggestive of hyperparathyroidism. Other causes of hypercalcemia (see later discussion) are usually associated with low or low-normal levels of PTH. Renal dysfunction, which may occur secondary to hypercalcemia or may be a primary disorder, may also elevate serum concentrations of PTH. If renal function is abnormal, serum PTH concentrations should be evaluated in conjunction with the serum ionized calcium concentration. Serum ionized calcium levels are increased with hyperparathyroidism, but are usually low to low-normal with renal failure (Table 22.6). Definitive diagnosis of primary hyperparathyroidism requires surgical exploration of the parathyroid glands. Dogs can have uniglandular or multiglandular disease. Systemic and local PTH concentrations decrease by more than 50% in all dogs after excision of the parathyroid gland(s), and serum calcium concentration eventually returns to normal, although rebound hypocalcemia is common.

Careful physical examination (including rectal examination), thoracic and abdominal radiography, abdominal ultrasonography,

TABLE 22.6 Serum Parathyroid and Calcium Levels With Primary Hyperparathyroidism and Renal Disease

	PTH	Ca^{2+a}
HPTH	↑	↑
Renal failure	↑	↓

[a]Serum ionized calcium.
Ca^{2+}, Calcium; *HPTH*, hyperparathyroidism; *PTH*, parathyroid.

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skeletal radiography, routine bloodwork, and/or lymph node aspirations should be performed in hypercalcemic animals to identify neoplastic causes of hypercalcemia of malignancy (e.g., lymphosarcoma, apocrine gland adenocarcinoma) before a diagnosis of hyperparathyroidism is pursued. Other causes of hypercalcemia include granulomatous disease, chronic renal failure, hypoadrenocorticism, and hypervitaminosis D. *Thyroglossal cysts* (formed when the embryonic thyroglossal duct fills with fluid) may be confused with parathyroid masses on palpation.

NOTE Paraneoplastic hypercalcemia of malignancy is a more common cause of hypercalcemia than primary hyperparathyroidism. Hypercalcemia of malignancy can rapidly cause renal failure if diagnosis and therapy are delayed, but hypercalcemia caused by hyperparathyroidism is often not as high as that seen in primary hyperparathyroidism and is less likely to cause renal failure.

MEDICAL MANAGEMENT

Hypercalcemia may be treated by diuresis (see later discussion on Preoperative Management). Surgical removal of the neoplastic parathyroid tissue is the definitive treatment for primary hyperparathyroidism. Glucocorticoid therapy is usually transiently effective in lowering the serum calcium concentration in animals with lymphosarcoma and may also lower the calcium concentration in animals with other disorders. In confusing cases, administration of L-asparaginase may be used as a therapeutic trial to rule out occult lymphosarcoma as a cause of hypercalcemia.

SURGICAL TREATMENT

Parathyroidectomy is the treatment of choice for hyperparathyroidism caused by parathyroid neoplasia and primary hyperplasia. If the parathyroid glands are uniformly enlarged, secondary hyperparathyroidism should be suspected and other diagnostic tests performed to identify the cause (e.g., renal secondary hyperparathyroidism); however, enlargement of all four glands may occur with primary hyperplasia. If one or several glands are slightly enlarged, parathyroid adenomas or primary hyperplasia should be suspected. Most dogs with primary hyperparathyroidism have a single parathyroid adenoma. If the parathyroid glands appear normal, ectopic parathyroid tissue may be located adjacent to the thyroid or as far caudal as the base of the heart. Alternatives to surgical parathyroidectomy include percutaneous ultrasound-guided ethanol ablation and percutaneous ultrasound-guided heat ablation.

NOTE If you cannot find any other cause of hypercalcemia in a patient with increased PTH concentrations, and if the parathyroid glands appear normal, look for ectopic parathyroid tissue.

Preoperative Management

Before induction of anesthesia, diuresis should be instituted with 0.9% saline solution to help lower serum calcium concentrations (Box 22.22). In severe hypercalcemia, salmon calcitonin can be used to lower the serum calcium. Fluids should be used with caution in animals with severe renal dysfunction. Once the animal has been appropriately hydrated, furosemide administration may promote further calciuresis. Electrolytes should be monitored to prevent iatrogenic hypokalemia. Volume status and electrolytes should be normal before anesthesia.

BOX 22.22 Diuresis of Hypercalcemic Dogs

0.9% Physiologic Saline Solution
90 mL/kg/d IV

Furosemide (Lasix)
2–4 mg/kg IV q8–12h, or can give as CRI (load with 0.66 mg/kg bolus, then give 0.66 mg/kg/h for 4–5 h; alternatively, can estimate the IV or PO dose to be given over the course of the next 24 h, then give that amount as CRI over 24 h. Be sure patient is volume replenished before administering), or give total daily dose as CRI.

CRI, Constant-rate infusion; *IV,* intravenous; *PO,* orally.

Anesthesia

Theoretically, notable hypercalcemia may cause bradycardia, peripheral vasoconstriction, and hypertension. Hypotension may occur during anesthesia associated with relaxation of peripheral vascular tone. Hypercalcemia also may predispose to cardiac arrhythmias. Anesthetic agents that potentiate arrhythmias (e.g., thiobarbiturates, halothane) should be avoided. Respiratory and metabolic acidosis should be avoided because elevations in pH will increase the free fraction of calcium, worsening hypertension and bradycardia.

Surgical Anatomy

A discussion of the anatomy of the parathyroid glands is provided on p. 616.

Positioning

The animal is placed in dorsal recumbency with the neck slightly hyperextended and forelimbs pulled caudally. The entire ventral neck and cranioventral thorax should be prepared for aseptic surgery.

SURGICAL TECHNIQUE

All four parathyroid glands should be carefully inspected. If the external parathyroid gland is involved, the gland can be removed without removal of the thyroid gland; however, removal of the internal parathyroid gland requires that thyroidectomy be performed (see p. 617). The external parathyroid gland should be spared when the internal parathyroid gland is neoplastic, if possible. Visualization of the abnormal parathyroid gland may be facilitated by infusion of IV methylene blue in saline solution (see Box 22.13). Abnormal parathyroid tissue may stain dark blue with this procedure. A potential side effect of methylene blue administration is hemolytic anemia caused by Heinz body formation. Severe and occasionally fatal Heinz body anemia has been reported after the use of methylene blue. If carcinoma is suspected on the basis of apparent invasiveness of the tumor, complete thyroidectomy and removal of draining lymph nodes are indicated.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Small, fine instruments, such as iris scissors and Bishop-Harmon thumb forceps, facilitate removal of the parathyroid glands.

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Bipolar cautery forceps are advantageous for providing hemostasis because they allow finer control of coagulation than do unipolar forceps. Sterile Q-tips are useful for dissecting the parathyroid glands from the thyroid glands.

POSTOPERATIVE CARE AND ASSESSMENT

Hypocalcemia is the most common postoperative complication in dogs; it may be less common in cats. Hypocalcemia may occur after removal of a single parathyroid adenoma because negative feedback from high circulating levels of PTH suppresses function in the other normal glands. PTH has a functional half-life of 20 minutes; therefore PTH levels fall rapidly once neoplastic tissue has been removed. Hypocalcemia may be most pronounced in animals with higher preoperative serum calcium levels and in those with notable skeletal demineralization. However, prediction of the severity of postoperative hypocalcemia using preoperative factors has proven challenging.[26,27] Treatment of hypocalcemia is presented in Box 22.21; chronic treatment should not be necessary in these patients. Renal function should be monitored postoperatively in patients with hypercalcemia.

PROGNOSIS

The prognosis for long-term survival after parathyroidectomy for hyperparathyroidism secondary to adenomas or hyperplasia is excellent if severe renal damage has not occurred. Following surgical removal of parathyroid carcinomas, 1-, 2-, and 3-year survival rates were estimated to be 72%, 37%, and 30%, respectively.[28] In recent studies, percutaneous ultrasound-guided radiofrequency heat ablation was successful in 69% of dogs, whereas percutaneous ultrasound-guided ethanol ablation was successful in 85% of dogs.[29,30]

THYROID CARCINOMAS IN DOGS

DEFINITIONS

Thyroid neoplasms may be carcinomas (malignant) or adenomas (benign). **Carcinomas** may arise from follicular cells and may be classified as follicular, compact, papillary, or mixed, or they may arise from parafollicular or C cells (medullary thyroid carcinomas).

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Thyroid neoplasms make up slightly more than 1% of all canine tumors. Canine thyroid carcinomas are more common than adenomas, whereas functional adenomas prevail in cats (see the discussion of feline hyperthyroidism on p. 617). Carcinomas and adenocarcinomas represent nearly 90% of thyroid cancers; adenomas represent approximately 9% in dogs. Carcinomas are further divided into differentiated follicular cell carcinomas and medullary thyroid carcinomas, although outcomes following surgical removal are comparable between the two.[31] Carcinomas are generally rapidly growing, highly invasive tumors that frequently metastasize to the draining lymph nodes and lungs. Reportedly, large tumors (i.e., those larger than 100 cm^3) are typically associated with pulmonary metastasis. Although histologic classification of thyroid tumors based on the predominant microscopic pattern has been done (e.g., compact cellular or solid, follicular, mixed solid follicular, anaplastic), the histologic pattern has been thought to correlate poorly with prognosis. However, medullary thyroid carcinomas are more apt to be well circumscribed and resectable and to have gross and histologic characteristics of a less malignant nature compared with other thyroid carcinomas. Ectopic thyroid tumors have been reported at the heart base, caudal mediastinum, and tongue.

NOTE Warn owners that surgical excision of canine thyroid tumors is usually difficult because of the invasiveness of the tumors and the tendency toward substantial hemorrhage.

Tumors arising in cystic remnants of the thyroglossal duct are rarely reported in dogs. They are usually well-circumscribed, fluctuant, movable enlargements in the ventral midline cervical region. Histologically, they are usually well-differentiated papillary carcinomas. Because more than a third of dogs with thyroid carcinomas have multiple malignancies, thorough staging should be performed before surgery.[32]

DIAGNOSIS

Clinical Presentation

Signalment

Dogs between 10 and 15 years of age have a significantly increased chance of developing thyroid cancer. Breeds most commonly affected are golden retrievers, beagles, and Siberian huskies. A gender predisposition is not apparent.

History

Affected animals are often presented for evaluation of a palpable cervical enlargement, dysphagia, dyspnea, coughing, voice change, and/or exercise intolerance. Respiratory abnormalities may be the result of tracheal compression or pulmonary metastasis, and regurgitation may be caused by compression and/or invasion of the esophagus. In rare cases, hyperthyroidism (i.e., polydipsia, polyuria, weakness, restlessness, and a propensity to seek cool places) is caused by canine thyroid carcinomas.

Physical Examination Findings

A ventral cervical mass is often palpable. Carcinomas usually appear firm and poorly encapsulated; adenomas are typically small and freely movable. Abnormal lung sounds may occur secondary to pulmonary metastasis. Bilateral ptosis and prolapse of the nictitating membrane may be associated with paralysis of the extraocular and intraocular muscles, secondary to thyroid adenocarcinoma invasion of the cavernous sinuses in dogs.

Diagnostic Imaging

Cervical radiography or ultrasonography may reveal diffuse cervical edema and soft tissue swelling caudal to the mandible and surrounding the trachea. The mass may be partly mineralized. Thoracic radiographs should be taken to identify pulmonary metastasis. Thyroid imaging (see p. 618) may reveal abnormal thyroid gland uptake (heterogeneous uptake with "hot" and "cold" regions compared with normal thyroid or salivary gland uptake) and focal accumulations of the radiopharmaceutical in the lungs, indicative of pulmonary metastasis. CT angiography can be beneficial in the differentiation of thyroid versus nonthyroid neck masses, aids in the staging of thyroid carcinomas, and is used to determine the degree of invasiveness to aid surgical planning.

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Laboratory Findings

Cytologic evaluation of a fine-needle aspirate of the cervical mass may reveal bizarre, pleomorphic cells consistent with neoplasia. Nondiagnostic samples may be obtained if the sample is contaminated with blood or is hypocellular. Additionally, neoplastic follicular epithelial cells are fragile and are often broken during sample preparation. Hyperthyroidism and hypothyroidism are occasionally associated with thyroid carcinomas; therefore measurement of serum fT4 and endogenous canine TSH concentrations is warranted. Hematologic and serum biochemical results are often normal. Hypocalcemia has been reported in a dog with a thyroid medullary carcinoma.

DIFFERENTIAL DIAGNOSIS

Cervical swelling caused by thyroid neoplasia must be differentiated from abscesses, lymphadenopathy, or sialadenopathy. This can usually be done by cytologic evaluation of fine-needle aspirates.

MEDICAL MANAGEMENT

Dogs with thyroid carcinomas, particularly if hyperthyroid, may be palliated with radioactive iodine (^{131}I); however, much larger doses of ^{131}I appear to be necessary in dogs than in cats with thyroid adenomas, and this option is not routinely used. Chemotherapy with doxorubicin may benefit animals in which complete excision is not possible. External beam RT appears beneficial for reducing tumor volume in animals after debulking procedures; however, large doses are required. Fractionated, definitive RT using multiple, moderate doses of radiation may be effective in providing local control of invasive thyroid carcinoma in dogs. Linear accelerators have replaced cobalt therapy for treatment of these tumors.

SURGICAL TREATMENT

Surgical excision of thyroid adenomas is the treatment of choice. Surgical removal of thyroid carcinomas is often difficult because of their invasive nature and pronounced vascularity (Fig. 22.25) but should be considered if metastasis is not evident and if the lesion is localized. Marginal excision (i.e., outside the tumor pseudocapsule) in tumors that are freely movable results in fewer complications than more extensive resection and does not appear to affect the local recurrence rate. Adjunctive RT and/or chemotherapy may be warranted after marginal excision, or if complete surgical excision is not possible. Chemotherapy may be indicated if debulking is done in animals with metastasis.

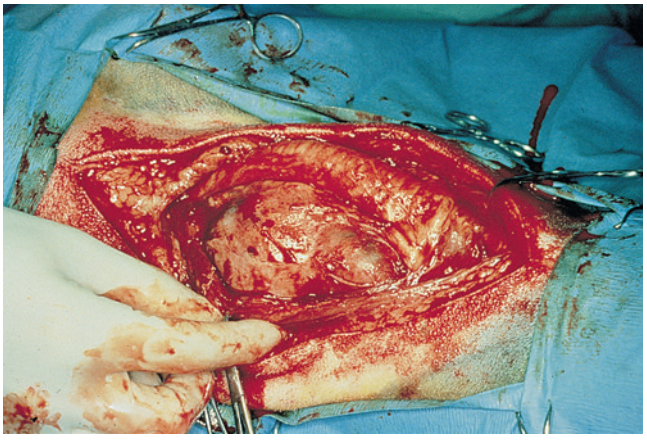


FIG. 22.25 Thyroid carcinoma in a dog. Note the invasiveness of the tumor.

NOTE Have blood available during surgery because hemorrhage is often excessive.

Preoperative Management

Substantial electrolyte and acid-base abnormalities should be corrected before surgery. Fluid therapy should be initiated before surgery in geriatric patients with reduced renal function and in those that are dehydrated.

Anesthesia

In human beings, life-threatening thyroid storms are reported intraoperatively and postoperatively for thyroid tumors. Clinical signs of tachycardia or arrhythmias may occur because of catecholamine release, and treatment should be anticipated. It may be wise to avoid drugs that are arrhythmogenic (e.g., barbiturates, halothane) in these patients. This complication is more likely to occur in cats than in dogs. Most thyroid tumors in dogs are nonsecreting; however, it is beneficial to be prepared to treat hypertension and tachycardia with β-blockers in the perioperative period.

Surgical Anatomy

The surgical anatomy of the thyroid glands is discussed on p. 616. Important structures that may adhere to or surround the tumor include the carotid artery, the internal jugular vein, the recurrent laryngeal nerve, and the esophagus. These structures should be identified and preserved, if possible, during the dissection.

Positioning

The animal is placed in dorsal recumbency with the neck slightly hyperextended. The front limbs should be tied back, away from the neck. The entire neck, cranial thorax, and caudal intermandibular space should be clipped and prepared for aseptic surgery.

SURGICAL TECHNIQUE

Make a ventral midline incision over the thyroid glands. Identify the neoplastic mass and adjacent structures. If necessary, ligate the carotid artery and jugular vein. Remove the mass (thyroid and parathyroid glands) by a combination of sharp and blunt dissection. Identify and remove abnormal cervical lymph nodes. Use electrocautery, vascular clips, Ligasure, and/or ligation to provide hemostasis. Inspect the contralateral thyroid and biopsy or remove if indicated. Close the incision routinely. Submit tissue for histologic evaluation (Fig. 22.26).

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

These tumors are frequently very vascular, and electrocautery is useful for obtaining hemostasis.

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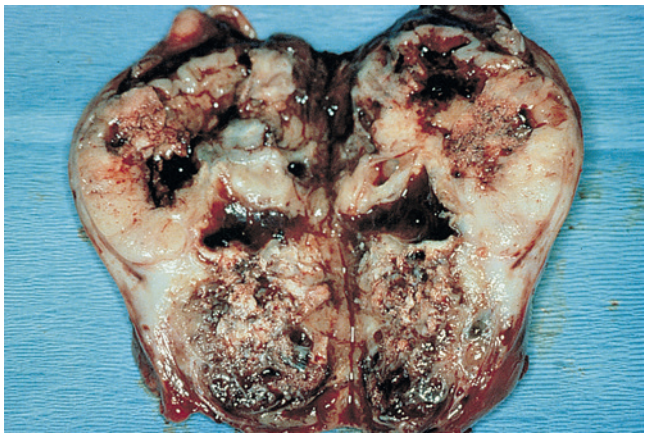


FIG. 22.26 A well-encapsulated thyroid carcinoma in a dog. Note the areas of necrosis in the gland.

POSTOPERATIVE CARE AND ASSESSMENT

A light pressure wrap may be used postoperatively to help reduce hemorrhage and swelling; however, it should be placed with care and monitored to prevent airway obstruction. Hematocrit should be monitored postoperatively and transfusions given as necessary. If unilateral thyroparathyroidectomy is performed, the animal should be observed for hypocalcemia or hypothyroidism, but supplementation is not usually necessary. If bilateral thyroparathyroidectomy is performed, vitamin D, calcium, and thyroid supplementation should be initiated postoperatively (see p. 624).

PROGNOSIS

The prognosis is good following surgical treatment of mobile thyroid tumors and irradiation of fixed thyroid carcinomas, with median survival times greater than 3 years. Median survival time for dogs with local or regional tumors (i.e., stage II or III) was significantly longer (839 days) than median survival time for dogs with metastasis (366 days). Vascular invasion, either grossly or histologically, has been shown to be a negative predictor for disease-free interval.[31] Following simultaneous bilateral thyroidectomy for discrete, mobile thyroid tumors, median survival was reported as 38.3 months; parathyroid gland tissue was preserved or reimplanted in 40% of dogs.[33]

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23

Surgery of the Hemolymphatic System

Surgery of the Lymphatic System

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Enlarged peripheral lymph nodes is most accurately termed **lymphadenomegaly,** but *lymphadenopathy* is often used. Tissue for histopathologic examination of lymph nodes may be obtained by removing the entire node (**lymphadenectomy**) or by excising a portion of it. **Lymphangiomas** are benign tumors of peripheral lymphatics; **lymphangiosarcomas** are malignant tumors of peripheral lymphatics. **Lymphangiomatosis** is a condition of multiple tumors (lymphangiomas) or cysts that grow in the lymphatic system.

PREOPERATIVE MANAGEMENT

Lymphadenomegaly is one of the more common lymphatic abnormalities of dogs and cats and may be caused by infection, nonseptic inflammation, neoplasia (metastatic or primary), or various other systemic diseases. It is important to distinguish generalized lymphadenomegaly from localized (regional) disease. With localized lymphandenomegaly, areas drained by the node should be examined for evidence of infection, inflammation, or neoplasia. Conversely, one must remember that lymph node size does not necessarily correlate with disease importance, and neoplastic cells may be retrieved from normal-sized lymph nodes. Although cytology of lymph node and splenic aspirates is very *specific* for neoplastic and fungal diseases (i.e., you can trust the diagnosis if you find such cells), it is not *sensitive* (i.e., not finding cancer or organisms never eliminates such diseases). Nonetheless, fine-needle aspiration (FNA) and/or core needle biopsies should be done before incisional or excisional lymph node or splenic biopsy because finding neoplasia or fungal elements by these less-invasive techniques makes the more-invasive techniques unnecessary. FNA cytology of enlarged lymph nodes is relatively noninvasive and inexpensive. Although most cytology samples are stained with routine Diff-Quick or Wright-Giemsa stains, sometimes special stains, immunophenotyping by flow cytometry, or polymerase chain reaction for antigen receptor rearrangement may be helpful for further evaluation. The reader is referred to a relevant medical text for an in-depth discussion of lymph node cytology.

NOTE If several lymph nodes are enlarged, avoid biopsying the mandibular node because this node tends to be reactive.

The mandibular, superficial cervical (prescapular), superficial inguinal, and popliteal lymph nodes are palpable in most animals (Fig. 23.1). The tonsils may be visualized in the oral cavity, and facial lymph nodes may be found in some normal animals. In dogs, the maxillary, accessory axillary, cervical, femoral, and retropharyngeal lymph nodes are usually palpable only when enlarged; however, the axillary node may be located readily in cats, even when it is only moderately enlarged. Unless the animal is extremely thin or cachectic, sublumbar and mesenteric lymph nodes must be at least moderately enlarged to be detected on rectal or abdominal palpation. The texture of the enlarged node and sensitivity to pressure or manipulation should be noted. Acute enlargement (i.e., suppurative lymphadenitis) can be associated with pain, but lymphoid neoplasia usually causes painless enlargement. Metastatic neoplasia and fungal infections sometimes cause nodes to become fixed to surrounding tissue. Clinical signs may result from lymphadenomegaly (e.g., coughing caused by tracheal compression by enlarged hilar lymph nodes, constipation resulting from sublumbar lymphandenomegaly).

Lymphangiomas are rare, nonmalignant abnormalities originating from lymphatic capillaries that present as fluid-filled spaces or masses in the skin or deeper tissues. In humans, they are generally diagnosed before the age of 2 years and are most commonly found on the head and neck. Lymphangiomas have been reported in both young and old dogs. They are typically developmental and associated with failure of primitive lymphatic sacs to establish venous communications; however, traumatic lymphangiomas occur.[1] In the developmental form, endothelial sprouts continue to grow and infiltrate surrounding tissues causing pressure and subsequent necrosis. This causes formation of cystic structures that manifest as large, fluctuant swellings that are noticed incidentally or because they interfere with normal structures as they grow. In dogs, they have been identified arising from skin, subcutaneous tissue, nasopharynx, and retroperitoneal space; in cats, they have been found in the liver and mediastinum. Treatment for lymphangioma consists of complete surgical excision or marsupialization; however, injection of the sclerosing agent Picibanil (OK-432), a lyophilized mixture of group A *Streptococcus pyogenes*, has been successful in treating human lymphangiomas.[2] Lymphangiomatosis has been reported in a 13-year-old, neutered male miniature dachshund that presented for bilateral symmetrical swelling in the pinnae and carpal, cubital

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ABSTRACT

Lymphandenomegaly is one of the more common diseases of the lymphatic system and is commonly caused by infection or neoplasia. Focal or diffuse enlargement of the spleen is also common in dogs and cats and is often associated with benign or malignant neoplasms or torsion. Prompt attention to the management of these animals is important; although the prognosis for some malignant tumors (e.g., hemangiosarcoma) of the spleen is relatively poor, benign enlargements such as hematomas and hemangiomas have a more favorable prognosis.

KEYWORDS

lymphatic
lymph node
lympadenomegaly
lympadenopathy
spleen
hemangiosarcoma
splenomegaly
lymphangioma
lymphangiosarcoma

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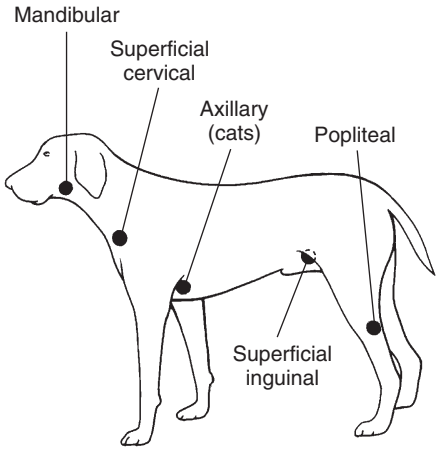


FIG. 23.1 Location of palpable lymph nodes.



FIG. 23.2 Massive head and neck swelling in a dog with diffuse lymphangiosarcoma.

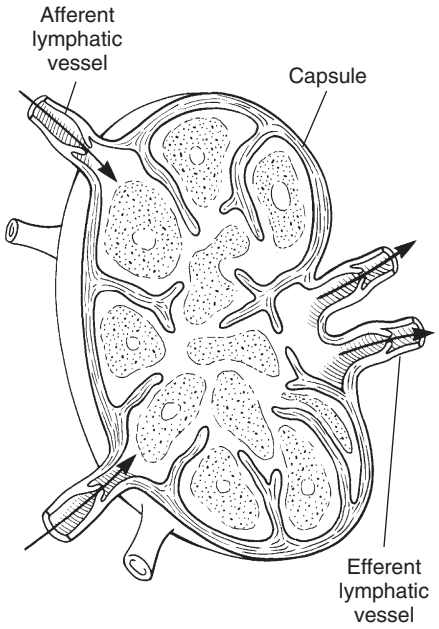


FIG. 23.3 Structure of a lymph node, showing afferent and efferent drainage.

and tarsal joints, and swelling in the tail.[3] Treatment with prednisolone improved the symptoms.

Lymphangiosarcomas (Fig. 23.2) are malignant tumors that arise from lymphatic capillaries. They are locally aggressive, and metastasis to regional lymph nodes, lungs, spleen, kidneys, and bone marrow has been reported. Even without metastasis, the local invasiveness of this tumor may necessitate amputation or euthanasia.[4] Reported most often in medium- to large-breed dogs, they may be found in dogs and cats of any size or age. The majority of affected dogs have presented for a mass and/or swelling in the cervical, trunk, or limb regions.[5] Tissue biopsies are recommended for progressive, edematous lesions of unknown origin. In dogs with lymphangiosarcoma, clinical improvement is typically noted with multimodal therapy; however, survival times may not be prolonged. In one study, survival ranged from 60 to 876 days for dogs with palliative treatment (n = 3); 90 days with prednisone only (n = 1); 182 days with chemotherapy alone (n = 1); 240 to 941 days with surgery (n = 5); and 574 days with surgery, radiation, and chemotherapy (n = 1).[5] In another study, doxorubicin treatment resulted in a 6-month recurrence-free interval in an affected dog. At relapse, the dog was treated with metronomic chemotherapy using chlorambucil and meloxicam, which failed to adequately control the disease. Toceranib phosphate resulted in almost complete regression of the mass.[6] Lymphangiosarcoma with systemic metastases has been reported in a young cat.[7]

Diagnosis may be made by histopathology, electron microscopy, immunohistochemistry, tissue culture, and/or endothelial expression of glycoconjugates. Histologically, lymphangiomas and lymphangiosarcomas are composed of vascular spaces lined by endothelial cells and focal lymphoid aggregates divided by connective tissue stroma. Unlike hemangiomas, the cystic spaces of these tumors are not filled with blood.

ANESTHESIA

Superficial (e.g., popliteal) nodes can be excised using local anesthetic and sedation if necessary; however, short-duration general anesthesia usually facilitates extirpation.

ANTIBIOTICS

Perioperative antibiotics are seldom indicated in animals undergoing lymph node biopsy or removal.

SURGICAL ANATOMY

Lymph nodes are bean-shaped structures with a convex surface and a small flat or concave hilus (Fig. 23.3). They are usually found encased in fat at flexor angles or joints, in the mediastinum and mesentery, and in the angle formed by the origin of larger blood vessels.

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DIAGNOSTIC IMAGING

Survey radiographs may detect internal lymphadenomegaly. Thoracic films should be examined for evidence of mediastinal, hilar, and sternal lymphadenomegaly; abdominal radiographs may reveal ventral deviation of the descending colon caused by sublumbar lymphadenomegaly or ill-defined mass effects in the midabdomen caused by mesenteric lymphadenopathy. Computed tomography (CT) scans may identify enlarged nodes in the chest, abdomen, or both. Magnetic resonance imaging may be of value in differentiating benign and malignant mediastinal lymphadenopathy. Positron-emission tomography (PET) scanning is not helpful as a screening tool because both benign and malignant conditions may cause intense uptake; however, PET scans are of benefit in the staging of lymphomas once a diagnosis is made.

Ultrasound is useful in detecting mesenteric, gastric, and hepatic lymphadenomegaly. Parameters that can be evaluated using ultrasound include lymph node size and margins, echotexture and echogenicity, presence and distribution of vascular flow, vascular flow indices, and acoustic transmission. Recent advances in ultrasound technology (e.g., contrast-enhanced ultrasound, contrast-enhanced endoscopic ultrasound, and real-time elastography) have shown potential in people in improving the accuracy for differentiating benign and malignant lymph nodes.[8]

SURGICAL TECHNIQUE

Lymph node biopsy is easy and relatively inexpensive, and it provides valuable information. No absolute contraindications to lymph node biopsy are known. Significant hemostatic disorders should be corrected preoperatively if possible, and care must be taken to ligate blood vessels properly.

Selection of a lymph node for a biopsy is based on clinical findings. With generalized lymphandenomegaly, the popliteal, inguinal, and prescapular lymph nodes are the preferred sites; biopsies should be obtained from at least two nodes. Mandibular lymph nodes and nodes draining the gastrointestinal tract can be diagnostic, but they are undesirable because their morphologic appearance is often distorted by reactive hyperplasia caused by constant antigenic exposure.

Fine-Needle Aspiration

Two main techniques may be used. *The first is to attach a 23- to 25-gauge needle to a 6- or 12-mL syringe. Insert the needle into the lymph node and briefly pull back the plunger to 5 or 10 mL of negative pressure several times (stop if blood is seen in the hub of the needle). Then blow the contents of the barrel of the needle out onto a clean glass slide. Make both horizontal and vertical pull-apart preparations.*

The second technique is to stick a 23- to 25-gauge needle repeatedly through a lymph node. Then attach the needle to a syringe filled with 5 mL of air and blow the contents out onto a clean glass slide. The second technique is less likely to involve blood contamination and should be done if the first technique is unsuccessful. It is also the preferred technique for aspiration of splenic masses.

Both techniques may have to be modified, depending on the patient and the tissue. Some lymphosarcomas have very fragile cells that will readily rupture. Being less aggressive in aspirating or passing a needle through the node and/or being more gentle and careful in making the cytology smear may allow one to obtain a diagnostic smear with a 20-gauge needle.

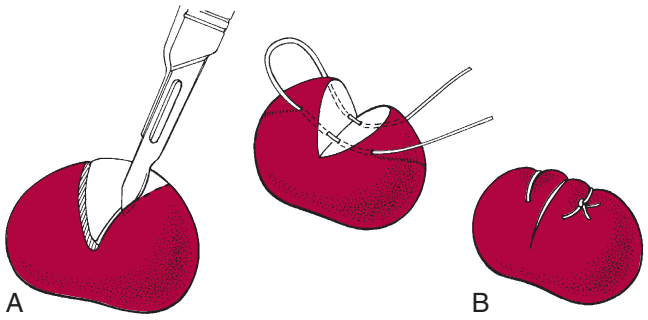


FIG. 23.4 (A) Incisional (wedge) biopsy of lymph nodes occasionally is performed. Use a No. 15 scalpel blade to remove a wedge-shaped section of the parenchyma. (B) Place a horizontal mattress suture of absorbable suture material to close the incision.

TruCut Biopsy

If the lymph node is large enough, a 14- to 16-gauge TruCut-type needle can be used to obtain a core of tissue.

NOTE Be sure to consider how deep a core you will obtain and what is behind the lymph node so that you do not traumatize or lacerate structures on the other side of the node.

Incisional Biopsy

Incisional (wedge) biopsy of lymph nodes is indicated when lymphadenectomy may be difficult because of a node's size or location (e.g., nodes that are located close to major vessels or nerves). *Use a No. 15 scalpel blade to remove a wedge-shaped section of the parenchyma (Fig. 23.4A), and place the sample in a buffered formalin solution. To provide hemostasis, place a horizontal mattress suture of absorbable suture material (e.g., 3-0 chromic catgut; Monocryl, Biosyn, or Caprosyn) to close the incision (Fig. 23.4B).*

Lymphadenectomy

Prepare the skin overlying the lymph node for aseptic surgery. Immobilize the lymph node firmly in one hand, and make an incision in the overlying skin. Bluntly dissect the node from surrounding tissue. Generally, a vessel near the hilus of the node requires ligation to prevent postoperative hemorrhage. *Handle the node gently to prevent damage and distortion of the lymph node tissue. Section the node to provide samples for aerobic and anaerobic cultures, fungal cultures, and histopathologic and cytologic evaluations. Make impression smears by lightly blotting the cut edge of the node with absorbent paper and touching the sample lightly to a glass slide before placing it in formalin. Close dead space and suture the skin routinely.*

HEALING OF THE LYMPHATIC SYSTEM

Lymphatics usually heal rapidly. Lymphedema is rare after lymphadenectomy because collateral pathways form. If lymphedema occurs, it is usually transient and seldom requires specific therapy. Frequently, prolonged obstruction of lymphatics may cause lymphaticovenous anastomoses to open or form, providing an alternate pathway for lymph flow.

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SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Special instruments are not required for lymph node biopsy or removal. Absorbable suture should be used in the lymph node parenchyma.

POSTOPERATIVE CARE AND ASSESSMENT

After lymphadenectomy, the patient should be observed for swelling at the surgical site. Swelling is usually associated with hematoma formation as a result of inadequate hemostasis or with seroma formation if dead space was not obliterated.

COMPLICATIONS

Manipulation of a tumor during biopsy procedures may transiently increase the number of neoplastic cells present in the lymphatic and vascular systems; however, finding cancer cells in a lymph node typically means that a diffuse neoplasia (e.g., lymphoma) is present, or that metastasis has already occurred. Metastasis caused by lymph node biopsy has seldom been substantiated. A hematoma may develop if vessels are not ligated adequately.

SPECIAL AGE CONSIDERATIONS

The age and physical condition of animals with lymphadenomegaly must be considered. Increased lymph node size may be expected in young animals as part of an appropriate immunologic response. As an animal ages, lymph nodes usually decrease in size, making nodes difficult to palpate. Loss of fat that normally surrounds the nodes in cachectic patients may make the nodes prominent.

SPECIFIC DISEASES

LYMPHEDEMA

DEFINITIONS

Lymphedema is an accumulation of fluid in the interstitial space. **Primary lymphedema** is caused by an abnormality or disease of lymph vessels or lymph nodes; **secondary lymphedema** occurs as a result of lymphatic obstruction of the nodes or vessels by neoplasia, filariasis, lymphoproliferative disorders, or some other infiltrative process or surgery.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Lymphedema results from a disturbance of the equilibrium between the amount of fluid in the interstitial space that needs to be cleared (capillary filtrate) and the capacity of the lymphatic and venous systems to remove this fluid. Possible causes include (1) overload of the lymphatic system, (2) inadequate collection by the lymphatic terminal buds, (3) abnormal lymphatic contractility, (4) insufficient lymphatics, (5) lymph node obstruction, and (6) central vessel (i.e., thoracic duct) defects. Regardless of the cause, edema results when capillary filtration exceeds the combined resorptive capabilities of the venous and lymphatic systems. This edema is relatively protein rich (2–5 g/dL). Because of the resultant high osmotic pressure, additional fluid is pulled into the interstitial space, worsening the edema. If the lymphatic system cannot adequately drain this interstitial fluid, collagen deposition and fibrosis may result. Hence, although the early stages are reversible, chronic edema is associated with thickening and fibrosis of tissue, making treatment difficult. The domestic species most commonly reported to have lymphedema is the dog.

Primary lymphedema is caused by an abnormality or disease of lymph vessels or lymph nodes. In humans, most patients developing primary lymphedema have no family history of lymphedema or awareness of abnormal lymphatic development before the onset of swelling; however, a hereditary form of lymphedema, Milroy's disease, has been identified. Genetic testing for this disease and other disorders (lymphedema-distichiasis) is available, and preemptive treatment is occasionally initiated prior to development of overt clinical signs. Two young whippet siblings were reported with idiopathic chylothorax and lymphedema, suggesting the possibility of a congenital or hereditary origin of the disorder in these dogs.[9]

Secondary lymphedema occurs as a result of lymphatic obstruction of the nodes or vessels by neoplasia, infection (e.g., filariasis), lymphoproliferative disorders, or some other infiltrative process or surgery. Filariasis (e.g., with *Wuchereria bancrofti*) is the most common cause of secondary lymphedema in people in tropical climates. Lymph node extirpation and/or radiation of lymph node beds associated with neoplasia is the most common cause in developed countries. Efforts to spare lymph nodes during cancer surgery and to protect from incidental damage during radiation therapy has reduced lymphedema by nearly 80% in people. Recurrent cellulitis may be the cause, or the result, of lymphedema. Secondary malignant lymphedema has been reported after mastectomy in two dogs.[10] Both dogs had large edematous lesions associated with lameness in the right hindlimb following surgery. Obstruction of lymph flow in the lymphatics of the right hindlimbs was shown with lymphangiography in one dog and lymphoscintigraphy in the other.

Distinguishing between primary and secondary lymphedema is difficult. Lymph node obstruction, although more commonly associated with secondary lymphedema, has been associated with primary lymphedema. Many of the dogs reported with primary lymphedema have had small or absent lymph nodes. Perhaps the initial defect in some dogs with lymphedema is fibrosis of the lymph nodes, which leads to secondary obstructive changes in the lymphatic vessels. As the vessels dilate, they lose contractility, and the lymphatic valves become permanently nonfunctional. Secondary lymphedema may be caused by conditions that increase the rate of interstitial fluid formation as a result of altered capillary permeability (e.g., trauma, heat, irradiation, or infection). Venous congestion (e.g., because of heart failure) may also cause lymphedema by reducing fluid resorption.

Lymphedema remains a frustrating and complex management challenge for clinicians and owners alike. Despite recent advances in the clinical understanding of this disease, as well as new developments regarding the genetic and molecular basis for lymphedema in humans, little is known regarding the cause in dogs. In both species, the condition remains incurable. Fortunately, many animals can be managed conservatively.

DIAGNOSIS

Clinical Presentation

Signalment

Age of onset of clinical signs does not help distinguish between primary and secondary lymphedema because middle-aged animals

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may have acute signs of lymphedema that occur secondary to congenital lymphatic abnormalities. Although primary lymphedema is usually noted at birth or shortly thereafter, older animals may develop lymphedema associated with congenital abnormalities. In these animals, the lymphatic system functions normally until a precipitating cause (e.g., infection, trauma, or surgery) overwhelms the marginal lymphatic system. Congenital, hereditary lymphedema has been reported in bulldogs and poodles. A gender predisposition is not evident.

History

The age of onset, progression of disease, extent of involvement (unilateral or bilateral, pelvic limb or forelimb), and history of previous surgery, trauma, or exposure to infectious agents should be ascertained. Lymphedema typically manifests as a spontaneous, painless swelling of the extremities with pitting edema. The onset may be insidious. The rear limbs are more commonly affected, and the swelling may be unilateral. Lymphedema usually begins in the distal extremity and progresses proximally. In severely affected animals, all four limbs and the trunk may be edematous. Although the patient may be less active than normal because of the weight of the limb or may carry the limb when ambulating, lameness and pain are uncommon without massive enlargement or cellulitis.

Physical Examination Findings

The diagnosis of lymphedema is usually made on the basis of clinical signs (Fig. 23.5). The limb is generally not excessively warm or cool. Although the condition is often bilateral, the degree of swelling is frequently greater in one limb. Occasionally the swelling may be precipitated by minor trauma or superficial skin infections. Fibrosis occurs as the edema becomes chronic, and the edema tends to progressively pit less until pitting is absent. With chronic edema, massage and rest do not appreciably reduce the size of the limb.



FIG. 23.5 A 2-year-old boxer with lymphedema. Note the massive swelling of the right rear limb.

Laboratory Findings

No specific laboratory abnormalities are found with lymphedema. It is important to note that these animals are not hypoalbuminemic; hypoalbuminemia is always an important differential for edema, even localized edema.

DIFFERENTIAL DIAGNOSIS

The key differential diagnosis is abnormality of the venous system, such as venous stasis or arteriovenous fistula. The clinical signs are usually adequate to differentiate lymphedema from edema caused by venous obstruction. Typical changes with edema caused by venous obstruction include varices, stasis hyperpigmentation, and cutaneous ulceration. Arteriovenous fistulae are vascular abnormalities in which a direct communication exists between an adjacent artery and vein. They may be congenital or acquired (e.g., after trauma, neoplasia, infection, or iatrogenic ligation of an artery and vein together). Clinical signs with arteriovenous fistulae vary depending on the location; however, palpation of strong pulsatile vessels, often of a fremitus or thrill, and auscultation of a machinery murmur (i.e., bruit) are classic findings. Angiography is necessary to confirm the diagnosis and determine the size, extent, and location of the fistula. The physical examination should eliminate systemic causes of bilateral edema, including heart failure, renal failure, cirrhosis, and hypoproteinemia. Other differential diagnoses include trauma, neoplasia, and foreign bodies.

DIAGNOSTIC IMAGING

The classic diagnostic tool for lymphedema has been direct lymphography, although lymphoscintigraphy may now be more common in veterinary medicine. Oil-based contrast media are contraindicated in patients with primary lymphedema because the high volume of contrast medium necessary to visualize the lymphatics and the propensity for extravasation may further damage existing lymphatics. Lymphoscintigraphy involves intradermal injection of high-molecular-weight, radiolabeled colloids. A gamma camera is used to obtain images of the affected limb over time to observe the progression of radioactivity through the lymphatics. Primary lymphedema typically shows slow absorption of the radiopharmaceutical, reduced visualization of lymphatic vessels and lymph nodes, and no interstitial activity. Secondary lymphedema typically shows poorly visualized primary lymph vessels and dilated secondary lymph vessels. There is also significant interstitial activity. These techniques rarely provide information that helps manage animals with spontaneous lymphedema, but they occasionally define the underlying lymphatic abnormality (e.g., hypoplasia, aplasia, or hyperplasia of lymphatics). They are tools that must be correlated with other historical and physical findings. The technique for lymphangiography is described on p. 637.

MEDICAL MANAGEMENT

In the early stages of lymphedema, before the development of fibrosis, nonsurgical therapy may reduce the swelling and make the patient more comfortable. Nonsurgical therapy consists of bandages that exert gentle pressure on the limb, meticulous care of the skin to prevent infection, weight control, and appropriate use of antibiotics to treat and prevent cellulitis and lymphangitis.

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Drugs that have been used to treat human patients with lymphedema include steroids, diuretics, anticoagulants, and fibrinolysin inhibitors. For the most part, the proposed benefits of these pharmaceuticals have not been substantiated. Long-term treatment of lymphedema with diuretics is contraindicated. Diuretics act by removing fluid from the tissue; proteins that are not reabsorbed become increasingly concentrated, further damaging tissues.

Classic massage therapy is generally contraindicated in extremities affected with lymphedema because it increases the arterial blood flow. This results in an increase in blood capillary pressure and subsequent increase in ultrafiltration of water in the area of the capillaries, which leads to an accumulation of fluid in the interstitial space and increased lymphatic load. In addition, superficial lymphatics are vulnerable to external pressure and traditional massage techniques may cause focal damage to anchoring filaments and the endothelial lining of the lymph vessels.

Manual lymph drainage, which is a technique where affected tissues are gently stroked in a manner that moves fluid towards the heart, may be used in combination with compression therapy and skin care. If applied correctly, manual lymph drainage increases the activity of lymph vessels and moves interstitial fluid, exerts little pressure on the skin, and does not cause an increase in local arterial blood flow.

Benzopyrones (Box 23.1) are a group of drugs that have been used to successfully treat experimental lymphedema in dogs and spontaneous lymphedema in human beings. All the drugs in this group appear to reduce high-protein edema. Their main action appears to be stimulation of macrophages, which promotes proteolysis. Protein fragments can then be reabsorbed into the blood. These drugs are active orally and topically, are inexpensive, and are relatively free of side effects. Included in this category of drugs are coumarin (5,6 benzo-[a]-pyrone), O-(β-hydroxyethyl)-rutosides, diosmin, and rutin.

NOTE Medical therapy is often ineffective in substantially reducing swelling caused by lymphedema; however, medical management has received little attention in veterinary medicine. Amputation is a reasonable alternative for unilateral lymphedema, particularly when it interferes with limb function and when initial medical management is ineffective.

SURGICAL TREATMENT

Except for amputation, no current surgical treatment offers a cure for lymphedema. Numerous therapies have been described in human patients, including lymphangioplasty, bridging procedures, lymphaticovenous shunts, omental transposition, superficial to deep anastomosis, and excision (with or without skin grafting). These techniques have not been adequately evaluated in dogs with spontaneous lymphedema. Biopsy samples of affected tissues should be submitted because lymphangiosarcoma (see earlier) has been reported to occur in human beings with long-standing lymphedema.

BOX 23.1 Benzopyrones for the Treatment of Lymphedema

Rutin[a]
50–100 mg/kg PO q8h

[a]This is one drug in the family of benzopyrones; other drugs may be as or more effective. The dose is an extrapolation of that used in human beings; side effects appear rare, but controlled studies have not been performed.
PO, Orally.

Preoperative Management

Perioperative antibiotics are indicated in patients undergoing lymphangiography because the risk of subsequent lymphangitis is high. Before performing other preoperative procedures, inject 1 mL of 3% Evans blue dye between the second and third, or third and fourth, digits to aid in the visualization and cannulation of lymphatics.

Anesthesia

General anesthesia is required for direct lymphangiography.

Surgical Anatomy

The lymphatic system of the extremities can be divided into two parts: superficial and deep (i.e., muscular) lymphatics. The superficial system appears to be the one most commonly involved in lymphedema. This is the system of lymphatics observed during pedal lymphangiography (Fig. 23.6). These lymphatics empty into a valved group of vessels found at the junction of the dermis and subcutaneous tissue. Lymph then drains into afferent lymphatics in the subcutaneous fat. The superficial lymphatics of the pelvic limb consist of a larger medial group and a smaller lateral group. Lymphatics follow the branches of the medial saphenous vein and drain into the superficial inguinal lymph nodes. Efferent lymphatics from the lymph nodes drain into the larger lymphatic ducts. The deeper lymphatics drain the fascial planes surrounding skeletal muscles (lymphatics are not found in skeletal muscle bundles), joints, and synovium. Deep lymphatic collector vessels accompany the main blood vessels of the extremities. Controversy continues as to whether the two



FIG. 23.6 Pedal lymphangiogram in a dog. The superficial lymphatic system is filled with contrast.

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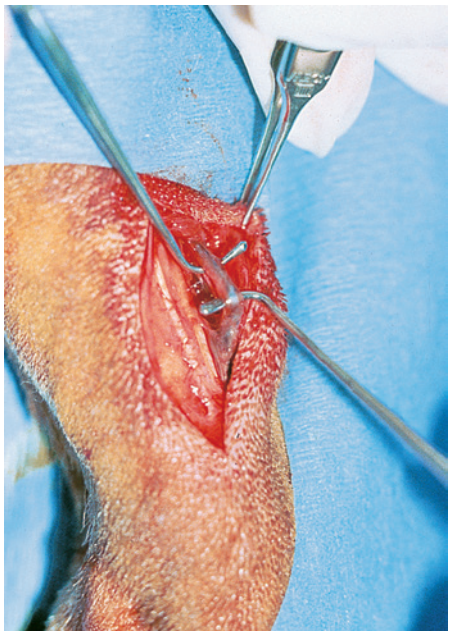


FIG. 23.7 Pedal lymphangiography. Evans blue dye is injected interdigitally before pedal lymphangiography. A 5-cm longitudinal incision is made on the dorsomedial aspect of the metatarsus, and a combination of sharp and blunt dissection is used to identify the blue-stained superficial metatarsal lymphatic vessel. Use small, blunt probes to meticulously clear the lymphatic vessel system of all subcutaneous tissue.

lymphatic systems (superficial and deep) communicate; however, communications usually appear to be a result of a lymphatic pathologic condition or a response to abnormal lymph flow. The lumbar lymph trunks receive vessels from the pelvic limbs, the abdominal lymph nodes, and the intestines before uniting to form the cisterna chyli.

Positioning

Position the animal on the radiology table in lateral recumbency with the affected limb down and the opposite limb retracted from the radiographic field. Prepare and drape the dorsomedial aspect of the metatarsus for aseptic surgery.

SURGICAL TECHNIQUE

Direct Lymphangiography

Make a 5-cm skin incision over the mid-dorsomedial metatarsal region. Use sharp and blunt dissection until a blue-stained, superficial metatarsal lymphatic vessel is identified (Fig. 23.7). Meticulously dissect the lymphatic free from surrounding tissue with fine, blunt dissection probes, and cannulate the lymphatic using a lymph duct cannulator or a 27- or 30-gauge over-the-needle catheter. Inject a small amount of sterile saline into the catheter or cannulator to verify patency. Then manually infuse an aqueous-based radiographic contrast agent into the lymphatic vessel. Take radiographs immediately after the injection (Fig. 23.8); additional radiographs may be taken, depending on the rate of lymphatic transport of the contrast agent, which varies from patient to patient. Upon completion of the lymphangiogram, withdraw the cannulator, ligate the lymphatic vessel, and close the incision in a routine fashion.



FIG. 23.8 Lymphangiogram of a dog with lymphedema. Note the dilated, tortuous lymphatics. Compare this with the lymphangiogram of a normal dog in Fig. 23.6.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

A commercial lymphatic duct cannulator, such as a Tegtmeyer lymph duct cannulator, may facilitate cannulation of the lymphatic vessel.

POSTOPERATIVE CARE AND ASSESSMENT

If the lymphatics appear abnormal in character or quantity, or if an obvious obstruction is not noted on the lymphangiogram, medical therapy or amputation should be considered. The patient should be observed for swelling or worsening of edema after the procedure.

PROGNOSIS

Primary lymphedema seldom resolves spontaneously. Neoplastic change in chronic lymphedematous tissue has been reported in dogs.

Surgery of the Spleen

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Splenomegaly is enlargement of the spleen arising from any cause. **Splenectomy** is surgical removal of the spleen. **Splenosis** is the congenital or traumatic presence of multiple nodules of normal splenic tissue in the abdomen. **Siderotic plaques** are brown or rust-colored deposits of iron and calcium that may be found on the splenic surface (Fig. 23.9). **Splenorrhaphy** is suturing of a ruptured spleen.

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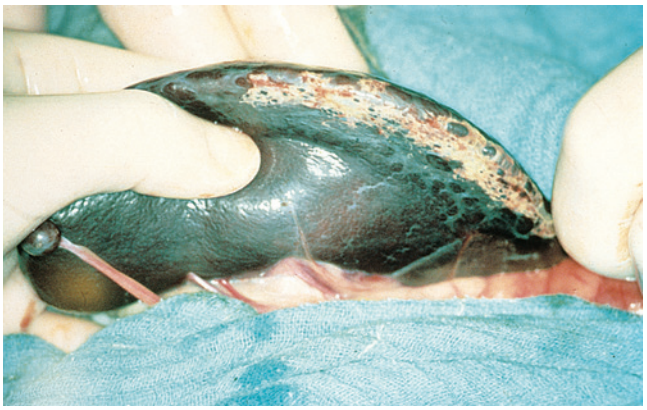


FIG. 23.9 Siderotic plaques on the splenic surface.

PREOPERATIVE MANAGEMENT

Animals with surgical diseases of the spleen often have diffuse or focal splenomegaly. Diffuse (symmetric) splenomegaly may be attributed to congestion (e.g., splenic torsion, right-sided heart failure, gastric dilatation volvulus [GDV], drugs) or infiltration due to infection (e.g., fungal, bacterial, rickettsial), splenic foreign body, immune-mediated disease (e.g., immune-mediated thrombocytopenia, immune-mediated hemolytic anemia), or neoplasia (e.g., lymphosarcoma, histiocytic sarcoma, feline mastocytosis).

Focal (asymmetric) splenomegaly may be caused by benign processes (e.g., nodular regeneration, hematoma [see p. 646], trauma) or neoplastic processes (e.g., hemangiosarcoma [see p. 645], lymphoma). Infiltrative splenomegaly resulting from neoplasia is one of the most common causes of spontaneous (noniatrogenic) splenomegaly in dogs and cats.

Anemia may be present because of acute hemorrhage associated with splenic trauma, rupture of a hematoma, or hemorrhage from an underlying disease (e.g., chronic infection, immune-mediated disease, or disseminated intravascular coagulation [DIC]). Coagulation profiles should be done in animals with bleeding that is not thought to be the result of trauma. Whole blood or packed red blood cells (RBCs) should be available when splenectomy is performed as many affected dogs require transfusions; in one study of dogs with splenic masses, transfusions were administered to 240 of 542 dogs (44%).[11] Normally hydrated animals with a packed cell volume below 20% or a hemoglobin level less than 5 to 7 g/dL may benefit from preoperative blood transfusions (see Box 4.1 and Table 4.5, respectively) or administration of purified hemoglobin; the latter may be available in some countries (e.g., Oxyglobin). If DIC is suspected, administration of plasma with or without heparin therapy may be helpful (Box 23.2). Intravenous fluids should be administered to dehydrated animals before surgery.

BOX 23.2 Therapeutics for Disseminated Intravascular Coagulation (DIC)

Plasma (Fresh Frozen)[a]

10–20 mL/kg, then reassess the plasma ATIII activity. Repeat as needed to increase the ATIII to near-normal concentrations.

Aspirin[b]

0.05–2.0 mg/kg PO q24h

Heparin-Activated Plasma[c]

Place the first heparin dose (50–100 U/kg) into the plasma and incubate for 30 minutes before administration. Once ATIII levels are above 60%, continue the heparin subcutaneously. If additional plasma is needed, incubation with heparin is not necessary.

Note: Adequate levels of ATIII are critical in these patients.

Heparin (Unfractionated)[c]

Initial dose: 35–200 U/kg SC q8–12h (35 U/kg is for prophylaxis; 200 U/kg is for treating established DIC. Subsequent doses depend on monitoring.)

Low-Molecular-Weight Heparin—Dalteparin (Fragmin)[c]

Dogs: 100–150 U/kg SC q8h

Cats: 150–175 U/kg SC q4h or 180 U/kg SC q6h

Note: Heparin is no longer considered to be an optimal treatment for DIC. Readers are referred to a relevant medical text for advances in the treatment of DIC.

Low-Molecular-Weight Heparin—Enoxaparin (Lovenox, Xaparin, Clexane)[c]

Dogs: 0.8 mg/kg SC q6h

Cats: 1.0 mg/kg SC q12h up to 1.25 mg/kg SC q6h

[a]Replacing clotting factors and ATIII is probably the best therapy for DIC.
[b]Has been effective in DIC associated with immune-mediated hemolytic anemia; efficacy in DIC caused by other diseases is unproven. High doses may be associated with toxicity in cats.
[c]Heparin is controversial in the treatment of DIC. Low-molecular-weight heparin is probably more effective than unfractionated heparin. Substantial differences have been noted between the two forms. The reader is referred to a relevant medical text for a more complete discussion, including differences in monitoring the effectiveness of therapy.
ATIII, Antithrombin III; *PO,* orally; *SC,* subcutaneous.

ANESTHESIA

Anemic patients should be given oxygen before induction of anesthesia and during recovery. Anticholinergic drugs may be used to prevent bradycardia. Barbiturates cause splenic congestion and should be avoided. Acepromazine should also be avoided in these patients because of the possibility of RBC sequestration, hypotension, and impact on platelet function. When doing a laparoscopic splenectomy, it is advantageous to avoid splenic enlargement. Acepromazine, thiopental, and propofol have been shown to cause splenic enlargement; however, hydromorphone and dexmedetomidine do not. Induction agents such as ketamine-diazepam or alfaxalone may be considered in these patients. If propofol is used, premedication with hydromorphone plus either midazolam or dexmedetomidine will decrease the amount of propofol required. A hypotensive episode may occur as a result of volume depletion after splenectomy, and the arterial blood pressure should be monitored carefully during surgery. See the anesthesia sections for GDV on p. 422 and for small intestinal surgery on p. 435 for anesthetic recommendations. See also Table 18.4 for anesthetic recommendations for the acute abdomen.

ANTIBIOTICS

Antibiotic use in animals with splenic disease is dictated by the nature of the underlying disease. The merit of perioperative prophylactic antibiotics for splenectomy in dogs is unclear and depends on the animal's age and concurrent disease and the length of surgery. Perioperative antibiotics in healthy animals are usually unnecessary but may be given at induction of anesthesia and discontinued within 24 hours (see Chapter 9). Longer

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term antibiotic therapy may be warranted in immunosuppressed or severely debilitated animals. Multiple abscessation, septicemia, and death associated with splenectomy performed in conjunction with dental cleaning and extraction has occurred in dogs.

SURGICAL ANATOMY

The spleen is situated in the left cranial abdominal quadrant. It usually lies parallel to the greater curvature of the stomach; however, its exact location depends on its size and the position of other abdominal organs. When the stomach is contracted, the spleen usually lies within the rib cage. However, with massive gastric enlargement, it may be found in the caudal abdomen. The splenic capsule is composed of elastic and smooth muscle fibers. The parenchyma consists of white pulp (lymphoid tissue) and red pulp (venous sinuses and cellular tissue filling the intravascular spaces). Large numbers of α-adrenergic receptors are responsible for splenic contraction. When the spleen is contracted, it feels firm in consistency. The spleen is normally red, but siderotic plaques (see Fig. 23.9) or fibrin deposits may change its appearance.

The arterial supply of the spleen is usually the splenic artery, a branch of the celiac artery. The splenic artery is generally more than 2 mm in diameter and gives off three to five long primary branches as it courses in the greater omentum toward the ventral third of the spleen. The first branch is usually to the pancreas and is the main supply of the left limb of that organ. The two remaining branches run toward the proximal half of the spleen, where they send 20 to 30 splenic branches that enter the parenchyma. The branches then continue in the gastrosplenic ligament to the great curvature of the stomach, where they form the short gastric arteries (which supply the fundus) and the left gastroepiploic artery (which supplies the greater curvature of the stomach) (Fig. 23.10). Other branches supply the splenocolic ligament and the greater omentum. Venous drainage occurs via the splenic vein into the gastrosplenic vein, which empties into the portal vein.

SURGICAL TECHNIQUE

The spleen is typically approached via a ventral midline abdominal incision that extends from the xiphoid to a point caudal to the umbilicus or via laparoscopy. For the former, the incision may need to be lengthened for large lesions or to allow complete abdominal exploration. Complete abdominal exploration should be performed in any animal suspected of having neoplasia.

Splenic Aspiration

Place the animal in right lateral or dorsal recumbency using manual restraint or mild sedation. Avoid using phenothiazine tranquilizers or barbiturates because the resultant splenic congestion may result in a nondiagnostic sample as a result of blood dilution. Surgically prepare a small area on the left side of the abdomen, and isolate the spleen. Penetrate the abdominal wall with a small needle (23- or 25-gauge, 1–1.5 inches [2.54–3.81 cm]) and pass the needle into the spleen several times, being careful not to lacerate splenic arteries. If you start to see blood in the hub of the needle, stop. Remove the needle from the abdomen, and blow out the contents of the needle onto a clean glass slide. Although uncommon, major hemorrhage can occur after FNA of the spleen (not only splenic hemangiosarcomas but also spleens with diffuse lymphosarcoma). Ultrasound can be used to guide placement of the needle.

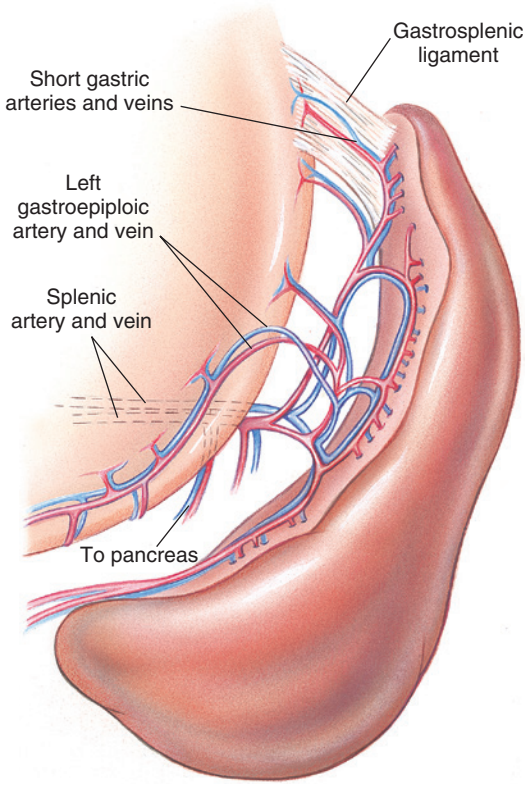


FIG. 23.10 Splenic vasculature. Note the short gastric arteries, which often are avulsed in dogs with gastric dilatation volvulus.

NOTE When cavitary lesions are identified with ultrasound scans, FNA should be performed with care or not at all. Cavitary lesions may rupture during aspiration, which could be fatal, especially in animals with coagulopathies.

Splenic Biopsy

Splenic biopsies are indicated to ascertain the cause of clinically significant splenomegaly or suspected metastatic lesions to the spleen. They may be obtained percutaneously (i.e., FNA or core biopsy) laparoscopically, or at surgery. Some authors have suggested that combined ultrasound-guided FNA and needle core biopsy may be complementary in dogs with suspected splenic neoplasia.[12] Ultrasound-guided biopsies improve the likelihood of obtaining diagnostic samples percutaneously. Percutaneous biopsies are often diagnostic for diffuse lesions (e.g., mastocytosis, lymphosarcoma); however, focal or nodular lesions may be missed. FNA can be specific (e.g., if malignant cells or yeast forms are found) but is not particularly sensitive. Aspirate cytology is much less sensitive for splenic lesions than for lymph node lesions, and it is particularly insensitive for differentiating hemangiosarcoma from hematoma (see p. 647). Laparoscopic biopsy of the splenic parenchyma using rigid biopsy forceps is often tolerated; such biopsy of splenic masses is associated with additional risk, which must be weighed against the potential benefits.[13]

Larger tissue samples should be placed in 10 volumes of formalin to one part tissue for routine histopathologic examination. Special preservatives may be required if additional staining techniques are desired (e.g., Bouin's fixative is preferred for identification of viral inclusions). Samples larger than 5 cm should be scored (cut into) before placement in formalin to allow the

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samples to fix properly. Large splenic masses should be scored at several sites but left intact to allow orientation of the entire lesion by the pathologist. If this is not possible, several representative samples should be submitted from diverse sites, including the margin of the abnormal- and normal-appearing tissue. If the lesion is cavitary (e.g., splenic mass, cyst, or abscess), it should be ruptured before it is placed in formalin.

Laparoscopic Biopsy

Establish a CO_2 pneumoperitoneum (10–12 mm Hg) using a Veress needle or place the first access port using the Hassan technique. First, insert a partially-threaded trocar so that it enters the abdomen at or near the umbilicus (Fig. 23.11A), then insert a 30-degree laparoscopic telescope to evaluate the abdomen. Next, place two accessory ports under laparoscopic guidance; insert one on the midline and the other 1 cm cranial to the prepuce (see Fig. 23.11B). After rotating the dog on its right side, insert a portal approximately 3 to 5 cm lateral and caudal to the second port. Introduce an atraumatic grasping forceps through that port and a LigaSure V through the second port. Immediately localize the splenic mass, if present, to avoid lacerating it during splenic manipulation. Elevate the splenic tail using grasping forceps and observe the ventral aspect of the spleen. Use laparoscopic cup biopsy forceps to grasp and twist the splenic capsule to allow access to the portion to be sampled. Then use the biopsy forceps to obtain a sample. Place a gelatin sponge in the biopsy site to achieve hemostasis.[13] *Close each port site in three layers.*

Surgical Biopsy

During celiotomy, biopsies of focal lesions may be taken by FNA or with a TruCut (see p. 544), Jamshidi, modified Franklin-Silverman, or punch biopsy. *To remove focal lesions near the center of the spleen, make a rectangular or oval incision through the capsule and into the parenchyma to sufficient depth to remove the lesion. Close the defect by placing simple interrupted or mattress sutures of absorbable material (3-0 or 4-0) in the splenic capsule.* Partial splenectomy may be performed if more diffuse lesions are present (see later discussion).

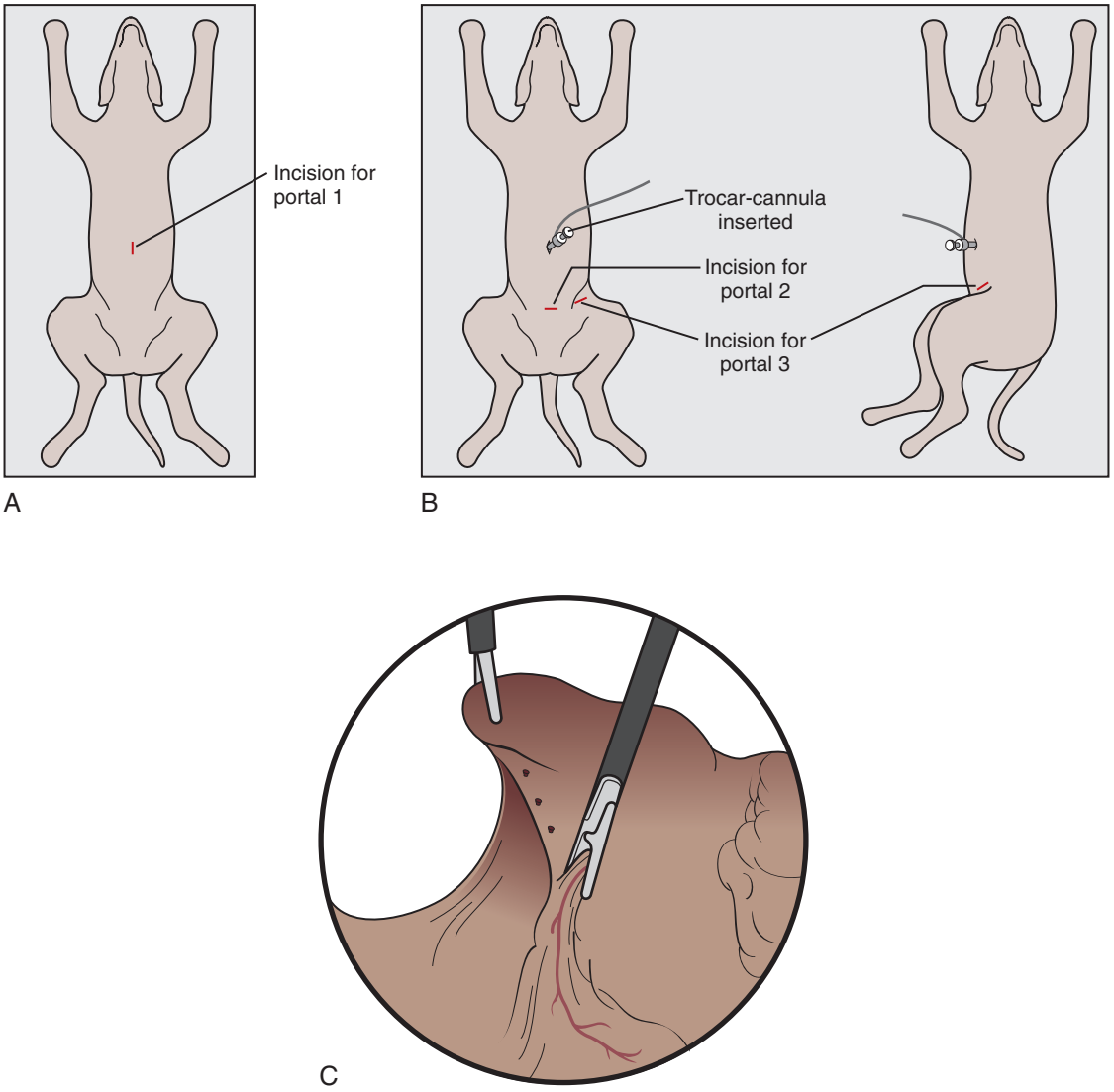


FIG. 23.11 (A) Portal placement 1 and (B) 2 for laparoscopic splenic biopsy or partial splenectomy. (C) Insert grasping forceps and a LigaSure through portal 3.

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Repair of Lacerations and Splenic Rupture

Trauma-associated splenic rupture/laceration (e.g., due to vehicular trauma) typically resolves without the need for repair or splenectomy. Iatrogenic splenic laceration (e.g., due to placement of Veress needles and laparoscopic trocars/cannulas [see p. 642]) is typically self-limiting and seldom requires intervention (unless it is grossly excessive, such as by completely impaling the spleen with the trocar/cannula assembly). If bleeding is excessive, transfusion of fresh whole blood or packed RBCs is typically sufficient to resolve the problem. An important exception is the patient with splenomegaly associated with portal hypertension. An engorged spleen in a hypertensive patient may have a particularly fragile capsule that cannot easily be sutured because the sutures tend to tear out, causing more bleeding. Sometimes even applying pressure to the site will cause further capsular tearing. Even small defects may result in sustained, uncontrollable hemorrhage and may require splenectomy to stop the bleeding.

Splenorrhaphy is indicated to provide hemostasis in superficial traumatic lesions of the splenic capsule. *Explore the lesion and ligate any large traumatized vessels. Place simple interrupted or mattress sutures of absorbable material (3-0 or 4-0) in the splenic capsule.* Gelfoam may be applied over smaller lacerations. *Apply gentle pressure to the area for several minutes. If bleeding continues, ligate the splenic branches supplying the lesion as close to the hilus of the spleen as possible.* Small areas of ischemia revascularize as a result of collateralization.

The LigaSure vessel sealing system (Covidien) is a surgical device that applies pressure and bipolar energy to the tissue, causing vessel sealing. Collagen and elastin in the vessel walls fuse into a single structure, obliterating the vessel lumen. A LigaSure may be used successfully to cauterize vessels smaller than 7 mm. It may be a fast and efficient way to control hemorrhage in a ruptured spleen.

Surgical Partial Splenectomy

Partial splenectomy is indicated in animals with traumatic or focal lesions of the spleen to preserve splenic function. *Define the area of the spleen to be removed and double ligate and incise the hilar vessels supplying the area (Fig. 23.12A). Note the extent of ischemia that develops, and use this as a guideline for the resection. Squeeze the splenic tissue at this line between a thumb and forefinger, and milk the splenic pulp toward the ischemic area. Place forceps on the flattened portion, and divide the spleen between the forceps (see Fig. 23.12B). Close the cut surface of the spleen adjacent to the forceps in a continuous pattern using absorbable suture (3-0 or 4-0) (see Fig. 23.12C). As an alternative, place two rows of mattress sutures in a continuous overlapping fashion at the line of demarcation. If hemorrhage continues, oversew the end of the spleen with a continuous suture of absorbable suture material.*

Automated stapling devices (e.g., transverse anastomosis staplers) may also be used for partial splenectomy; however, some risk exists that if the staples are not secured in sufficient tissue, they will loosen, allowing hemorrhage to occur from the splenic stump. Use of 3.5 or 4.8 size stainless steel staples is recommended. When properly performed, surgical stapling for partial splenectomy significantly reduces surgical time and omental adhesion to the spleen.

Laparoscopic Partial Splenectomy

Place the ports as described previously for laparoscopic splenic biopsy. Dissect the splenic attachments, sealing and dividing attachments and vessels as close as possible to the parenchyma. Place a specimen retrieval bag (e.g., Endo Catch) through the second port. Place only the portion of the spleen with the mass in the specimen retrieval bag because of the small bag size. Discontinue insufflation, remove the port, and enlarge the incision to facilitate removal of the specimen retrieval bag. Submit the spleen for histologic evaluation. Close the incisions in three layers.

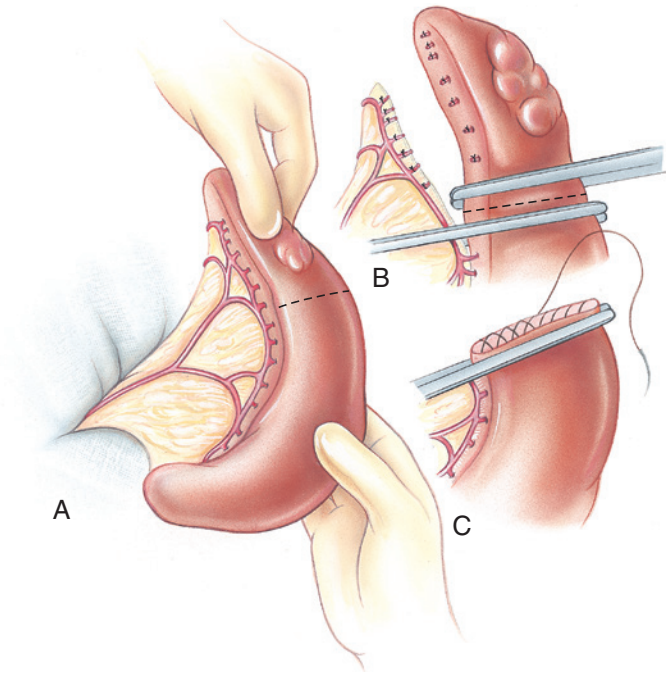


FIG. 23.12 Partial splenectomy preserves splenic function in animals with traumatic or focal lesions. (A) Define the area of the spleen to be removed, and double ligate and incise the hilar vessels supplying the area. (B) Transect the spleen between forceps. (C) Close the cut surface in a continuous suture pattern.

Total Splenectomy

Total splenectomy is most commonly performed in animals with splenic neoplasia, torsion (stomach or spleen), or severe trauma that is causing life-threatening hemorrhage that cannot be stopped. Splenectomy was previously advocated for immune-mediated hematologic disorders refractory to medical therapy (e.g., thrombocytopenia, hemolytic anemia). Splenectomy may be associated with an improved outcome in select dogs with immune-mediated thrombocytopenia or immune-mediated hemolytic anemia refractory to medical management. Immunosuppressive drugs (e.g., cyclosporine, azathioprine) and corticosteroids have reduced the need for splenectomy; however, splenectomy is reasonable if drug therapy is unsuccessful or causes unacceptable side effects.

The spleen normally contains a reservoir of RBCs, has hematopoietic capabilities, has important phagocytic functions, and is helpful in maintaining immunocompetence; total splenectomy eliminates these beneficial actions. Elective splenectomy has been performed in dogs used as blood donors, so that subclinical infection with infectious agents (e.g., *Anaplasmosis, Babesia, Bartonella*) will become obvious, and the dog can be eliminated from the program. Dogs and cats used as blood donors should be screened for both vector-borne and non–vector-borne

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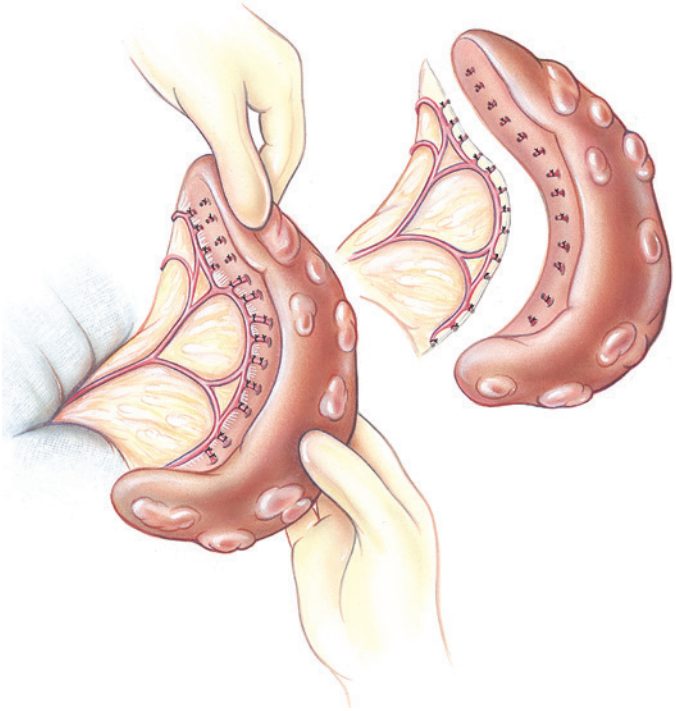


FIG. 23.13 For total splenectomy, double ligate and transect all vessels at the splenic hilus. If possible, preserve the short gastric branches supplying the gastric fundus.

pathogens.[14] Although life-threatening sepsis has occurred in splenectomized people, this has not been observed in dogs. Nevertheless, partial splenectomy may be preferred over total splenectomy when possible. Splenectomy is contraindicated in patients with bone marrow hypoplasia in which the spleen is a main site of hematopoiesis.

After exploring the abdomen, exteriorize the spleen and place moistened abdominal sponges or laparotomy pads around the incision under the spleen. Double ligate and transect all vessels at the splenic hilus with absorbable (preferred) or nonabsorbable suture material (Fig. 23.13), or use a vessel sealant device such as the LigaSure (see Chapter 8). If possible, preserve the short gastric branches supplying the gastric fundus.

As an alternative, open the omental bursa and isolate the splenic artery. Identify the branch or branches supplying the left limb of the pancreas. Double ligate and transect the splenic artery distal to this vessel (or these vessels) (Fig. 23.14).

Laparoscopic-Assisted Splenectomy

Laparoscopic-assisted splenectomy is feasible in dogs. After port placement (multiple laparoscopic ports or a single multichannel port), a minilaparotomy is done and a wound retraction device is inserted with progressive exteriorization of the spleen and sealing of hilar vessels.[15] *Place a multichannel port or standard laparoscopic port caudal to the umbilicus, then insufflate the abdomen. To facilitate abdominal exploration if a standard port was used, place a second port cranial and lateral to the first port. Replace the initial port with a wound retraction device (e.g., Alexis Wound Retractor) to circumferentially dilate the abdominal incision. Lengthen the incision in the linea alba, insert the flexible ring into the abdomen against the body wall, and ensure no abdominal tissue was entrapped between the ring and body wall. Evaluate the*

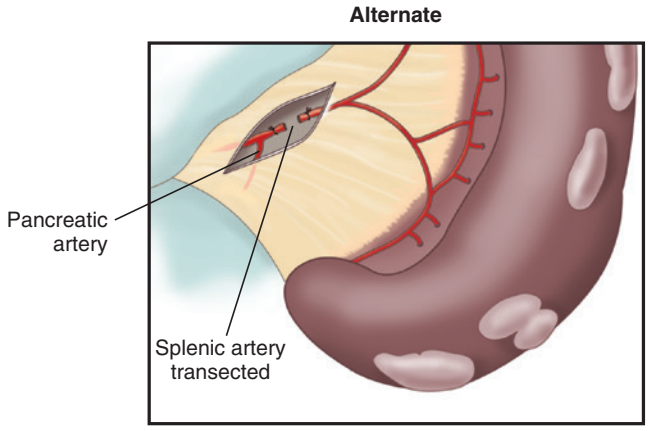


FIG. 23.14 Splenectomy may be performed by double ligating the splenic artery distal to the branches that supply the left limb of the pancreas.

gastrointestinal tract by exteriorizing and replacing each segment through the wound retractor.

NOTE It is uncertain whether dogs undergoing splenectomy are at increased risk of GDV. In one study, 219 dogs undergoing splenectomy for reasons other than splenic torsion in which a gastropexy was not performed (172 dogs) were compared to control dogs (47 dogs undergoing enterotomy). No difference in the subsequent development of GDV was found between groups in this study.[16] In another study, dogs with previous splenectomy were found to be 5.3 times more likely to develop GDV.[17]

SUTURE MATERIALS AND SPECIAL SURGICAL INSTRUMENTS

Other than a general soft tissue pack, special instruments are not required for surgical procedures of the spleen; however, a large number of clamps should be available for splenectomy. Absorbable suture material, bipolar sealing derives, or vascular clamps may be used to ligate the splenic vessels. If generalized peritonitis is present, monofilament, synthetic absorbable suture should be used to ligate vessels.

POSTOPERATIVE CARE AND ASSESSMENT

After splenic biopsy or splenectomy, the animal should be closely observed for 24 hours for evidence of hemorrhage. The hematocrit should be evaluated every few hours until the animal is stable. Nasal oxygen should be administered to anemic patients (see Chapter 4) and analgesics given if necessary (see Tables 13.1 and 13.2). Hemorrhage may indicate technical failure or DIC (which can be associated with neoplastic lesions or torsed spleens). Fluid therapy should be continued until the animal is able to maintain its own hydration, and electrolyte and acid-base abnormalities should be corrected. Mild postoperative leukocytosis may occur after splenectomy in dogs because the spleen influences bone marrow leukocyte production; however, steep or prolonged elevations may indicate infection (e.g., splenic abscess or peritonitis). An increase in the numbers of Howell-Jolly bodies,

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nucleated erythrocytes, target cells, and/or platelets may be noted after splenectomy but is not a cause for concern.

COMPLICATIONS

The major complication of splenic surgery is hemorrhage. This is more of a problem with splenic biopsy or partial splenectomy than with total splenectomy, providing proper technique is used for vessel ligation. Reported complications of splenectomy in dogs include abscessation, traumatic pancreatitis, and gastric fistulation due to impairment of gastric blood flow. The risk of septic complications after splenectomy appears to be significant only in animals that are immunosuppressed before surgery (e.g., those undergoing immunosuppressive therapy for immune-mediated hemolytic anemia). Previous subclinical infection with hemoparasites (e.g., *Babesia*, *Ehrlichia*, *Mycoplasma*) may become obvious after splenectomy. Interference with blood flow through the pancreatic branch of the splenic artery may cause pancreatic ischemia and pancreatitis. With laparoscopic-assisted splenectomy, complications are typically limited to minor intraoperative hemorrhage.

SPECIAL AGE CONSIDERATIONS

Splenic surgery is most commonly performed in middle-aged or older animals. Special care must be taken to meet the metabolic and nutritional needs of these patients. Physical examinations and laboratory analyses must be thorough to determine whether concurrent disease exists that may influence the surgery or postoperative care.

SPECIFIC DISEASES

SPLENIC TORSION

DEFINITION

Splenic torsion is the twisting of the spleen on its vascular pedicle.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Splenic torsion most often occurs in association with GDV; isolated splenic torsion occurs rarely in dogs (Fig. 23.15) and to our knowledge has not been reported in cats. Importantly, splenic torsion is rarely associated with neoplasia. Typically, the thin-walled splenic vein is occluded, although the splenic artery remains partly patent, resulting in congestive splenomegaly. Vascular thrombosis (particularly of the splenic vein) may occur. In some dogs the clinical signs are acute; in others the torsion is presumably intermittent, and abnormalities are noted weeks before diagnosis.

The cause of isolated splenic torsion is unclear. It may be related to congenital abnormalities or traumatic disruption of the gastrosplenic or splenocolic ligament. It has also been hypothesized that splenic torsion may occur after partial gastric torsion (i.e., an intermittently malpositioned stomach), in which case the spleen remains torsed despite repositioning of the stomach. Primary splenic torsion may be acute or chronic. The chronic form is difficult to diagnose because clinical signs are often vague and may be intermittent; however, chronic splenic torsion was recently reported in two dogs that had had clinical signs from between 2 weeks and 4 months; clinical signs resolved in these dogs after splenectomy.[18]

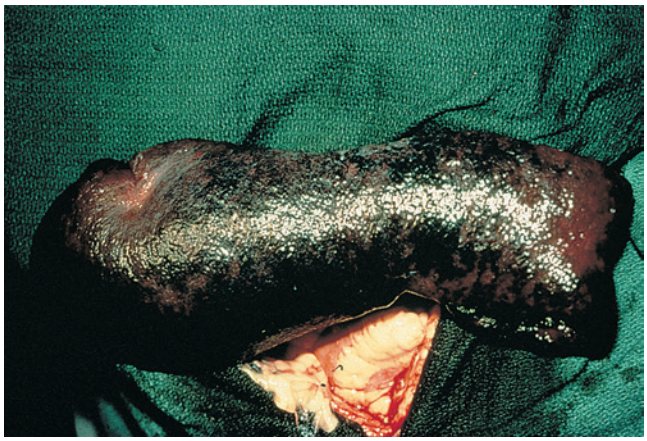


FIG. 23.15 Torsed spleen in a dog with acute cardiovascular collapse and shock. Note the dark, congested appearance of the spleen.

Splenic infarction may be associated with other disease, such as hepatic disease, renal disease, hyperadrenocorticism, neoplasia, or thrombosis associated with cardiovascular disease. In these cases, splenic infarction appears to be a sign of altered blood flow and coagulation rather than of the primary disease. In such patients, splenectomy should be reserved for animals with life-threatening complications, such as hemoabdomen or sepsis. If bacteria gain access to a torsed spleen, they can rapidly proliferate in the devitalized, necrotic tissue. If not resolved, septicemia and systemic inflammatory response syndrome may result.

NOTE Splenic torsion can be acute and life threatening, requiring prompt diagnosis and treatment.

DIAGNOSIS

Clinical Presentation

Signalment

Splenic torsion is most commonly reported in large-breed or giant-breed, deep-chested dogs, particularly great Danes and German shepherd dogs, but English bulldogs may also be over-represented. In a 2016 study of 102 affected dogs, these three breeds accounted for 50% of the cases.[19] This same study found castrated males to be more commonly affected than females or intact males.

History

Most animals are presented for treatment because of some combination of vomiting, weakness or depression, icterus, hematuria or hemoglobinuria, abdominal pain, and/or diarrhea. Clinical signs may be acute or chronic. Some owners have reported chronic intermittent signs up to 3 weeks before evaluation. Torsion has been attributed to a diaphragmatic hernia that occurred several years previously. Acute torsion may cause signs of cardiovascular collapse and shock.

Physical Examination Findings

The most prominent physical examination finding is splenic enlargement or a midabdominal mass. Abdominal pain, fever,

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dehydration, pale mucous membranes, or icterus (or all of these) is sometimes found. Dogs with cardiovascular collapse and shock have tachycardia, pale mucous membranes, prolonged capillary refill times, and/or weak peripheral pulses.

Diagnostic Imaging

The most common radiographic findings are diminished visceral detail associated with peritoneal effusion and displacement of the small intestine by an enlarged spleen. The splenic outline is often difficult to discern. Sometimes, obvious splenomegaly is seen. If the dorsal extremity (head) or the body of the spleen is not observed in its normal position, splenic torsion is suggested. Occasionally, gas bubbles are present within the splenic parenchyma, presumably formed by gas-producing bacteria (e.g., *Clostridium* spp.) in the devitalized spleen. Gastric dilatation may occur.

Ultrasonographically, the splenic parenchyma may be normal, hypoechoic, or anechoic with interspersed linear echoes; however, a hypoechoic spleen is most commonly seen.[19] Finding a notably enlarged spleen that is diffusely hypoechoic, with linear echoes separating large, anechoic areas (Fig. 23.16) may be unique to splenic torsion. Enlargement of hilar splenic vessels may also suggest this condition. B-mode evaluation of the splenic veins for intraluminal echoes and spectral or color Doppler evaluation for absent velocity flow may be important assessments to make in dogs with splenic torsion and/or infarction. Visible splenic vein intraluminal echogenicities compatible with thrombi may be seen ultrasonographically in dogs with splenic torsion that have vascular congestion and compression and thrombosis of the splenic vein. Spectral Doppler and color Doppler imaging of the splenic vein will show an absence of flow in affected dogs.

Laboratory Findings

Laboratory analysis may reveal anemia, leukocytosis, hemoglobinuria, elevated serum alkaline phosphatase activity, thrombocytopenia, and/or elevated alanine transaminase activity. Proactive monitoring of coagulation parameters to allow for early recognition of signs of coagulopathy and prompt administration of appropriate treatment may help decrease the mortality rate associated with this condition.[19]

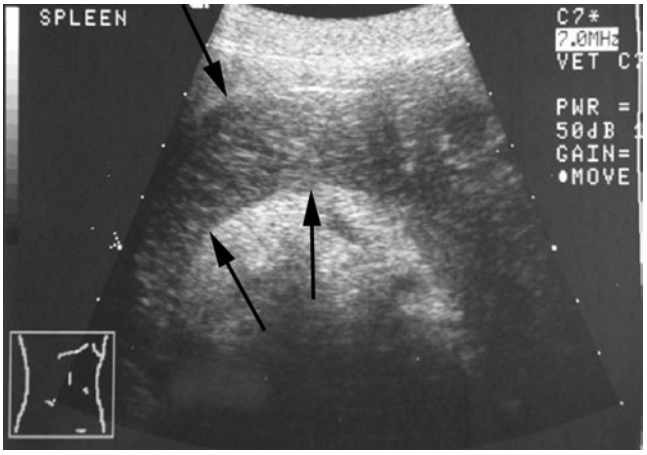


FIG. 23.16 Ultrasound image of the spleen in a dog with splenic torsion. Note the hypoechoic appearance of the splenic parenchyma with hyperechoic lines throughout *(arrows)*.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include other causes of splenomegaly (e.g., neoplasia, trauma, hematoma, abscess, or immune-mediated disease), peritoneal effusion (e.g., peritonitis or ascites), other midabdominal masses (e.g., gastrointestinal, pancreatic, renal, or lymph node enlargement), and GDV.

MEDICAL MANAGEMENT

Splenic torsion is a surgical disease; medical management is usually limited to stabilizing the animal for surgery (see later discussion under "Preoperative Management"). If the animal is shocky, intravenous fluids and antibiotic therapy should be initiated.

SURGICAL TREATMENT

The timing of surgical therapy is influenced by the animal's status at presentation. An animal presented for treatment with signs of shock should be operated on as quickly as possible after its condition has been stabilized. Surgery may be reasonably delayed for a short period in animals with chronic disease; however, prompt surgical intervention is recommended.

Preoperative Management

If possible, fluid deficits and electrolyte and acid-base abnormalities should be corrected before surgery. Whole blood or packed RBC administration is warranted in animals with a hematocrit below 20% (see Box 4.1). Perioperative antibiotic therapy (e.g., cefazolin 22 mg/kg IV) is recommended because vascular occlusion and necrosis may allow proliferation of bacteria in the spleen. Electrocardiograms are warranted to determine whether cardiac arrhythmias are present that may require therapy before induction of anesthesia or during surgery. Blood transfusion products should be available because the enlarged and congested spleen may rupture with handling, causing abdominal hemorrhage.

Anesthesia

See the anesthesia sections for GDV on p. 422 and small intestinal surgery on p. 435 for anesthetic recommendations. See also Table 19.1 for anesthetic recommendations for the acute abdomen.

Surgical Anatomy

See p. 639 for surgical anatomy of the spleen.

Positioning

For laparotomy, the animal is positioned in dorsal recumbency, and the entire ventral abdomen is prepared for aseptic surgery. The ventral incision should extend from the xiphoid and should be long enough to allow the enlarged spleen to be manipulated and exteriorized.

SURGICAL TECHNIQUE

Splenectomy (see p. 641) is the treatment of choice in dogs with acute splenic torsion because no good way to secure the spleen in its normal position is known, and torsion may recur. Furthermore, derotating the spleen may allow necrotic debris and toxins to enter the systemic circulation and is not recommended; thus splenectomy is the only viable option in animals with chronic torsion, and is recommended in those with acute torsion.

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SUTURE MATERIALS AND SPECIAL INSTRUMENTS

See p. 642 for the suture materials and special instruments used for splenectomy.

POSTOPERATIVE CARE AND ASSESSMENT

Most animals recover quickly after removal of the torsed spleen. Intravenous fluid therapy should be continued until the animal is able to maintain its own hydration. Vomiting may occur postoperatively, associated with pancreatic ischemia and pancreatitis. The blood supply to the left limb of the pancreas arises from the splenic artery, and the vascular obstruction may extend to this pancreatic branch. Previous subclinical infections with hemoparasites (e.g., *Babesia*, *Ehrlichia*, Hemotrophic *mycoplasma* [formerly *Haemobartonella*]) may become obvious after splenectomy.

PROGNOSIS

The prognosis is generally good after surgical management of splenic torsion. Delayed diagnosis may result in splenic necrosis, sepsis, peritonitis, and/or DIC. Perioperative risk factors that may increase the risk of death prior to hospital discharge include presence of septic peritonitis at initial examination, intraoperative hemorrhage, and postoperative development of respiratory distress.[19] Bipolar vessel sealing devices may decrease surgery time, reduce blood loss, and allow for less manipulation of the spleen, which may decrease complication rates.[20]

SPLENIC NEOPLASIA

DEFINITIONS

Hemangiosarcomas (HSAs) are malignant neoplasms that arise from blood vessels; **hemangiomas** are benign tumors of dilated blood vessels. A **hematoma** is a swelling or mass of blood (usually clotted) confined to an organ, tissue, or space that is caused by any reason. HSA is also known as *angiosarcoma* and *hemangioendothelioma*.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The spleen is composed of a variety of tissues, and splenic neoplasia may arise from blood vessels, lymphoid tissue, smooth muscle, or the connective tissue that makes up the fibrous stroma. In a 2012 study of 249 dogs with splenic masses, nearly half were found to have nonmalignant disease.[21] The most common splenic tumor in dogs is HSA; this tumor is more commonly diagnosed in large dogs (>27.8 kg) than small dogs.[22] The pathogenesis of canine HSA may be related to miRNA dysregulation.[23] Other malignant tumors of the spleen in dogs include liposarcoma, fibrohistiocytic nodules, lymphoma, blastoma, and adenocarcinoma (Box 23.3 and Fig. 23.17). Nonmalignant masses include nodular hyperplasia, hemangioma, hematoma, and splenitis (see Box 23.3 and Fig. 23.18). Dogs with hemoperitoneum have a higher frequency of splenic neoplasia; similarly, cats with hemoperitoneum commonly have abdominal neoplasms, and HSA is the most common malignant splenic tumor in cats.

BOX 23.3 Differential Diagnoses for Splenic Enlargement, Nodules, or Masses (in Alphabetical Order)

Neoplastic Disease

Benign

- Fibroma
- Hemangioma
- Lipoma
- Myelolipoma

Malignant

- Chondrosarcoma
- Fibrosarcoma
- Hemangiosarcoma
- Histiocytosis
- Liposarcoma
- Lymphosarcoma
- Mast cell tumor
- Mesenchymoma
- Metastatic neoplasia
- Myxosarcoma
- Osteosarcoma
- Rhabdomyosarcoma
- Undifferentiated/anaplastic sarcoma

Nonneoplastic Disease

- Abscessation
- Engorgement due to portal hypertension
- Extramedullary hematopoiesis
- Hematoma
- Iatrogenic (drugs)
- Thrombosis/infarction
- Torsion

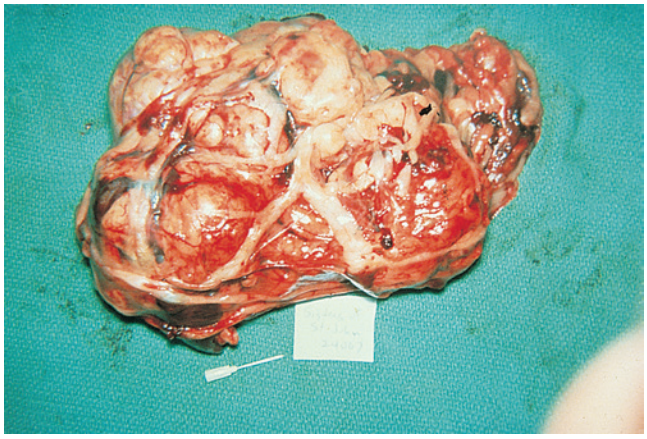


FIG. 23.17 Splenic leiomyosarcoma in a 6-year-old mixed-breed dog.

NOTE Incidentally found, nonruptured splenic masses or nodules without associated hemoperitoneum are most commonly benign.[24] Surgery is warranted in these dogs as the prognosis is often fair or good.

Canine splenic HSA may be seen in more than one-third of dogs presenting with acute nontraumatic hemoabdomen. Because HSAs arise from blood vessels, they may form in several different sites in the body (e.g., spleen, right atrium, subcutaneous tissue, liver). The incidence of concurrent splenic and right atrial HSA

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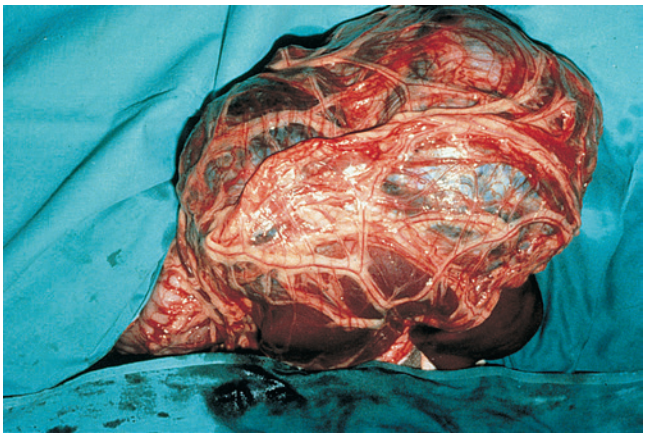


FIG. 23.18 Splenic hematoma in a 10-year-old labrador retriever. note the similar appearance of this benign mass to the malignant tumor in Fig. 23.17.

is unknown, but was recently reported to be as low as 8.7%.[25] Splenic HSAs are aggressive tumors that frequently metastasize to the liver, omentum, mesentery, and brain. Most dogs with HSA have gross evidence of metastatic disease on initial presentation.

Splenic hematomas vary in size and are encapsulated, blood- and fibrin-filled masses that are often grossly and ultrasonographically indistinguishable from HSAs. Histologically, the cavities are surrounded by congestion, fibrosis, and areas of necrosis. They may result from trauma, may occur spontaneously, or may develop secondary to other diseases (e.g., nodular hyperplasia). Hemangiomas and HSAs may be difficult to distinguish histologically, but because the prognosis for these lesions is very different, it is important that they be accurately differentiated. Splenic masses with evidence of malignant neoplastic endothelial cell proliferation can be easily identified as HSAs. However, multiple sections of a malignant mass may be studied without finding malignant cells. More importantly, a proliferation of plump endothelial cells that resemble neoplastic endothelium, but do not have evidence of mitotic activity, may be misdiagnosed as HSA. Hyperplastic nodules are also a common finding at necropsy.

Mastocytoma, lymphosarcoma, myeloproliferative disease, and HSA are the most common neoplasms of the feline spleen. Splenic involvement is a consistent finding in cats with noncutaneous systemic mastocytosis. It is not associated with the feline leukemia virus and is primarily a disease of older cats. Mast cell infiltrates may be recognized in other organs (e.g., liver, lymph nodes, and bone marrow), and circulating mastocytosis may be present in 50% of affected cats. Splenomegaly is one of the most common gross findings in feline mast cell disease, which is usually diagnosed by finding neoplastic cells in the circulation or on bone marrow examination. Splenic HSA is less commonly recognized in cats than in dogs. Extraabdominal metastasis of mast cell tumors, particularly to the myocardium, appears to be common.

DIAGNOSIS

Clinical Presentation

Signalment

Splenic tumors may occur in large-breed or small-breed dogs. Dogs weighing less than 27.8 kg may be less likely to be diagnosed with HSA than larger dogs.[22] There are conflicting reports on the effect of sex on disease prevalence. Canine splenic HSAs occur mainly in older animals. A predisposition is described for German shepherd and Labrador retriever dogs; boxers, Bernese mountain dog, German shorthaired pointers, and flat coated retriever may also have a high relative risk.[26] No breed or sex predisposition has been reported in cats with HSA, and no obvious breed or gender predilection has been observed in dogs with nonangiogenic and nonlymphomatous splenic sarcomas. The mean age of occurrence of HSA in dogs is 8 to 13 years and from 8 to 10.5 years in cats.

History

Dogs with HSA may be presented because of abdominal enlargement, anorexia, lethargy, depression, and/or vomiting, or they may have acute signs of weakness, depression, anorexia, and hypovolemic shock caused by splenic rupture and hemorrhage. The clinical signs of splenic hematoma are similar, except that rupture leading to collapse and anorexia are less common because large masses frequently become apparent before rupture occurs. The most common clinical signs of disease with other types of sarcomas include diminished appetite, abdominal distention (as a result of peritoneal effusion or tumor mass or both), polydipsia, vomiting, and/or lethargy. In contrast to dogs with HSA, splenic rupture and hemorrhage are uncommon in dogs with nonangiogenic and nonlymphomatous splenic tumors. The most common presenting signs in cats are a palpable abdominal mass and anorexia.

Physical Examination Findings

The physical examination findings include lethargy, weakness, abdominal distention, and possibly splenomegaly or a splenic mass. If a splenic mass is palpated, it should be handled gently to prevent iatrogenic rupture. If abdominal effusion is present, it is not always possible to palpate the enlarged spleen. If rupture occurs, the animal may have signs of hypovolemic shock (tachycardia, pale mucous membranes, and weak peripheral pulses). Hemoabdomen is more commonly associated with HSA than with hemangioma or hematoma. In fact, dogs with nonruptured splenic masses or nodules, without associated hemoperitoneum, are more likely to have benign splenic lesions than malignant tumors.[24] Some dogs with HSA will have cutaneous metastasis (dark reddish-purple mass). Sometimes a murmur is heard in patients with HSA in the right atrium; pericardial effusion due to such an HSA may cause muffled heart sounds or a jugular pulse.

Diagnostic Imaging

Abdominal masses are usually detected radiographically in dogs with HSA and nonangiogenic and nonlymphomatous sarcomas; however, peritoneal fluid may make locating the lesion in the spleen difficult. Masses involving the tail of the spleen are typically identified in the cranial ventral abdomen on the lateral radiographic projection (Fig. 23.19). Thoracic radiographs (or thoracic CT) should be taken in animals with splenic masses to detect pulmonary or thoracic neoplasia.

Ultrasonography is more definitive than radiography in locating lesions in the spleen and in detecting abdominal metastases; however, differentiation of hematomas from neoplastic lesions is unreliable. Finding internal septation and encapsulation or apparent metastasis may help differentiate hematoma from HSA. Contrast-enhanced ultrasound can improve the characterization of focal or multifocal lesions of the spleen. The most useful

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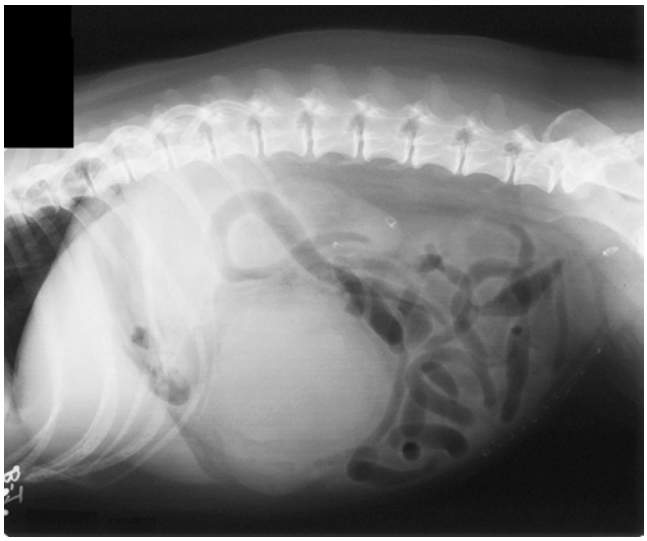


FIG. 23.19 Lateral radiograph of a dog with a large soft tissue mass in the cranial ventral abdomen. This mass arose from the tail of the spleen.

criterion was the hypoechogenicity of the lesion in the washout phase combined with the presence of tortuous feeding vessels, which is observed in association with malignancy. Magnetic resonance imaging may be a useful tool for differentiating benign and malignant splenic lesions in dogs. CT and PET/CT (see Chapter 14) may also be useful diagnostic imaging modalities for evaluation of focal canine splenic masses.

NOTE Before surgery, it may be wise to obtain an echocardiogram of the heart to look for right atrial HSA in dogs with splenic masses.

Laboratory Findings

Neutrophilic leukocytosis may be present in some dogs. Mild or moderate anemia associated with chronic disease or hemoperitoneum is also common. Other hematologic abnormalities caused by HSA may include numerous nucleated RBCs (inappropriate numbers for the degree of anemia), Howell-Jolly bodies, poikilocytosis, acanthocytosis, schistocytosis, and/or thrombocytopenia. Dogs with hemoabdomen and HSA may have lower total solid concentrations and platelet counts on admission than dogs with other causes of hemoabdomen. Hemostatic disorders, particularly thrombocytopenia caused by DIC, are common in dogs with splenic tumors. Abdominal effusion is generally serosanguinous or hemorrhagic. Cytologic analysis of abdominal fluid rarely reveals tumor cells.

DIFFERENTIAL DIAGNOSIS

Splenic hematoma and hemangioma must be differentiated from HSA and other neoplastic diseases of the spleen (see earlier comments and Box 23.3). Elastography and dual-phase CT have not been found to differentiate between benign and malignant splenic tumors; however, thymidine kinase 1, a soluble biomarker associated with DNA synthesis in serum, may help discriminate between benign disease and HSA in dogs with hemoabdomen and a splenic mass.[27] If cavitary lesions are identified ultrasonographically, FNA should be performed cautiously or not at all. Diagnosis of HSA is difficult with FNA because the cells exfoliate poorly. In addition, cytologic differentiation of hematoma and HSA with samples obtained by FNA is often impossible because large numbers of neoplastic cells are frequently necessary to make an accurate diagnosis. Furthermore, cavitary lesions may rupture during aspiration, and this could be fatal. Exploratory surgery is usually indicated; however, it is difficult to differentiate these lesions by direct visualization. Although the presence of hepatic nodules may indicate metastasis and malignancy in dogs with splenic masses, hepatic nodules may also represent extramedullary hematopoiesis or nodular hyperplasia in animals with benign or malignant tumors. If desired, one may perform laparoscopy first to see whether hepatic, omental, or peritoneal metastasis is apparent before deciding whether to do surgery. This is a potentially specific technique for finding metastatic disease undetectable by imaging, but its sensitivity is dependent on the size of the metastatic lesions. Other causes of hemoperitoneum include hepatocellular carcinoma, rodenticide poisoning, and trauma. CT may not differentiate malignant and nonmalignant hepatic or splenic masses.

NOTE Histologic diagnosis of splenic HSA may require review of several sections of the mass.

MEDICAL MANAGEMENT

Surgical resection is the mainstay of therapy in dogs with splenic HSA; however, postoperative chemotherapy or immunotherapy may prolong survival. Readers are referred to an oncology text for discussion of protocols and treatment regimens used in dogs with HSA.

SURGICAL TREATMENT

Splenectomy is the treatment of choice for animals with splenic hematoma and hemangioma. It is also the treatment of choice for animals with HSA in which evidence of extensive metastasis or other organ failure does not preclude the short-term benefits of removing the enlarged or ruptured spleen. Laparoscopy is a more sensitive method of detecting visceral HSA metastasis than imaging and can be done before surgery to decide whether surgery is appropriate for a given patient. Splenectomy may not be warranted in dogs with concurrent right atrial tumor; therefore careful preoperative examination (e.g., echocardiogram) of patients is necessary. Dogs with splenic lymphoma and clinical signs associated with massive splenomegaly, splenic rupture, and hemoperitoneum may also benefit from splenectomy. Gastropexy may be performed concurrently (see previous discussion on splenectomy).

NOTE Importantly, large splenic masses may be more likely to be benign than malignant. Dogs with benign splenic masses often have larger and heavier spleens than dogs with HSA.

Preoperative Management

Anemic animals may require blood transfusions (see Box 4.1) before surgery, and they should be preoxygenated. An electrocardiogram should be performed to determine whether ventricular arrhythmias requiring preoperative or intraoperative therapy

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are present. Ventricular arrhythmias are present in some dogs with splenic masses, and anemia and hemoabdomen may be strongly associated with arrhythmia development. Hydration, electrolyte, and acid-base abnormalities should be corrected before induction of anesthesia, but it must be remembered that fluid therapy may result in worsening of previously mild anemia; the hematocrit must be reexamined shortly before anesthesia. Perioperative antibiotics (e.g., cefazolin 22 mg/kg IV) may be indicated in some animals undergoing splenectomy (see p. 638).

Anesthesia

See the anesthesia sections for GDV on p. 422 and small intestinal surgery on p. 435 for anesthetic recommendations. See also Table 18.4 for anesthetic recommendations for the acute abdomen.

Surgical Anatomy

See p. 639 for the surgical anatomy of the spleen.

Positioning

The animal is placed in dorsal recumbency for a ventral midline celiotomy (see p. 516).

SURGICAL TECHNIQUE

Splenectomy is described on p. 641. Total splenectomy, rather than partial splenectomy, is warranted in animals with malignant tumors or large benign masses. Laparoscopy has been used to remove splenic HSA; however, only surgeons proficient in interventional laparoscopy should attempt this procedure (see p. 642 for a description of the technique). A hepatic biopsy (see p. 546) is likely to be of low-yield in a dog with HSA that has a normal appearing liver. In one study, no dogs with grossly normal livers had metastasis detected on liver pathology; however, nodules (whether multiple, dark-colored, and/or actively bleeding) were highly associated with malignancy.[28]

NOTE It is difficult to differentiate HSA from hematoma. Several samples of the mass must be submitted for histopathologic evaluation.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Surgical instruments for diseases of the spleen are described on p. 642. Patients with neoplasia (particularly debilitated animals) may heal poorly; therefore care should be taken in closing abdominal incisions, and strong, monofilament absorbable or nonabsorbable suture should be used.

POSTOPERATIVE CARE AND ASSESSMENT

Animals with splenic HSA should be closely observed for DIC after splenectomy. Fluid therapy should be continued until the animal can maintain its own hydration. The hematocrit should be monitored and blood transfusions provided if the packed cell volume falls below 20%. Septic complications after splenectomy appear to be rare, and antibiotic therapy can be discontinued within 24 hours in most animals.

NOTE Be prepared to treat cardiac arrhythmias, particularly if hemoabdomen or anemia or both are present.

PROGNOSIS

Life expectancy of dogs with splenic masses, as one might expect, is determined largely on whether the mass is benign or malignant. In one study, the median life expectancy of dogs with benign and malignant lesions was 436 and 110 days, respectively.[24] In the aforementioned study, the median life expectancy of dogs with HSA was 132 days; only seven of these 18 dogs received adjunctive chemotherapy. Vascular tumors of intermediate malignancy, termed *hemangioendotheliomas,* appear to have a better prognosis than HSAs. Because most tumors of the spleen cannot be differentiated on gross inspection alone and survival of dogs with hematomas is much longer than that of dogs with HSA-associated lesions, surgery should not be denied for dogs in which a definitive diagnosis of HSA has not been made.

Survival of dogs with HSA may be influenced by clinical stage; dogs with hemoperitoneum at the time of diagnosis may have a shortened survival. Dogs that are anemic or thrombocytopenic at presentation, or that develop ventricular arrhythmias during surgery, may have a worse prognosis.[29] Affected dogs typically die from uncontrolled bleeding from metastatic lesions and thrombotic and coagulation disorders. Although the histologic pattern of growth does not appear to affect survival, dogs with cavernous tumors have a shorter survival because of the increased propensity for these tumors to rupture and bleed.[26]

Median survival time of dogs with splenic HSA treated with splenectomy alone was 1.6 months in a 2015 study.[30] When the entire follow-up period was considered, there was no significant difference in survival time between dogs treated with surgery alone and dogs treated with surgery and chemotherapy. However, during the first 4 months of follow-up, after adjusting for the effects of clinical stage, survival time was significantly prolonged among dogs receiving any type of chemotherapy and among dogs receiving both conventional and metronomic chemotherapy.[30]

Over 50% of dogs with splenic lymphoma treated by splenectomy alone will survive at least 1 year, and animals surviving to a year are unlikely to die of this disease. Marginal zone lymphoma and mantle cell lymphoma were the most common histologic B-cell lymphoma subtypes in one study.[31] Preoperative or postoperative adjuvant chemotherapy is unlikely to provide a survival benefit.[31]

Median survival time after splenectomy in cats was 197 days, with a range of 2 to 1959 days, in one study.[32] Preoperative weight loss was the only factor that had prognostic significance for survival following splenectomy in the aforementioned study. For cats with weight loss, the time was 3 days; for those without weight loss, the time was 293 days.

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24

Surgery of the Kidney and Ureter



GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Nephrectomy is excision of the kidney; **nephrotomy** is a surgical incision into the kidney. **Nephrostomy** is the creation of a permanent fistula leading into the pelvis of the kidney; temporary nephrostomy tubes (**nephropyelostomy**) are occasionally used to divert urine when obstructive uropathy occurs or when the proximal ureter has been avulsed from the kidney. **Pyelolithotomy** is an incision made into the renal pelvis and proximal ureter to remove calculi; a **ureterotomy** is an incision into the ureter. **Neoureterostomy** is a surgical procedure performed to correct intramural ectopic ureters; **ureteroneocystostomy** involves implantation of a resected ureter into the bladder. **Renal disease** refers to when there are morphologic or function lesions in one or both kidneys; it does not refer to severity or extent. **Chronic kidney disease** (CKD), the most common renal disease of dogs and cats, refers to patients with a permanent loss of functioning nephrons that have had renal damage, with or without a decreased glomerular filtration rate (GFR) for at least 3 months, or that have had a 50% reduction of GFR for at least 3 months. A staging system for canine and feline CKD has been proposed by the International Renal Interest Society (Box 24.1), reflecting degrees of creatinine concentration with substages regarding proteinuria and blood pressure. **Renal failure** is when the kidneys are not able to maintain excretory, regulatory, and/or endocrine function, with subsequent retention of waste products and abnormalities in fluid, electrolyte, and/or acid-base homeostasis. This generally happens when 75% or more of the nephrons become nonfunctional. **Azotemia** is defined as an increased concentration of nitrogenous waste products in the blood. **Uremia** is the clinical syndrome associated with sufficient loss of functional nephrons. **Chronic renal failure** (CRF) is a progressive disease that occurs in patients with CKD with significant clinical signs (polyuria, polydipsia, weight loss, decreased appetite) and laboratory findings (azotemia, anemia, proteinuria). **Acute renal failure** (also referred to as **acute kidney injury** [AKI]) refers to patients that have an abrupt decline in renal function, which leads to retention of nitrogenous wastes. Acute renal failure is usually caused by ischemic, toxic, or infectious insults. Sometimes this becomes confusing; animals with CRF or CKD can become acutely worse if reasons for acute injury are superimposed upon the chronic condition (i.e., acute on chronic kidney failure).

PREOPERATIVE MANAGEMENT

Renal, ureteral, or urethral disease may cause AKI or CKD, or disease in these organs may be associated with AKI or CKD from other causes. The minimum database for patients with urinary dysfunction includes measurement of hematocrit, blood urea nitrogen (BUN), phosphorus, calcium, creatinine, total protein, albumin, electrolytes, total carbon dioxide levels, urinalysis, and blood pressure, and an electrocardiogram (ECG) if electrolyte values are not readily available. Renal patients may have significant metabolic derangements besides azotemia. CKD and especially AKI may be associated with varying degrees of dehydration. Oliguria may be present in AKI or in severe CKD; however, both AKI and CKD may be associated with polyuria. Preoperative intravenous (IV) fluid therapy is necessary to restore circulating blood volume and urine production; however, fluids must be administered judiciously to prevent overloading of these patients. Diuretics may also be helpful for enhancing urine production in animals that are adequately hydrated. Urine production of hydrated animals on maintenance fluids that do not have abnormal extrarenal losses should be at least 50 mL/kg per day or more than 2 mL/kg per hour.

Various electrolyte and acid-base abnormalities may occur, depending on the severity and duration of the renal, urethral, or ureteral disease. Hyperkalemia may be present in AKI due to obstructive disorders, uroabdomen, and parenchymal dysfunction, as well as in patients with severe, end-stage CRF. Hypokalemia may occur with CKD and diuretic therapy. Hyperkalemia and hypokalemia predispose the patient to cardiac arrhythmias and should be addressed before surgery. Severe hypermagnesemia and hypomagnesemia can cause cardiac conduction disturbances and central nervous system aberrations. Clinically important hypocalcemia is occasionally associated with AKI. Hypercalcemia may cause AKI or may be due to CKD. Metabolic acidosis may be present in animals with acute kidney disease or CKD but tends to be worse with acute injury.

> **NOTE** Abnormalities in the serum potassium level may lead to cardiac arrhythmias and death; correct these abnormalities before surgery.

Animals with later stages of CKD are typically anemic owing to diminished renal erythropoietin production. Elevated circulating parathormone concentrations may also have a negative effect on erythropoietin concentrations. Gastritis with bleeding or increased red cell fragility may occur in uremic patients. Coagulation profiles are warranted in animals with severe kidney disease. Normally hydrated animals with a packed cell volume below 20% or a hemoglobin level less than 5 g/dL may benefit from preoperative blood transfusions (see Box 4.1).

ANESTHETIC CONSIDERATIONS

Animals with CKD may pose unique challenges for anesthesia. The CKD patient may have chronic electrolyte abnormalities

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ABSTRACT

The primary goal of surgery on the kidneys or ureters is to preserve renal function, particularly in the scenario of obstruction of the renal pelvis or ureter. Renal biopsy may be indicated to determine an underlying cause and reversibility of renal disease. Removal of a kidney and associated ureter should be reserved for cases of renal neoplasia, irreparable blunt, penetrating,or iatrogenic trauma to the kidney and ureter, or congenital anomalies that cannot be definitively addressed. Ectopic ureters, resulting in urinary incontinence, are the most common congenital abnormality of the upper urinary tract.

KEYWORDS

kidney
ureter
nephrectomy
nephrotomy
pyelotomy
ureterotomy
neoureterostomy
ureteroneocystostomy
acute kidney injury
chronic kidney disease
azotemia
uremia
calculi
ectopic ureter

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BOX 24.1 Staging System for Chronic Kidney Disease

Step 1: Staging Is Initially Based on Fasting Plasma Creatinine Assessed on at Least Two Occasions in the Stable Patient

Canine

- Stage 1 (100% renal function remaining) = plasma creatinine <125 µmol/L or <1.4 mg/dL
- Stage 2 (33% renal function remaining) = plasma creatinine = 125–179 µmol/L or 1.4–2.0 mg/dL
- Stage 3 (25% renal function remaining) = plasma creatinine = 180–439 µmol/L or 2.1–5.0 mg/dL
- Stage 4 (<10% renal function remaining) = plasma creatinine ≥440 µmol/L or ≥5.0 mg/dL

Feline

- Stage 1 (100% renal function remaining) = plasma creatinine <140 µmol/L or <1.6 mg/dL
- Stage 2 (33% renal function remaining) = plasma creatinine = 140–249 µmol/L or 1.6–2.8 mg/dL
- Stage 3 (25% renal function remaining) = plasma creatinine = 250–439 µmol/L or 2.9–5.0 mg/dL
- Stage 4 (<10% renal function remaining) = plasma creatinine ≥440 µmol/L or ≥5.0 mg/dL

Step 2: Cases Are Then Substaged Based on Proteinuria and Blood Pressure

Urine Protein/Creatinine Ratio

- Nonproteinuric = 0–0.2
- Borderline proteinuric = 0.2–0.5 (dogs), 0.2–0.4 (cats)
- Proteinuric >0.5 (dogs), >0.4 (cats)

Risk of End Organ Damage From Hypertension (Systolic Blood Pressure)

Stage 0: Minimal risk = 130–149 mm Hg
Stage 1: Moderate risk = 150–159 mm Hg
Stage 2: Moderate risk = 160–179 mm Hg
Stage 3: High risk >180 mm Hg

that should generally be corrected slowly. Hypokalemia predisposes to cardiac arrhythmias. Very severe hypokalemia may cause ECG changes (e.g., flattened T waves, U waves) and skeletal muscle weakness. Severe hypomagnesemia sometimes causes dysrhythmias, weakness, or hypotension. Patients may be tachycardic owing to hypovolemia, which may mask concurrent hypertension or anemia. Affected animals may be malnourished and hypoproteinemic, which may affect how they respond to medications. Altered pH and hypoproteinemia in azotemic patients may alter drug binding as well as the blood-brain barrier; thus they are often particularly susceptible to medications that cause sedation. Therefore the lower end of drug doses should be used.

Patients with AKI due to acute ureteral obstruction are often otherwise healthy. The mildly affected animal can be treated similarly to an otherwise healthy patient, except that special attention should be paid to limiting IV fluids before relieving the urinary obstruction. The obstructed patient with AKI may have sodium and potassium disturbances and possibly life-threatening ECG changes associated with hyperkalemia, hypovolemia, hypotension, weakness, and hypothermia. These patients can usually tolerate only limited doses of drugs causing further sedation or hypotension. Another group of AKI patients consists of those with CKD that have acute disease superimposed, and therefore present in an AKI crisis. Laboratory data are extremely important in determining the animal's electrolyte status and in anticipating the animal's response to anesthesia.

Several generalities can be made about patients with renal disease. First, the decompensated patient that is hypovolemic and depressed usually needs minimal to no premedication. Opioids may need to be given to effect during surgery, instead of as a premedication. Second, many drugs (e.g., induction agents, pain medications) have to be given slowly and in lower doses than in healthy animals. Third, when possible, slowly correct electrolyte abnormalities, hypovolemia, anemia, and hypothermia before surgery. Patients should be diligently monitored intraoperatively and postoperatively, including oxygen saturation via pulse oximeter, blood pressure, ECG, respiratory rate, and temperature. In addition, monitoring of end-tidal carbon dioxide is very helpful in titrating narcotics intraoperatively and in assessing adequacy of ventilation.

Severely anemic patients should be transfused if the hematocrit is low (<18% in cats; <20% in dogs) before induction of anesthesia; these animals will benefit from oxygen before, during, and after anesthesia (see p. 29). Anticholinergic drugs should be given as needed to prevent or treat bradycardia. Medications that should be avoided in patients with renal compromise include nonsteroidal antiinflammatory drugs, aminoglycosides, and some IV radiographic contrast agents. Compound A, a byproduct of sevoflurane, causes renal failure in rats; however, research does not support this finding in dogs, cats, and humans. In the human literature, it is recommended to use sevoflurane at an oxygen flow rate greater than 2 L/min to protect against nephrotoxicity in compromised patients, or to use isoflurane. Ketamine at low doses or as a low-dose constant-rate infusion is generally not contraindicated in animals with renal disease. However, in chronically ill or compromised patients, ketamine can cause problems when used at induction doses. Ketamine stimulates the sympathetic nervous system and inhibits reuptake of norepinephrine. The effect of ketamine relies on adequate catecholamine stores to support blood pressure during induction. In chronically ill or severely compromised patients, catecholamine stores may be depleted, resulting in marked hypotension and bradycardia when this drug is used.

Systemic arterial blood pressure should be maintained between 70 and 80 mm Hg to ensure adequate renal perfusion, and urine output should be monitored during surgery, with a urine output goal of at least 0.5 mL/kg per hour. Because of the intrinsic properties of the kidney to autoregulate, renal blood flow tends to remain constant despite variations in systemic arterial pressure between 75 and 160 mm Hg. However, hypotension during surgery may cause renal vasoconstriction, diminished renal blood flow, and subsequent renal damage. Hypotensive drugs (e.g., acepromazine) should not be used in animals with renal impairment. If a cat is oliguric but normotensive, mannitol 0.25 to 0.5 g/kg IV may be used. If both oliguria and hypotension are present, dopamine (dogs: 5–20 µg/kg per min IV; cats: 2 µg/kg per min IV) or dobutamine (2 µg/kg per min IV) may be administered. Once oliguria is established, dopamine is minimally effective at creating a polyuric state. Isoflurane is the inhalation agent of choice in arrhythmic patients. If nephrectomy is to be performed, function in the remaining kidney should be protected by ensuring adequate blood flow during and after surgery.

The following general anesthetic principles should be considered in animals with renal disease (Table 24.1). The patient may be premedicated with an anticholinergic if bradycardic (i.e., atropine

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TABLE 24.1 Anesthetic Considerations for Animals With Renal Disease[a]

Preoperative Considerations

Associated conditions	• Anemia • Dehydration • Electrolyte abnormalities • Gastritis/gastric erosions • Malnutrition • Hypertension • Dysrhythmias • Ascites/edema (due to hypoalbuminemia)
Bloodwork	• HCT • Electrolytes • BUN • Cr • TP • Albumin • Calcium • Phosphorus • Blood gas
Physical examination	• May be dehydrated, tachycardic or bradycardic, hypotensive or hypertensive, and/or hypothermic
Other diagnostics	• Blood pressure • ECG

Premedications	• Rehydrate over 4–6 h if possible
	• Blood transfusion if HCT <20% in dogs, <18% in cats
	• Correct electrolyte abnormalities
	• Avoid sedatives in sick patients
	• Avoid alpha-2 agonists and acepromazine
	• If patient is anxious, administer midazolam (0.1–0.2 mg/kg IV, IM)
	• If patient is not decompensated, administer:
	  • Hydromorphone[b] (0.05–0.2 mg/kg IV, IM in dogs; 0.025–0.1 mg/kg IV, IM in cats), *or*
	  • Morphine[c] (0.1–0.2 mg/kg IV, or 0.2–0.4 IM), *or*
	  • Oxymorphone (0.1–0.2 mg/kg IV, IM), *or*
	  • Buprenorphine[d] (0.005–0.02 mg/kg, IV, IM)

Intraoperative Considerations

Induction	• If dehydrated, give the following:
	  • Etomidate (0.5–1.5 mg/kg IV) *or*
	  • Propofol (1–4 mg/kg IV) slowly, *or*
	  • Alfaxalone (2–3 mg/kg IV)
	• If hydrated, give the following:
	  • Propofol (2–6 mg/kg IV), *or*
	  • Alfaxalone (3–5 mg/kg IV)

Maintenance	• Isoflurane *plus*
	  • Fentanyl (1–10 µg/kg IV PRN in dogs; 1–4 µg/kg IV PRN in cats), *plus* PRN
	  • Fentanyl CRI (1–5 µg IV loading dose, then 1–10 µg/kg/h IV), *or*
	  • Hydromorphone[b] (0.05–0.2 mg/kg IV PRN in dogs; 0.025–0.1 mg/kg IV PRN in cats), *or*
	  • Buprenorphine[d] (0.005–0.02 mg/kg IV PRN), *or*
	  • Ketamine (low dose) (0.5–1 mg/kg IV)
	  • For hypotension (to keep MAP 60–80 mm Hg), give phenylephrine, ephedrine, dopamine as needed

Fluid needs	• 20 mL/kg first hour if not anuric, then 5–10 mL/kg/h plus 3× EBL
Monitoring	• Blood pressure • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature • U/O
Blocks	Epidural: • Morphine[c] (0.1 mg/kg preservative free), *or* • Buprenorphine[d] (0.003–0.005 mg/kg diluted in saline) • Avoid local anesthetics for spinals and epidurals in hypotensive patients Incisional: • Lidocaine (<5 mg/kg in dogs; 2–4 mg/kg in cats), *or* • Bupivacaine (<2 mg/kg)

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TABLE 24.1 Anesthetic Considerations for Animals With Renal Disease[a]—cont'd

Postoperative Considerations

Analgesia	• Fentanyl CRI (1–5 µg/kg IV loading dose, then 2–15 µg/kg/h IV), *or* • Morphine[c] (0.1–0.5 mg/kg IV or 0.1–1 mg/kg IM q2–8h in dogs; 0.025–0.2 mg/kg IV or 0.05–0.3 mg/kg IM q2–8h in cats if no hypotension), *or* • Hydromorphone[b] (0.05–0.2 mg/kg IV, IM q3–8h in dogs; 0.025–0.1 mg/kg IV, IM q3–8h in cats), *or* • Hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs), *or* • Oxymorphone (0.1–0.2 mg/kg IV, IM q2–4h), *or* • Buprenorphine[d] (0.005–0.02 mg/kg IV, IM q4–8h or 0.01–0.02 mg/kg OTM q6–12h in cats) • NSAIDs are contraindicated
Monitoring	• SpO_2 • Blood pressure • HR • Respiratory rate • Temperature • U/O
Blood work	• HCT • Electrolytes • BUN • Cr • +/– Blood gas
Estimated pain score	Moderate to severe

[a]Patients with renal disease may have decreased volume of distribution and decreased clearance of many drugs. It is recommended to start with low doses and titrate slowly to effect.
[b]Monitor for hyperthermia in cats.
[c]Give slowly to prevent histamine release.
[d]Buprenorphine is a better analgesic than morphine in cats.
BUN, Blood urea nitrogen; *Cr*, creatinine; *CRI*, constant-rate infusion; *EBL*, estimated blood loss; *ECG*, electrocardiogram; *$EtCO_2$*, end-tidal CO_2; *HCT*, hematocrit; *HR*, heart rate; *IM*, intramuscular; *IV*, intravenous; *MAP*, mean arterial pressure; *NSAID*, nonsteroidal antiinflammatory drug; *OTM*, oral transmucosal; *PRN*, as needed; *SpO_2*, hemoglobin saturation with oxygen; *TP*, total protein; *U/O*, urine output.

or glycopyrrolate) and an opiate. If the animal has minimal renal compromise, propofol or alfaxalone can be used for induction. Ketamine should be avoided in chronically ill dogs and cats with renal compromise. If a dog is severely depressed, hydromorphone plus diazepam (see Table 24.1) may allow intubation. If additional drugs are needed (see Table 24.1), a reduced dose of etomidate, propofol, or alfaxalone may be administered IV.

Water should not be withheld preoperatively in patients with renal disease, and careful attention should be paid to hydration status. IV fluids should be given to almost every animal with renal disease.

ANTIBIOTICS

Animals with renal calculi, ectopic ureters, or urinary tract obstruction may have concurrent infections and should be given appropriate antibiotics based on urine culture and susceptibility testing; alternatively, antibiotics can be withheld until appropriate intraoperative cultures have been taken. Potentially nephrotoxic antibiotics (i.e., aminoglycosides, tetracycline [except doxycycline], and sulfonamides) should be avoided. Penicillins and cephalosporins (e.g., ampicillin, amoxicillin, cefazolin, cephalexin; Box 24.2) are concentrated in urine. They are effective against most gram-positive organisms; cephalosporins also have an enhanced gram-negative spectrum. Fluoroquinolones (e.g., enrofloxacin; see Box 24.2) have broad activity against aerobic gram-negative bacteria. Drug doses or dosing frequency should be altered as required by the degree of renal compromise.

BOX 24.2 Selected Antibiotics of Use in Animals With Renal Disease[a]

Ampicillin
22 mg/kg IV, IM, SC q8h

Amoxicillin Plus Clavulanate (Clavamox)
Dogs: 12.5–25 mg/kg PO q12h
Cats: 62.5 mg PO q12h

Cefazolin (Ancef, Kefzol)
22 mg/kg IV or IM q8–12h

Cephalexin
22 mg/kg PO q8h

Enrofloxacin (Baytril)
5 mg/kg PO or IV q24h for simple UTI
Dogs: 7–20 mg/kg PO or IV q24h for pyelonephritis[a]
Cats: 5 mg/kg PO q24h; do not exceed 5 mg/kg/d due to risk of blindness

[a]If giving enrofloxacin IV, dilute 1:1 with saline and administer slowly over 20–30 minutes.
IM, Intramuscular; *IV*, intravenous; *PO*, orally; *SC*, subcutaneous; *UTI*, urinary tract infection.

SURGICAL ANATOMY

The kidneys lie in the retroperitoneal space lateral to the aorta and the caudal vena cava. They have a fibrous capsule and are held in position by subperitoneal connective tissue. The cranial pole of the right kidney lies at approximately the level of the 13th rib, opposite the first three lumbar vertebrae. The cranial one-third is covered by the caudate process of the caudate lobe of the liver. The cranial pole of the left kidney lies opposite the second to fourth lumbar vertebrae, farther caudal than the right kidney by approximately half a kidney length. The renal pelvis is the

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funnel-shaped structure that receives urine and directs it into the ureter. Generally, five or six diverticula curve outward from the renal pelvis. The renal artery normally bifurcates into dorsal and ventral branches; however, variations in the renal arteries and veins are common. The ureter begins at the renal pelvis and enters the dorsal surface of the bladder obliquely by means of two slitlike orifices. The blood supply to the ureter is provided from the cranial ureteral artery (from the renal artery) and the caudal ureteral artery (from the prostatic or vaginal artery).

Circumcaval ureters are an uncommon embryologic malformation resulting in dorsal displacement of the ureter to the caudal vena cava. Although thought to be a rare abnormality, recent evidence suggests that it is common in cats and may be associated with benign ureteral obstruction.[1,2]

NOTE The anatomy of the renal vasculature varies considerably, so use care when ligating these vessels during nephrectomy.

SURGICAL TECHNIQUE

To address the kidney, a ventral midline abdominal incision is performed from the xiphoid to caudal to the umbilicus. If the distal ureter is to be transected (i.e., nephrectomy), or if a cystotomy is necessary, the incision should extend to the pubis. Balfour retractors are used to retract the abdominal wall and expose the kidney. The entire abdominal contents should be inspected before the urinary tract is explored. The right kidney is exposed by elevating the duodenum and displacing the other loops of intestine toward the animal's left side. Similarly, the left kidney is exposed by elevating the mesocolon so that the small intestine is retracted to the animal's right side. The kidney can be isolated from the remaining abdominal contents with moistened laparotomy sponges.

Renal Biopsy

Renal biopsy may be indicated to determine a definitive diagnosis in dogs and cats with renal disease, or to provide an estimate of the severity or reversibility of renal damage. It may be performed at surgery, or it may be done percutaneously by ultrasonography, laparoscopy, or digitally using a keyhole abdominal incision. Ultrasound-guided and laparoscopic biopsies are the preferable percutaneous techniques. Renal biopsy should be avoided in patients with bleeding disorders, large intrarenal cysts, perirenal abscesses, severe hydronephrosis, and obstructive uropathy. Administering fluids before, during, and shortly after biopsy to initiate and maintain a mild diuresis may reduce formation of blood clots in the renal pelvis, which could cause obstruction and further loss of GFR. Spring-loaded biopsy needles, biopsy guns, or manual devices (e.g., Tru-Cut) may be used to obtain a sample, but manual devices are not recommended because they can easily produce fragmented tissue samples. Advantages of the spring-loaded devices are that they can be manipulated with one hand and often obtain better samples with less artifact, especially when guided by ultrasound. Sixteen-gauge needles are often used, but 18-gauge needles are typically adequate and may be easier to use in smaller patients. Attention to detail is critical; a good quality sample obtained with an 18-gauge needle is more useful than a fragmented sample from a 16-gauge needle. Needle biopsies are often inadequate when juvenile-onset renal disease (i.e., renal dysplasia) is evaluated because of nonuniform distribution of lesions; wedge biopsies are preferred in such cases.

NOTE It is extremely easy to obtain tissue samples that are nondiagnostic because they primarily consist of medullary tissue, are too small, have too few glomeruli, or have too many artifacts.

For patients with glomerular disease, at least two samples larger than 10 mm or three samples smaller than 10 mm are recommended. These samples should be divided and placed in appropriate solutions for light microscopy, immunofluorescence, and electron microscopy immediately after they are obtained. Consider submission of samples to experienced nephropathologists, a developing subspecialty, to obtain an accurate diagnosis in animals with glomerulonephropathies. One currently available center providing such services is the International Veterinary Renal Pathology Service, a joint collaboration of Texas A&M University and The Ohio State University. The minimum numbers of glomeruli considered necessary for accurate diagnosis of glomerular disease are as follows: 5 to 10 for light microscopy, 1 to 2 for electron microscopy, and 3 to 5 for immunofluorescence. The smaller number is adequate if all glomeruli are similar, but the larger number is needed if they are dissimilar (a common situation). For patients with AKI, two samples larger than 7 mm submitted in formalin for light microscopy is sufficient. Obtaining multiple biopsies of the kidney does not appear to produce more damage than a biopsy with only a single pass, provided that the biopsy needle remains in cortical tissue.

Ultrasound-Guided Percutaneous Biopsy

Ultrasound-guided percutaneous biopsies require heavy sedation or preferably general anesthesia. *Place the patient in ventrodorsal recumbency.* If generalized diffuse disease is suspected, as occurs with glomerular disease, the right kidney is the preferred side for a biopsy because it is technically easier to perform. However, either kidney may be sampled from this position. *Clip the hair over the biopsy site and aseptically prepare it. Place a sterile sleeve over the ultrasound probe. Using sterile ultrasound coupling gel, identify and examine the kidney. Once the site of entry for the biopsy has been determined, make a small stab incision in the skin using a No. 15 Bard-Parker scalpel blade.* The safest approach is to pass the biopsy instrument through the most lateral aspect of the kidney cortex; this is the area farthest from the major vasculature. *Insert the biopsy device (e.g., an 18- or 16-gauge, single spring–fired biopsy core needle [e.g., E-Z Core Single Action Biopsy Device] or an automatic spring-loaded biopsy gun [e.g., Bard Biopty or Monopty single-use spring-loaded disposable biopsy instrument]) through the nick in the skin, and advance it until it just penetrates the kidney capsule (Fig. 24.1A). Position the biopsy needle in such a way that a biopsy is taken of glomerular tissue in the renal cortex, rather than of medullary tissue (Fig. 24.1B). Place the tip of the needle through the renal capsule before activating it to prevent sliding of the needle along the capsule and to avoid tearing the capsule. Fire the biopsy device and retract the needle through the skin. Gently manipulate the sample from the biopsy needle and place it onto a glass slide with physiologic saline. Cut the sample into three sections; place one in 10% neutral buffered formalin solution for light microscopy, one in glutaraldehyde for electron microscopy, and freeze one for immunofluorescence.*

NOTE Do not place the biopsy needle too deep in the kidney or too few glomeruli may be obtained in the sample. The risk for transection of a large arcuate vessel is also present (see Fig. 24.1C).

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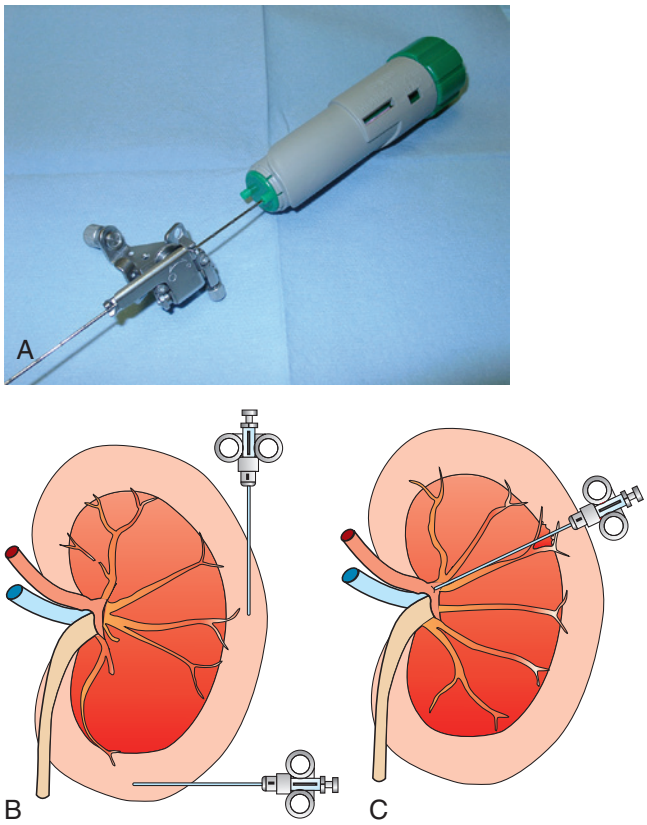


FIG. 24.1 (A) Monopty biopsy needle (Bard Medical) with biopsy guide attached. (B) To obtain a biopsy of the kidney, advance the biopsy instrument until it penetrates the capsule. Position the biopsy needle to take a biopsy of the renal cortex. (C) Do not advance too deeply to avoid transecting the large arcuate vessel.

Laparoscopically-Guided Biopsy

Laparoscopy for renal biopsy requires general anesthesia. Single-cannula laparoscopy is sufficient for insufflation and visualization; however, double cannulas will provide an instrument portal to facilitate manipulation of the viscera and stabilization of the kidney during biopsy.

Using a No. 15 Bard-Parker scalpel blade, make a third small skin incision for penetration of a 14-, 16-, or 18-gauge core biopsy needle. Use the manipulation instrument to position and secure the kidney, and place a double spring–fired biopsy needle against the renal capsule and direct it tangentially before discharge. After the biopsy, withdraw the needle and apply pressure via the manipulating instruments. Be sure the samples consist of cortical tissue and are of sufficient length, then process as described previously.

A 5-mm cup biopsy instrument is an alternative to a core biopsy needle for laparoscopic renal biopsy, and has been shown to have minimal risk and may yield more glomeruli than a 16-gauge biopsy needle in healthy dogs.[3]

Surgical Biopsy

Surgical biopsies may be performed using an automatic biopsy instrument (see previous discussion), a manual device (Tru-Cut or Franklin modified Vim-Silverman biopsy needle), or a wedge resection with a scalpel blade (Fig. 24.2). Spring-loaded devices are recommended because they are easier to use than manual devices, can be manipulated with one hand, and often produce superior tissue samples. Wedge resection allows a larger sample to be obtained than can be obtained with the use of needles or guns. With either technique, it is important to ensure that adequate amounts of cortical tissue are obtained.

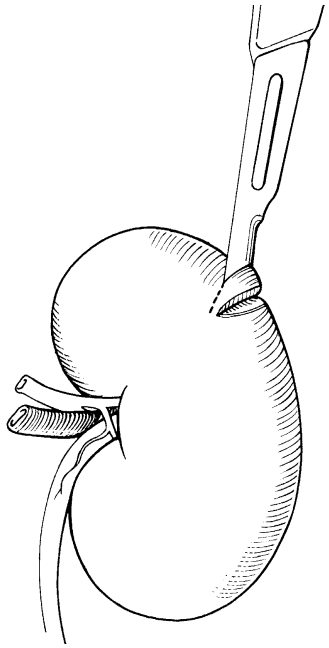


FIG. 24.2 Wedge biopsies provide larger samples than needle biopsies.

Needle Biopsy

Perform a needle biopsy with a Tru-Cut-type instrument by placing the tip of the instrument on the kidney capsule with the obturator specimen rod fully retracted within the outer cannula. Position the biopsy needle as described previously (see Fig. 24.1). Push the specimen rod into the lesion by advancing the plastic handle or by triggering the firing mechanism. With manual devices, advance the outer sheath of the needle into the tissue to sever the biopsy sample. Withdraw the needle with the outer sheath over the specimen rod. Apply digital pressure to the site to control hemorrhage. Be sure the sample is long enough (see previous discussion) and primarily consists of cortical tissue. Process the sample as described previously.

NOTE Avoid taking a biopsy near the renal pelvis to prevent extravasation of urine. To make sure that you get a diagnostic sample, position the biopsy device across the cortex, as indicated previously.

Wedge Biopsy

For a wedge biopsy, make an incision into the renal parenchyma with a No. 11 or a No. 15 scalpel blade. Make another incision at an angle to the first incision to remove a wedge-shaped piece of parenchyma (see Fig. 24.2) and apply digital pressure. Be sure to include cortex in the sample. Close the incision with a mattress suture of 3-0 or 4-0 absorbable suture material across the capsule.

Nephrectomy

Nephrectomy (ureteronephrectomy) is indicated for renal neoplasia, uncontrollable renal hemorrhage, persistent urine

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leakage, pyelonephritis resistant to medical therapy (e.g., associated with nephroliths), hydronephrosis, and ureteral abnormalities that defy surgical repair (e.g., avulsion, stricture, rupture, obstruction due to calculi). Before nephrectomy, renal function in the opposite kidney should be assessed by determining the GFR through administration of an exogenous marker or by nuclear scintigraphy. Bilateral renal dysfunction may warrant a guarded prognosis. If renal neoplasia is suspected, radiography or computed tomography (CT) (thoracic and abdominal) and ultrasonography should be used to help rule out metastasis, including to the opposite kidney.

NOTE To avoid unintentional transection, the opposite ureter should always be identified before the affected kidney is removed; this is particularly important when large neoplasms are removed.

Grasp the peritoneum over the kidney and incise it. Using a combination of blunt and sharp dissection, free the kidney from its sublumbar attachments. Elevate the kidney and retract it medially to locate the renal artery and vein on the dorsal surface of the renal hilus (Fig. 24.3). Identify all branches of the renal artery. Double ligate the renal artery with absorbable suture (e.g., polydioxanone, polyglyconate, glycomer 631, poliglecaprone 25) or nonabsorbable suture (e.g., cardiovascular silk) close to the abdominal aorta to ensure that all branches have been ligated. Consider placement of a transfixation suture if the artery is larger than 3 to 4 mm in diameter. Identify the renal vein and ligate it similarly. The left ovarian and testicular veins drain into the renal vein and should not be ligated in intact dogs. Avoid ligating the renal artery and vein together to prevent the formation of an arteriovenous fistula. *Ligate the ureter near the bladder with a simple encircling ligature. Remove the kidney and ureter and, after procuring appropriate culture specimens, submit them for histologic examination.*

Transperitoneal laparoscopic ureteronephrectomy has been reported in nine dogs for hydronephrosis, renal dysplasia, and renal masses; conversion to open laparotomy was required in two cases due to either uncontrollable hemorrhage or poor anatomic visualization of the distal ureter.[4]

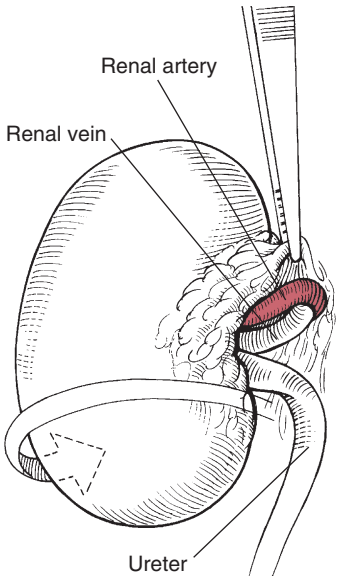


FIG. 24.3 During nephrectomy, elevate the kidney and retract it medially to locate the renal artery and vein on the dorsal surface of the renal hilus.

Partial Nephrectomy

Partial nephrectomy is occasionally warranted for focal renal lesions, particularly if preservation of renal function is necessary because of bilateral renal dysfunction. However, in most cases total nephrectomy is usually easier and carries less risk of postoperative hemorrhage. If partial nephrectomy is performed, electrocoagulation of bleeding vessels should be avoided because it causes excessive parenchymal damage. Avoid partial nephrectomy in animals with clinically significant coagulopathies; excessive blood loss may occur after this procedure.

If possible, strip the renal capsule from the area of the kidney to be excised. Use absorbable suture (No. 0 or 1) with two long, straight needles attached. Thread the needles into the kidney at the proposed resection site (Fig. 24.4A–B). Tie the thread into three separate ligatures, but avoid damaging the renal vessels or ureter (Fig. 24.4C). Excise the renal tissue distal to these ligatures. Ligate any bleeders and suture the exposed diverticula with absorbable suture (2-0 or 3-0). Approximate the capsule over the end of the kidney (Fig. 24.4D), and anchor it to the sublumbar tissues to prevent rotation of the kidney. As an alternative, clamp the renal vessels with vascular forceps and excise the kidney parenchyma. Ligate the parenchymal vessels and close the renal pelvis and diverticula. Suture the capsule as described previously and remove the clamps from the renal vessels.

Nephrotomy

Nephrotomy is occasionally performed to remove calculi (see p. 672) lodged in the renal pelvis, but it may also be performed to explore the renal pelvis for neoplasia or hematuria. Nephrotomy should be avoided in patients with severe hydronephrosis because ample parenchyma may not be available to prevent postoperative urine leakage. In addition, nephrotomy may temporarily diminish renal function by 25% to 50%. Although bilateral nephrotomies can be performed, this could precipitate AKI if renal function is sufficiently compromised preoperatively. Staged procedures are indicated in such patients. Nephrotomy may be performed by bisecting the kidney or by using an intersegmental approach whereby the plane of dissection follows the terminal branches of the posterior and anterior renal arteries. The interlobar arteries are not transected; this theoretically minimizes nephron destruction. Neither technique affects GFR in normal dogs, but because the bisection approach requires less surgical manipulation and time, it is the preferred technique.

Nephrotomy incisions may be closed without sutures or with transparenchymal horizontal mattress sutures. The latter may cause increased vascular strangulation, pressure necrosis, infarction, and postoperative hemorrhage. Cyanoacrylate adhesive provides rapid hemostasis; however, if the adhesive enters the renal diverticula, calculi may form.

Locate the renal vessels and temporarily occlude them with vascular forceps, a tourniquet, or an assistant's fingers. Mobilize the kidney to expose the convex lateral surface. Make a sharp incision along the midline of the convex border of the kidney capsule, then bluntly dissect through the renal parenchyma, ligating renal vessels as necessary (Fig. 24.5). Culture the renal pelvis. Remove the calculi and flush the kidney with warm saline or lactated Ringer's solution. Assess the ureter for patency by placing a 3.5-Fr soft rubber catheter down the ureter and flushing it with warm fluids. Close the nephrotomy by apposing the cut tissues and applying digital pressure

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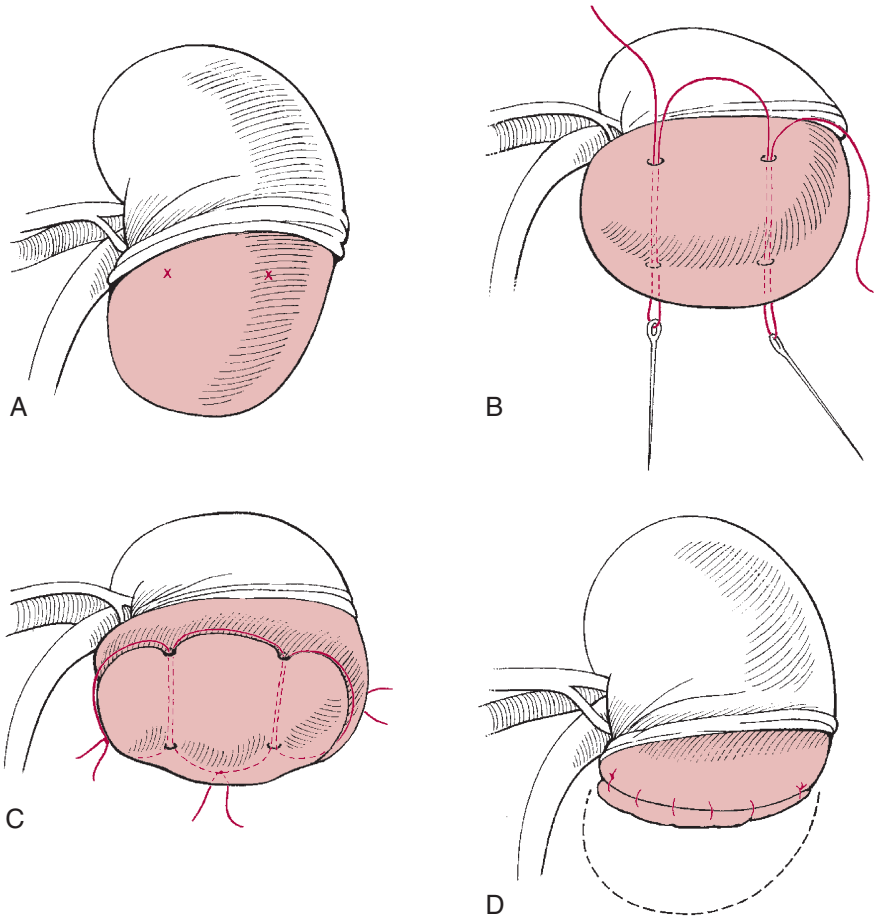


FIG. 24.4 For partial nephrectomy, use large, absorbable suture with two long, straight needles attached. (A–B) Thread the needles into the kidney at the proposed resection site. (C) Tie the thread into three separate ligatures, but avoid damaging the renal vessels or ureter. Excise the renal tissue distal to these ligatures. (D) To prevent rotation of the kidney, approximate the capsule over the end of the kidney and anchor it to the sublumbar tissue.

for approximately 5 minutes while restoring blood flow through the renal vessels (sutureless technique). As an alternative, appose the capsule with a continuous pattern of absorbable suture material (see Fig. 24.5). If adequate hemostasis is not achieved, or if urine leakage is a concern, place absorbable sutures through the cortex in a horizontal mattress fashion (see previous comments and Fig. 24.5). Then, suture the capsule in a continuous pattern with absorbable suture. Replace the kidney in its original location. Sutures may be placed in the peritoneum where the kidney was elevated to help stabilize it.

Pyelolithotomy

Pyelolithotomy may be performed to remove renal calculi if the proximal ureter and the renal pelvis are sufficiently dilated. This procedure prevents trauma to the renal parenchyma associated with nephrotomy. Pyelolithotomy is extremely difficult if the ureter is not dilated. *Dissect the kidney from its sublumbar attachments and expose the dorsal surface. Identify the ureter and renal vessels (Fig. 24.6A). Make an incision over the dilated pelvis and proximal ureter and remove the calculi (Fig. 24.6B). Flush the renal pelvis and diverticula with warm saline to remove small debris. Next, flush the ureter to ensure its patency. Close the incision in a continuous pattern with 5-0 or 6-0 absorbable suture (Fig. 24.6C).*

Ureterotomy

Ureterotomy is occasionally performed to remove obstructive calculi. The procedure carries the risk of postoperative leakage and stricture formation and should be performed with care. If obstruction is not present, dietary dissolution of struvite calculi may be attempted. However, removal of calculi is indicated if obstruction occurs or seems likely (e.g., hydroureter, hydronephrosis). Depending on the animal's size, removal of the stones with a ureteroscope may be possible. Some stones in the distal ureter may be flushed or pulled into the bladder through a cystotomy, making a ureterotomy unnecessary. Although ureteral mucosa regenerates over a stent if the mucosa has not been completely disrupted, the use of stenting catheters is controversial because they may promote stricture formation and infection. If stents are used, they should be smaller than the diameter of the ureter. In some animals, ureteral stents may be placed so that they exit the urethral orifice and are sutured to the exterior. Transverse or longitudinal incisions may be made in the ureter; however, tension on transverse ureterotomies may be less, and therefore they may heal more readily.

Make a transverse or longitudinal incision in the dilated ureter proximal to the calculi and remove them (Fig. 24.7A and Video 24.1). Place a small, soft rubber catheter into the ureter proximal and distal to the incision, and flush the ureter with warm fluid.

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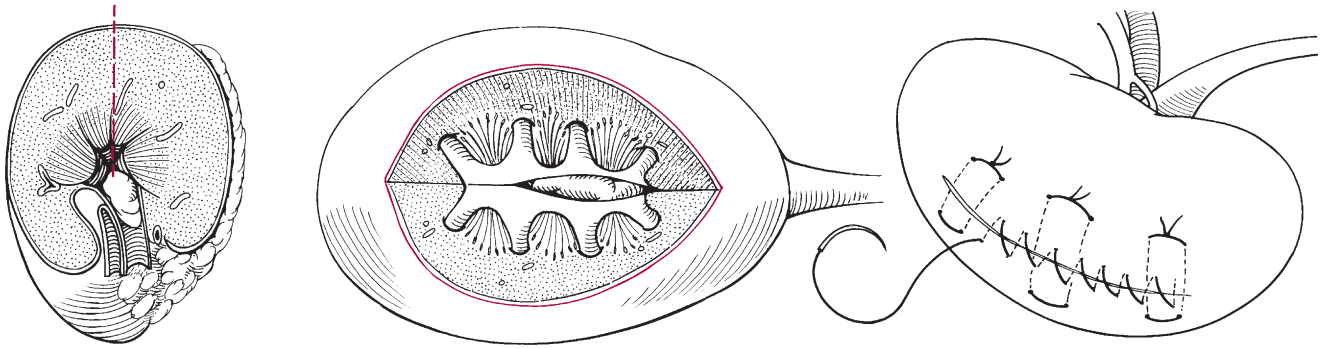


FIG. 24.5 Nephrotomy is usually performed to remove calculi lodged in the renal pelvis. Make a sharp incision along the capsule of the convex border of the kidney, and bluntly dissect the renal parenchyma to the renal pelvis. Remove the calculi and close the nephrotomy by apposing the cut tissue and suturing the capsule in a continuous pattern with absorbable suture material (see text for sutureless technique). If adequate hemostasis is not achieved, or if urine leakage is a concern, place absorbable sutures through the cortex in a horizontal mattress fashion. Then suture the capsule in a continuous pattern with absorbable suture material.

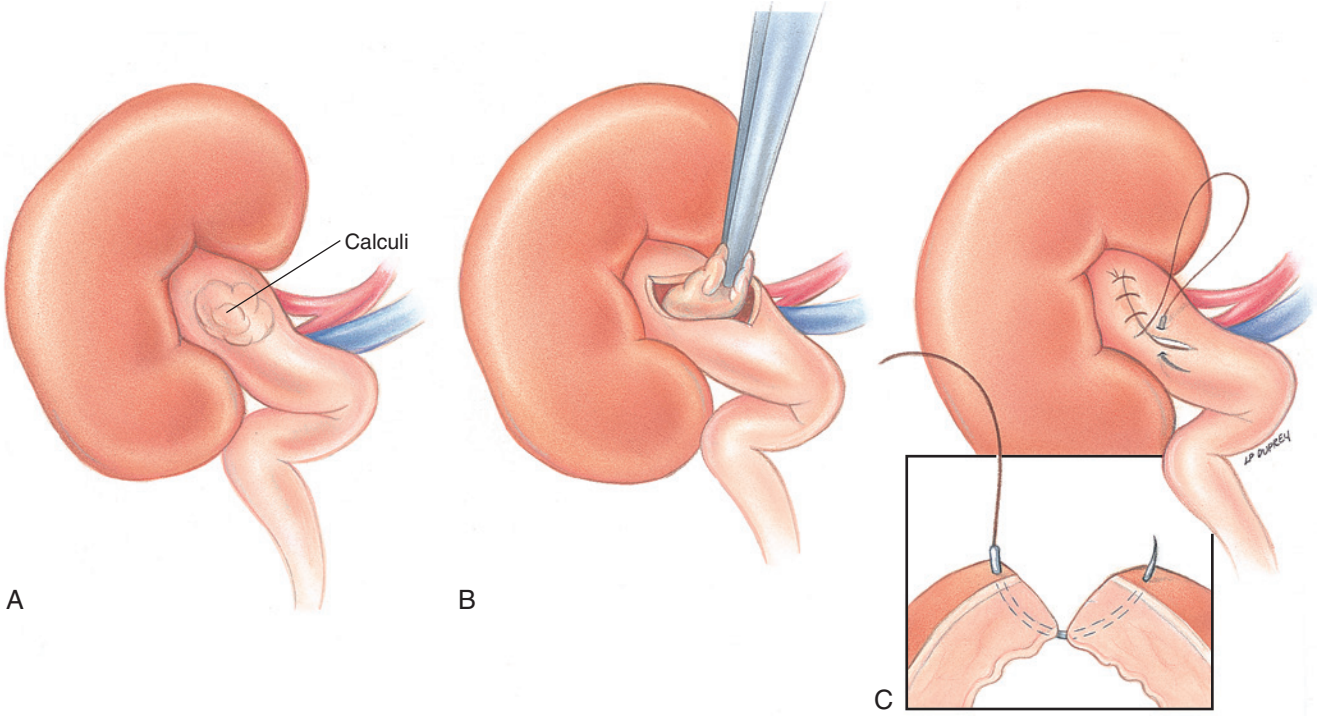


FIG. 24.6 Pyelolithotomy may be performed when the proximal ureter is dilated. (A) Expose the dorsal surface of the kidney and identify the ureter and renal vessels. (B) Make an incision over the dilated pelvis and proximal ureter and remove the calculi. (C) Close the incision in a continuous pattern with absorbable suture.

Make sure that all calculi have been removed and that the ureter is patent. Close the incision in a simple interrupted pattern with 5-0 to 7-0 absorbable suture (see Fig. 24.7B). As an alternative, if the ureter is not dilated and if stricture formation seems likely, make a longitudinal incision over the calculi and close the incision in a transverse fashion (see Fig. 24.7C). If the ureter has been damaged, perform a resection and anastomosis (see p. 659) or a proximal urinary diversion via a nephropyelostomy tube (see p. 660).

NOTE Ureterotomy, ureteral reimplantation, and ureteral anastomosis are greatly facilitated by wearing loupes for magnification or by using an operating microscope.

Ureteral Reimplantation

The diameter of the feline ureter is approximately 0.4 mm at the level of the bladder, and standard ureteroneocystostomy techniques often cause ureteral obstruction. Microsurgical

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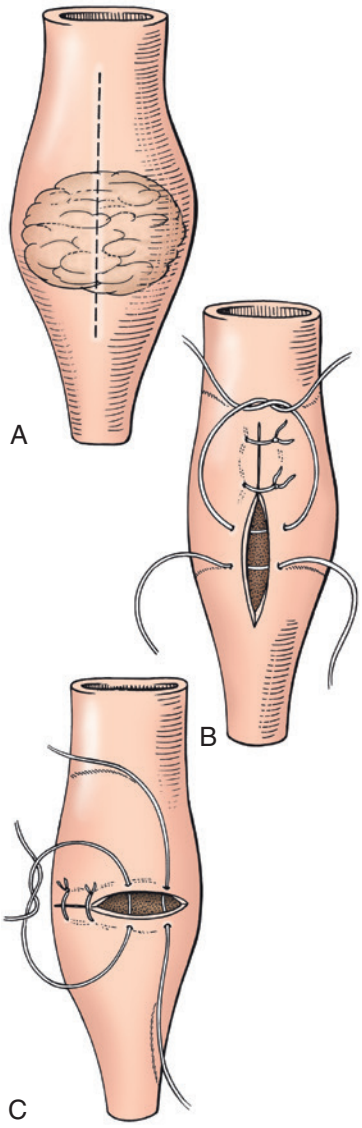


FIG. 24.7 Ureterotomy is occasionally performed to remove obstructive calculi. (A) Make a transverse or longitudinal incision in the dilated ureter proximal to the calculi and remove them. (B) Close the incision in a simple interrupted pattern with absorbable suture. (C) As an alternative, make a longitudinal incision over the calculi and close the incision in a transverse fashion with absorbable suture.

techniques may be necessary to prevent ureteral obstruction in cats, particularly when the ureter is not dilated owing to disease (e.g., renal transplantation). A "drop-in" technique was originally described for ureteroneocystostomy of cats undergoing renal transplantation; however, granuloma formation and hemorrhage were common. Extravesical techniques have largely replaced intravesical techniques in people. Studies in cats have suggested that an extravesical technique using a simple interrupted suture pattern is preferred over an extravesical technique using a continuous suture pattern or an intravesical mucosal apposition technique. Although all three techniques resulted in renal pelvis dilation, the dilation resolved more rapidly with the extravesical technique using a simple interrupted pattern, and this technique was associated with consistently lower serum creatinine concentrations during the first postoperative week.

Another technique for ureteroneocystostomy in cats undergoing renal transplantation is ureteral papilla harvest and transplantation.[5] In this technique, the entire ureteral papilla, including a 2- to 3-mm cuff of the bladder wall, is harvested from the donor and implanted in the recipient. Proposed benefits of this technique are that the anastomosis is less technically challenging and that there is decreased risk of ureteral obstruction and urine leakage.

To perform an extravesical simple interrupted suture pattern technique, make a partial-thickness incision through the muscularis and submucosa of the ventral aspect of the apex of the urinary bladder to expose the mucosa (Fig. 24.8A). Spatulate the distal end of the ureter, and make an incision equal in length to the spatulation incision in the ureter through the bladder mucosa in the caudal aspect of the muscularis incision. Place a simple interrupted suture (6-0 or 8-0 nylon) between the proximal ureter at the end of the spatulation and the cranial aspect of the bladder mucosal incision, and a second interrupted suture between the distal end of the ureter and the caudal aspect of the bladder mucosal incision (Fig. 24.8B). Place a 4-0 polypropylene stent into the lumen of the ureter to ensure patency. Place the stent after the first two sutures are tied, and remove it before tying the final sutures of the mucosal layer. Place two additional interrupted sutures between the ureteral and bladder mucosae on one side of the incision (Fig. 24.8C). Close the seromuscular incision (Fig. 24.8D).

Ureteral Anastomosis

AP Ureteral anastomosis is technically difficult in small patients (i.e., small dogs and cats) and has a high rate of postoperative obstruction. If the ureter is transected or damaged near the bladder, ureteroneocystostomy may be performed (see p. 667). If the ureter is avulsed from the renal pelvis, urinary drainage can be performed by placing a catheter through the renal parenchyma into the ureter (Fig. 24.9) with the end of the catheter exteriorized through the body wall. However, if function in the contralateral kidney is adequate, nephrectomy appears to result in less morbidity and death than ureteral repair and may be considered to prevent leakage, stricture, or infection. In preparation for ureteral anastomosis, minimal dissection should be done around the ureter to prevent compromising its blood supply. Stay sutures should be placed for manipulation, and traumatic forceps should be avoided to prevent damaging the ureter. The amount of tension that can be placed on the ureter without causing stricture formation is unknown; therefore tension across the anastomotic site should be avoided. Various synthetic materials have been used to replace the ureter, but most are unacceptable because they promote fibrosis, formation of calculus, and/or infection. A bladder-flap ureteroplasty has been described for ureteral trauma near the bladder (Fig. 24.10). With this technique, a flap is elevated from the ventral surface of the bladder and the ureter is reimplanted into the flap. The flap is then closed as a tube. As with ureterotomy, stenting catheters should be used with caution because they may promote stricture formation.

For ureteral anastomosis, suture the ureter directly or spatulate it by making a longitudinal incision on opposite sides of each end of the ureter (Fig. 24.11A). Preplace absorbable monofilament sutures (6-0 to 8-0) at the apex of the spatulated incisions and align the ureteral ends (Fig. 24.11B). Appose the ureteral ends in a simple interrupted pattern using the preplaced sutures. Close

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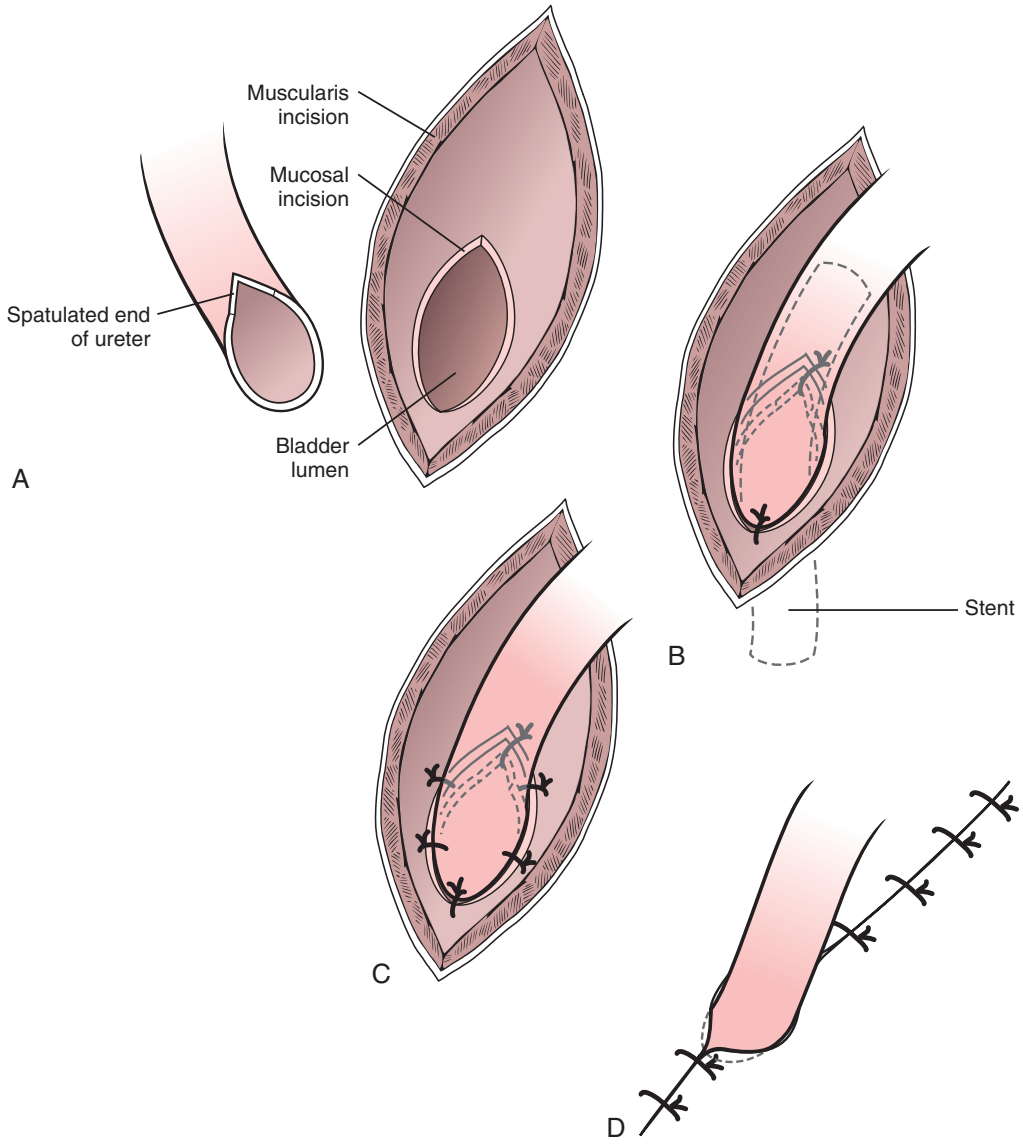


FIG. 24.8 To perform an extravesical simple interrupted suture pattern technique, (A) make a partial thickness incision through the muscularis and submucosa of the ventral aspect of the apex of the urinary bladder to expose the mucosa. Spatulate the distal end of the ureter and make an incision equal in length to the spatulation incision in the ureter through the bladder mucosa in the caudal aspect of the muscularis incision. (B) Place a simple interrupted suture (6-0 or 8-0 nylon) between the proximal ureter at the end of the spatulation and the cranial aspect of the bladder mucosal incision, and a second interrupted suture between the distal end of the ureter and the caudal aspect of the bladder mucosal incision. Place a 4-0 polypropylene stent into the lumen of the ureter to ensure patency. Place the stent after the first two sutures are tied, and remove it before tying the final sutures of the mucosal layer. (C) Place two additional interrupted sutures between the ureteral and the bladder mucosa on one side of the incision. (D) Close the seromuscular incision.

the remainder of the ureter with simple interrupted sutures (Fig. 24.11C). Ensure that the ends of the ureter are not twisted and that sufficient sutures have been placed to prevent leakage (Fig. 24.11D).

Tension on the anastomosis can be relieved by freeing up the kidney from its peritoneal attachments and repositioning it farther caudally. The kidney is then pexied to the body wall to prevent renal torsion. Alternatively, the apex of the bladder can be moved cranially and laterally toward the kidney and sutured to the psoas muscle dorsally. If necessary, place a nephrostomy tube.

Nephrostomy Tube Placement

Nephrostomy tubes may be placed percutaneously (see p. 671) or at surgery. Nephrostomy tubes are prone to poor drainage,

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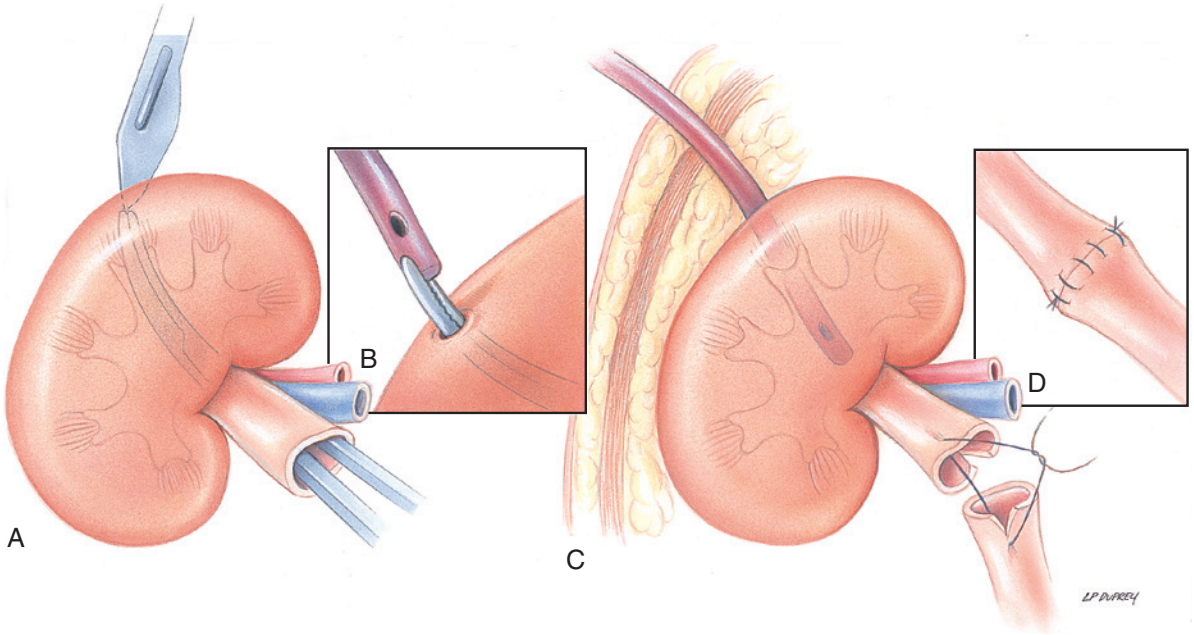


FIG. 24.9 Urinary drainage can be performed by placing a catheter through the renal parenchyma into the renal pelvis. (A) Place a hemostat into the renal pelvis via the ureter and incise over its tip with a scalpel blade. (B) Grasp a catheter with the hemostat. (C) Pull the catheter into the renal pelvis. (D) Anastomose the torn ureteral ends.

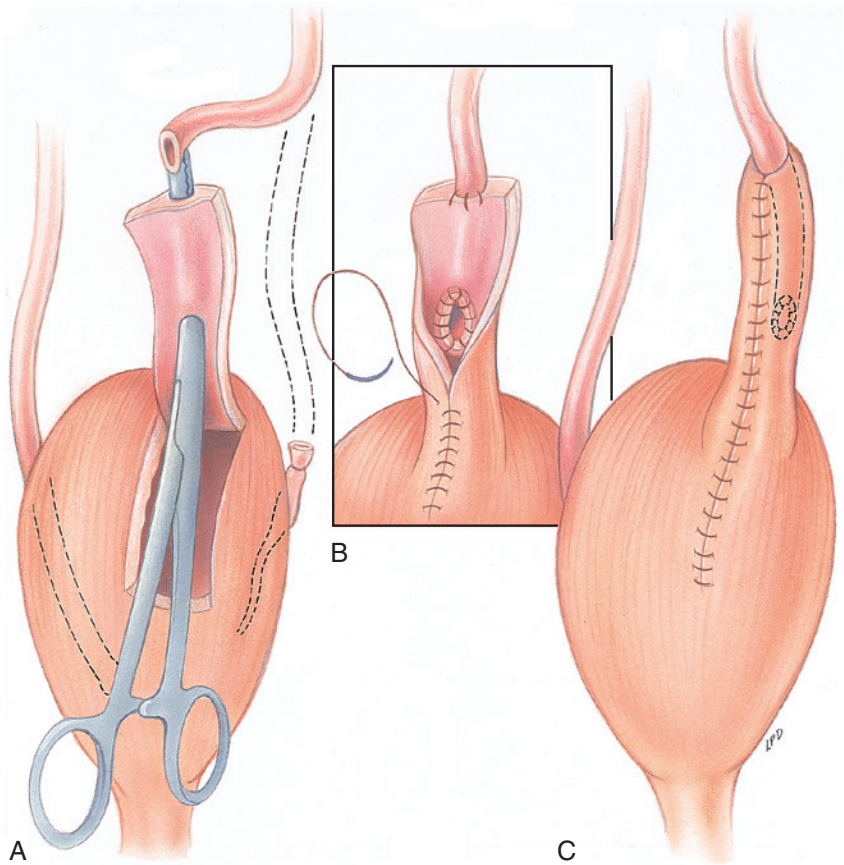


FIG. 24.10 A bladder-flap ureteroplasty may be performed when ureteral trauma occurs near the bladder. (A) Elevate a flap from the ventral surface of the bladder. (B) Reimplant the ureter into the flap. (C) Close the flap as a tube.

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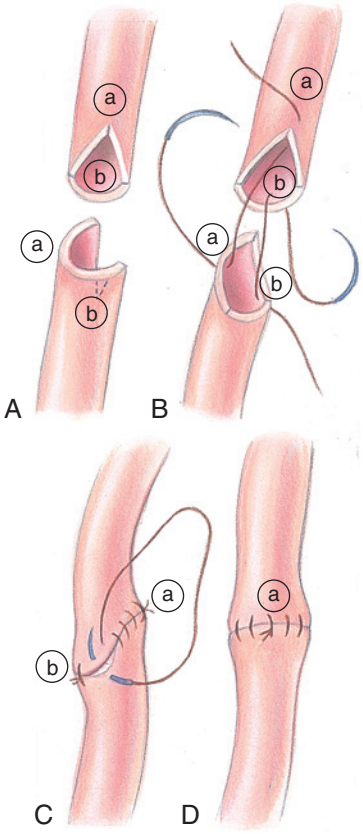


FIG. 24.11 For ureteral anastomosis, suture the ureter directly or (A) spatulate it by making a longitudinal incision on opposite sides of each end of the ureter. (B) Preplace absorbable sutures at the apex of the spatulated incisions and align the ureteral ends. Appose the ureteral ends with simple interrupted absorbable sutures using the preplaced sutures. (C–D) Close the remainder of the ureter with simple interrupted sutures.

tube dislodgment, and urine leakage; thus their use should be reserved for cases in which urine leakage is considered highly likely after ureterotomy or ureteral anastomosis. A tube specifically designed for use as a nephrostomy tube that has a flanged or pigtail end will likely stay in the renal pelvis better than a red rubber tube (locking-loop pigtail catheter).

After proximal ureterotomy, insert a 20-gauge over-the-needle IV catheter through the ureterotomy incision, and advance it through the renal pelvis. Alternatively, insert a small hemostat and incise over the tip with a scalpel blade (see Fig. 24.9A). Exit the catheter or hemostat through the renal cortex. Tie a length of suture material to a 5-Fr red rubber or Foley catheter and pass the other end of the suture through the IV catheter. Slowly withdraw the catheter and suture, pulling the tip of the nephrostomy tube into the ureterotomy incision. If using a hemostat, grasp the end of the tube and pull it into the renal cortex (see Fig. 24.9B–C). Cut the suture and position the tip of the catheter in the renal pelvis. Perform a nephropexy by placing sutures between the renal capsule and the body wall where the tube exits. Secure the tube to the skin and underlying musculature, and connect it to a closed drainage system.

Alternatively, use a nephrostomy tube with a flanged or pigtail end. Place the catheter using the dilator and stylet supplied in the kit. Secure as described previously.

Subcutaneous Ureteral Bypass System

A subcutaneous ureteral bypass (SUB) device is a permanent, implantable system designed to circumvent ureteral obstruction in cats and dogs (see p. 673).

Neoureterostomy

Neoureterostomy (see p. 667) is performed for intramural ectopic ureters. Although some ectopic ureters completely bypass the bladder, most travel under the bladder mucosa before exiting and opening into the urethra or vagina.

Ureteroneocystostomy

Ureteroneocystostomy is performed for extraluminal ectopic ureters and to repair ureters that are damaged near the bladder. The ureter is resected or debrided and is reimplanted into the bladder lumen (see p. 667).

HEALING OF THE KIDNEY AND URETER

Mild contusions or fractures of the kidney parenchyma heal primarily by synthesis of fibrous connective tissue. Although scar production occurs and may obliterate some functional nephrons, wound contraction is usually minimal. However, renal pelvis and collecting ducts may undergo wound contraction and scar tissue formation, resulting in strictures. Uroepithelium has enormous proliferative potential and may seal a damaged area within 48 hours. If at least 50% of the ureteral circumference remains, the ureter will heal by epithelialization, fibrous connective tissue synthesis, and longitudinal rather than circumferential wound contraction. Peristalsis is absent in the distal segment of a transected ureter for at least 10 days after repair. This may promote hydroureter in the proximal segment and subsequent hydronephrosis. Immobilizing the ureter to surrounding structures also inhibits peristalsis and diminishes urine flow. Ureteral defects measuring 5 cm will heal over a stent; however, the ureter is typically narrow, and the wall is composed of fibrous tissue.

NOTE The uroepithelium has enormous regenerative capacity, but improper technique may result in strictures.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Absorbable suture material, such as polydioxanone (PDS), polyglyconate (Maxon), poliglecaprone 25 (Monocryl), or glycomer 631 (Biosyn), should be used in the kidney, ureter, and bladder. Nonabsorbable suture material may promote formation of calculus and infection. Although PDS and Maxon maintain tensile strength and are more slowly absorbed than is desirable for most urinary surgery, they have less tissue drag than multifilament suture. The use of pediatric or ophthalmic instruments facilitates surgery of the ureter. These instruments tend to be smaller and more delicate and may cause less tissue trauma than larger instruments. Surgical loupes or an operating microscope is recommended for ureteral surgery, particularly in cats and small dogs.

POSTOPERATIVE CARE AND ASSESSMENT

The hematocrit should be monitored postoperatively and ultrasound-guided abdominocentesis performed if hemorrhage

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or leakage is suspected. Alternatively, a peritoneal drain can be placed during surgery to facilitate monitoring of abdominal fluid. Significant hemorrhage may require blood transfusions (see Box 4.1) or repeat surgery. Severely anemic animals should have nasal oxygen during the anesthetic recovery period. Central venous pressure and urine output may be monitored to evaluate hydration postoperatively. Indwelling urinary catheters allow measurement of urine output. Patients should be closely monitored for urethral obstruction after repair of ectopic ureters or after ureteroneocystostomy performed for other reasons. Ureteral obstruction may occur as a result of surgical swelling or stomal stenosis; however, unless the surgery was performed bilaterally, this typically goes undetected unless abdominal radiographs or ultrasonography documents significant hydroureter or hydronephrosis. Urinary leakage may be diagnosed by abdominocentesis and subsequent comparison of fluid and serum creatinine and potassium levels. With uroperitoneum, creatinine and potassium levels in the abdominal fluid are higher than serum levels (see p. 699). Electrolyte and acid-base abnormalities should be monitored and corrected postoperatively. Postoperative analgesics should be given as necessary (see Table 13.2).

COMPLICATIONS

The major complications of surgery of the kidney are renal failure, hemorrhage, and urinary leakage. Urinary leakage or obstruction due to stenosis or stricture is common after ureteral surgery. Complications of renal biopsy are not typically life-threatening but have varied in frequency from 1% to 18%, with severe hemorrhage being most common. Older patients, patients weighing less than 5 kg, and patients with severe azotemia may be more likely to have complications. Other complications include microscopic hematuria, gross hematuria, arteriovenous fistula formation, cyst formation, perirenal hematoma, intrarenal hematoma, lacerated renal artery or vein, intraabdominal hemorrhage caused by laceration of an organ or vessel, infarction or thrombosis, infection, scar formation and fibrosis, or hydronephrosis that occurs secondary to the formation of blood clots in the renal pelvis. Severe hemorrhage requiring blood transfusion may occur in some animals.

SPECIAL AGE CONSIDERATIONS

Older animals often have some degree of renal compromise and require careful monitoring during any surgical procedure. Hypotension should be avoided during surgery and the postoperative period to prevent further renal damage. If cardiac disease is also present, fluids should be used judiciously to prevent overhydration while renal blood flow is maintained.

SPECIFIC DISEASES

ECTOPIC URETER

DEFINITIONS

Ectopic ureter, or *ureteral ectopia,* is a congenital anomaly in which one or both ureters empty outside the bladder. **Extraluminal (extramural) ectopic ureters** are those that completely bypass the bladder; **intraluminal (intramural) ectopic ureters** course submucosally in the bladder to open in the urethra or vagina. **Ureteroceles** are focal, cystic dilations of the distal ureter that may be ectopic or intravesical (orthotopic).

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The ureter normally enters the dorsolateral caudal surface of the bladder and empties into the trigone after a short intramural course (Fig. 24.12). Ectopic ureters are the most common congenital cause of urinary incontinence in dogs and result from abnormal positioning of the metanephric duct along the mesonephric duct in utero. The trigger for this abnormal development is unknown. Abnormalities in embryogenesis of the urinary system may also cause associated abnormalities (e.g., urethral sphincter incompetence, bladder hypoplasia, renal agenesis, renal aplasia, renal dysplasia, hydroureter, vestibulovaginal abnormalities, and/or ureteroceles; Fig. 24.13). Anatomic correction of

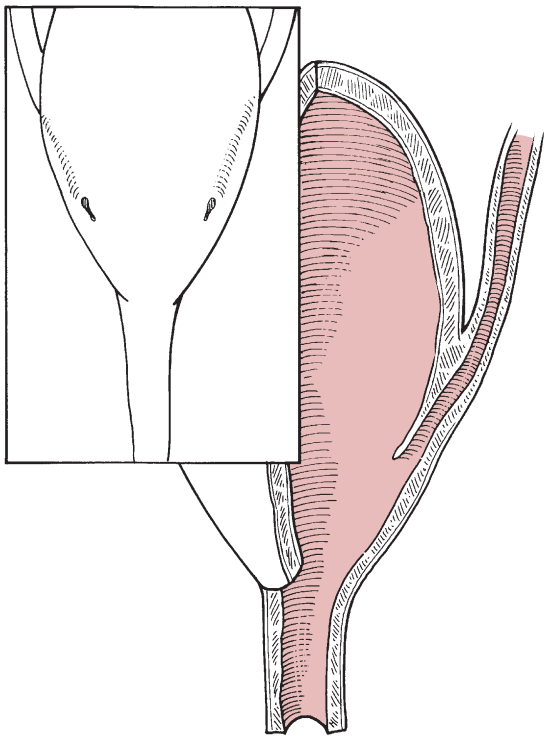


FIG. 24.12 The ureters normally enter the dorsolateral caudal surface of the bladder and empty into the trigone after a short intramural course.

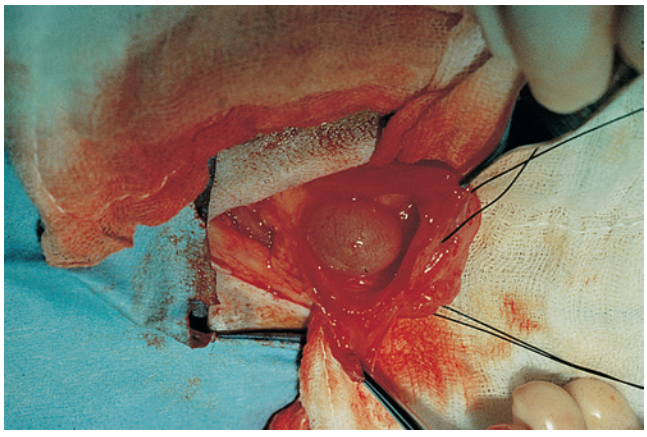


FIG. 24.13 A ureterocele in the bladder of a dog. The contralateral ureter was ectopic.

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ureteral ectopia is recommended; however, the presence of other abnormalities increases the likelihood of continued postoperative incontinence.

The most common location for termination of ectopic ureters is in the urethra, although termination in the uterus and vagina can occur. Ectopic ureters are classified as *intramural* (the ureter enters the bladder wall at a normal anatomic position, but a portion of the ureter extends submucosally within the bladder wall before it enters the urethral lumen; Fig. 24.14A) or *extramural* (the ureter bypasses the bladder to enter the urethral lumen; Fig. 24.14B). Bilateral ectopic ureters occur in more than one-third of dogs (some reports have suggested more than 90%). Other abnormalities noted in some dogs include double ureteral openings (i.e., where the ureter opens in the bladder plus more distally; Fig. 24.14C) and ureteral troughs (Fig. 24.14D). Ureteral ectopia is much less common in cats.

Upper and lower urinary tract infections (UTIs) are common in dogs with ureteral ectopia. Small kidneys may be caused by end-stage pyelonephritis, congenital dysplasia, or congenital cystic disease. Hydronephrosis may be caused by chronic pyelonephritis or ureteral obstruction (e.g., stenosis or absence of a functional opening). Hydroureter, the most common urogenital abnormality in dogs with ureteral ectopia, may be caused by chronic infection, obstructed urine outflow, or primary lack of ureteral peristalsis. Hypoplastic bladders or intrapelvic bladders may be congenital or may result from lack of normal filling of the bladder. With unilateral ectopia, hydroureter and hydronephrosis may occur in the contralateral ureter as a result of chronic ascending UTIs.

DIAGNOSIS

Clinical Presentation

Signalment

Ectopic ureters are more commonly diagnosed in female than in male dogs. Male dogs are also affected but may be less commonly diagnosed because the opening of the ectopic ureter is

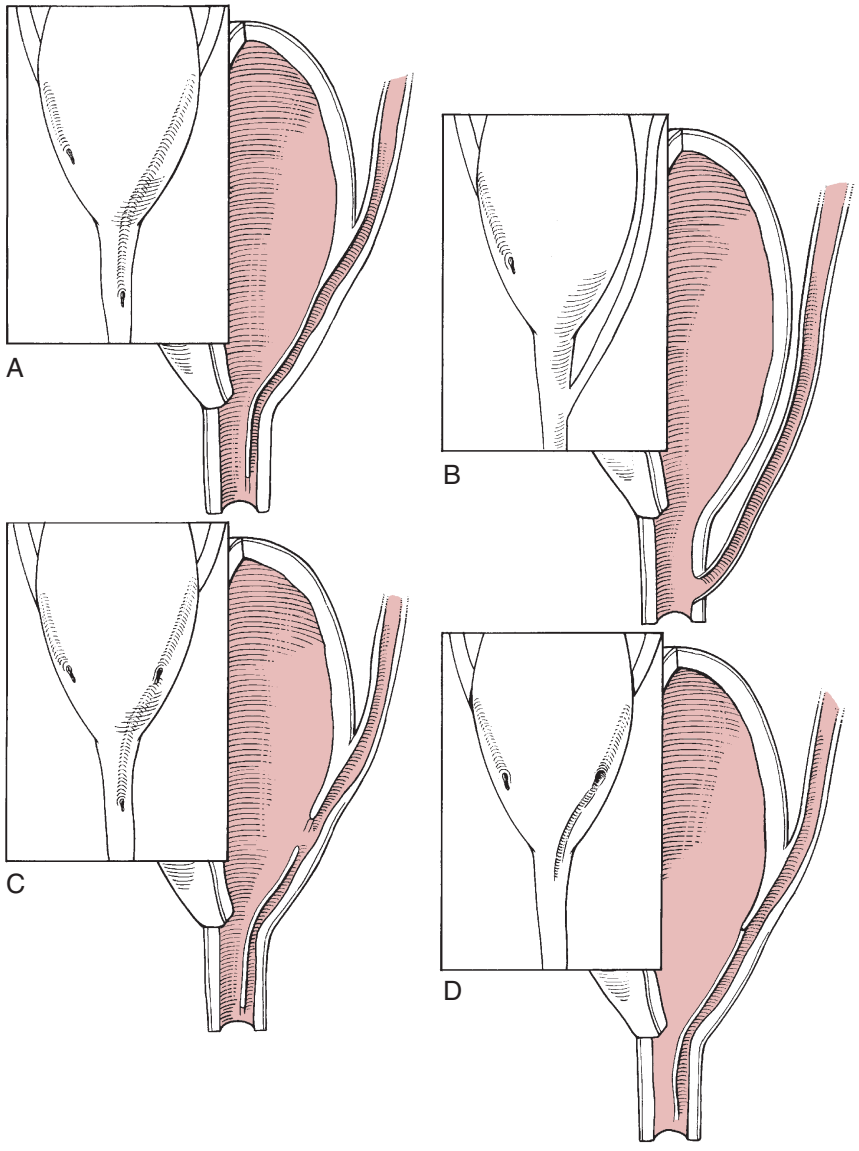


FIG. 24.14 The different types of ureteral ectopia. (A) Intramural. (B) Extramural. (C) Double ureteral openings. (D) Ureteral troughs.

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closer to the bladder than to the tip of the penis, and distal urethral pressures may prevent urine dribbling. Female dogs are usually diagnosed at a young age (median age, 10 months); however, males with ectopic ureters tend to be older at the time of diagnosis (12–24 months). Siberian huskies, golden retrievers, Labrador retrievers, Newfoundlands, English bulldogs, miniature poodles, Swiss mountain dogs (in particular, Entlebuchers and Appenzellers), fox terriers, and soft-coated wheaten terriers seem to have an increased incidence. Ureteral ectopia should be suspected in any young animal that has a history of incontinence (intermittent or continuous) since birth or weaning; however, this disease must be included as a differential in older animals with lifelong urinary incontinence, as well as in those that are poorly responsive to medications for incontinence. There are also isolated reports of ectopic ureters found in adult, continent dogs. Ectopic ureteroceles also cause urinary incontinence or may be associated with recurrent UTI without incontinence; ureteroceles have been intermittently reported in dogs and cats.

NOTE Ectopic ureters should not be excluded as a possible diagnosis even if urinary incontinence is intermittent or if the animal seems to urinate normal volumes. Affected dogs with ectopic ureters opening near the sphincter may have some response to medical management, mimicking dogs with acquired urinary incontinence.

History

Urinary incontinence is classically described as a constant dribbling, but it sometimes has a partial response to drugs or may be intermittent. Many affected animals are able to urinate normally, particularly if the condition is unilateral, the ureters open near the trigone and retrograde filling of the bladder occurs, or the bladder is of sufficient size to act as a reservoir. Some animals with bilateral ectopia may have normal voiding associated with intermittent urine dribbling. Dogs with ectopic ureteroceles typically have a history that is similar to those with ectopic ureters. Dogs with intravesicular ureteroceles may have incontinence, dysuria, hematuria, chronic UTIs, and complete or partial urinary obstruction, or they may be incidental findings in animals with no clinical signs.

Physical Examination Findings

Physical examination findings include wetness of perivulvar hair, odor, and irritation or urine scalding of surrounding skin. Some dogs may have a persistent hymen that is detected digitally or with vaginoscopy. Other findings are unremarkable unless pyelonephritis is present.

Diagnostic Imaging

The size and shape of the kidneys, bladder, and prostate should be assessed with survey abdominal radiographs and abdominal ultrasound. Skilled ultrasonographers may be able to diagnose ectopic ureters by identifying and following the ureter or ureters past the level of the trigone (Fig. 24.15). Excretory urography was historically used as the primary method for confirming ectopic ureters (Fig. 24.16) and defining associated urogenital abnormalities (i.e., hydronephrosis, hydroureter, hypoplastic bladder, and ureteroceles; see Fig. 24.13). Radiographs should be taken both early and late after contrast administration because extramural ectopia is best identified before the bladder completely fills with contrast. Alternatively, a pneumocystogram may be performed before excretory urography to facilitate visualization of the ureters when they are filled with positive contrast. The colon should be emptied to enhance visualization of the ureters and their site of termination. However, contrast radiography may not accurately identify all ectopic ureters and often fails to differentiate between intramural and extramural lesions. Retrograde cystography, pneumocystography (with the animal in the dorsoventral position to allow gas contrast to rise adjacent to ureters), and vaginoscopy may help correctly define ectopic ureter morphology.

Cystoscopy is probably a reliable, sensitive, and specific method for diagnosing ectopic ureters in females (Fig. 24.17). Cystoscopy

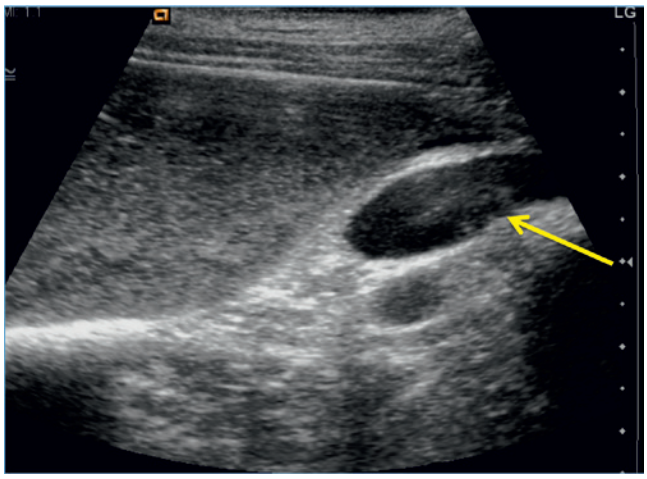


FIG. 24.15 Caudal abdominal ultrasound identifying an extramural ectopic ureter *(arrow)* in a 9-month-old dog.



FIG. 24.16 Excretory urogram in a dog with an ectopic ureter. Note the ureter filled with contrast adjacent to the neck of the bladder and extending past the trigone *(arrows)*. This is the ectopic ureter.

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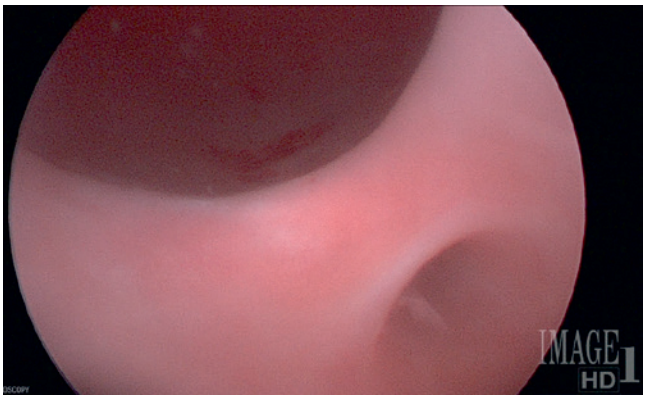


FIG. 24.17 Cystoscopic identification of unilateral ureteral ectopia.

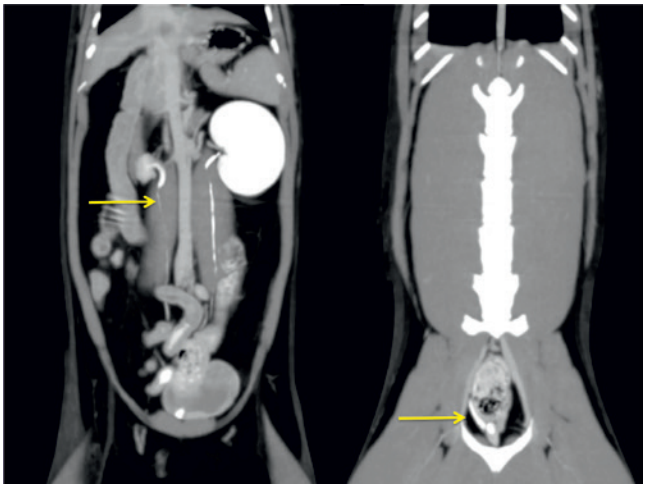


FIG. 24.18 Coronal computed tomography image of ureteral ectopia in a 6-month-old cat *(arrows)*.

is also beneficial because it allows one to visualize the vestibule and genitourinary mucosa for other abnormalities (Video 24.2).

CT excretory urography is the most sensitive method for diagnosis of ectopic ureters, and if available, should be considered the imaging modality of choice (Fig. 24.18). In a recent study comparing CT findings with cystoscopic and/or surgical findings, reviewers correctly identified 20 of 20 (100%) ureters as normal or ectopic based on the CT findings.[6] Common abnormalities observed in dogs with intramural ectopic ureters include lack of a normal ureterovesicular junction, a urethral-ureteral orifice location, and lack of ureteral divergence.

Preoperative urodynamic evaluation (urethral pressure profile) may be helpful in predicting the likelihood of postoperative continence. The chance of persistent incontinence following surgery is approximately 50%. This information could be helpful in determining whether additional procedures (e.g., colposuspension, cystourethropexy) should be performed at the time of surgery, or in counseling owners about medical treatment for incontinence after surgery.

NOTE Excretory urography is not as sensitive as cystoscopy or CT and does not accurately differentiate all intramural and extramural lesions.

BOX 24.3 Drugs Used for Urinary Sphincter Leakage

Phenylpropanolamine[a]
Dogs: 1–2 mg/kg PO q8–12h[a]
Cats: 1 mg/kg PO q12h

Ephedrine
Dogs: Start at 0.4 mg/kg and gradually increase to 4 mg/kg PO q8–12h[b]
Cats: 2–4 mg/cat PO q8–12h

Diethylstilbestrol
Dogs: 0.1–1 mg/kg PO q24h (dose proportional to size of dog), then same dose once every 3–7 days as needed, with maximum of 0.2 mg/kg/wk[c]

[a]Phenylpropanolamine may be available through selected veterinary pharmaceutical companies; it is best to start at the low end of the dose and gradually increase.
[b]Toxicity typically starts at 5 mg/kg, and death can readily occur at 10 mg/kg.
[c]If benefit is not noted after 5 days of daily therapy, then it is unlikely that further administration will benefit the patient and other drugs should be used.
PO, Orally.

Laboratory Findings

Complete blood cell count, serum chemistry profile, and urinalysis (with aerobic microbial culture) should be performed. Concomitant UTI is common. Renal failure may be present because of chronic pyelonephritis, obstructive uropathy, or concurrent congenital abnormalities (see p. 650).

DIFFERENTIAL DIAGNOSIS

Ureteral ectopia should be seriously considered in any young animal presented for incontinence or any older animal which has had incontinence since it was young. Behavioral incontinence is also common in young animals because of exaggerated submissiveness. Other causes of incontinence include urge incontinence (associated with inflammation or infection), neurogenic disorders (e.g., lower and upper motor neuron disorders or reflex dyssynergia), anatomic outflow obstruction (e.g., paradoxic incontinence), and urethral sphincter incontinence (e.g., hormone-responsive incontinence). Behavioral, urge, neurogenic, and hormone-responsive incontinence should be eliminated before tests for ectopia are considered in older animals.

MEDICAL MANAGEMENT

Incontinence may persist after surgical correction if concomitant urethral sphincter incompetence is a factor. Alpha-adrenergic agonists (e.g., phenylpropanolamine) or diethylstilbestrol (Box 24.3) may be used to increase urethral sphincter tone. Pseudoephedrine may be less effective than phenylpropanolamine for treating urinary incontinence in female dogs. See also Chapter 25.

SURGICAL TREATMENT

Surgical correction is the treatment of choice for ectopic ureters even if marginal improvement occurs with medical management. Surgery should be performed as soon as possible to limit secondary abnormalities (i.e., hydroureter and hydronephrosis) caused by ascending UTIs or outflow obstruction. Intramural ectopic ureters can be corrected with neoureterostomy, ureteroneocystostomy, or laser ablation. Cystoscopic laser ablation using either

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a diode laser or holmium:yttrium aluminum garnet laser has been reported for treatment of ectopic ureters in both female and male dogs with results comparable with surgical procedures; however, advanced equipment and training are necessary to perform this procedure. If the ureter is extraluminal, ureteroneocystostomy must be performed by resecting the ureter as distal as possible and reimplanting in the bladder lumen. Nephroureterectomy may be performed for unilateral ectopic ureters if significant morphologic or functional abnormalities of the kidney and ureter are present.

Preoperative Management

Hydration, acid-base, and electrolyte abnormalities should be corrected before surgery (see p. 650). Appropriate antibiotics should be administered as indicated by urine culture and susceptibility testing. If antibiotic therapy has not been initiated before surgery, antibiotics (e.g., cefazolin) should be administered after intraoperative cultures have been taken. Individual renal function should be determined before surgery if hydronephrosis or renal fibrosis is present. Nonfunctional kidneys should be removed if it is safe to do so (see later discussion).

NOTE Be sure to determine the function of each kidney before surgery if nephroureterectomy is being considered, even if the kidney to be removed appears to be nonfunctional.

Anesthesia

If renal impairment is not present, many different anesthetic regimens can be used safely. If renal impairment is present, see p. 650 for suggested anesthetic protocols.

Surgical Anatomy

The surgical anatomy of the kidney and ureter is described on p. 653.

Positioning

The animal is placed in dorsal recumbency, and the abdomen is prepared for a ventral midline incision. The prepared area should extend from above the xiphoid to below the pubis.

SURGICAL TECHNIQUE

The entire urinary system should be explored before the ureter is repaired. Nonfunctional kidneys and their ureter should be removed; otherwise, the ureter and kidney should be preserved. If nephrectomy is considered, bilateral ectopia should be ruled out first. If nephrectomy is done, the end of the ectopic ureter should be ligated as close as possible to its termination.

Neoureterostomy

Handle the bladder tissue with extreme care and use stay sutures whenever possible. Once the bladder has been emptied of urine, use sterile, cotton-tipped swabs rather than a sponge to absorb urine to prevent abrading the mucosal surface. Pediatric instruments may help reduce tissue trauma. Swelling or hyperemia makes the ureters difficult to locate beneath the mucosa. *Make an incision into the ventral bladder near the urethra (Fig. 24.19A). Place stay sutures to facilitate retraction of the bladder wall edges. Inspect the trigone for ureteral openings. Identify a submucosal swelling or ridge within the bladder wall; this may be facilitated by digitally occluding the urethra to cause ureteral dilation. Use a No. 11 or 15 scalpel blade to make a 3- to 5-mm longitudinal incision through the bladder mucosa into the ureteral lumen. Using 5-0 to 7-0 absorbable suture material, suture the ureteral mucosa to the bladder in a simple interrupted pattern (Fig. 24.19B). Place a 3.5- or 5-Fr catheter into the distal ureter (Fig. 24.19C). Just distal to the new stoma, pass one or two nonabsorbable sutures (3-0 or 4-0) from the serosal surface circumferentially around the tube, staying beneath the mucosa (Fig. 24.19D). Be sure the suture does not penetrate the bladder lumen. Use this suture to ligate the distal ureter after the catheter is removed.* Alternatively, the distal urethral segment can be resected; however, this has not been shown to decrease the incidence of postoperative incontinence. A novel technique for addressing the distal ureteral segment involves neoureterostomy with transection of the distal ureter, leaving the distal ureter in situ.[7]

Close the proximal urethra with single-layer simple interrupted or simple continuous sutures, but ensure that the urethral diameter is not compromised. Close the bladder in such a manner as to ensure a watertight seal (i.e., simple continuous or inverting suture pattern, see p. 685).

Ureteroneocystostomy

If the ureter is extraluminal, or if the submucosal tunnel of intramural ectopic ureters is difficult to identify, the ureter is resected and reimplanted into the bladder lumen. In dogs, the ureter may be implanted into the bladder with the use of a simple transverse pull-through or an intramural tunnel (3:1 tunnel length–to–ureteral orifice diameter). The latter technique may cause less fibrosis and quicker return of normal ureteral function.

Perform a ventral cystotomy as described previously for neoureterostomy. Ligate the ureter and transect it, preserving as much length as possible (Fig. 24.20). Place a stay suture on the proximal end of the transected ureter. Incise the bladder mucosa, and create a short, oblique submucosal tunnel in the bladder wall. Use the stay suture to draw the ureter into the bladder lumen to prevent damaging the ureter. Once the ureter is within the bladder lumen, the distal end should be excised; the remaining end of the ureter should be spatulated and then sutured to the bladder mucosa using 5-0 synthetic absorbable suture (e.g., poliglecaprone 25, glycomer 631) in a simple interrupted appositional pattern.

Cystoscopically-Guided Laser Ablation

With the dog in dorsal recumbency, examine the vagina and the lower urinary tract using a rigid cystoscope of appropriate size with passive flow of 0.9% saline. Following identification of unilateral or bilateral ectopic ureters, use a diode laser or a radiofrequency surgical device to transect the tissue separating the ectopic segment of ureter from the urethra and the bladder. Incise the tissue from the ureteral orifice extending cranially into the bladder lumen.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Absorbable suture material such as polydioxanone (PDS), polyglyconate (Maxon), poliglecaprone 25 (Monocryl), or glycomer 631 (Biosyn) should be used in the bladder because nonabsorbable suture materials may promote formation of calculus or infection. Multifilament suture may be rapidly degraded in infected urine. Small suture (i.e., 4-0 or 5-0) is preferred to suture the ureter to the bladder mucosa. Nonabsorbable suture could be considered for ligation of the distal ureter because incontinence may recur as a result of recanalization.

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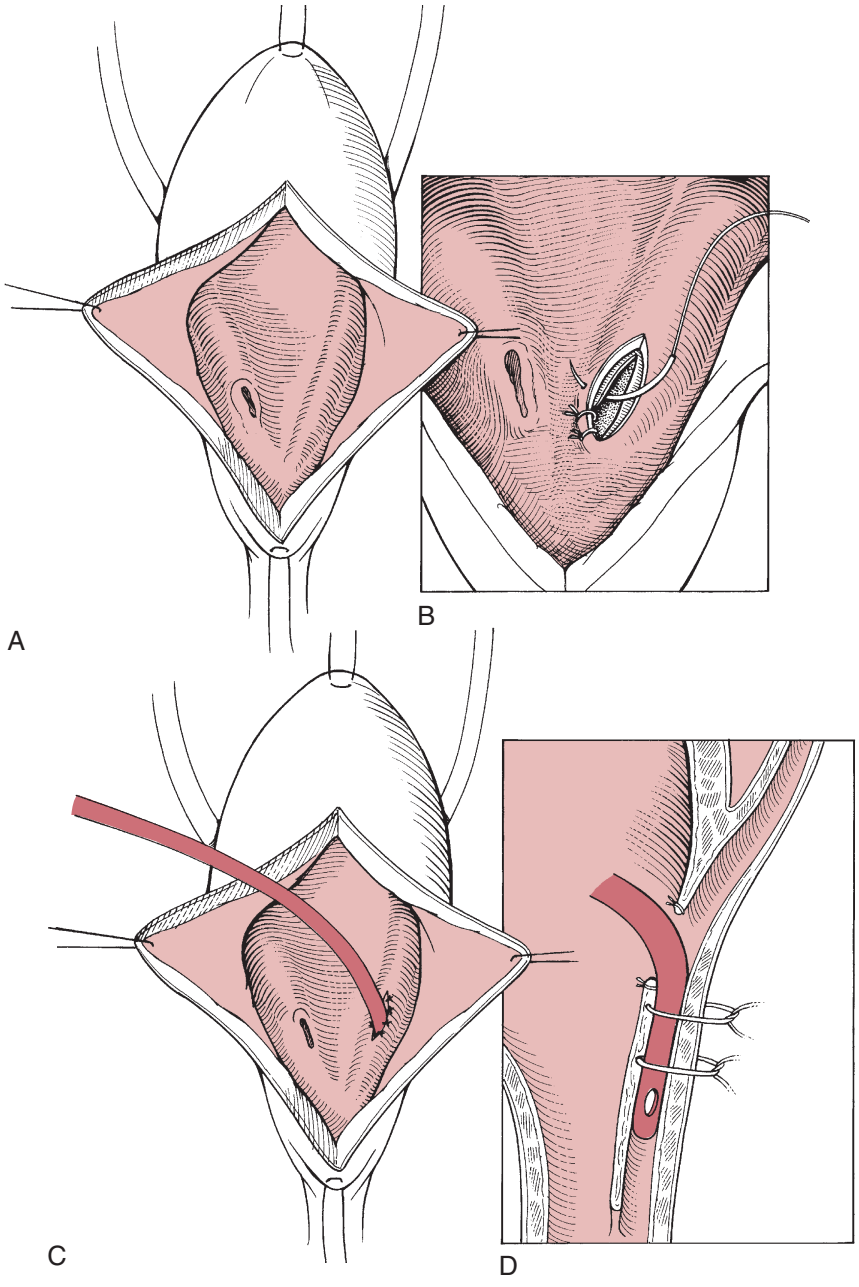


FIG. 24.19 Neoureterostomy is performed for intramural ectopic ureters. (A) Perform a cystotomy and make a 3- to 5-mm longitudinal incision through the bladder mucosa into the ureteral lumen. (B) Using absorbable suture material, suture the ureteral mucosa to the bladder in a simple interrupted pattern. (C) Place a 3.5- or 5-Fr catheter into the distal ureter. (D) Distal to the new stoma, pass one or two nonabsorbable sutures from the serosal surface circumferentially around the tube, staying beneath the mucosa.

POSTOPERATIVE CARE AND ASSESSMENT

The animal should be observed closely after surgery for signs of urinary obstruction or leakage. If urethral obstruction occurs because of postoperative swelling, an indwelling urinary catheter should be placed for 3 to 4 days until normal voiding occurs. If bilateral ectopia is corrected during the same surgery (or if significant renal impairment exists in the contralateral kidney with unilateral surgery), the animal should be monitored for renal failure due to ureteral swelling and subsequent obstruction. Technetium-99m–labeled diethylenetriamine pentaacetate (^{99m}Tc-DTPA) may be a useful aid for evaluating ureteral function in dogs after ureteroneocystostomy. If incontinence continues for longer than 2 to 3 months postoperatively, an excretory urogram, cystoscopy, CT, or urodynamic evaluation should be performed. Occasionally the distal end of the ligated ureter is patent, or a bilateral ectopia was missed.

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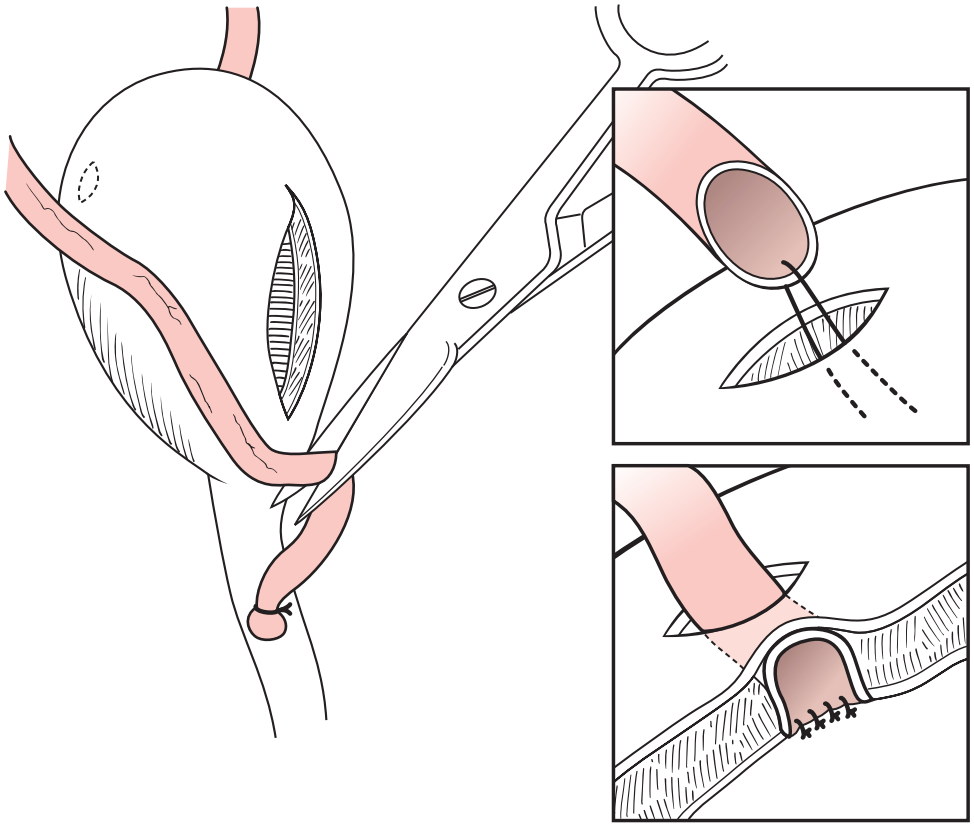


FIG. 24.20 Ureteroneocystostomy is performed when the ureter is extraluminal. Ligate the ureter and transect it. Place a stay suture on the proximal end of the transected ureter. Incise the bladder mucosa and create a short, oblique, submucosal tunnel in the bladder wall. Spatulate the ureter end and suture it to the bladder mucosa with absorbable suture.

NOTE Ureteral stoma swelling is probably common after this surgery and may cause some obstruction to ureteral flow, but it usually goes undetected and resolves without therapy.

PROGNOSIS

Past reports suggest that as few as 30% of patients are fully continent postoperatively; however, 72% of male and female dogs with surgically-corrected ectopic ureters were continent in a 2012 study.[8] Labrador retrievers appear to have a better prognosis when compared with other breeds. Many dogs with ectopic ureters have functional abnormalities of the urinary bladder or urethra. Obtaining urethral pressure measurements before surgery and after initiating diethylstilbestrol, estriol, or phenylpropanolamine therapy (see p. 666) may help predict the likelihood of continence after surgery. Siberian huskies are particularly prone to postoperative incontinence because of a high incidence of concurrent urethral sphincter incompetence. These dogs may respond to diethylstilbestrol, estriol, alpha-adrenergic agonists, or imipramine. If bladder hypoplasia is present, incontinence may continue until the bladder enlarges and properly functions as a reservoir. Dogs with ureteral troughs may have a poorer prognosis than dogs with nondistended intramural ectopic ureters. Failure to resect a ureterocele may result in continued incontinence and UTI.

RENAL AND URETERAL CALCULI

DEFINITIONS

Urolithiasis refers to the condition of having urinary calculi or uroliths (kidney, ureter, bladder, or urethra). The condition of having renal or ureteral calculi (i.e., **nephroliths** or **ureteroliths**) is **nephrolithiasis** or **ureterolithiasis**, respectively. **Nephrolithotomy** is performed to remove renal calculi from the renal pelvis by incising through kidney parenchyma; **pyelolithotomy** is an incision into the renal pelvis and proximal ureter. **Ureterolithotomy** is the removal of calculi from the ureter by incision (**ureterotomy**). A **staghorn calculus** is one that occurs in the renal pelvis and extends into the diverticula. Nephrolithotomy is also known as *lithonephrotomy*.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Less than 5% of canine uroliths are found in the kidney and ureter. However, the incidence of feline ureteral stones has markedly increased over the last 15 to 20 years, 90% to 100% of which consist of calcium oxalate. Nephroliths pose a unique diagnostic and therapeutic challenge because they may be clinically silent, or affected animals may have pain, fever, and/or renal failure attributable to urinary outflow obstruction, fibrosis, or

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infection. Clinical signs of ureteroliths may also be nonspecific and discovered incidentally. Renal and ureteral calculi may be unilateral or bilateral. Bilateral renal or ureteral calculi may be present in as many as 25% of affected cats and 20% of dogs. Less than 10% of cats typically have associated cystic calculi.

Struvite (magnesium ammonium phosphate) and calcium oxalate uroliths are the most common types in dogs; other types include urate, silicate, cystine, and mixed stones. Calcium oxalate nephroliths and ureteroliths are the most common types in cats. The pathogenesis of calcium oxalate uroliths is poorly understood, but a high association has been noted between calcium oxalate nephroureteroliths and CKD.

Whether all renal or ureteral stones should be removed is controversial. Removal should be considered if renal stones are associated with refractory infection or hematuria, or if ureteral stones are causing complete obstruction. Removal of uninfected stones from the kidney may result in more damage than is caused by the stones themselves. Other factors to be considered in deciding about surgery include the effectiveness of medical therapy in dissolving the stone, renal function in the affected and contralateral kidney, the animal's overall health, and the presence of obstructive uropathy (i.e., hydronephrosis, hydroureter, or renal failure).

Although nonsurgical removal of renal calculi is common in people (i.e., lithotripsy), these techniques are less available and less effective in dogs and cats. Lithotripsy has been used to fragment ureteroliths in dogs; however, it is not recommended in cats because the feline kidney is more sensitive to shockwave-induced injury. Medical dissolution may be effective with some stones, but calcium oxalate nephroliths and ureteroliths are not amenable to dissolution. Any stone that is surgically removed should be submitted for analysis (Fig. 24.21) because knowledge of the stone's mineral composition directs the appropriate treatment to help prevent recurrence. Because of the relationship between infection and calculi, aerobic microbial cultures of urine (and calculi, if available) are obligatory for patients with uroliths. Factors contributing to the formation of uroliths include a favorable urine pH, infection, high concentrations of crystalloids in the urine, and diminished concentrations of urine crystallization inhibitors (see p. 701 for a more detailed discussion of stone formation and treatment). In general, it is difficult or impossible to eliminate UTIs if calculi are present.

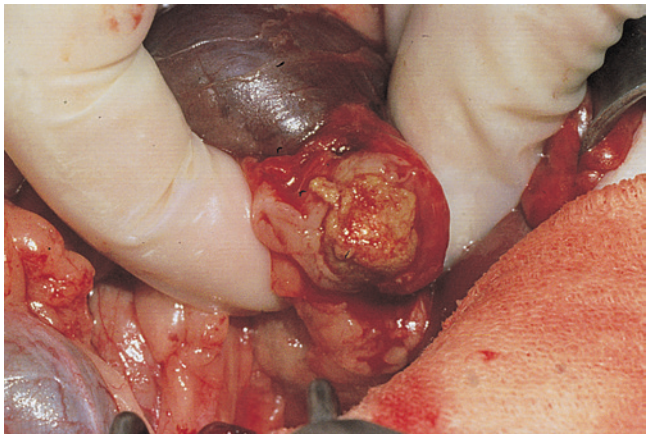


FIG. 24.21 Renal calculus in a dog.

DIAGNOSIS

Clinical Presentation

Signalment

Some animals have a higher incidence of urolithiasis because of breed predispositions, metabolic abnormalities, or underlying disease processes (see Chapter 25). Breeds at highest risk for developing renal calculi include miniature schnauzers, shih tzus, lhasa apsos, Yorkshire terriers, and female pugs. Also at high risk are male Dalmatians and male basset hounds. Among small-breed dogs, females are generally at higher risk of developing renal calculi than males. Middle-aged to older animals have a higher rate of upper tract urolithiasis than young animals. The median age of cats with ureteroliths has been reported to be 7 years, but young cats may also be affected. No apparent breed or sex predisposition has been noted in cats.

History

Historical information varies, depending on whether the stone has caused obstruction or whether concurrent infection is present. Nephroureteroliths are often found incidentally. Clinical signs may be intermittent, particularly if the animal has been treated with antibiotics. Stranguria, hematuria, and pollakiuria are relatively common signs. A previous history of urolithiasis is common if stone analysis was not performed or if appropriate therapy was not instituted after previous surgery. The most common clinical signs in cats with ureteral calculi are nonspecific (e.g., anorexia or inappetence, vomiting, weight loss, polyuria, polydipsia). Other less common clinical signs may include stranguria, hematuria, pollakiuria, signs of abdominal pain, and hypersalivation.

NOTE Because clinical signs in cats with ureteroliths are typically nonspecific, it may be wise to perform survey radiographs in all cats with ill-defined disease, regardless of whether they have evidence of acute kidney disease or CKD.

Physical Examination Findings

Renal calculi may be asymptomatic, or they may be associated with hematuria, signs of UTI (e.g., pollakiuria, stranguria), signs of CKD or AKI (i.e., anorexia, depression, vomiting, and polyuria-polydipsia), flank pain, and/or renomegaly. Hematuria is often the clinical sign noted in cats with nephrolithiasis; these cats may be erroneously diagnosed as having feline idiopathic cystitis. Polyuria-polydipsia, lethargy, depression, fever, and anorexia are consistent with pyelonephritis. Infection may cause substantial renal destruction and uremia (e.g., anorexic, depressed, dehydrated, and vomiting). Dysuria or stranguria may occur with concurrent lower UTI. Signs associated with ureteral calculi are usually caused by concurrent pyelonephritis or obstructive uropathy (i.e., uremia). Physical examination findings in cats with ureteral calculi may be nonspecific (e.g., pain on abdominal palpation and weight loss), or urinary tract abnormalities (e.g., hematuria, stranguria, pollakiuria) may be noted.

Diagnostic Imaging

Renal and ureteral calculi may be incidental radiographic or ultrasonographic findings. Most renal and ureteral calculi are radiopaque and appear as increased opacities in the renal

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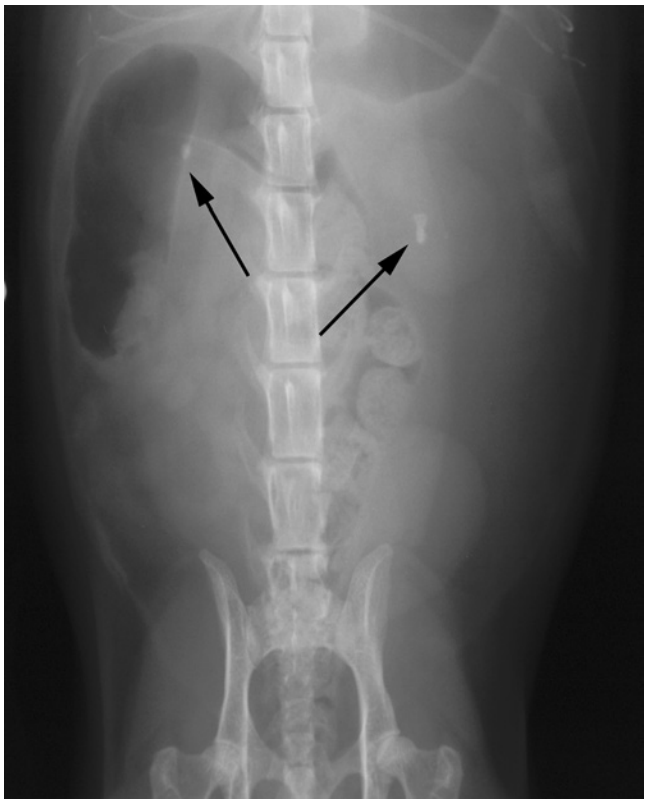


FIG. 24.22 Radiopaque renal calculi in a cat *(arrows)*.

pelvis or ureter (Fig. 24.22). The ureters, bladder, and urethra should be examined carefully for calculi anytime nephroliths are found. Associated abnormalities (e.g., hydronephrosis or hydroureter) may be assessed by ultrasonography (preferred) or excretory urography (see p. 665). Most cats with ureteral calculi also have renal calculi, and some have cystic calculi. Most cats with ureteral calculi have evidence of ureteral obstruction (e.g., dilation of the ureter and/or renal pelvis) on ultrasonographic evaluation.

Advanced imaging (CT or magnetic resonance imaging) may also be useful for evaluating ureteral obstruction in cats. Excretory urography is less successful for identifying obstruction owing to poor study quality, because the degree of opacification is dependent on GFR and renal concentrating ability, which are frequently abnormal in animals with ureteral calculi. Percutaneous antegrade pyelography has been described as an alternative to traditional excretory urography and may be more sensitive; however, contrast leakage and the potential for hemorrhage make this imaging modality less optimal than ultrasound or advanced imaging.

Laboratory Findings

Complete blood cell count, serum chemistry profile, urinalysis, and urine culture should be performed. Concomitant UTI is common. Renal failure may be caused by chronic pyelonephritis or obstructive uropathy (see p. 650). Evidence of hepatic disease or portosystemic shunting (e.g., low BUN, hypocholesterolemia, hypoalbuminemia, increased serum bile acids, hyperammonemia) may be present in animals with urate calculi. Feline ureterolithiasis is commonly associated with azotemia, hyperphosphatemia, anemia, and hyperkalemia. Occasionally, hypercalcemia is found, but hypocalcemia is more common. Unilateral ureterolithiasis is often associated with azotemia and hyperphosphatemia in cats, suggesting impairment of renal function in the contralateral kidney.

DIFFERENTIAL DIAGNOSIS

Uroliths are possible in any animal presented for AKI, CKD, uroabdomen, UTI, hematuria, stranguria, or pollakiuria.

MEDICAL MANAGEMENT

Possible underlying causes of renal or ureteral calculi should be identified and treated (e.g., UTI). Some stones can be managed with dietary therapy or pharmacologic agents (see the discussion on bladder calculi, p. 701). Fluid diuresis alone or in combination with diuretic drugs may resolve intraluminal causes of obstruction. In cats, it has been anecdotally suggested that glucagon (0.1 mg/cat IV twice daily) may cause relaxation of the ureteral smooth muscle and promote passage of ureteral calculi, but there may be adverse side effects, and its use is not recommended. If dietary therapy is used to dissolve renal calculi, the stones may become small enough to enter the ureter and cause obstruction; these animals should be monitored carefully for evidence of ureteral obstruction during such therapy. In severely ill cats with ureteral obstruction, percutaneous placement of a nephropyelostomy tube may allow treatment of azotemia before surgery. This technique may also help determine whether the obstructed kidney has any remaining function before surgery.

Percutaneous Nephropyelostomy Tubes

Using ultrasound guidance, place a 16-gauge over-the-needle catheter through the body wall from lateral to medial adjacent to the kidney, and pass it through the kidney into the renal pelvis. Cut the tip off a 3.5-Fr red rubber catheter and pass it down the IV catheter. Position the tip in the renal pelvis. Withdraw the IV catheter and seat it onto the hub of the red rubber catheter. Secure the nephrostomy tube with a deep suture that incorporates skin and underlying musculature to minimize dislodgment. Attach the catheter to a closed drainage system.

SURGICAL TREATMENT

Surgical removal of renal and ureteral calculi should be considered when they are infected or cause obstruction. To prevent irreversible renal damage, surgery should be performed as soon as possible once the animal's condition has been stabilized. However, many animals with pyelonephritis due to nephrolithiasis have chronic renal disease and are such poor anesthetic and surgical risks that chronic, palliative medical management may be chosen. Alternatives to surgical removal of ureteral calculi include ureteral stenting and placement of an SUB.

If possible, hydration, acid-base, and electrolyte abnormalities should be corrected before surgery. Appropriate antibiotics should be administered as indicated by urine culture and susceptibility testing. If antibiotic therapy has not been initiated before surgery, antibiotics (e.g., cefazolin) should be administered after intraoperative cultures have been taken. Renal function of both kidneys should be determined before surgery. Nephrectomy, rather than stone removal, is indicated in nonfunctional kidneys; otherwise, the kidney and the ureter should be preserved.

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NOTE Make sure you determine how functional each kidney is before surgery. Sometimes a kidney that grossly appears to be end-stage is the kidney with most of the remaining function.

Anesthesia

If renal impairment is not present, many anesthetic regimens can be used safely. If substantial renal impairment is present, see p. 650 for suggested anesthetic protocols. Prevent hypotension to protect blood flow to the remaining kidney during nephrectomy.

Surgical Anatomy

The surgical anatomy of the kidney and ureter is discussed on p. 653.

Positioning

The animal is placed in dorsal recumbency, and the abdomen is prepared for a ventral midline incision. The prepared area should extend from above the xiphoid to caudal to the pubis. If nephrectomy is performed, the incision must be extended caudally to allow the ureter to be ligated near the bladder.

SURGICAL TECHNIQUE

The entire urinary system, including the contralateral kidney and ureter, should be explored before the calculi are removed. Occasionally, multiple ureteral and renal calculi are found. Stones may also be found in the bladder or urethra. Renal calculi can be removed via nephrotomy (see p. 656) or pyelolithotomy (see p. 657). If the renal pelvis and the proximal ureter are sufficiently dilated, a pyelolithotomy is preferred because it avoids renal parenchymal incision and subsequent damage. However, if the stone is large and involves the diverticula and the pelvis, nephrotomy may usually be necessary. Occasionally soft stones can be crushed and removed through the renal pelvis, but care is necessary to prevent ureteral damage that may cause subsequent stricture formation.

NOTE Always submit the stones for analysis and culture.

Bilateral nephrotomy puts the patient at risk for postoperative renal failure. If possible, staged procedures should be considered. The renal pelvis or ureter should be cultured. Stones should be analyzed and cultured. Fungal elements are rarely seen on cytologic analysis of material (grit, exudate) found in the renal pelvis, but when present suggest systemic aspergillosis, a severe, potentially fatal disease.

Ureterotomy may be performed in animals with ureteral calculi; however, a combination of microsurgical techniques and intensive postoperative care is necessary to minimize morbidity. If stones are located in the distal two-thirds of the ureter, they may be removed by partial ureterectomy and ureteroneocystostomy (see p. 667). If they are located in the proximal third of the ureter, stones are removed by ureterotomy. Very proximal ureteroliths may be reached through pyelotomy. If ureterotomy is performed, a nephrostomy tube may decrease postoperative urine leakage, but significant complications are associated with this procedure. Alternatively, a peritoneal drain can be placed to address transient urine leakage.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Absorbable suture material such as polydioxanone (PDS), polyglyconate (Maxon), poliglecaprone 25 (Monocryl), or glycomer 631 (Biosyn) should be used in the kidney and ureter. Pediatric or ophthalmic instruments, as well as loupes or an operating microscope, may facilitate ureteral surgery.

POSTOPERATIVE CARE AND ASSESSMENT

The animal should be observed closely after surgery for signs of urinary obstruction or leakage. Renal failure may occur if bilateral nephrotomy was performed or if significant renal impairment was present in the contralateral kidney before surgery. See p. 662 for postoperative care of patients with renal disease.

COMPLICATIONS

The major complications of renal surgery are renal failure, hemorrhage, and urine leakage. Urine leakage or obstruction caused by stenosis or stricture is common with ureteral surgery. Nephrotomy is infrequently associated with urine leakage, persistent hematuria, renal pelvis dilation, renal mineralization, nephrolithiasis, suppurative pyelitis, bacterial UTI, and/or hydronephrosis.

SPECIAL AGE CONSIDERATIONS

Many animals (especially those older than 5 years) have some degree of renal disease and require careful monitoring during any anesthetic procedure. Hypotension should be prevented during and after surgery to avoid further renal damage. If cardiac disease is present, fluids should be used judiciously to prevent overhydration while renal blood flow is maintained.

PROGNOSIS

Most uroliths recur if the underlying disease, infection, or metabolic abnormality is not treated, often within a few months but sometimes within weeks. The effect of nephrotomy on renal function is inconsistent. Nephrotomy may decrease renal function as the result of a combination of direct nephron damage plus changes caused by vascular occlusion (i.e., inflammation, edema, scar formation, and ischemic damage). The effect of nephrotomy on GFR in dogs and cats is poorly understood.

Surgical treatment of ureteral calculi in cats may afford a better prognosis than does medical treatment. Median survival in cats following ureteral surgery is reported as 1519 days, with a perioperative mortality rate of 8%.[9] The prognosis for recovering renal function following stone removal is dependent on the degree and duration of obstruction. Improvement in renal concentrating ability can be expected after complete unilateral ureteral obstruction if the obstruction is resolved within 1 week after onset.

URETERAL STENTING

Placement of unilateral or bilateral ureteral stents is a means to bypass obstruction, particularly in cats, regardless of the location of the obstruction or the number of stones present in the kidney or ureter. Stents commonly used for feline ureteral obstruction are double-pigtail 2.5-Fr polyurethane multifenestrated stents (Vet-Stent-Ureter, Infiniti Medical) (Fig. 24.23). Stents allow for

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FIG. 24.23 Ventrodorsal radiograph after ureteral stent implantation in a 6-year-old cat.

passive ureteral dilation, reestablishment of urine flow, and potential recovery of the affected kidney. Placement of ureteral stents can be performed endoscopically with cystoscopic and/or fluoroscopic guidance with placement of the stent through the ureterovesicular junction or from a ventral midline approach. From an open approach, stents can be placed normograde via pyelocentesis, retrograde via cystotomy through the ureterovesicular junction, or retrograde through ureterotomy incision. Complications associated with ureteral stents include stent migration, stent fracture, recurrent obstruction, recurrent UTI, and chronic urinary tract disease. In one study, 27% of cats required stent replacement due to a variety of complications.[10]

In direct comparison of cats undergoing ureteral surgery with or without ureteral stent placement, cats with ureteral stents had a lower perioperative mortality rate (8% vs. 22%) and were more likely to have resolution of azotemia prior to hospital discharge.[11] In a separate study, perioperative mortality rates and median survival times were similar between cats with ureteral stents (9%; 1575 days) and cats without (8%; 1519 days).[9]

SUBCUTANEOUS URETERAL BYPASS DEVICE

An SUB device is a permanent, implantable system designed to circumvent ureteral obstruction in cats and dogs. The system connects a locking pigtail nephrostomy tube to a fenestrated and cuffed cystostomy tube through a titanium access port placed



FIG. 24.24 Lateral radiograph following subcutaneous ureteral bypass device in a 9-year-old cat.

BOX 24.4 Types of Canine Renal Tumors

Malignant	Benign
Carcinomas	Adenomas
Hemangiosarcomas	Hemangiomas
Fibrosarcomas	Teratoma
Leiomyosarcomas	
Nephroblastomas	
Squamous cell carcinomas	
Undifferentiated carcinomas	

subcutaneously (Fig. 24.24). The purpose of the access port is to allow flushing and sampling of the system as needed, which contributes to long-term patency. Ports require flushing every 3 to 4 months initially to prevent encrustation and obstruction. Renal pelvic dilation of at least 5 mm is required for nephrostomy tube placement. Complications include urine leakage at any point in the system; hemorrhage during nephrostomy tube placement; system occlusion from blood clots, debris, or stones; kinking of the tubing; and UTI. Large outcome studies regarding the use of SUBs in cats with ureteral obstruction are not currently available; however, early data show that the rate of perioperative complications, rate of perioperative mortality, and potential for long-term survival are comparable with other surgical methods for addressing ureteral obstruction.[12]

RENAL AND URETERAL NEOPLASIA

DEFINITION

Nephroblastomas are rapidly developing, malignant mixed tumors that arise from embryonal elements of the kidney. They are also called *embryonal adenomyosarcoma, nephroma,* and *Wilms tumor*.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Primary renal tumors are uncommon in dogs and cats. Approximately 85% of renal tumors are malignant, and thoracic metastatic disease is common. Renal tumors are of four distinct origins (types): renal tubular, transitional cell, nephroblastic, and nonepithelial. In dogs, carcinomas (also known as *renal tubular carcinoma* and *renal tubular adenocarcinoma*) are most common, followed by sarcomas (Box 24.4). *Lymphoma* is the most common

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renal neoplasm in cats and may be primary or metastatic (associated with alimentary lymphoma). Nearly 10% of cats develop malignant tumors (especially lymphoma) after renal transplantation and immunosuppression. Generalized nodular dermatofibrosis, a skin disorder seen in German shepherd dogs, is associated with renal cystadenocarcinomas and other neoplasms. *Nephroblastomas* are rare tumors of juvenile and adult dogs that are associated with hypertrophic osteopathy (see p. 1297). Most are malignant, and these tumors are thought to arise from embryonic tissue or metanephric blastema (i.e., tissue that forms the proximal components of the nephron from the glomerulus to the distal convoluted tubule) that has persisted in a primitive state without fully differentiating into functional tissue.

Bilateral renal involvement may occur in up to 30% of dogs with primary renal neoplasia. Metastasis to the liver, adrenal glands, lungs, lymph nodes, bone, and brain is common with renal tumors. Pulmonary metastasis is detected radiographically in nearly half of dogs with renal carcinoma. Pulmonary metastasis occurs with some nephroblastomas but is infrequent for transitional cell tumors. Renal metastasis of other primary abdominal tumors is common. Renal neoplasia may cause local signs or systemic manifestations of renal failure. Tumors arising from the renal pelvis are more apt to cause hematuria or hydronephrosis than signs of renal failure. If the contralateral kidney is normal, unilateral renal damage may not cause renal failure even if it becomes nonfunctional. Large renal neoplasms may compress or invade the caudal vena cava, causing vascular obstruction. Collateral circulation usually develops in such cases, preventing signs of obstruction (e.g., rear limb edema or ascites).

NOTE Primary renal neoplasia may occur bilaterally.

Ureteral tumors are very uncommon; only 17 cases have been documented in the veterinary literature. Most ureteral tumors are benign; however, there are three reports of ureteral sarcoma in dogs.

DIAGNOSIS

Clinical Presentation

Signalment

Dogs with renal tumors are typically geriatric large-breed dogs (mean age, 8.1 years; mean weight, 24.9 kg). No specific breed predilection has been noted, with the exception of hereditary multifocal renal cystadenocarcinomas in German shepherd dogs. Although previous studies found that renal carcinomas occur more commonly in male than in female dogs, the most recent study did not identify a gender predisposition. However, among dogs with renal sarcomas, males were affected less commonly than females. Nephroblastomas and undifferentiated renal sarcomas occur most commonly in young dogs and cats; however, they may also occur in older animals. Teratomas are rare but may occur in the kidneys of young dogs.

History

Animals with primary renal tumors often have vague, nonspecific signs. The most common reported clinical signs are hematuria, inappetence, lethargy, and weight loss. Abdominal enlargement due to a renal mass may be the only sign. Renal failure is seen primarily with bilateral involvement (e.g., lymphoma in cats). Dyspnea related to pulmonary metastasis is occasionally noted.

Physical Examination Findings

An abdominal mass is often palpated in dogs and cats with renal neoplasia. The kidney may feel enlarged, firm, or nodular. Other findings are often nonspecific and may include weight loss, inappetence, lethargy, anemia, dyspnea, and pyrexia. Lameness has been associated with bony metastasis and hypertrophic osteopathy.

Diagnostic Imaging

Renal enlargement may be identified on survey abdominal radiographs; however, ultrasonography is more sensitive and specific. Excretory urography may localize the neoplasm and define parenchymal involvement. If vascular involvement is suspected, selective angiography can detect intravascular or extravascular (compressive) lesions. Cross-sectional imaging (i.e., CT and magnetic resonance imaging) may be employed to evaluate renal masses. Thoracic radiographs should be taken because nearly 20% of dogs with renal tumors have evidence of pulmonary metastasis.

Laboratory Findings

Laboratory findings are often nonspecific; however, anemia and azotemia are common. Complete blood cell count (including a platelet count), serum chemistry profile, and urinalysis are indicated. Polycythemia is rarely found. Gross hematuria may occur with mesenchymal tumors (e.g., anaplastic sarcomas, fibromas, hemangiosarcomas, lymphosarcomas) and transitional cell tumors; however, microscopic hematuria is more common. Proteinuria may be noted.

DIFFERENTIAL DIAGNOSIS

Renal neoplasia must be differentiated from other causes of renomegaly (e.g., hydronephrosis, polycystic disease, abscess) or abdominal enlargement (e.g., splenic or hepatic neoplasia). Animals with congenital portosystemic shunts may have bilateral nonpathologic renomegaly. Abdominal ultrasonography is the most useful diagnostic tool for renal and ureteral neoplasia. Ultrasound-guided biopsy (see p. 654) can be performed if the kidney does not appear fluid filled; however, biopsy may cause peritonitis or uncontrollable hemorrhage, or it may seed the abdomen with tumor cells.

Perirenal pseudocysts have been reported in cats and are formed when fluid accumulates between the parenchyma of the kidney and the renal capsule owing to underlying parenchymal disease. Resection of the pseudocyst wall is effective in eliminating signs but does not necessarily stop the progression of renal disease. The prognosis for cats with pseudocysts is related to the degree of renal dysfunction at the time of diagnosis.

MEDICAL MANAGEMENT

Preoperative medical management of animals with renal neoplasia is necessary if renal failure is present or if anemia is severe (i.e., the packed cell volume is <20%). Medical management of animals with renal failure is discussed on p. 650.

SURGICAL TREATMENT

Nephrectomy is indicated for malignant renal tumors if they are unilateral and without evident metastasis (Fig. 24.25). Dogs

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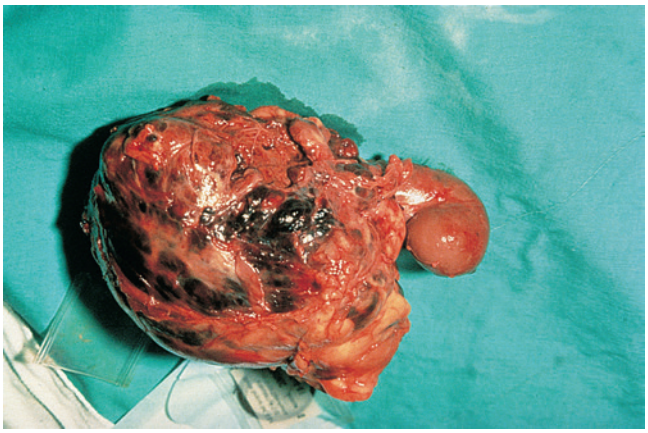


FIG. 24.25 Renal hemangioendothelioma in a 6-year-old dog.

with renal carcinoma occasionally live for years after removal of the affected kidney. However, metastasis is typically present at the time of diagnosis because of the late onset of clinical signs. Adjuvant chemotherapy or radiation therapy may prolong the lives of dogs and cats with malignant renal neoplasia, but few data are available on which to base recommendations. Cats with renal lymphoma may respond to chemotherapy for variable time periods.

Preoperative Management

Hydration, acid-base, and electrolyte abnormalities should be corrected before surgery, if possible. Perioperative antibiotic therapy is indicated in some patients (i.e., patients with large neoplasms that may be secondarily infected, or in those that are immunosuppressed or chronically debilitated). Animals with preexisting UTIs should be treated preoperatively with antibiotics. If the patient is substantially anemic, preoxygenation is beneficial. Preoperative blood transfusions should be considered in moderately to severely anemic patients, and blood should be available for intraoperative and postoperative transfusions if needed.

Anesthesia

If renal impairment is not present, many anesthetic regimens can be used safely. If renal impairment is present, see p. 650 for suggested anesthetic protocols.

Surgical Anatomy

The surgical anatomy of the kidney and ureter is discussed on p. 653.

Positioning

The animal is placed in dorsal recumbency, and the abdomen prepared for a ventral midline incision. The prepared area should extend from above the xiphoid to below the pubis.

SURGICAL TECHNIQUE

The entire abdomen should be explored for metastasis before a nephrectomy is performed (see p. 517). The other kidney should be palpated and biopsies performed if bilateral involvement is suspected. Intraoperative cytology or examination of frozen sections is helpful for determining whether the tumor is malignant. The adjacent ureter should be located to ensure that it is not inadvertently ligated. Occasionally the tumor will invade surrounding tissue (e.g., sublumbar musculature, the caudal vena cava), making complete removal difficult. The entire ureter should be removed with the kidney. Careful handling of the neoplastic kidney and ligation of the renal vein may help prevent seeding of neoplastic cells via the vasculature or directly into adjacent tissue. Liver and lymph node biopsies should be performed routinely.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Absorbable suture material such as polyglactin 910 (Vicryl), polydioxanone (PDS), polyglyconate (Maxon), poliglecaprone 25 (Monocryl), or glycomer 631 (Biosyn), or nonabsorbable cardiovascular silk, can be used to ligate the renal vessels and ureter.

POSTOPERATIVE CARE AND ASSESSMENT

See p. 662 for postoperative care of patients with renal disease.

COMPLICATIONS

The major complications of nephrectomy are hemorrhage and urine leakage. If the animal had preexisting CKD, AKI may occur postoperatively. With large renal tumors, inadvertent ligation of the opposite ureter is possible if care is not taken to determine its location intraoperatively.

SPECIAL AGE CONSIDERATIONS

Older animals often have some degree of CKD in the contralateral kidney and require careful monitoring during the surgical procedure. In addition, many animals have bilateral renal neoplasia. Renal neoplasia should not be excluded as a possible diagnosis in young animals with renomegaly.

PROGNOSIS

Because most renal tumors are aggressively malignant, they are seldom diagnosed before they have metastasized, making for a poor prognosis. However, if nephrectomy is performed before metastasis, long-term survival is possible. Median survival for dogs with renal carcinomas is reported at 16 months (range, 0–59 months), 9 months for dogs with sarcomas (range, 0–70 months), and 6 months for dogs with nephroblastomas (range, 0–6 months).[13] Although long-term survival (i.e., >2 years) with nephroblastoma is possible, dogs with renal tumors receiving adjunctive chemotherapy do not survive a significantly longer period than dogs not receiving chemotherapy. With benign neoplasia, nephrectomy is usually curative.

RENAL AND PERIRENAL ABSCESSES

DEFINITIONS

Perirenal, or *perinephric,* abscesses are abscesses located outside the renal capsule in the perinephric fascia. These infections often result from the extension of an intrarenal abscess. **Renal,** or *intrarenal,* abscesses occur within the renal parenchyma. **Pyonephrosis**, or *pyelonephrosis* or *renal pelvic abscesses*, occur when there is dilation of the renal pelvis with purulent material and is most often associated with ureteral outflow obstruction.

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GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Focal renal infections are classified as intrarenal or perirenal. Intrarenal abscesses are further divided into renal cortical abscesses and renal corticomedullary abscesses. The incidence of intrarenal and perirenal abscesses in humans ranges from 1 to 10 cases per 10,000 hospital admissions. The incidence of perirenal abscesses in dogs and cats is unknown, but they seem rare. Before the advent of antibiotics, most cases of renal abscesses in humans resulted from hematogenous seeding of the kidney from a distant focus of infection. Young males without an antecedent history of renal disease were predominantly affected. However, currently males and females are affected equally, and most cases are a complication of urinary tract obstruction. Although few cases have been reported in dogs, renal abscesses have been associated with pyelonephritis, hyperadrenocorticism, diabetes mellitus, and renal biopsy. Most renal cortical abscesses are unilateral and *Staphylococcus* spp. is the most commonly isolated causative agent in dogs and cats. Some renal cortical abscesses rupture through the renal capsule, thereby forming a perinephric abscess. Renal corticomedullary abscesses generally result from bacteriuria and ascending infection in patients with underlying urinary tract abnormalities. Enteric aerobic gram-negative bacilli (e.g., *Escherichia coli*, *Proteus* spp., *Klebsiella* spp.) seem most common.

DIAGNOSIS

Clinical Presentation

Signalment

No breed or sex predisposition has been identified in dogs or cats for either condition. Dogs and cats of any age may be affected.

History

The history of animals with renal or perirenal abscesses varies from acute pain and fever to chronic, mild, intermittent nonspecific signs. Occasionally, the only abnormality noted is abdominal enlargement associated with a renal mass.

Physical Examination Findings

Animals with renal or perirenal abscesses typically demonstrate pain on abdominal palpation. The kidney may feel enlarged, soft, and fluctuant. Other findings are often nonspecific and may include weight loss, anorexia, and pyrexia. Physical examination findings cannot differentiate renal from perirenal abscessation.

Diagnostic Imaging

Renal enlargement may be identified on survey abdominal radiographs; however, ultrasonography is more sensitive and specific for detection of focal masses. Ultrasound and CT may differentiate between renal and perirenal abscesses. Perirenal pseudocysts and subcapsular hematomas can typically be differentiated with advanced imaging modalities.

Laboratory Findings

Laboratory findings are often nonspecific; however, leukocytosis and renal azotemia may be found. Complete blood cell count (including a platelet count), serum chemistry profile, urinalysis, and urine culture should be performed. UTI is common with renal abscesses.

DIFFERENTIAL DIAGNOSIS

Perirenal and intrarenal abscesses must be differentiated from renal neoplasia and other causes of renomegaly (e.g., hydronephrosis, polycystic disease; see p. 674).

MEDICAL MANAGEMENT

Treatment of renal and perirenal abscesses have traditionally required surgical intervention. In people, some entities (e.g., renal cortical abscesses, acute focal bacterial nephritis) are now treated with antibiotics and generally do not require drainage procedures. Thus it may be reasonable to try an intensive trial of appropriate antibiotic therapy before considering surgical drainage or nephrectomy for lesions localized to the renal parenchyma. By contrast, resolution of acute multifocal bacterial nephritis generally requires some form of drainage procedure for patients with large abscesses or for those who respond slowly to antibiotics alone. Percutaneous drainage, rather than open surgical drainage, may be possible in some patients but should be done with care to prevent peritonitis. Ultrasound-guided percutaneous drainage of pyonephrosis may be performed in dogs. A 2015 study described the successful use of endoscopic- and fluoroscopic-guided double-pigtail ureteral stenting and renal pelvic lavage for renal-sparing treatment of obstructive pyonephrosis.[14]

Early surgical drainage of perinephric abscesses is imperative because antibiotic therapy alone is inadequate and should be viewed as adjunctive treatment to drainage. In human patients, perinephric abscesses have been drained by percutaneous tube placement, aspiration of pus, and antibiotic irrigation before definitive surgery (nephrectomy), but nephrectomy is typically indicated. Patients with a renal or perirenal abscess require prolonged courses of antibiotic therapy, generally lasting 4 to 6 weeks.

SURGICAL TREATMENT

Surgical drainage may be indicated in some patients with reduced function in the contralateral kidney; however, renal and perirenal abscesses generally require nephrectomy (see previous discussion in the Medical Management section).

Preoperative Management

If possible, hydration, acid-base, and electrolyte abnormalities should be corrected before surgery. Perioperative antibiotic therapy is indicated in most patients. If the patient is anemic, preoxygenation is beneficial.

Anesthesia

See p. 650 for suggested anesthetic protocols for animals with renal impairment.

Surgical Anatomy

The surgical anatomy of the kidney and ureter is discussed on p. 653.

Positioning

The animal is placed in dorsal recumbency, and the abdomen is prepared for a ventral midline incision. The prepared area should extend from above the xiphoid to below the pubis.

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SURGICAL TECHNIQUE

Nephrectomy (see p. 655) may be performed for renal and perirenal abscesses if the other kidney is sufficiently normal to sustain renal function after surgery. The entire abdomen should be explored for other evidence of infection, and the contralateral kidney should be palpated and determined to be normal before the diseased kidney is removed. The ureters should be palpated for evidence of obstructive disease (e.g., ureteroliths) that might have been predisposed to the infection. Renal tissue should be cultured and submitted for histopathology. A urine sample should be submitted for culture at the time of surgery.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Absorbable suture material, such as polyglactin 910 (Vicryl), polydioxanone (PDS), polyglyconate (Maxon), or poliglecaprone 25 (Monocryl), glycomer 631 (Biosyn) or nonabsorbable cardiovascular silk, can be used to ligate the renal vessels and ureter.

POSTOPERATIVE CARE AND ASSESSMENT

See p. 662 for postoperative care of patients with renal disease.

COMPLICATIONS

Major complications of nephrectomy are hemorrhage and urinary leakage. If the animal had preexisting renal dysfunction, AKI may occur postoperatively. With percutaneous drainage, peritonitis is a possible and serious complication.

SPECIAL AGE CONSIDERATIONS

Older animals may have some degree of CKD in the contralateral kidney and require careful monitoring during the surgical procedure.

PROGNOSIS

Perinephric abscesses are associated with significant mortality despite aggressive drainage of the abscess, surgical intervention, and antibiotics, possibly because the diagnosis is often delayed. Prompt diagnosis with nephrectomy of the abscessed kidney may lead to a positive outcome.

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25



Surgery of the Bladder and Urethra

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Cystotomy is a surgical incision into the urinary bladder, whereas **urethrotomy** is an incision into the urethra. **Cystectomy** is removal of a portion of the urinary bladder. **Cystolithiasis** and **cystolithectomy** refer to urinary bladder calculi and their removal, respectively. The **trigone** of the bladder is a smooth triangular portion of the mucous membrane at the base of the bladder (i.e., near the urethra) where the ureters empty. **Cystostomy** is the creation of an opening into the bladder; **prepubic catheterization** (e.g., temporary cystostomy, tube cystostomy) is usually performed to provide cutaneous urinary diversion in animals with urethral obstruction or trauma. **Uroabdomen** (uroperitoneum) is the presence of urine in the abdominal cavity; urine may be leaking from the kidneys, ureters, bladder, or urethra. **Urethrostomy** is the creation of a permanent fistula into the urethra; it is generally performed for irreparable or recurrent urethral stricture, or to prevent repeated obstruction (e.g., feline idiopathic cystitis [FIC]).

PREOPERATIVE MANAGEMENT

Cystolithiasis, neoplasia, and rupture are the most common abnormalities of the urinary bladder in small animals. Urinary obstruction may occur if calculi become lodged in the urethra or if a tumor obstructs the proximal urethra or trigone. Male cats with FIC may develop penile urethral obstruction (see p. 717). Obstruction to urinary flow may cause a distended urinary bladder, postrenal azotemia, and hyperkalemia. Bladder rupture primarily occurs after motor vehicular trauma but may also be caused by necrotic bladder (e.g., following damage to its blood supply or prolonged urethral obstruction) or as a complication of bladder surgery (Fig. 25.1). Urinary leakage into the abdominal cavity eventually causes uremia, dehydration, hypovolemia, hyperkalemia, and death if undiagnosed or untreated. Urinary obstruction and uroperitoneum are medical emergencies, not surgical emergencies. Hyperkalemia associated with these conditions makes the animal prone to cardiac arrhythmias; therefore fluid and electrolyte abnormalities should be corrected before anesthesia.

Hyperkalemia can cause "tented" or spiked T waves, absent or flattened P waves, prolongation of the P-R interval, widened QRS complexes, and/or bradycardia, in addition to predisposing to cardiac arrhythmias. Potassium concentrations greater than 7 mEq/L may cause irregular idioventricular rhythm, and potassium concentrations exceeding 9 mEq/L commonly cause atrial standstill. Mild or moderate hyperkalemia may be treated with intravenous (IV) fluids (i.e., 0.9% saline for dilution; Box 25.1). The type of fluid does not appear to have a clinical impact on the resolution of metabolic and electrolyte abnormalities as studies have shown lactated Ringer's solution (LRS), Normosol-R, and 0.9% saline to be effective in resolving metabolic acidosis, hyperkalemia, and postrenal azotemia associated with urethral obstruction in cats; however, 0.9% saline was less efficient in restoring acid-base and electrolyte imbalances both experimentally and in a retrospective study.[1,2] If the animal has concurrent hyponatremia, 5% dextrose solution and half-strength saline should be avoided. Fluid correction should be initiated immediately after IV catheter placement in order to support vascular volume, dilute serum potassium, and address acid-base abnormalities, particularly in severely affected cats.

Hyperkalemia from uroabdomen responds well to abdominal drainage plus intravenous fluid therapy. Hyperkalemia caused by urethral obstruction responds well to intravenous fluids plus elimination of the obstruction (e.g., urethral catheterization or decompressive cystocentesis). Although seldom required, life-threatening hyperkalemia may be treated with IV sodium bicarbonate. Bicarbonate therapy drives potassium into cells in exchange for hydrogen ions. Patients with life-threatening hyperkalemia are often moribund and poorly responsive. Sometimes these patients may have respiratory acidosis associated with poor ventilation, in which case they can be intubated and hyperventilated to correct respiratory acidosis. Similar to administering bicarbonate, hyperventilating the patient raises pH and drives potassium intracellularly. Alternatively, life-threatening hyperkalemia can be treated with insulin and dextrose administration (see Box 25.1). Insulin facilitates cellular uptake of potassium, whereas dextrose prevents hypoglycemia following insulin administration. If the hyperkalemia appears immediately life threatening, 10% calcium gluconate can be given slowly intravenously while watching the electrocardiogram (ECG); this may protect the heart until other therapy lowers the plasma potassium concentration.

Preventing reabsorption of electrolytes and waste products by abdominal drainage, urinary catheterization, and IV fluid therapy is the best way to treat hyperkalemia and azotemia in animals with uroperitoneum. Closed systems (e.g., 14-gauge over-the-needle catheter) are preferred for abdominal drainage because they can be attached to an empty fluid bag, allowing for a closed system and quantification of abdominal fluid loss to gauge the amount of IV fluid replacement (Fig. 25.2). The goal of abdominal drainage in these patients is to normalize serum electrolytes and decrease azotemia, making the animal a better candidate for general anesthesia. Fluid therapy, urethral

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ABSTRACT

Surgery of the bladder and urethra is most often performed to relieve obstruction caused by calculi. Other indications for surgery include blunt trauma that can result in rupture of the bladder, and biopsy or resection of lower urinary masses. Urethral stricture formation due to obstruction, traumatic catheterization, neoplastic invasion, or chronic inflammation may require urinary diversion that can be accomplished by a variety of methods depending on the location. One of the most common causes of urinary incontinence is urethral sphincter mechanism incompetence; several surgical methods to aid in return of continence are described in this chapter.

KEYWORDS

bladder
urethra
cystotomy
cystostomy
cystectomy
urethrotomy
urethrostomy
uroabdomen
calculi
neoplasia
urethral prolapse
urinary incontinence

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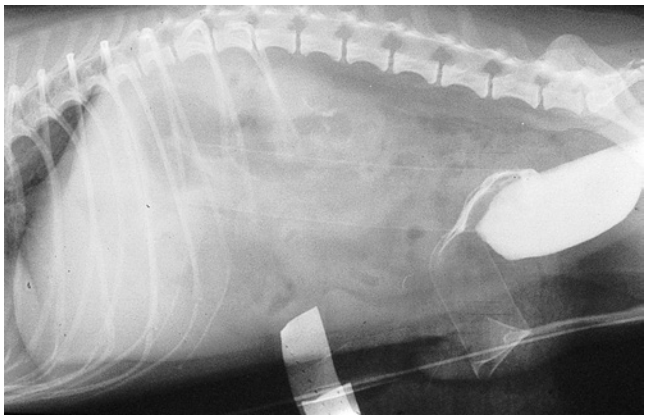


FIG. 25.1 Positive contrast cystourethrogram in a dog 3 days after a cystotomy was performed. Note contrast leaking from the incision at the dorsal aspect of the bladder.

BOX 25.1 Treatment of Hyperkalemia in Cats

1. Dilute by giving physiologic 0.9% saline solution IV or, once the obstruction is relieved, LRS IV.
2. If necessary to lower potassium immediately, give sodium bicarbonate (see Box 4.1) or regular insulin 0.2–0.4 U/kg IV plus dextrose (2 g/U of insulin).
3. If hyperkalemia is immediately life threatening, may give 10% calcium gluconate (0.2–1.5 mL/kg) for transient cardiac protection. Give slowly (over 5–10 min) while monitoring the patient's ECG.
4. Provide intubation and controlled hyperventilation to rapidly reduce respiratory acidosis.

ECG, Electrocardiogram; *IV,* intravenous; *LRS,* lactated Ringer's solution.

catheterization, and abdominal drainage for 6 to 12 hours are often adequate for this purpose. Peritoneal dialysis or hemodialysis may be useful when treating patients with concurrent renal dysfunction.

Urethral trauma (e.g., gunshot or bite wounds, rupture caused by vehicular trauma, obstruction with stones) or neoplasia may result in urinary obstruction. If the prostatic or penile urethra is torn, subcutaneous urine leakage may occur. Spontaneous rupture of the urethra is uncommon but may occur in dogs (Fig. 25.3). Initial signs of subcutaneous urine leakage are bruising and/or swelling, especially of the inguinal tissue of male dogs. The skin and subcutaneous tissue can eventually become necrotic if left untreated. Preoperative management of patients with urethral rupture may necessitate placement of an indwelling urinary catheter and/or cutaneous urinary diversion (tube cystostomy; see p. 685).

ANESTHESIA

Patients with acute obstruction are often otherwise healthy (Table 25.1). They may be presented early or late in the course of the urinary obstruction; thus clinical signs may range from a cat that is uncomfortable and straining, but has a normal serum biochemical profile, to a recumbent and very ill acute renal failure patient. In the cat with relatively normal bloodwork, IV fluids should be limited before the urinary obstruction is relieved. In the obstructed patient in acute renal failure that has major sodium and potassium disturbances with possibly

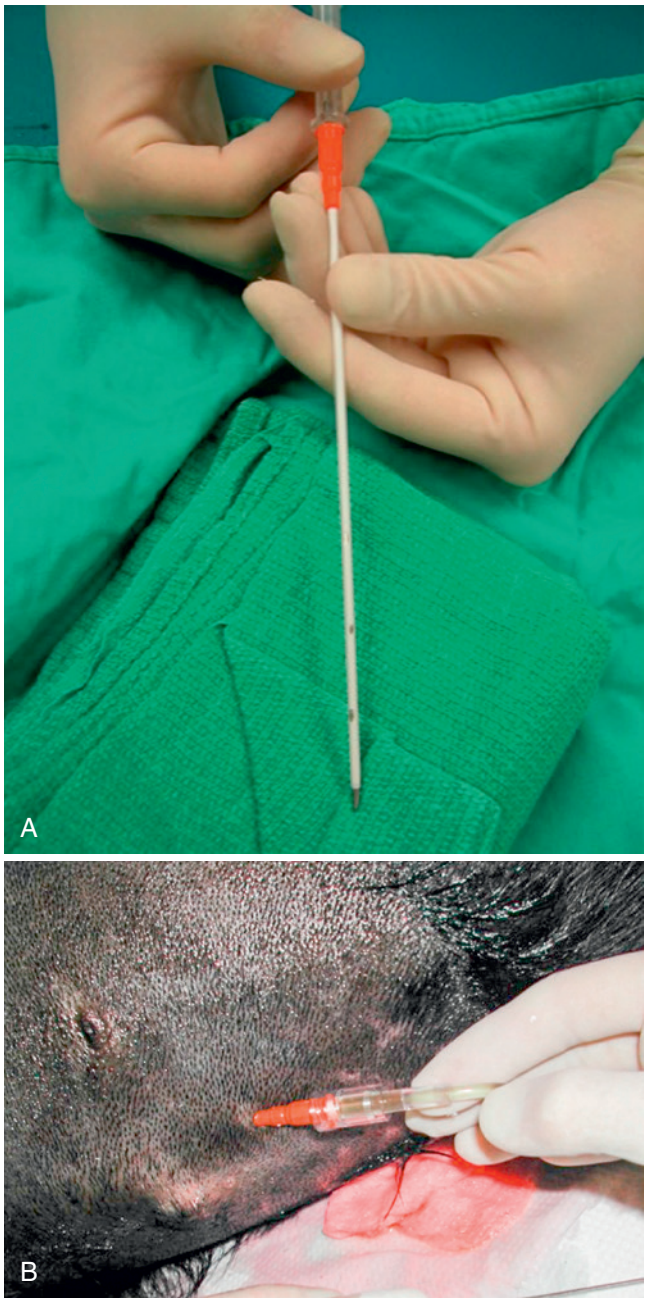


FIG. 25.2 (A) 14-gauge over-the-needle catheter with additional fenestrations (B) placed in the abdominal cavity of an 8-year-old Rottweiler with a uroabdomen.

life-threatening electrocardiographic changes due to hyperkalemia, hypovolemia, hypotension, weakness, and/or hypothermia, only limited doses of drugs can typically be tolerated without causing further sedation or hypotension (Table 25.2). In the depressed or recumbent obstructed patient, bloodwork is extremely important in determining electrolyte status and in anticipating the patient's response to anesthesia.

Electrolyte (i.e., hyperkalemia) abnormalities and acidosis in patients with urinary obstruction or leakage should be corrected before anesthetic induction (see previous discussion and pp. 699 and 718). IV fluids are given to restore hydration and combat postobstructive diuresis; relief of obstruction without appropriate parenteral fluids can result in hypovolemia and possibly death.

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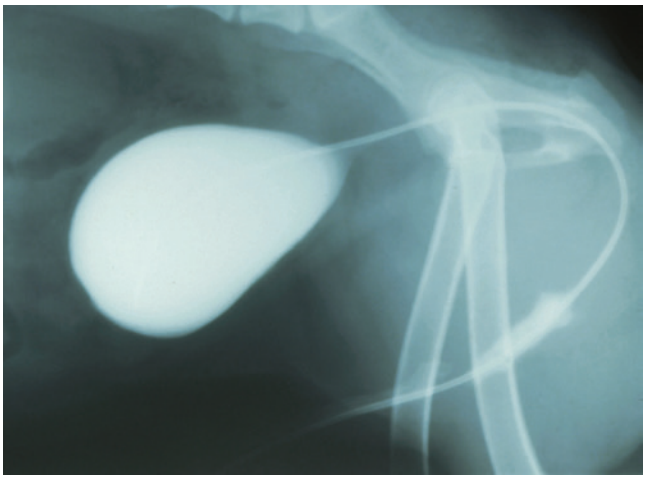


FIG. 25.3 Radiograph of a dog with spontaneous urethral rupture associated with a urinary tract infection. Note the leakage of contrast into the tissue just proximal to the os penis.

An ECG should be monitored before, during, and after surgery for cardiac arrhythmias. If the animal is hyperkalemic (potassium >7 mEq/L), 0.9% saline should be used for fluid therapy. If serum potassium is normal, a balanced electrolyte solution should be administered.

Anticholinergics are not routinely recommended as premedication for trauma patients because they may increase heart rate and oxygen consumption, and may cause a predisposition to cardiac arrhythmias (Table 25.3). If analgesia is needed, butorphanol, hydromorphone, oxymorphone, or buprenorphine may be given in small, incremental doses (see Table 13.2). Acepromazine is not recommended as a routine premedication and should be used only if volume replacement has been adequate and if shock or severe blood loss is unlikely. Combinations of opioids and benzodiazepines (e.g., diazepam, midazolam) do not cause severe vasodilation or myocardial depression and are useful for inducing anesthesia despite hypovolemia (see Tables 25.2 and 25.3). Etomidate may be used for induction because it maintains

Text continued on page 684

TABLE 25.1 Anesthetic Considerations in an Uncompromised, Obstructed Cat

Preoperative Considerations

Associated conditions	• Minimal; usually healthy
Bloodwork	• HCT • TP • Electrolytes • BUN • Cr
Physical examination	• May be anxious and straining • Minimal dehydration

Premedications	• If patient is anxious, give the following:
	  • Diazepam (0.2 mg/kg IV), *or*
	  • Midazolam (0.2 mg/kg IV, IM), *plus*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM), *or*
	  • Oxymorphone (0.05–0.2 mg/kg IV, IM), *or*
	  • Morphine[b] (0.1–0.2 mg/kg IV or 0.2–0.4 IM), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM)

Intraoperative Considerations

Induction	• If premedicated, give:
	  • Propofol (2–4 mg/kg IV), *or*
	  • Alfaxalone (2–3 mg/kg IV)
	• If not premedicated, then:
	  • Propofol (4–8 mg/kg IV), *or*
	  • Alfaxalone (2–5 mg/kg IV), *or*
	  • Ketamine (5.5 mg/kg IV) with diazepam (0.28 mg/kg IV)

Maintenance	• Isoflurane or sevoflurane *plus*
	  • Fentanyl (1–4 μg/kg IV PRN) for short-term pain relief, *plus* PRN
	  • Buprenorphine[b] (0.005–0.02 mg/kg IV PRN), *plus*
	  • Oxymorphone (0.05–0.1 mg/kg IV PRN), *or*
	  • Hydromorphone (0.05–0.2 mg/kg IV PRN), *or*
	  • Ketamine (low dose) (0.5–1 mg/kg IV once)

Fluid needs	• 5–10 mL/kg first hour
Monitoring	• Blood pressure • HR • Respiratory rate • SpO_2 • Temperature • $EtCO_2$
Blocks	Sacrococcygeal: • Lidocaine 2% (0.1–0.2 mL/kg), *or* • Bupivacaine 0.5% (0.1–0.2 mL/kg), *or* • Lidocaine 2% (0.05–0.1 mL/kg) with bupivacaine 0.5% (0.05–0.1 mL/kg)

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TABLE 25.1 Anesthetic Considerations in an Uncompromised, Obstructed Cat—cont'd

Postoperative Considerations

Analgesia	• Morphine[b] (0.05–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h), *or* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q3–4h), *or* • Oxymorphone (0.05–0.2 mg/kg IV, IM q3–4h), *or* • Switch to oral pain medications (e.g., butorphanol 0.5–2 mg/kg PO q6–8h *or* buprenorphine[b] 0.01–0.02 mg/kg OTM q6–12h) NOTE: NSAIDs are contraindicated in patients with hypotension, hypovolemia, or renal compromise
Monitoring	• SpO_2 • Blood pressure • HR • Respiratory rate • Temperature • U/O
Bloodwork	Repeat previously abnormal bloodwork
Estimated pain score	Usually mild to moderate once obstruction is relieved

[a]May cause hyperthermia in cats.
[b]Give slowly to prevent histamine release.
[c]Buprenorphine is a better analgesic than morphine in cats.
BUN, Blood urea nitrogen; *Cr,* creatinine; *$EtCO_2$,* end-tidal carbon dioxide; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *NSAID,* nonsteroidal antiinflammatory drug; *OTM,* oral transmucosal; *PO,* orally; *PRN,* as needed; *SpO_2,* oxygen saturation via pulse oximeter; *TP,* total protein; *U/O,* urine output.

TABLE 25.2 Anesthetic Considerations in the Compromised, Obstructed Cat

Preoperative Considerations

Associated conditions	• Dehydration • Electrolyte abnormalities • Hypotension • Dysrhythmias
Bloodwork	• HCT • Electrolytes • BUN • Cr • TP
Physical examination	• Often initially healthy patients • May be dehydrated, tachycardic or bradycardic, hypotensive, and/or hypothermic
Other diagnostics	• Blood pressure • ECG

Premedications	• Rehydrate over 4–6 h if possible; if emergent, may have to give more rapid boluses to expedite time to surgery
	• Correct electrolyte abnormalities
	• Avoid sedatives in sick or depressed patients
	• Avoid alpha-2 agonists and acepromazine
	• If patient is anxious, give midazolam (0.1–0.2 mg/kg IV, IM)
	• If patient is not depressed, then give:
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM in dogs; 0.05–0.1 mg/kg IV, IM in cats), *or*
	  • Oxymorphone (0.05–0.2 mg/kg IV, IM), *or*
	  • Morphine[b] (0.1–0.2 mg/kg IV and 0.02–0.4 mg/kg IM), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM)

Intraoperative Considerations

Induction	• If dehydrated, give the following:
	  • Etomidate (0.5–1.5 mg/kg IV); give after administering a benzodiazepine (diazepam [0.25 mg/kg IV] or midazolam [0.25 mg/kg IV]) to avoid myoclonus, *or*
	  • Propofol (1–4 mg/kg IV) slowly, *or*
	  • Alfaxalone (2–3 mg/kg IV) slowly
	• If hydrated, give the following:
	  • Propofol (4–8 mg/kg IV), *or*
	  • Alfaxalone (2–5 mg/kg IV), *or*
	  • Ketamine (5.5 mg/kg IV) with midazolam (0.28 mg/kg IV)

Maintenance	• Isoflurane *plus:*
	  • Fentanyl (2–10 µg/kg IV PRN in dogs; 1–4 µg/kg IV PRN in cats) for short-term pain relief, *plus*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Oxymorphone (0.05–0.2 mg/kg IV, PRN), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV PRN), *plus*
	  • Ketamine (low dose) (0.5–1 mg/kg IV)
	• For hypotension (to keep MAP 60–80 mm Hg) give phenylephrine, ephedrine, dopamine, norepinephrine, or vasopressin as needed

Continued

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TABLE 25.2 Anesthetic Considerations in the Compromised, Obstructed Cat—cont'd

Fluid needs	• 5–10 mL/kg/h if minimal EBL and minimal evaporative losses
Monitoring	• Blood pressure • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature • U/O
Blocks	Epidural: • Morphine (0.1 mg/kg preservative free), *or* • Buprenorphine (0.003–0.005 mg/kg diluted in saline) • Avoid local anesthetics for spinals and epidurals in hypotensive patients

Postoperative Considerations

Analgesia	• Morphine[b] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.05–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h in cats) if no hypotension, *or* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats), *or* • Oxymorphone (0.05–0.2 mg/kg IV, IM q3–4h), *or* • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM q4–8h or 0.01–0.02 mg/kg OTM q6–12h in cats) NOTE: Avoid NSAIDs
Monitoring	• SpO_2 • Blood pressure • HR • Respiratory rate • Temperature • U/O • ECG if electrolyte abnormalities
Bloodwork	• HCT • Electrolytes
Estimated pain score	Moderate

[a]Monitor for hyperthermia in cats.
[b]Give slowly to prevent histamine release.
[c]Buprenorphine is a better analgesic than morphine in cats.
BUN, Blood urea nitrogen; *CR,* creatinine; *CRI,* constant rate infusion; *EBL,* estimated blood loss; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *MAP,* mean arterial pressure; *NSAID,* nonsteroidal antiinflammatory drug; *OTM,* oral transmucosal; *PRN,* as needed; *SpO_2,* oxygen saturation via pulse oximeter; *TP,* total protein; *U/O,* urine output.

TABLE 25.3 Anesthetic Considerations in the Trauma Patient

Preoperative Considerations

Associated conditions	• Anemia • Dehydration • Electrolyte abnormalities • Hypotension • Dysrhythmias • Presence of other trauma (e.g., pelvic, abdominal, thoracic)
Bloodwork	• HCT • Electrolytes • BUN • Cr • TP • Albumin • +/– Blood gas
Physical examination	• May be dehydrated, in shock, hypotensive, tachycardic or bradycardic, and hypothermic • There will be an absence of pulmonary sounds in the dorsal lateral chest wall with auscultation if there is a pneumothorax; absence of pulmonary sounds in the ventral chest wall and muffled heart sounds if there is hemothorax; the abdomen may be painful if the animal has a uroabdomen or hemoabdomen
Other diagnostics	• Blood pressure • ECG • Radiographs • SpO_2 • +/– Ultrasound • +/–Thoracocentesis

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TABLE 25.3 Anesthetic Considerations in the Trauma Patient—cont'd

Premedications	• Oxygen by face mask or flow-by
	• Give shock dose of fluids if necessary to stabilize
	• Give blood transfusion if HCT <20% in dogs, <18% in cats (see p. 32)
	• Avoid sedatives in depressed patients
	• Avoid all respiratory depressants in dyspneic patients including but not limited to opioids, xylazine, medetomidine, and dexmedetomidine
	• If hypotensive, avoid acepromazine, xylazine, medetomidine, and dexmedetomidine
	• If tachycardic, avoid atropine, glycopyrrolate, and induction doses of ketamine
	• Administer GI protectant of choice
	• In anxious patients, give midazolam (0.2 mg/kg IV, IM)
	• In patients that are not depressed or dyspneic, give the following:
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM in dogs, 0.05–0.1 mg/kg IV, IM in cats), *or*
	  • Oxymorphone (0.05–0.2 mg/kg IV, IM), *or*
	  • Morphine[b] (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg, IV, IM)
	• After a benzodiazepine is given but prior to induction, clip hair from surgical area

Intraoperative Considerations	

Induction	Preoxygenate for 3–5 min with face mask or flow-by oxygen
	• If dehydrated, give the following:
	  • Etomidate (0.5–1.5 mg/kg IV) (give after administering a benzodiazepine: diazepam [0.25 mg/kg IV] or midazolam [0.25 mg/kg IV] to avoid myoclonus)
	• If hydrated, give the following:
	  • Propofol (2–6 mg/kg IV); if dyspneic, administer rapidly to expedite intubation and ventilation, *or*
	  • Alfaxalone (2–5 mg/kg IV)

Maintenance	• Isoflurane or sevoflurane *plus*
	  • Fentanyl (2–10 µg/kg IV PRN in dogs; 1–4 µg/kg IV PRN in cats) for short-term pain relief, *plus*
	  • Fentanyl CRI (1–5 µg/kg IV loading dose, then 2–30 µg/kg/h IV), *or*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Oxymorphone (0.05–0.2 mg/kg IV, IM), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV PRN), *plus*
	  • Ketamine (low dose) (0.5–1 mg/kg IV), *or*
	  • Ketamine CRI (0.5 mg/kg IV loading dose, then 10 µg/kg/min IV)
	• For hypotension (to keep MAP 60–80 mm Hg), give phenylephrine, ephedrine, dopamine, norepinephrine, or vasopressin as needed
	• If pulmonary involvement, ventilation parameters:
	  • SpO_2 >95%
	  • Small tidal volumes: 5–7 mL/kg
	  • Respiratory rates: 10–30
	  • Peak airway pressures <12–15 mm Hg if able to achieve adequate tidal volumes

Fluid needs	• 5–10 mL/kg/h if minimal evaporative losses • Plus 3× EBL • Consider colloids if persistent hypotension
Monitoring	• BP • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature • +/– Arterial line • U/O
Blocks	Epidural: • Morphine[b] (0.1 mg/kg preservative free), *or* • Buprenorphine (0.003–0.005 mg/kg diluted in saline) • Avoid local anesthetics for spinals and epidurals in hypotensive patients. Incisional: • Lidocaine (<5 mg/kg in dogs; 2–4 mg/kg in cats), *or* • Bupivacaine (<2 mg/kg)
Postoperative Considerations	
Analgesia	• Fentanyl CRI (1–10 µg/kg IV loading dose, then 2–20 µg/kg/h IV), *or* • Morphine[b] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.05–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h in cats) if no hypotension, *or* • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM q4–8h or 0.01–0.02 mg/kg OTM q8–12h in cats), *or* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats), *or* • Oxymorphone (0.05–0.2 mg/kg IV, IM q3–4h), *or* • Hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs), *plus* • +/– Ketamine CRI (2 µg/kg/min IV); if no previous loading dose, give 0.5 mg/kg IV prior to CRI • Avoid NSAIDs if hypotensive

Continued

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TABLE 25.3 Anesthetic Considerations in the Trauma Patient—cont'd

Monitoring	• SpO_2 • Blood pressure • ECG • HR • Respiratory rate • Temperature • U/O
Bloodwork	• HCT • TP • Electrolytes • +/– Blood gas • +/– Albumin
Estimated pain score	Moderate to severe

[a]Monitor for hyperthermia in cats.
[b]Give slowly to prevent histamine release.
[c]Buprenorphine is a better analgesic than morphine in cats.
BP, Blood pressure; *BUN,* blood urea nitrogen; *Cr,* creatinine; *CRI,* constant rate infusion; *EBL,* estimated blood loss; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *GI,* gastrointestinal; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *MAP,* mean arterial pressure; *NSAID,* nonsteroidal antiinflammatory drug; *OTM,* oral transmucosal; *PRN,* as needed; *SpO_2,* oxygen saturation via pulse oximeter; *TP,* total protein; *U/O,* urine output.

cardiovascular stability and is not arrhythmogenic. Alternatively, propofol or alfaxalone may be administered at reduced dosages.

Cats may be premedicated using low doses of butorphanol, buprenorphine, oxymorphone, or hydromorphone and induced with etomidate. Because cats excrete the active form of ketamine in their urine, it should be avoided or used in lower doses if urinary obstruction or renal dysfunction is present. Isoflurane and sevoflurane in oxygen are the least cardiodepressant inhalation anesthetics and may be used for anesthetic maintenance. Compound A, a byproduct of sevoflurane, causes renal failure in rats but does not appear to do so in dogs, cats, or humans. However, in the human literature, it is recommended that sevoflurane should be used at an oxygen flow rate greater than 2 L/min to protect against the possibility of nephrotoxicity in renal compromised patients. The alternative is to use isoflurane and avoid the theoretical possibility of worsening kidney function.

ANTIBIOTICS

Perioperative antibiotic therapy should be considered in animals with urinary obstruction or leakage because infection prolongs healing and promotes stricture formation. Animals with cystic or urethral calculi often have concurrent infections and should be treated with appropriate antibiotics based on urine culture and susceptibility, or antibiotics can be withheld until intraoperative cultures are taken. When culture of urine obtained by cystocentesis is negative, aerobic cultures of a bladder mucosal biopsy and/or cystic calculi should be obtained. *Escherichia coli* has been found to be the most common isolate in dogs with recurrent or persistent urinary tract infections (UTIs); however, mixed bacterial infections are common. Potentially nephrotoxic antibiotics (e.g., aminoglycosides) should be avoided in patients with urinary obstruction or possible pyelonephritis (see p. 653).

NOTE Organisms may be cultured from the bladder mucosa or from a urolith in dogs with urolithiasis from which a negative urine culture was previously obtained; therefore animals undergoing cystotomy should have cultures of bladder mucosa and uroliths.

SURGICAL ANATOMY

The urinary bladder location varies depending on the amount of urine it currently contains; when empty, it lies primarily within the pelvic cavity. In a 12-kg dog, the bladder holds up to 120 mL of urine without becoming overly distended. The bladder is divided into the trigone, which connects it to the urethra, and the body. The bladder receives its blood supply from the cranial and caudal vesical arteries, which are branches of the umbilical and urogenital arteries, respectively. Sympathetic innervation is from the hypogastric nerves, whereas parasympathetic innervation is via the pelvic nerve. The pudendal nerve supplies somatic innervation to the external bladder sphincter and striated musculature of the urethra (see p. 653). The urethra in male dogs and cats is divided into prostatic, membranous (pelvic), and penile portions (Fig. 25.4).

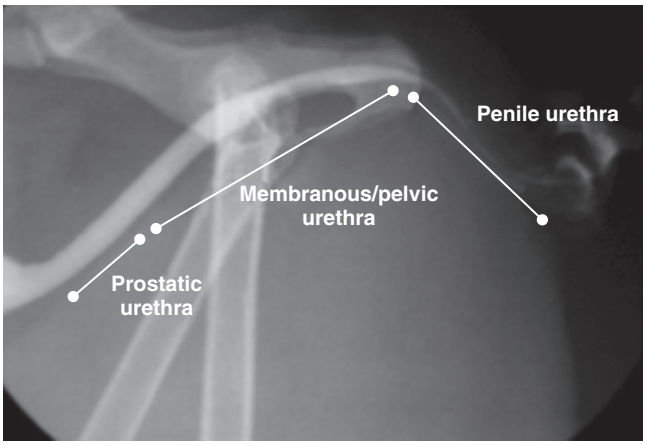


FIG. 25.4 Labeled radiograph of a normograde positive contrast cystourethrogram in a cat demonstrating the anatomic regions of the feline urethra.

SURGICAL TECHNIQUE

For the bladder, an abdominal incision is made from the umbilicus caudal to the pubis (Fig. 25.5). The proximal urethra (i.e., prostatic urethra) can be reached by this approach; however, pelvic osteotomy (see p. 689) or symphysiotomy is required for adequate exposure of the membranous urethra (i.e., from the caudal edge of the prostate to the ischial arch; Figs. 25.6 and 25.7). The penile urethra begins at the ischial arch and extends to the external urethral penile orifice. The penile urethra may be approached

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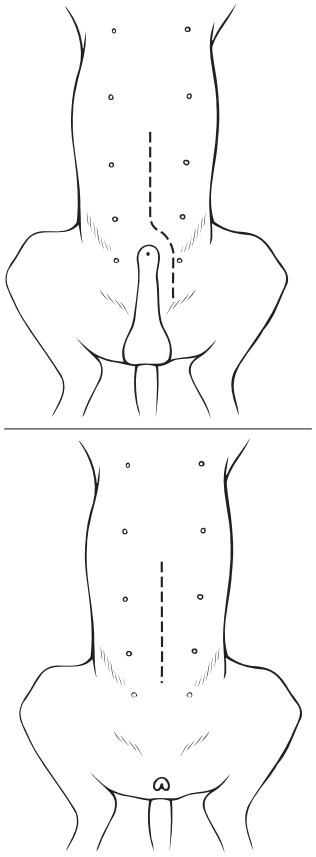


FIG. 25.5 To expose the bladder, make an incision from the umbilicus to the pubis.

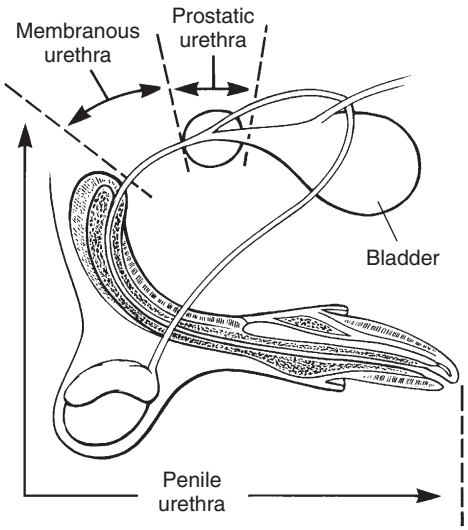


FIG. 25.6 The urethra of male dogs is composed of prostatic, membranous (pelvic), and penile portions.

in the perineal (perineal urethrotomy) or scrotal (scrotal urethrotomy) region, or between the scrotum and the external urethral orifice (prescrotal urethrotomy). The skin overlying the site is prepared for aseptic surgery in standard fashion before either approach is used. If the prepuce is to be left in the surgical field, a preputial flush with chlorhexidine or dilute Betadine should be part of the surgical preparation.

Cystotomy

Cystotomy may be performed for removal of cystic and urethral calculi (see p. 700), identification and biopsy of masses (see p. 707), repair of ectopic ureters (see p. 663), or evaluation of UTI resistant to treatment. The longitudinal incision is generally made on the ventral or dorsal surface of the body of the bladder, away from the urethra; however, ventral exposure is preferred owing to ease of access and should be performed if identification or catheterization of ureteral openings is necessary. The goal of cystotomy closure is to obtain a watertight seal that will not promote formation of calculi. This has traditionally been accomplished using a single- or double-layer appositional pattern, or by inverting suture patterns using absorbable suture material. A single-layer appositional closure is always preferred if the bladder wall is thick. Even in normal bladders, a single-layer appositional suture pattern (simple continuous [preferred] or simple interrupted) is adequate. A retrospective study found no difference in complication rates between urinary bladders closed with a double-layer inverting pattern versus a single-layer appositional pattern.[3] Luminal penetration is common in thin-walled bladders, but this is not believed to be associated with formation of calculus if absorbable monofilament suture is used. If hemorrhage is expected to be severe, suturing the bladder mucosa as a separate layer (in a simple continuous suture pattern) may be considered to decrease postoperative bleeding (see p. 696 for a discussion of suture materials).

Isolate the bladder from the rest of the abdominal cavity by placing moistened laparotomy pads beneath it. Place stay sutures on the bladder apex and trigone to facilitate manipulation (Fig. 25.8A). Make a longitudinal incision in the ventral aspect of the bladder, away from the ureters and urethra, and between major blood vessels. Remove urine by suction or perform intraoperative cystocentesis before cystotomy if suction is not available. Excise a small section of the bladder mucosa adjacent to the incision to submit for aerobic culture. Check the bladder apex for a diverticulum, and excise it if necessary. Examine the mucosa for defects or lesions, and pass a catheter down the urethra to check for patency. Close the bladder in a single layer using a continuous suture pattern with absorbable suture material (see previous discussion and Fig. 25.8B). If the dog has severe bleeding tendencies, consider suturing the mucosa as a separate layer with a simple continuous suture pattern.

NOTE When removing cystic calculi, be sure to catheterize the urethra and flush until you are certain that the urethra is free of calculi. Leaving stones in the urethra is a common error. Perform postoperative abdominal radiographs following removal of radiodense uroliths to confirm that no stones have been left behind.

Cystostomy (Prepubic Catheterization)

Temporary cystostomy or prepubic catheterization is performed to provide cutaneous urinary diversion in animals with urinary obstruction or with traumatized or surgically repaired urethras. It also may be advisable for animals with bladder atony secondary to neurologic disease or for prevention of overdistention of the bladder after surgery. Cystostomy may be performed by placing a Foley catheter (6–20 Fr, depending on the size of the animal)

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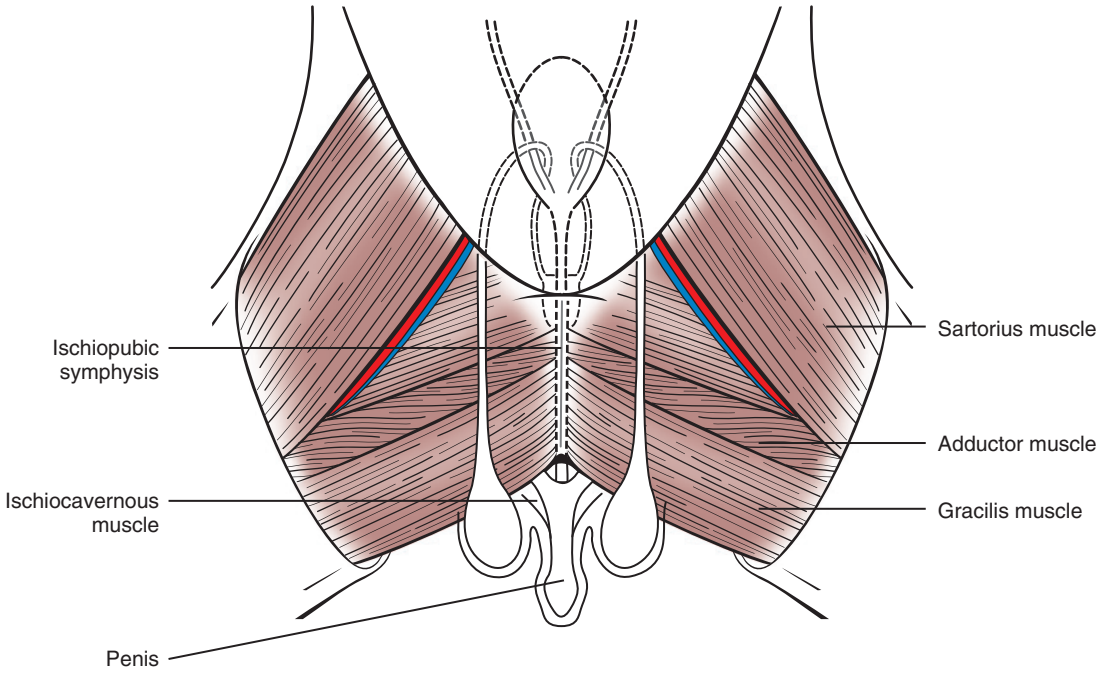


FIG. 25.7 Anatomy of the bladder, urethra, and reproductive system in male cats.

via a small midline abdominal incision, through a muscle-splitting inguinal approach, by laparoscopy, or percutaneously. Stamey Malecot catheters (10–14 Fr) are used for percutaneous placement; however, premature removal of this catheter may occur. Therefore surgically placed Foley catheters are often preferred for long-term catheterization in ambulatory patients. Use of low-profile gastrostomy tubes for long-term or permanent cystostomy in dogs and cats has been described and appears to be well tolerated. Regular gastrostomy (Pezzer; see p. 98) tubes have also been used for prepubic catheters. All catheter types can be placed using local analgesia with or without sedation depending on the stability of the animal; however, surgical placement is preferred. The Stamey Malecot catheter can be removed by gentle traction within 3 or 4 days after placement without risk of urinary leakage; however, it is recommended that a Foley catheter be left in at least 5 to 7 days.

To place a Foley catheter (Fig. 25.9A), make a small midline incision caudal to the umbilicus in females or adjacent to the prepuce in males. Alternatively, consider a 2- to 3-cm oblique inguinal approach directly over the bladder. Locate the bladder, and place stay sutures and a purse-string suture into the bladder wall (Fig. 25.10A). Place the tip of the Foley catheter into the abdominal cavity through a separate stab incision in the abdominal wall (see Fig. 25.10B). Make a small stab incision into the bladder (within the purse-string suture), and place the Foley catheter into the bladder lumen. Inflate the balloon with saline, and secure the catheter within the lumen by tying the purse-string suture (see Fig. 25.10C). Pexy the bladder to the body wall with several interrupted absorbable sutures (see Fig. 25.10D). Close the initial incision, and secure the catheter to the skin using a Chinese finger-trap suture (see Fig. 30.10).

For a Stamey Malecot catheter (see Fig. 25.9B), place the dog in right- or left-lateral recumbency and prepare the ventrolateral aspect of the caudal abdominal wall. Do not evacuate the bladder before catheter placement. Make a small skin incision over the bladder, and with the stylet securely fixed within the catheter (with the Malecot wings twisted flat), direct it through the stab incision. Thrust the catheter into the bladder lumen, making sure that the entire flanged portion of the catheter is within the bladder lumen (once urine is obtained, advance the catheter 1 cm farther). Release the Luer-Lok to open the Malecot wings, and remove the obturator. Secure the catheter to the skin.

Intrapelvic Urethral Anastomosis

The intrapelvic urethra may be torn secondary to pelvic fracture or other trauma, or it may be damaged during urethral catheterization or surgery. Urethrectomy associated with inadvertent prostectomy is an uncommon complication following abdominal cryptorchidectomy, yet requires urethral anastomosis for salvage. Primary suture repair of a completely transected urethra is indicated whenever possible. Dependent on size, small lacerations or partial ruptures may heal if urine is diverted through a urethral catheter or tube cystostomy for 7 to 21 days.

Perform a caudal ventral midline abdominal incision and, if necessary, a pubic symphysiotomy or bilateral pubic and ischial osteotomy (see later). Locate the transected ends of the urethra and debride them. Minimize dissection around the urethra and bladder to prevent damage to the vascular or nerve supply to these structures (Fig. 25.11). Suture the ends with six to eight absorbable interrupted sutures over a transurethral catheter (preferably a Foley catheter or other soft catheter). Leave the catheter in place for 7 to 10 days. If the urethral tissues do not hold suture because of prolonged urine extravasation and subsequent tissue devitalization, delayed repair is indicated. *Place a transurethral catheter to divert urine flow for 5 to 7 days. If a catheter cannot be placed from the penile orifice into the bladder, pass a catheter from the bladder into the traumatized tissue, tie it to a catheter placed from the penile urethral orifice, and use it to pull the penile catheter into the bladder. If the urethra does not heal completely in 7 to 10 days, or if stricture occurs, resect the urethral ends and suture them over a catheter, as described for primary repair.*

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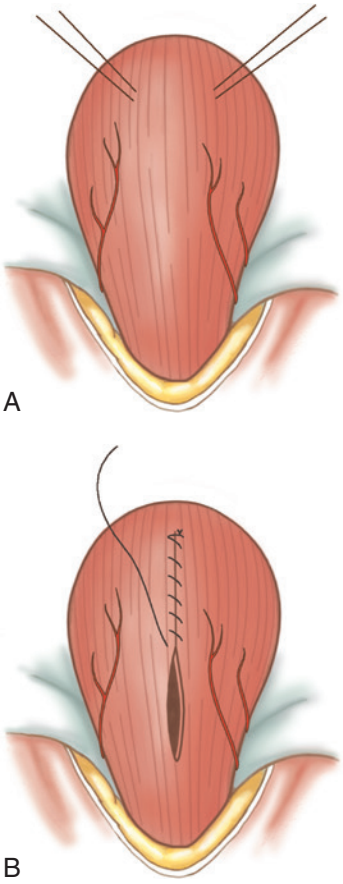


FIG. 25.8 Cystotomy is indicated to remove calculi, repair trauma, resect or biopsy neoplasms, or correct congenital abnormalities. (A) Isolate the bladder and place stay sutures in it to facilitate manipulation. Make the incision in the dorsal or ventral aspect of the bladder. (B) Use a simple continuous suture to close the incision. If the bladder is thin and if leakage occurs, a two-layer closure can be used, but this is seldom necessary.

Prepubic catheterization (tube cystostomy) can also be used to provide continuous urinary diversion while the urethra is healing, but one must ensure that bladder distention is not allowed, or urethral flow of urine will occur.

Adequate urethral exposure can be obtained in some dogs by splitting the pubic symphysis at the midline. In other dogs, the cranial aspect of the pubis may need to be removed. Bilateral pubic and ischial osteotomy allows exposure of the entire urogenital tract in female dogs. *Make a ventral midline incision from the umbilicus to the vulva. Perform a celiotomy from the umbilicus to the pubis, then sharply separate the adductor muscles on the midline of the pubis and ischium. Subperiosteally elevate the adductor muscles until the obturator nerves and half of the obturator foramen are exposed (Fig. 25.12A). Transect the prepubic tendon along the left pubis to the proposed pubic osteotomy site. Predrill holes in the pubis and ischium on both sides of the four proposed osteotomy sites and craniocaudally along the left pubis (see Fig. 25.12B). Osteotomize the pubis, and elevate the internal obturator muscle from the left pubis and ischium, allowing reflection of the entire central bony plate to the right (see Fig. 25.12C). To close the osteotomy sites, preplace orthopedic wire through the previously drilled holes on the right side. Then, before replacing the bone plate, place sutures through the line of holes in the left pubis and ischium, through the left internal obturator muscle, and back through the adjacent holes in the pubis or ischium. Place orthopedic wire through the left osteotomy sites, then secure the preplaced wires and sutures (see Fig. 25.12D). Reappose the adductor muscles and the prepubic tendon before closing the linea alba.*

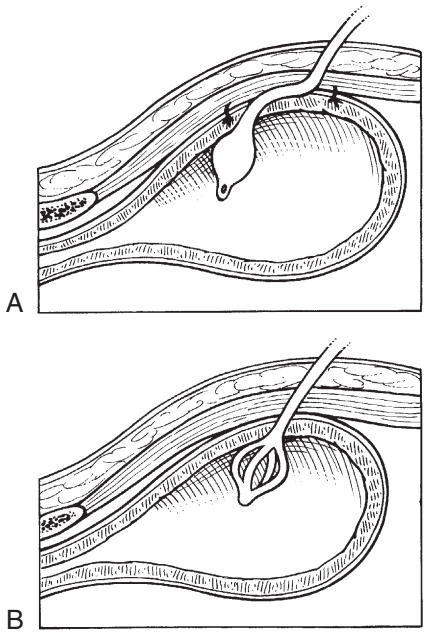


FIG. 25.9 Temporary cystostomy or prepubic catheterization may be performed by placing (A) a Foley catheter or (B) a Stamey Malecot catheter into the bladder.

Urethrotomy

Urethrotomy is performed in male dogs to remove urethral calculi that cannot be retrohydropropulsed into the bladder (see p. 703) and to facilitate placement of catheters into the bladder. Occasionally, urethrotomy is performed for a biopsy of obstructive lesions (i.e., strictures, scar tissue, and neoplasms). Prescrotal or perineal urethrotomy may be performed depending on the level of the obstructive lesion.

NOTE To prevent possible postoperative urethral stricture, cystotomy is preferred over urethrotomy if calculi can be advanced back into the bladder by retrohydropropulsion.

Prescrotal Urethrotomy

Prescrotal urethrotomy (Fig. 25.13) is used to remove calculi from the distal penile urethra in dogs, or to place Foley catheters into the urinary bladder if the catheter is of sufficient length and if the obstruction is distal to the proposed urethrotomy incision. Occasionally, urethrotomy can be performed under local anesthesia with opioid sedation in severely depressed or uremic patients. Prescrotal urethrotomies can be left to heal by secondary intention; however, hemorrhage should be expected from the surgical site for 3 to 5 days (particularly during urination). Therefore primary closure is preferred if the mucosa is healthy and if adequate apposition of the urethral mucosa can be achieved, as this decreases postoperative bleeding.

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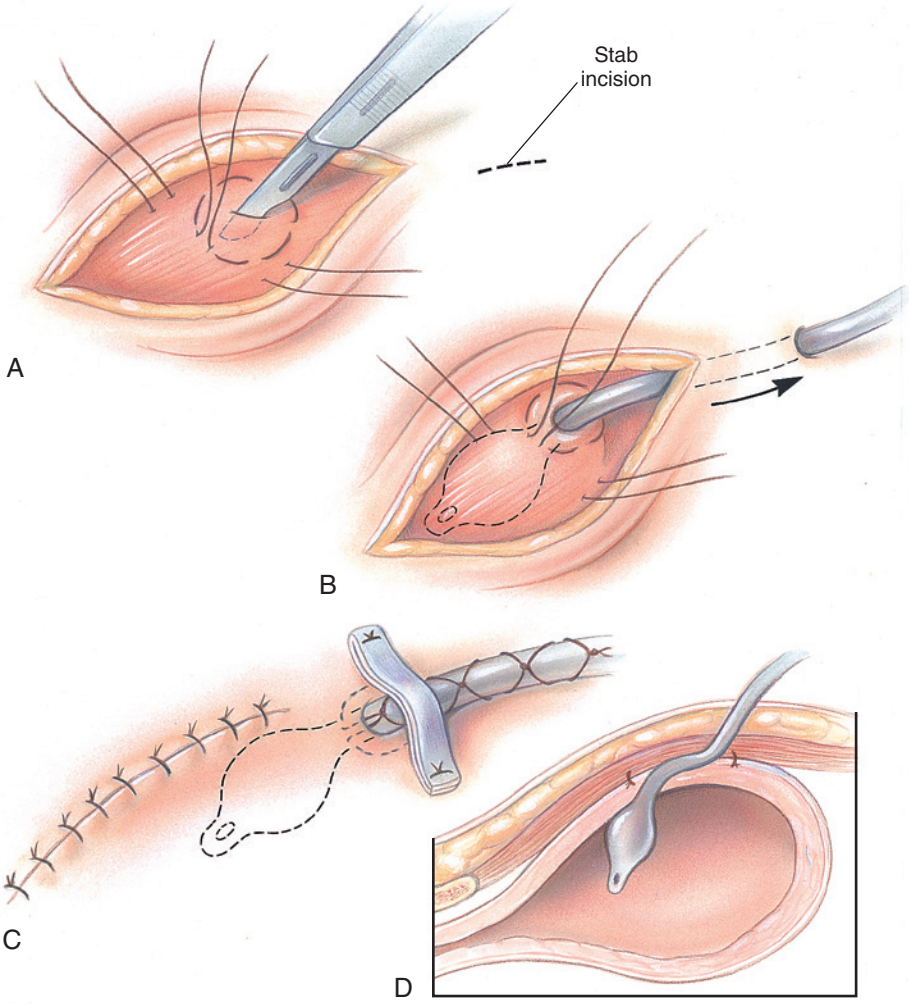


FIG. 25.10 (A) To place a Foley catheter, make a small incision and locate the bladder. Place stay sutures and a purse-string suture in the bladder. Place the tip of the Foley catheter into the abdominal cavity through a separate stab incision in the abdominal wall. (B) Make a small stab incision into the bladder, and place the Foley catheter into the bladder lumen. (C) Inflate the balloon with saline, and secure the catheter within the lumen by tying the purse-string suture around it with a Roman sandal suture (see Fig. 30.10). (D) Tack the bladder to the body wall with several absorbable sutures.

With the dog in dorsal recumbency, place a sterile catheter into the penile urethra to the scrotum or to the obstruction. Make a ventral midline incision through the skin and subcutaneous tissue between the caudal aspect of the os penis and the scrotum. Identify, mobilize, and retract the retractor penis muscle laterally to expose the urethra (Fig. 25.14). Using a No. 11 or 15 scalpel blade, make an incision into the urethral lumen over the catheter (Fig. 25.15). Use iris scissors to extend the incision, if necessary. Remove calculi with forceps, and gently flush the urethra with warm saline. Leave the incision to heal by secondary intention, or preferably close the urethra with simple interrupted or continuous appositional absorbable sutures (4-0 or 5-0). Place the first layer in the urethral mucosa and corpus spongiosum, then appose subcutaneous tissue and skin with simple interrupted sutures or a continuous subcuticular suture pattern.

Remove the urinary catheter following surgery, regardless of whether the urethra is sutured.

Perineal Urethrotomy

Perineal urethrotomy (Fig. 25.16) is occasionally used to remove calculi lodged at the ischial arch and to place catheters into the bladder of large male dogs. Perineal urethrotomy is less commonly indicated than urethrotomy at other sites. This urethrotomy site should be closed to prevent subcutaneous urine leakage.

Place a purse-string suture in the anus. Place a sterile catheter into the urethra to the level of the bladder or the site of the obstruction. With the dog in sternal recumbency and the rear limbs hanging over the edge of the table, make a midline incision over the urethra, midway between the scrotum and the anus. Identify the retractor penis muscle, elevate it, and retract it (Fig. 25.17A). Separate the paired bulbospongiosus muscles at their raphe to expose the corpus spongiosum, then incise the corpus spongiosum to enter the urethral lumen (see Fig. 25.17B–C). Close the incision as just described for prescrotal urethrotomy (see Fig. 25.17D).

Urethrostomy

Urethrostomy is indicated for (1) recurrent obstructive calculi that cannot be managed medically; (2) calculi that cannot be removed by retrohydropropulsion or urethrotomy; (3) urethral stricture; (4) urethral or penile neoplasia or severe trauma; and (5) preputial neoplasia requiring penile amputation. Depending on the site of the lesion, ureterostomy can be prescrotal, scrotal, perineal, or prepubic in dogs. Scrotal urethrostomy is preferred if castration is an option and if the lesion is distal to the scrotum. Perineal urethrostomy is routinely performed in cats; however, prepubic and subpubic urethrostomy procedures are also described.

Prescrotal Urethrostomy

Prescrotal urethrostomy is performed similarly to prescrotal urethrotomy, except that the urethral mucosa is sutured to the skin. *Make a 3- to 4-cm incision in the urethral mucosa as described*

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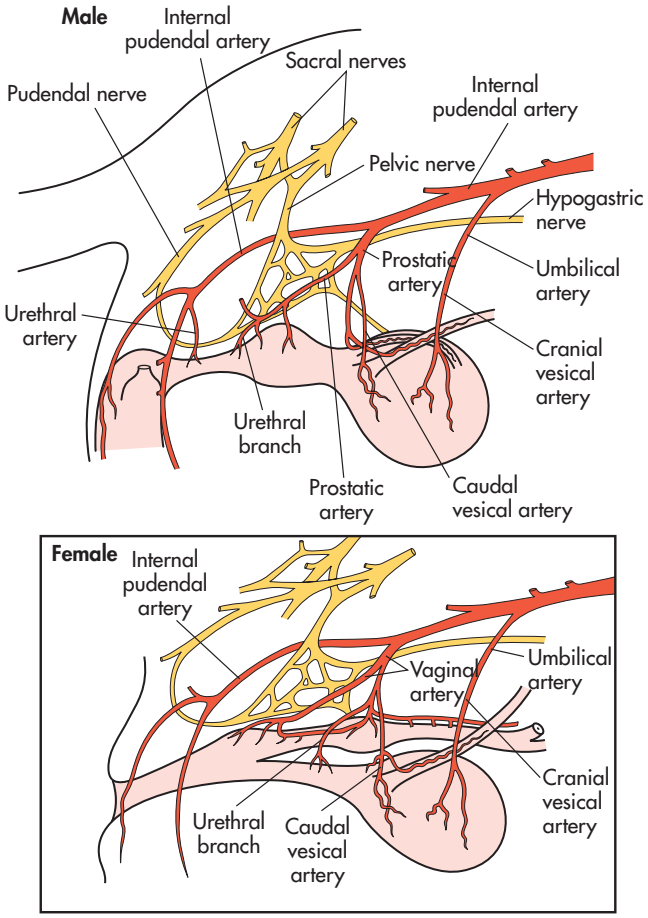


FIG. 25.11 Vascular and nerve supply to the bladder and urethra.

on p. 688. The length of the urethral incision should be six to eight times its luminal diameter. Periurethral sutures can be placed to the subcutaneous tissue using a simple continuous suture pattern of absorbable suture material. *Place simple interrupted absorbable sutures (3-0 to 5-0) from the urethral mucosa to the skin, beginning at the caudal aspect of the incision. Suture the remainder of the urethral mucosa to the skin with simple interrupted or simple continuous sutures (Fig. 25.18). Suture skin at either end of the incision with simple interrupted sutures.*

Scrotal Urethrostomy

Scrotal urethrostomy (Fig. 25.19) is preferred over perineal or prepubic urethrostomy because the urethra is wider and more superficial and is surrounded by less cavernous tissue here than at other sites. Therefore postoperative hemorrhage is often less than with other techniques, and stricture is less likely.

If the dog is intact, castrate him and excise the scrotum; otherwise, perform a scrotal ablation (Fig. 25.20A). Place a sterile catheter into the urethra to the level of the ischial arch or beyond. Make a midline incision over the urethra through the subcutaneous tissue. Identify the retractor penis muscle, mobilize it, and retract it laterally to expose the urethra. Using a No. 11 or 15 scalpel blade, make a 3- to 4-cm incision into the urethral lumen over the catheter (see Fig. 25.20B). Suture the urethra as described above for prescrotal urethrostomy (see Fig. 25.20C).

Canine Perineal Urethrostomy

Perineal urethrostomy often causes unacceptable urine scalding and is used only in dogs that have urinary problems that will not be solved by a scrotal or prescrotal urethrostomy. The surrounding cavernous tissue is large at this location, and hemorrhage can be profuse. Furthermore, the urethra is less superficial here,

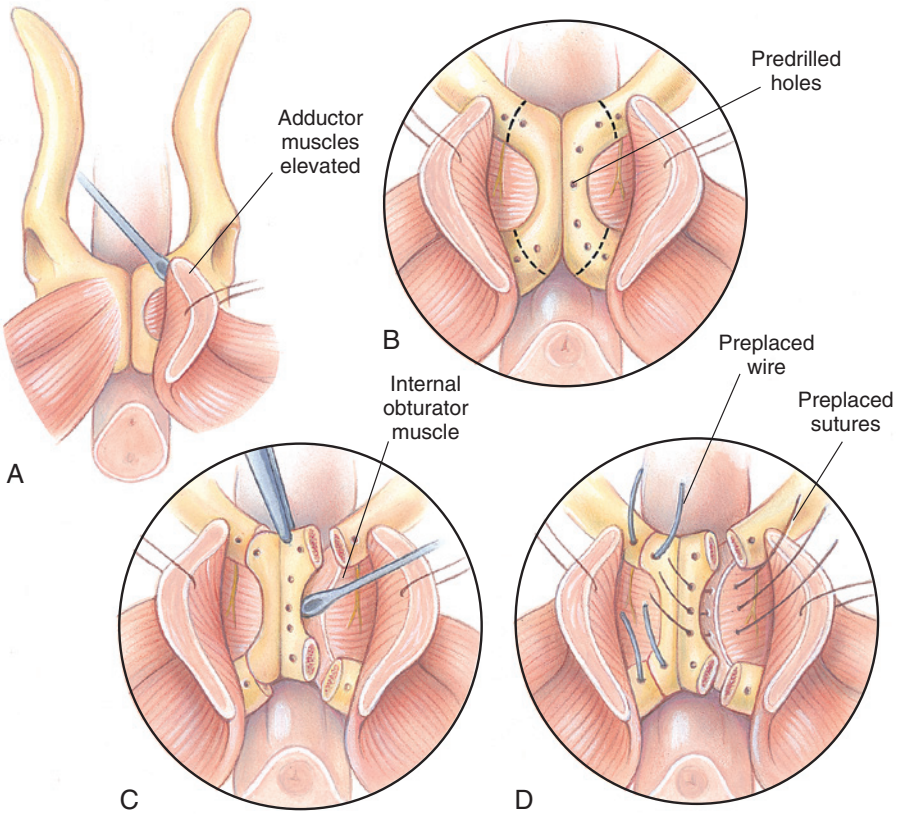


FIG. 25.12 (A) For bilateral pubic and ischial osteotomy, elevate the adductor muscles until the obturator nerves and half of the obturator foramen are exposed. (B) Predrill holes in the pubis and ischium on both sides of the four proposed osteotomy sites and craniocaudally along the left pubis. (C) Osteotomize the pubis and elevate the internal obturator muscle from the left pubis and ischium, allowing reflection of the entire central bony plate. (D) Close the osteotomy with orthopedic wire.

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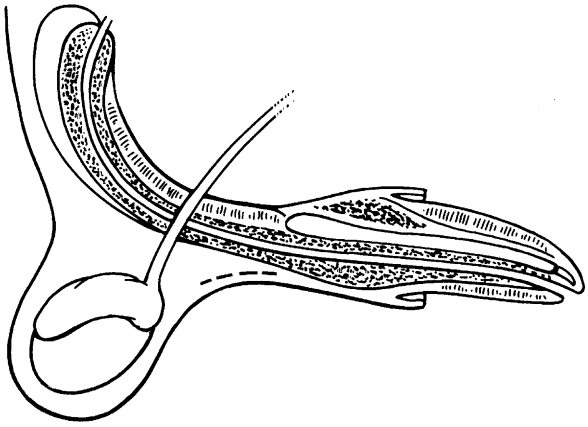


FIG. 25.13 Prescrotal urethrotomy.

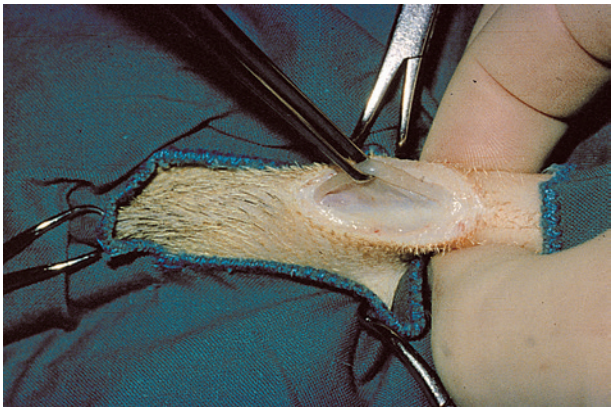


FIG. 25.14 For prescrotal urethrotomy, make a ventral midline incision through the skin and subcutaneous tissues, between the caudal aspect of the os penis and scrotum. Identify, mobilize, and retract the retractor penis muscle laterally to expose the urethra.

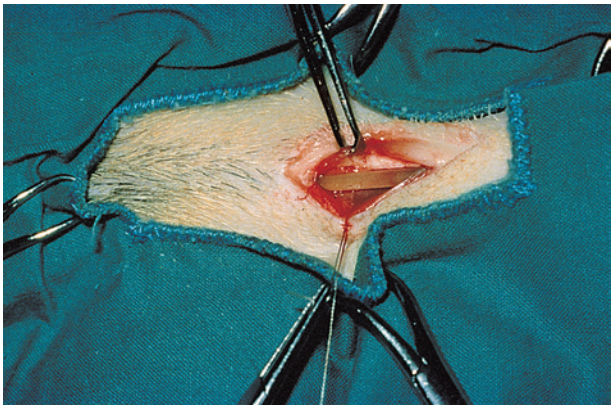


FIG. 25.15 Use a No. 11 or 15 scalpel blade to make an incision into the urethral lumen over the catheter.

and mobilizing it can result in excessive suture-line tension, causing dehiscence.

Make a 4- to 6-cm incision in skin and overlying tissue, and incise the perineal urethra as described for perineal urethrotomy. The urethral incision should be 1.5 to 2 cm in length. *Suture the urethral mucosa to the skin, as described for prescrotal urethrostomy (Fig. 25.21).*

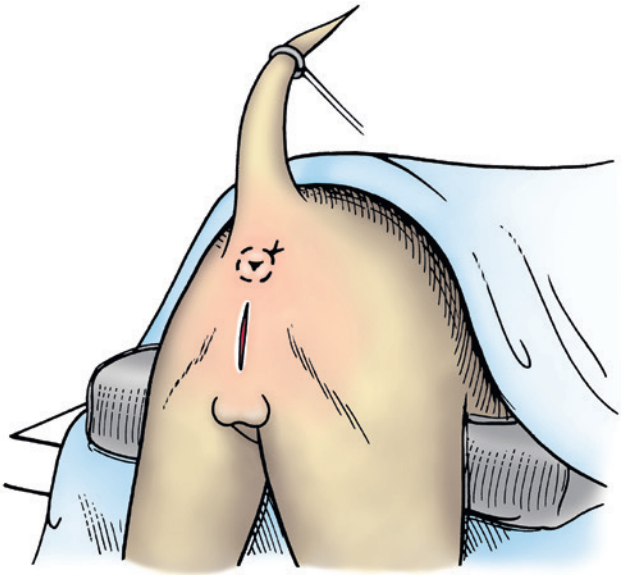


FIG. 25.16 Perineal urethrotomy.

Feline Perineal Urethrostomy

Perineal urethrostomy is indicated to prevent recurrence of obstruction in male cats or to treat obstruction that cannot be eliminated by catheterization. It is also useful for treating strictures secondary to urethral obstruction and catheterization.

Place a purse-string suture in the anus, and catheterize the penis if possible. Place the cat in dorsal or ventral recumbency. If the cat is placed in dorsal recumbency, pull the pelvic limbs forward to improve access to the perineal region. Dorsal recumbency allows for cystotomy and perineal urethrostomy without having to reposition the cat. *Make an elliptical incision around the scrotum and prepuce, and excise them. Place an Allis tissue forceps on the end of the prepuce or around the catheter to help manipulate the penis. Free the penis and the distal urethra from the surrounding tissue on either side (Fig. 25.22A). Extend the dissection ventrally and laterally toward the penile attachments at the ischial arch. Elevate the penis dorsally, and sharply sever the ventral penile ligament. Then, transect the ischiocavernosus muscles (see Fig. 25.22B) and the ischiourethralis muscles at their insertion on the ischium to avoid damaging branches of the pudendal nerves and to minimize hemorrhage. Reflect the penis ventrally to expose the dorsal surface. Locate the bulbourethral glands proximal and dorsal to the bulbospongiosus muscle and cranial to the severed ischiocavernosus and ischiourethralis muscles (see Fig. 25.22C).* Avoid excessive dorsal dissection to prevent damage to nerves and vessels supplying the urethral muscle. *Elevate and remove the retractor penis muscle over the urethra, and longitudinally incise the penile urethra using a No. 11 blade or sharp tenotomy scissors. Continue the urethral incision proximal to the pelvic urethra to the level of the cranial extent of the bulbourethral glands. Pass a closed Halsted mosquito hemostat up the urethra to ensure that the urethral width is adequate.* The hemostat should be able to be passed to the level of the box locks without resistance. Alternatively, a 5- to 8-Fr soft catheter can be passed through the urethrostomy incision. *Suture the urethral mucosa to the skin using 4-0 or 5-0 absorbable (polydioxanone [PDS], polyglyconate [Maxon]) or nonabsorbable*

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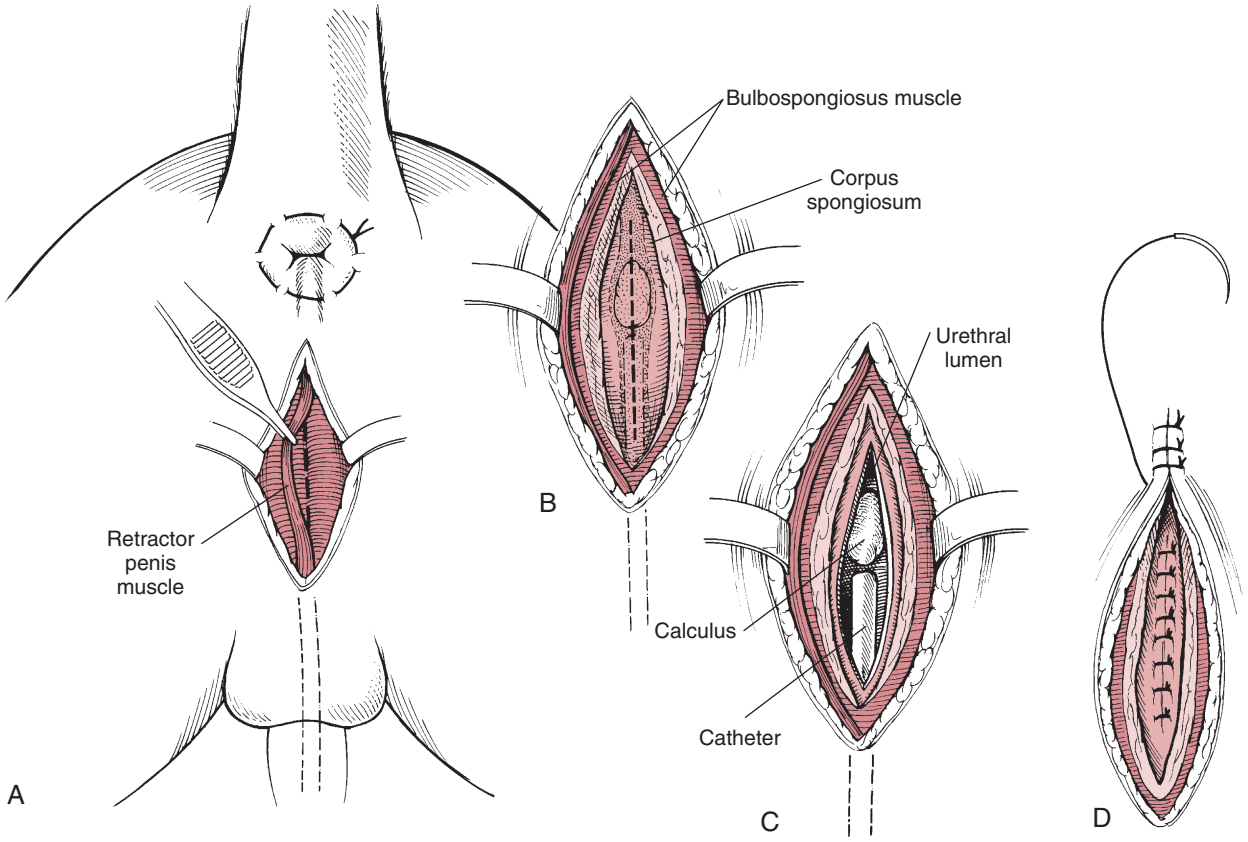


FIG. 25.17 For perineal urethrotomy, make a midline incision over the urethra, midway between the scrotum and the anus. (A) Identify the retractor penis muscle, elevate it, and retract it. (B) Separate the paired bulbospongiosus muscles at their raphe to expose the corpus spongiosum. (C) Incise the corpus spongiosum to enter the urethral lumen. (D) Close the urethra with simple interrupted absorbable sutures. Place the first layer in the urethral mucosa and corpus spongiosum; appose subcutaneous tissue and skin with simple interrupted sutures or a continuous subcuticular suture pattern.

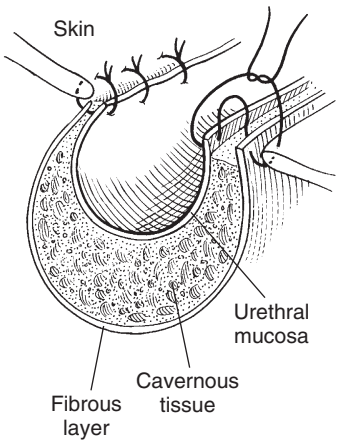


FIG. 25.18 For urethrostomy, place simple interrupted absorbable sutures from the urethral mucosa to the skin. To improve hemostasis, avoid incorporating cavernous tissue in the sutures.

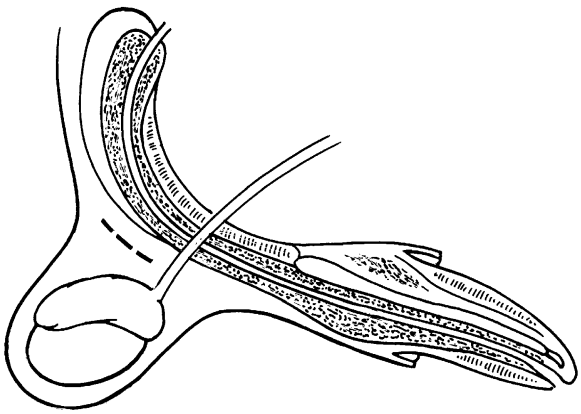


FIG. 25.19 Scrotal urethrostomy.

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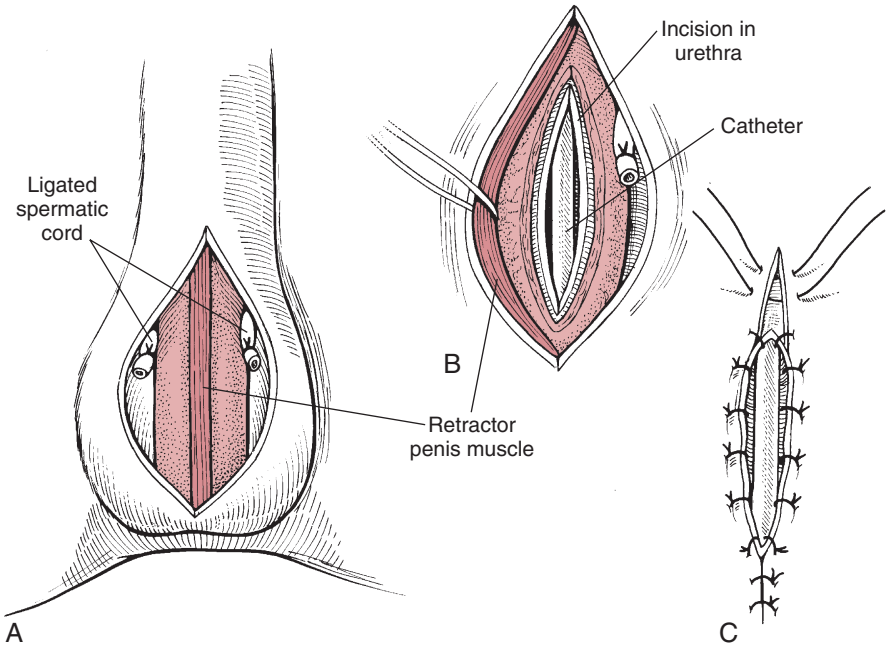


FIG. 25.20 Scrotal urethrostomy is preferred over other sites because less hemorrhage occurs. Perform a scrotal ablation. (A) Make a midline incision over the urethra through the subcutaneous tissue. Identify the retractor penis muscle and mobilize and retract it laterally to expose the urethra. (B) Using a No. 11 or 15 scalpel blade, make a 3- to 4-cm incision into the urethral lumen over the catheter. (C) Suture urethral mucosa to skin with simple interrupted sutures.

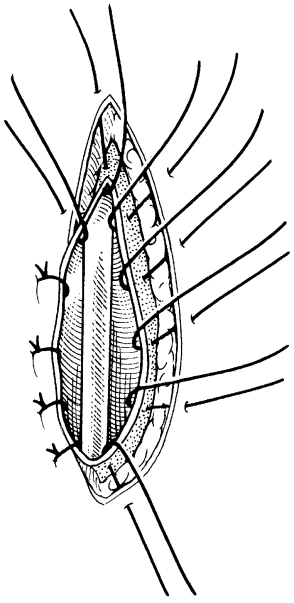


FIG. 25.21 For urethrostomy, place simple interrupted absorbable sutures from the urethral mucosa to the skin beginning at the caudal aspect of the incision. Suture the remainder of the urethral mucosa to the skin with simple interrupted sutures. Suture the skin at either end of the incision with simple interrupted sutures.

(nylon [e.g., Monosof], polypropylene [Prolene]) suture on a tapercut, swaged-on needle with a simple interrupted or a simple continuous suture pattern. Be sure to suture the urethral mucosa to the skin (it is sometimes difficult to identify mucosa). First, place the most proximal sutures at a 45-degree angle to the skin, then place the remainder of the sutures (see Fig. 25.22D). Suture the proximal two-thirds of the penile urethra to the skin, and amputate the distal end by placing a horizontal mattress suture through the skin and penile tissues and by severing the penis distal to this ligature. Close the remaining skin with simple interrupted sutures (Fig. 25.23).

Prepubic Urethrostomy

Prepubic (antepubic) urethrostomy is an uncommon salvage procedure performed when damage to the membranous or penile urethra is irreparable, or when removal of these tissues is necessary (i.e., neoplasia). Unless nerve damage occurs (e.g., following prostatectomy), most animals are continent following this procedure.

Make a ventral midline incision from the umbilicus to the pubis. Free the intrapelvic urethra from the pelvic floor using blunt dissection. Be sure to preserve the urethral artery and its branches. Sever the distal aspect of the intrapelvic urethra. It may be necessary to carefully dissect the prostate from the urethra to ensure that ample urethra is available to exteriorize in some male dogs. Preserve the blood supply to the neck of the bladder. In male dogs, exteriorize the urethra through a small stab incision 2 to 3 cm lateral to the prepuce or within the prepuce. In females, exteriorize the urethra through the ventral midline incision or 2 to 3 cm lateral to the linea alba (Fig. 25.24A). Spatulate the distal end of the urethra to increase the luminal diameter (see Fig. 25.24B), then suture the urethral mucosa to the skin with interrupted sutures of absorbable or nonabsorbable monofilament suture. Be sure that little tension is placed on the urethrostomy site and that the urethra is not bent sharply.

Subpubic Urethrostomy

Subpubic urethrostomy is similar to prepubic urethrostomy, except that the urethra is exteriorized caudal to the brim of the

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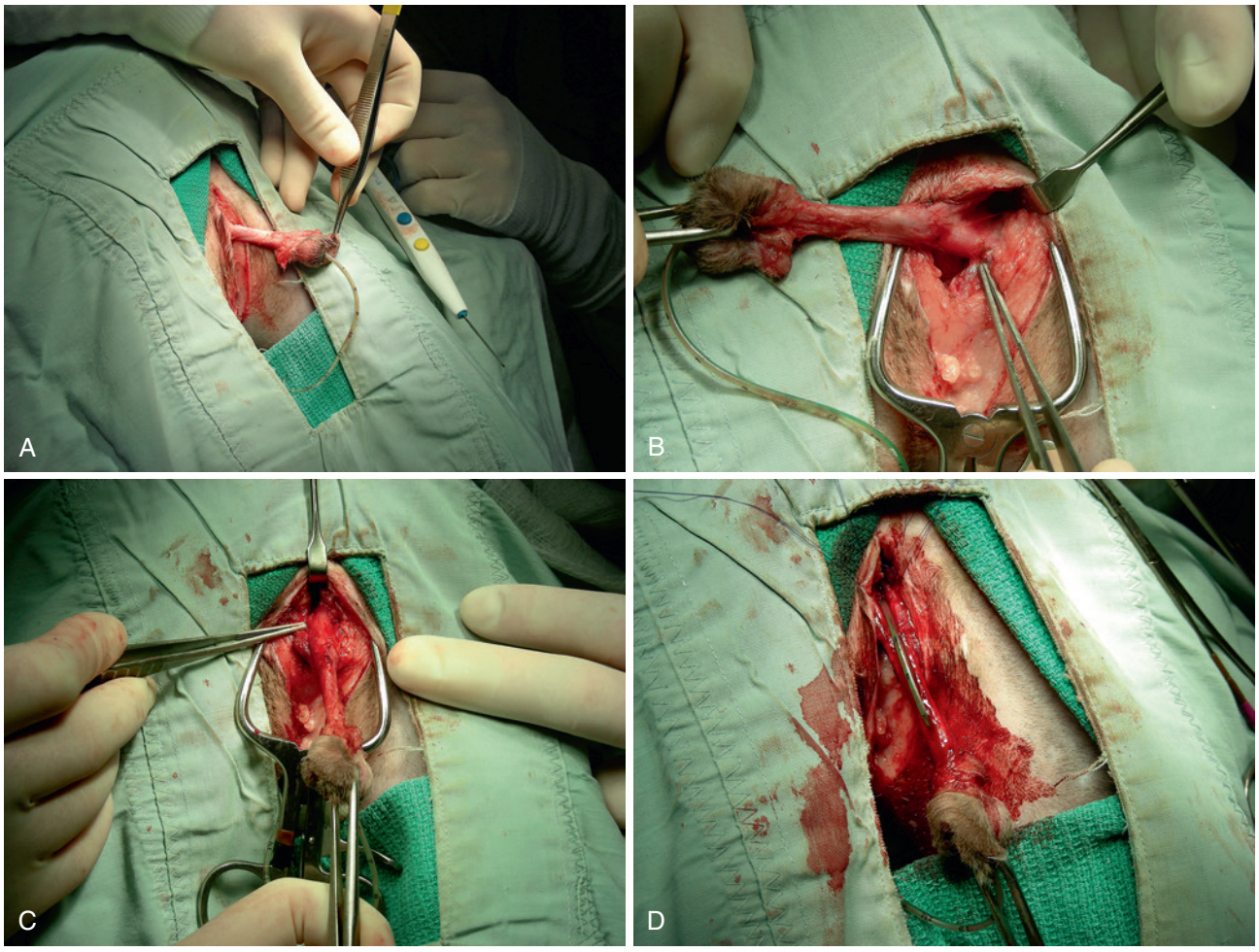


FIG. 25.22 Perineal urethrostomy may be performed in male cats with urinary obstruction. (A) Free the penis and the distal urethra from the surrounding tissues. (B) Identify and transect the ischiocavernosus muscles and the ischiourethralis muscles at their origin on the ischium to avoid damaging branches of the pudendal nerves and to minimize hemorrhage. (C) Locate the bulbourethral glands to use as a landmark for the level of the proximal extent of the urethral incision. (D) Identify the urethral mucosa, and suture the most dorsal aspect to the skin first; then place additional sutures from the proximal two-thirds of the penile urethra to the skin.



FIG. 25.23 Appearance of a completed perineal urethrostomy in a cat.

pubis. In cats, this procedure may be less likely to cause postoperative stricture, recurrent UTI, or chronic urine-scald dermatitis. It is indicated when repeated stricture occurs after perineal urethrostomy.

Perform this procedure similarly to the one described previously, but retract the skin caudally past the brim of the pubis. Expose the medial boundary of the obturator foramen by elevating the adductor muscle and the cranial portion of the gracilis muscle from the periosteum of the pubis. Partially incise the prepubic tendon, and reflect it laterally to expose the pubic rami (Fig. 25.25). Osteotomize the pubic rami 1.5 cm lateral to the pubic symphysis. Make a transverse incision through the body of the pubic bone and across the pubic symphysis. Rotate the pubic flap ventrally to visualize the intrapelvic urethra. Transect the urethra cranial to the lesion (i.e., stricture) and replace the pubic flap (Fig. 25.26A). Reappose the muscular aponeuroses of the gracilis and adductor muscles with interrupted or horizontal mattress sutures. Make a 1-cm stab incision 3 cm distal to the caudal extent of the abdominal incision. Tunnel through the subcutaneous tissue and exteriorize the urethra (see

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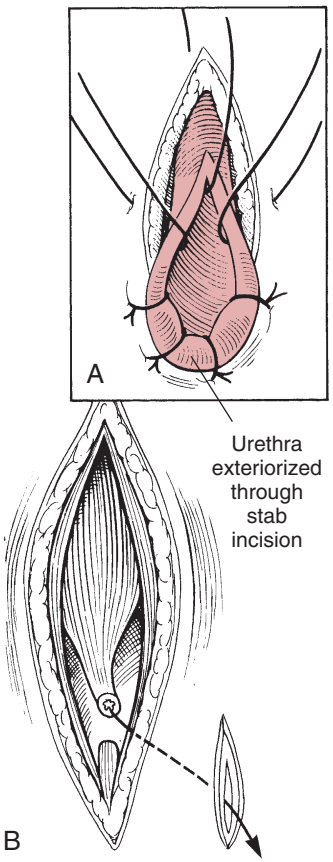


FIG. 25.24 Prepubic urethrostomy may be performed when distal urethral lesions are present. (A) Sever the distal aspect of the intrapelvic urethra and exteriorize it through a small stab incision 2 to 3 cm lateral to the linea alba. (B) Spatulate the distal end of the urethra to increase the luminal diameter, and suture the urethral mucosa to the skin with interrupted sutures.

Fig. 25.26B). Spatulate the urethral end, and suture it to the skin with 4-0 suture material. Close the abdominal incision, but leave the caudal 1 cm of the linea alba open to prevent crimping of the urethra as it passes over the pubic flap. If indicated, the strictured perineal urethrostomy site is resected, and the tissues are closed or left open to heal by secondary intention.

Transpelvic Urethrostomy

An alternative technique to prepubic or subpubic urethrostomy in male cats involves removal of a portion of the ischium to exteriorize the urethra ventrally. Good success has been reported in a limited number of cases.

Position the cat in dorsal recumbency with the feet secured to the surgical table in a cranial position. Make a small caudal ventral median celiotomy, and expose the urinary bladder 2 cm cranial to the cranial pubic margin. Make a small incision into the bladder, aspirate urine from it, and lavage the bladder with sterile saline. Pass a 6-Fr urinary catheter into the bladder, and advance it into the proximal urethra to the obstruction site. Secure the catheter to the bladder wall with a temporary purse-string suture. Excise the scrotum and prepuce using an elliptical skin incision that extends to the cranial margin of the pubis. Extend the penis caudally, and denude its ventral surface. Remove fat to expose the caudal and ventral aspects of the pubic symphysis (Fig. 25.27). Elevate muscles

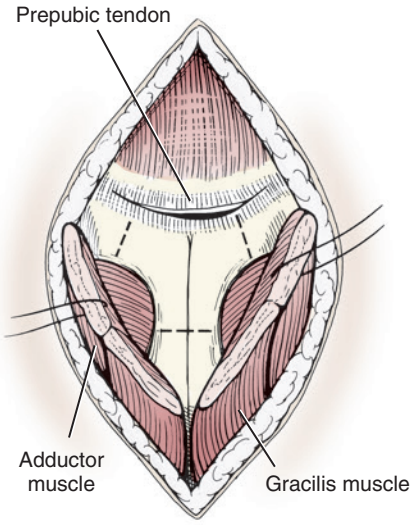


FIG. 25.25 For pubic osteotomy, partially incise the prepubic tendon and reflect it laterally to expose the pubic rami. Osteotomize the pubic rami 1.5 cm lateral to the pubic symphysis. Make a transverse incision through the body of the pubic bone and across the pubic symphysis.

medial to lateral on both sides of the ischium to expose its ventral aspect (approximately 1.5 cm wide and 1.5 cm long). Use bone rongeurs to remove the ischium in a caudocranial direction until an ostectomy area approximately 10 mm wide and 12 mm long has been created. Take care to avoid damage to underlying soft tissue. Palpate the indwelling urethral catheter, and make a ventral longitudinal urethral incision over the catheter from the bulbourethral glands to a point 2 to 3 mm from the cranial margin of the ostectomy. Place simple interrupted 4-0 monofilament sutures from the urethral mucosa to the skin margins. Amputate the portion of the penis distal to the bulbourethral glands. Place additional skin sutures to close the remaining wound. Remove the urinary catheter, and pass it into the urethrostomy to ensure patency. Close the cystotomy and celiotomy incisions routinely.

Permanent Urinary Diversion

AP Permanent urinary diversion may be indicated when neoplasia involves the bladder trigone. After cystectomy, the ureters may be anastomosed to an isolated bowel conduit or reservoir, or into the intact colon, jejunum, ileum, vagina, prepuce, remaining urethra, or ventral abdominal skin (Fig. 25.28).[4–6] Complications associated with ureteral anastomosis in the bowel include reabsorption of electrolytes and nitrogenous waste products, upper UTI, and neurologic dysfunction. Azotemia, hyperammonemia, hyperchloremia, and metabolic acidosis are also common after these procedures. Because it is a salvage procedure commonly associated with life-threatening complications, such reconstructions should be avoided whenever possible. Stenting is typically done to avoid this technique. Urethral stenting has been reported in dogs with both benign and malignant obstructions (Fig. 25.29).[7,8] The most common complication following stent placement is urinary incontinence, although stent migration and tissue ingrowth through the stent may also occur.

Ureterocolonic anastomosis is the most commonly performed technique for permanent urinary diversion. The patient should be fasted for 48 hours, and saline enemas should be given 12 to

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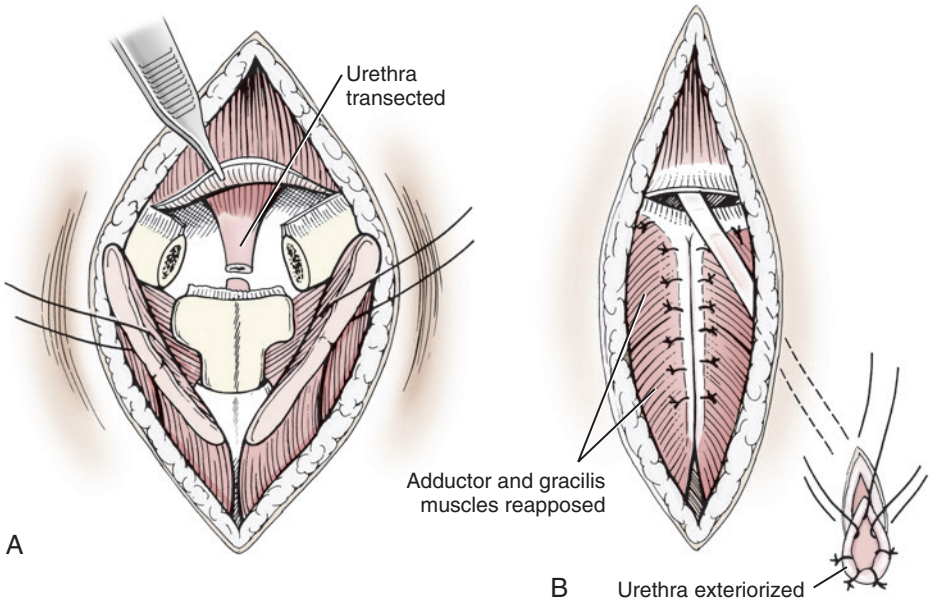


FIG. 25.26 For subpubic urethrostomy, perform a pubic osteotomy (see Fig. 25.24) and rotate the pubic flap ventrally to visualize the intrapelvic urethra. (A) Transect the urethra cranial to the lesion and replace the pubic flap. (B) Exteriorize the urethra through a stab incision, spatulate the urethral end, and suture it to the skin.

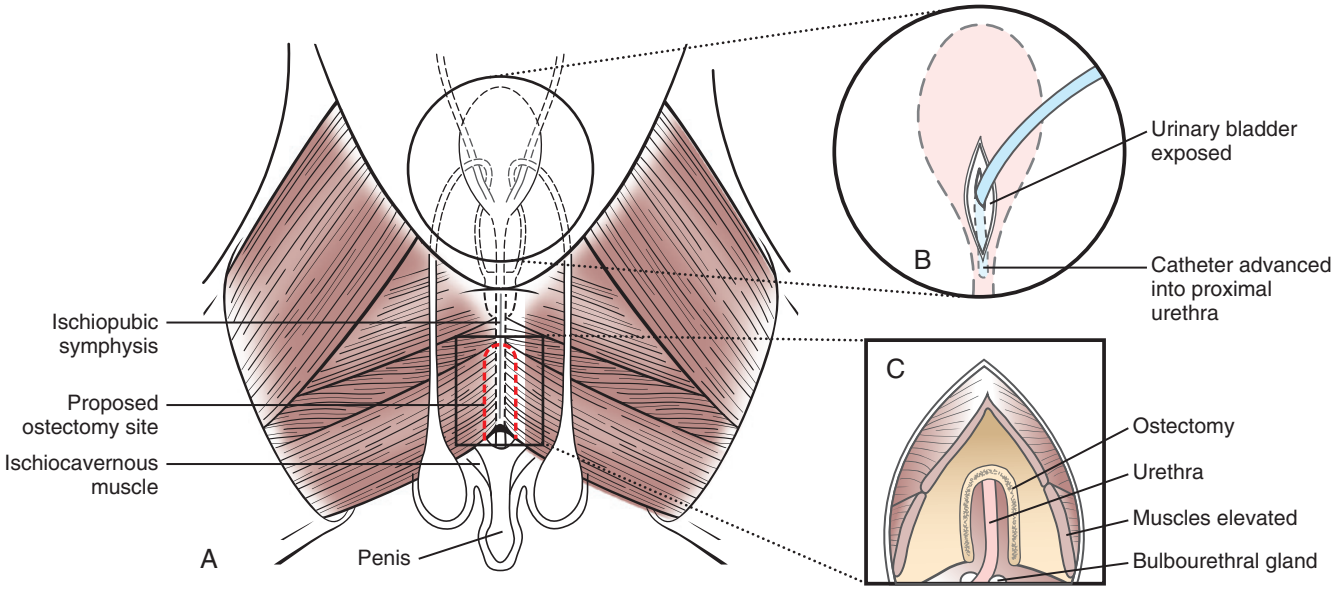


FIG. 25.27 For transpelvic urethrostomy, position the cat in dorsal recumbency. (A) Perform a caudal abdominal laparotomy to expose the bladder and extend the incision to access the ventral aspect of the pelvis. (B) Make a small incision in the urinary bladder to facilitate normograde passage of a urinary catheter. (C) Expose the ischium by elevating the muscles on either side, and perform an ostectomy to access the urethra below.

24 hours before surgery. Prophylactic antibiotics are given and should be continued for at least 8 weeks after surgery.

Excise the bladder and the proximal urethra (1–2 cm distal to the suspected area of neoplasia), and ligate and transect the ureters. Dissect the ureters from their retroperitoneal attachments. Determine the length of the ureters, and choose a site for each to be implanted into the colon. Stagger the sites for anastomosis of the right and left ureters so that they are at different sites in the colon. Express feces from the proposed site of ureteral anastomoses, and place atraumatic forceps on the colon. Make a three-sided seromuscular colonic flap for each ureter (Fig. 25.30A), then create a 4-mm circular defect in the colonic mucosa with tenotomy scissors (Fig. 25.30B). Transect the end of the ureter, spatulate it, and tunnel the ureters through the seromuscular flap into the colonic lumen. Suture the ureter to the colonic mucosa with simple interrupted sutures (5-0 or 6-0 absorbable suture material; Fig. 25.30C).

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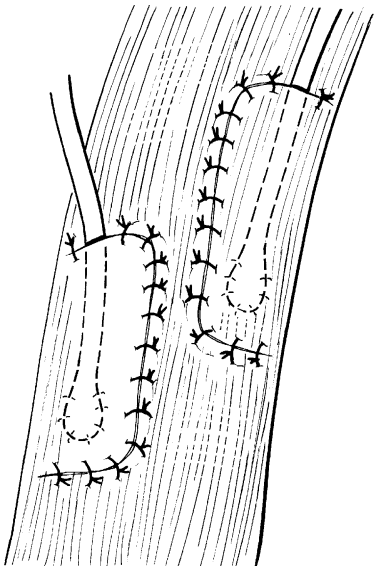


FIG. 25.28 Permanent urinary diversion may be performed by anastomosing the ureters into the intact colon, jejunum, or ileum.

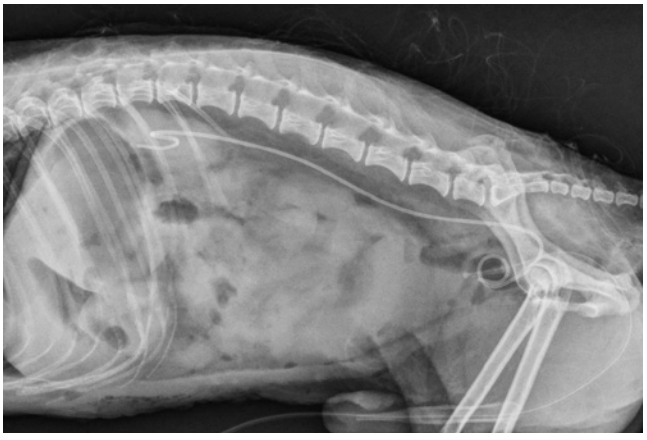


FIG. 25.29 Lateral radiograph of a 12-year-old dog following placement of a urethral stent for malignant obstruction.

Close the flap over the ureter, but be sure to avoid compromising the ureteral lumen (Fig. 25.30D).

HEALING OF THE BLADDER AND URETHRA

Compared with other organs, the urinary bladder heals quickly, regaining 100% of normal tissue strength in 14 to 21 days. Complete reepithelialization of the bladder occurs in 30 days. Substantial portions of the bladder can be safely resected. As long as the trigone is undamaged, the bladder will expand (owing to epithelial regeneration, scar tissue formation and remodeling, hypertrophy, and proliferation of smooth muscle) until it again functions as an effective reservoir in a few weeks. Although numerous materials have been described for bladder replacement, use of porcine small intestine submucosa grafts for replacement of large portions of the bladder has been associated with good success and minimal complications.

If urethral continuity is not completely disrupted, the urethra can heal by regeneration of urethral mucosa in as little as 7 days. Urine extravasation (particularly if infected) delays wound healing and promotes periurethral fibrosis and stricture. Urinary diversion via a urethral catheter or tube cystostomy is therefore indicated for small urethral lacerations (see p. 685). Differences are not typically noted when an indwelling transurethral catheter, a cystostomy catheter, or a combination of the two is used for urinary diversion after transection and anastomosis of the intrapelvic urethra. When complete transection of the urethra occurs, fibrous tissue proliferation is evident in the gaps between the severed ends. Contraction of the fibrous tissue often leads to stricture and urinary obstruction. Primary anastomosis over an indwelling catheter (or proximal urinary diversion) should be performed to decrease the likelihood of stricture formation. The catheter should be left in place for 3 to 5 days.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Absorbable suture materials (e.g., polydioxanone [PDS], polyglyconate [Maxon], polyglactin-910 [Vicryl], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], polyglytone 6211 [Caprosyn]) are preferred for bladder and urethral surgery. Most sutures appear to lose tensile strength faster in alkaline urine (such as that seen with *Proteus* infections) than in infected acidic urine or sterile urine. Polyglycolic acid, polyglactin 910, and poliglecaprone 25 are rapidly degraded in infected urine; polydioxanone, polyglyconate, and glycomer 631 are acceptable for use in sterile bladders and in those infected with *E. coli*. However, use of any suture that is degraded via hydrolysis may be risky when the bladder is infected with *Proteus* spp. (see also p. 61) because monofilament absorbable sutures have been shown to degrade within 7 days in *Proteus mirabilis*-inoculated urine in vitro.

NOTE Nonabsorbable sutures should be avoided in the urinary bladder or urethra as these may promote formation of calculi.

POSTOPERATIVE CARE AND ASSESSMENT

Urination should be closely monitored in patients after urethral surgery to detect obstruction caused by tissue swelling, fibrosis, or necrosis. Following removal of the urinary obstruction, intravenous fluid therapy should be maintained until postobstructive diuresis ceases. Electrolytes should be monitored (particularly potassium) because hypokalemia may occur secondary to diuresis or medical therapy of hyperkalemia. Patients should be monitored for postoperative pain and analgesics provided as necessary (see Chapter 13). Elizabethan collars should be used in patients with indwelling urinary catheters, urethrotomies, or urethrostomies to prevent early catheter removal or self-mutilation. Patients with urethrotomies and urethrostomies should be observed for postoperative hemorrhage. Digital pressure on the surgical site may be necessary to stop bleeding immediately after surgery or after urination (for 3–5 days). Bladder atony may occur after as little as 12 hours of obstruction if the animal is sedated or is given narcotic analgesics postoperatively, or does not void because of pain. The bladder should be kept decompressed by manual expression or by catheterization until the patient is urinating normally.

For cats with urethrostomies, paper instead of gravel litter should be used until the wound is healed, and urine cultures should be performed to check for UTI. An indwelling catheter may promote stricture formation and UTI in cats following surgery; therefore catheter use is not recommended in this

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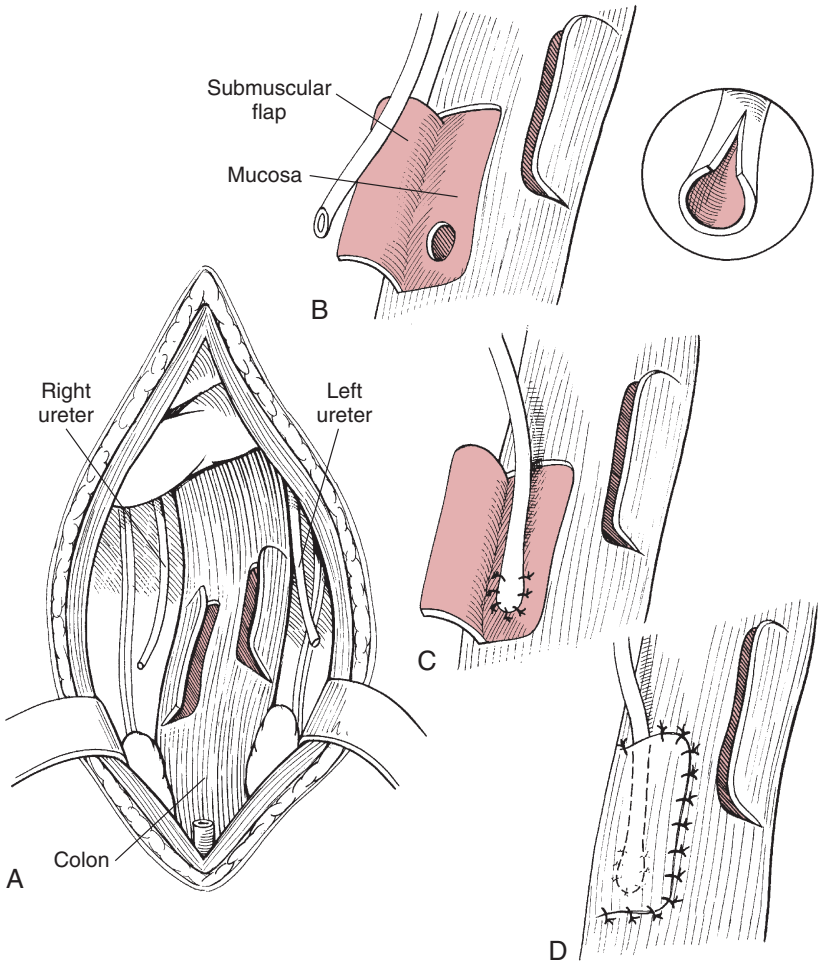


FIG. 25.30 Colonic urinary diversion. (A) Make a three-sided seromuscular flap for each ureter. (B) Create a 4-mm circular defect in the colonic mucosa with tenotomy scissors. Transect the end of the ureter, spatulate it, and tunnel the ureters through the seromuscular flap into the colonic lumen. (C) Suture the ureter to the colonic mucosa with simple interrupted sutures. (D) Close the flap over the ureter, but avoid compromising the ureteral lumen.

situation. Animals with ureterocolonic anastomoses should be checked regularly for pyelonephritis. Inappetence in patients with ureterocolonic anastomoses may cause increased absorption of urine as the result of lack of fecal bulk; therefore animals should be encouraged to eat as soon as possible after surgery.

COMPLICATIONS

Major complications following routine cystotomy are uncommon, although 37% to 50% of animals may have transient hematuria and dysuria.[3] The most common complications of urethral wound repair are stricture formation and urinary leakage. Indwelling catheters may allow ascending bacterial infection or may cause fibrosis and stricture. Oversized catheters (those that distend the urethra) should be avoided. Complications of prepubic catheterization (temporary cystostomy) may include bowel perforation from improper percutaneous placement, UTI, transient hematuria, uroabdomen, premature catheter removal, and breakage or incomplete removal of the catheter. Stricture formation in cats following perineal urethrostomy is generally a result of making the stoma too small (i.e., making the stoma in the proximal penile urethra instead of in the distal pelvic urethra) or of postoperative subcutaneous urine leakage and subsequent granulation tissue formation (see p. 690). Urinary and fecal incontinence may occur if the nerves are damaged during dissection around the pelvic urethra. Perineal urethrostomy is associated with a high prevalence of postoperative UTI. Prepubic, subpubic, and transpelvic urethrostomies should be considered only as salvage procedures because they are associated with many complications, including UTI, peristomal skin irritation or necrosis, and urinary incontinence. Rectal prolapse has also been reported following perineal urethrostomy in cats. Pyelonephritis, renal failure caused by end-stage kidney disease, neurologic dysfunction, hyperchloremic metabolic acidosis, and diarrhea with subsequent perineal irritation are common complications of ureterocolonic urinary diversion (see p. 694).

SPECIAL AGE CONSIDERATIONS

Older animals may have preexisting cardiac or renal dysfunction and should be monitored closely. Young animals may have very

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small urethras, making surgical repair of complete transections difficult.

SPECIFIC DISEASES

UROABDOMEN

DEFINTIONS

Uroabdomen or *uroperitoneum* is an accumulation of urine in the peritoneal cavity. Urine may leak from the kidney, ureter, bladder, and/or proximal urethra.

GENERAL CONSIDERATIONS AND CLINCIALLY RELEVANT PATHOPHYSIOLOGY

Bladder rupture is the most common cause of uroabdomen in dogs and cats. It may occur spontaneously (associated with tumor, severe cystitis, or urethral obstruction), may be caused by blunt or penetrating abdominal trauma, or may be iatrogenic following cystocentesis, bladder catheterization, or manual expression of the bladder. Urinary tract leakage may also be a complication of surgery. Animals brought in after a vehicular trauma should be assessed for possible urinary tract trauma. The impact of the collision may cause the bladder, urethra, or ureter to rupture, avulse, or necrose. Sharp ends of pelvic fractures may lacerate the urethra. Diagnosis may be delayed because clinical signs are rarely present at initial examination (see below). Initial assessment of the abdominal cavity performed with a FAST (focused assessment using sonography for trauma; see p. 536) protocol may detect free abdominal fluid before the animal develops clinical signs.

Immediate surgery is contraindicated in animals with uroabdomen that are hyperkalemic or uremic. In general, emergency surgery is rarely appropriate for animals diagnosed with uroabdomen. Patients should first be treated medically to normalize electrolytes and acid base, and to decrease circulating nitrogenous waste products. Intravenous fluids should be given and abdominal drainage and urethral catheterization performed (see p. 699). A large over-the-needle catheter (14-gauge) can be placed in the ventral abdomen under local anesthesia (sedate if necessary) to allow drainage for 6 to 12 hours. This will stabilize most animals with previously normal renal function.

When urine leaks into the abdominal cavity, some nitrogenous waste products and electrolytes are reabsorbed across the peritoneal membrane and reenter the circulation. Whether molecules are reabsorbed depends on their size. Urea rapidly equilibrates across the peritoneal surface, whereas larger molecules (e.g., creatinine) cannot pass back into the bloodstream, and they remain concentrated in the abdominal fluid. Abdominal fluid creatinine concentrations must substantially exceed serum concentrations (>2:1) for a diagnosis of uroabdomen (Box 25.2). Because urea rapidly equilibrates across the peritoneum, blood urea nitrogen (BUN) may be approximately the same in both abdominal fluid and serum, regardless of the cause of the abdominal effusion. Potassium is also helpful in diagnosing uroabdomen. A potassium abdominal fluid-to-blood ratio greater than 1.4 to 1 is predictive of uroabdomen in dogs.

NOTE Creatinine does not equilibrate across the peritoneal surface, whereas BUN does. Compare creatinine or potassium concentrations in the fluid and serum to diagnose uroabdomen.

BOX 25.2 Diagnosis of Uroabdomen in Dogs and Cat

Diagnostic
Creatinine: Abdominal fluid: peripheral blood ≥2

Suggestive
Creatinine: Abdominal fluid: peripheral blood >1 but <2
Potassium: Abdominal fluid > peripheral blood

DIAGNOSIS

Clinical Presentation

Signalment

Urinary bladder rupture has been suggested as being more frequent in male versus female dogs because the long, narrow male urethra cannot dilate rapidly; however, ruptured bladders are common in females that have sustained vehicular trauma. Traumatic urethral rupture in female dogs is uncommon. Male dogs and cats with obstruction due to calculi or FIC are at risk for bladder rupture if the obstruction is not alleviated promptly (see pp. 701 and 717).

History

Clinical signs of urinary tract trauma are often vague and may be masked by other signs of trauma. Urinary trauma often goes clinically undetected in many dogs with pelvic trauma and concurrent urinary tract trauma; a 2016 study of dogs with traumatic pelvic fractures found concurrent uroabdomen in 3.6%, whereas concurrent hemoabdomen was found in 32.5%.[9] An animal may have uremia (e.g., vomiting, anorexia, depression, and lethargy) or hematuria, dysuria, abdominal pain, and/or abdominal swelling. Abdominal and perineal bruising is common with vehicular trauma, particularly if pelvic fractures occur. Bruising in this region, however, may also indicate subcutaneous urine leakage. It is important to remember that animals with ruptured bladder or unilateral ureteral trauma may urinate normal volumes, without obvious hematuria. If the rupture is located dorsally or is small, leakage may occur only when the bladder becomes distended. Similarly, the ability to retrieve fluid while performing bladder catheterization does not preclude a diagnosis of ruptured bladder.

NOTE Do not rule out bladder rupture in animals that urinate normally.

Physical Examination Findings

Abdominal palpation should be performed to determine the size and shape of the bladder. The animal should be closely examined for abdominal swelling or fluid accumulation. Urine quantity and character (e.g., hematuria and dysuria) and bruising on the ventral abdomen or perineum should be monitored.

Diagnostic Imaging

Survey radiographs may show reduced size or absence of the urinary bladder, decreased visceral detail, and/or increased size of the retroperitoneal space. FAST ultrasound will identify free abdominal fluid and will guide abdominocentesis for fluid retrieval

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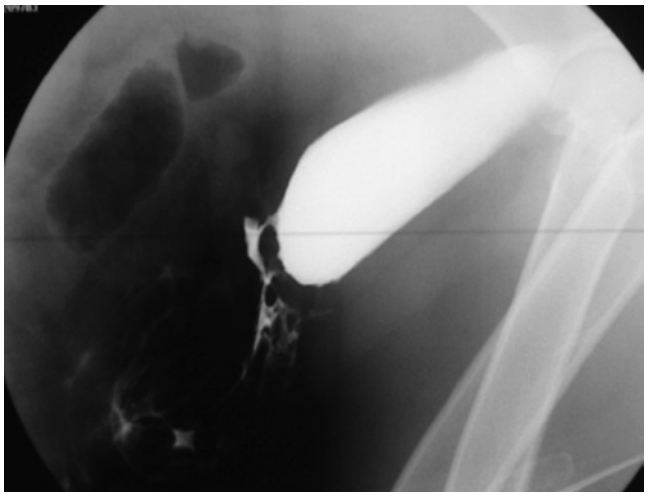


FIG. 25.31 Positive contrast cystourethrogram in a dog with a ruptured bladder. The radiograph was taken while the contrast agent was being injected and shows a "jet" lesion of contrast agent from the bladder.

(see p. 536). If a ruptured bladder is suspected, a positive contrast cystogram can be performed; however, leakage of contrast medium into the peritoneal space during cystography does not necessarily mean that the animal needs exploratory surgery. If the leak is small and no clinical evidence of uroabdomen is noted, conservative management of the patient may be appropriate as small tears may heal spontaneously.

In *cystography,* a balloon-tipped catheter is placed into the urinary bladder; for a *cystourethrogram* in a male dog, the catheter is placed into the distal urethra (just past the os penis), and the balloon is inflated. While the bladder is palpated for distention, approximately 2.2 mL/kg of diluted (1 part contrast medium to 2 parts sterile saline) aqueous organic iodide contrast medium is injected into the catheter. A radiograph is obtained as the last few milliliters of contrast is being injected. Fluoroscopy is advantageous for determining when the bladder is distended. It is critical to adequately distend the urinary bladder before determining that the study is normal because small lesions may not leak while the bladder wall is flaccid. In addition, care should be taken to avoid completely occluding the neck of the urinary bladder with the bulb of the catheter because this may prevent leakage from a rupture in this area. Obtaining a radiograph as the contrast agent is being injected may show a "jet" lesion of contrast agent from the bladder (Fig. 25.31). Free contrast agent in the abdominal cavity will coat and highlight abdominal organs. If a lesion is not identified in the bladder after adequate distention of the urethra, and if the animal is well hydrated, an excretory urogram can be performed (see p. 665). Contrast leakage into the retroperitoneal space (for proximal lesions) or abdomen (for distal lesions) occurs with ureteral rupture or laceration (Fig. 25.32). If periureteral fibrosis has occurred, obstruction rather than leakage may be noted. Leakage of contrast from the renal capsule may be noted with renal parenchymal trauma. Parenchymal trauma of the right kidney should be suspected in dogs with uroabdomen and fracture of the 13th right rib.

Laboratory Findings

Complete blood count (CBC) and serum biochemical profile with electrolytes should be performed. Hyperkalemia, acidosis, and azotemia may be noted. Analysis of abdominal fluid should be performed if urinary tract rupture is suspected. With uroabdomen, creatinine levels of abdominal fluid will be twice those of blood (see Box 25.2). Renal failure may be present if obstruction preceded the rupture (see p. 650). Bladder rupture secondary to UTI causes septic peritonitis (see p. 527).

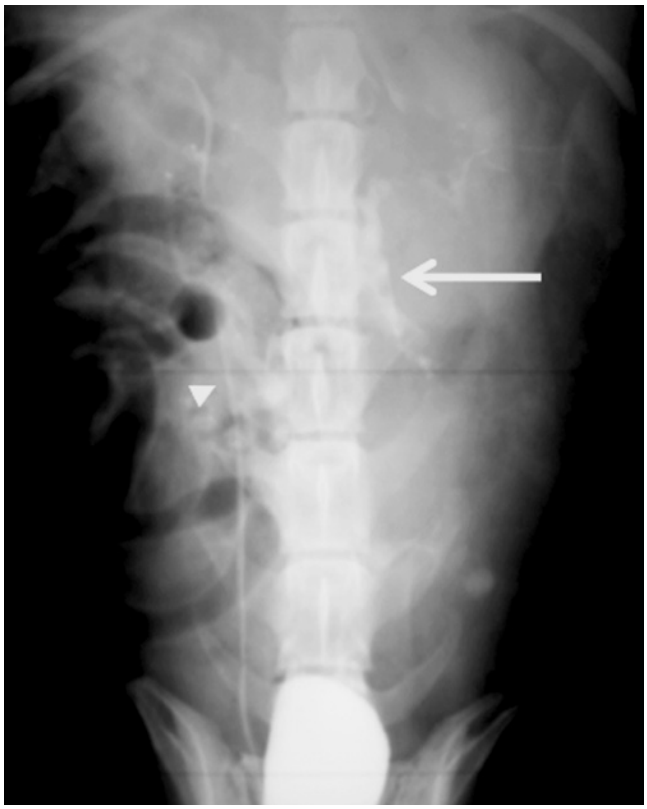


FIG. 25.32 Excretory urogram in a dog with a ruptured ureter. Note the accumulation of contrast around the caudal pole of the left kidney *(arrow)* with a normal right ureter *(arrowhead).*

DIFFERENTIAL DIAGNOSIS

Other causes of abdominal effusion or azotemia should be considered. Peritonitis may cause vomiting, dehydration, and prerenal azotemia. Vomiting may be caused by pancreatic, peritoneal, renal, splenic, hepatobiliary, or gastrointestinal abnormalities. In animals with abdominal effusion subsequent to trauma, hemoabdomen, uroabdomen, bile peritonitis, and septic peritonitis should be considered.

MEDICAL MANAGEMENT

If the animal is not hyperkalemic or azotemic (e.g., uroabdomen is diagnosed within 12–18 hours after rupture), IV fluid therapy with 0.9% saline, LRS, or Normosol-R is instituted, and immediate surgical repair could be considered. Occasionally, concurrent trauma (e.g., traumatic myocarditis, pulmonary contusions) will delay surgery. In such patients, abdominal drainage and/or urinary diversion (i.e., urethral catheter and/or tube cystostomy; see p. 685) may be necessary until the animal is more stable. With delayed diagnosis, correction of electrolytes, hydration, and acid-base balance should be performed before surgery.

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Antibiotics may be administered on the basis of culture results or of bacterial morphology if a UTI is present, or prophylactically if abdominal drains are placed.

NOTE Animals with acid-base and electrolyte abnormalities are poor anesthetic candidates. These abnormalities are easily corrected before surgery.

SURGICAL TREATMENT

Urethral trauma may be repaired by primary anastomosis (immediate or delayed), or the urethra may be allowed to heal over a urinary catheter if it is not completely transected. Ureteral rupture may be repaired by anastomosis (see p. 659) or reimplantation into the bladder (see p. 658), depending on the location of the damage. Bladder rupture generally occurs near the apex. Although small ruptures may heal if the bladder is kept decompressed, surgical exploration and repair are indicated in most patients. The entire abdomen should be explored to determine the reason for rupture and/or to identify concurrent trauma. If bladder rupture is secondary to severe cystitis, tumor, or obstruction, the bladder may be extremely friable, or large areas may be necrotic, making excision and primary closure of the rent difficult. In such cases, prolonged urinary diversion may be beneficial. If cystitis or tumor is present, a biopsy of the bladder mucosa should be submitted for culture and histologic examination. In animals with rupture caused by obstruction from calculi, the urethra should be carefully checked for calculi and its patency verified before the bladder defect is repaired. One should also check the peritoneal cavity for free calculi.

Preoperative Management

An ECG should be evaluated for arrhythmias. If possible, hydration, acid-base, and electrolyte abnormalities should be corrected before surgery (see pp. 678 and 699). If antibiotic therapy has not been initiated before surgery, perioperative antibiotics (e.g., cefazolin, ampicillin) may be administered at induction.

Anesthesia

If renal impairment is not present, many different anesthetic regimens may be used safely. If renal impairment is present, see p. 650 for suggested anesthetic protocols. If the animal is vomiting, avoid mask or chamber induction.

Surgical Anatomy

Refer to p. 684 for surgical anatomy of the bladder and urethra.

Positioning

The animal is placed in dorsal recumbency, and the abdomen is prepared for a ventral midline incision. For bladder rupture, the entire ventral abdomen should be prepared to allow complete exploration of the abdomen.

SURGICAL TECHNIQUE

Cystotomy is described on p. 685.

Excise devitalized or necrotic bladder tissue, and suture the rent with a simple continuous suture pattern. If tissues are friable, and if a watertight seal is not achieved, provide a serosal patch over the incision line (see p. 447).

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Absorbable suture material (e.g., polydioxanone [PDS], polyglyconate [Maxon], polyglactin-910 [Vicryl], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], polyglytone 6211 [Caprosyn]) is preferred for bladder and urethral surgery (see p. 696).

POSTOPERATIVE CARE AND ASSESSMENT

Intravenous fluids should be given until the animal is able to drink adequate fluids to maintain hydration. The patient should be observed closely after surgery for signs of urinary obstruction or peritonitis. If bladder atony is present, the bladder should be kept decompressed by intermittent urinary catheterization or by manual expression once the bladder incision has healed. UTI is common with indwelling or repeated catheterization. An alpha blocker (e.g., phenoxybenzamine; Table 25.4) and/or a somatic muscle relaxant (e.g., diazepam) can help decrease urethral sphincter tone. Bethanechol is a cholinergic drug that increases detrusor contractility and may aid voiding, but side effects can be severe, depending on the dose administered. Cisapride may provide some benefit in improving bladder tone. The bladder should be manually expressed following surgery (particularly in patients with friable bladder secondary to infection or obstruction); this must be done carefully to avoid disrupting the suture line.

NOTE Be sure that no excessive resistance to urinary or fecal outflow is present before using bethanechol. Use care when expressing a bladder in which you have recently performed a cystotomy.

COMPLICATIONS

The major complication of bladder surgery is urinary leakage, especially if a watertight seal is not achieved or if devitalized tissues are sutured and subsequently dehisce. Occasionally, peritonitis may occur from infected urine or secondary to surgically induced contamination.

PROGNOSIS

The prognosis is excellent for animals with traumatic bladder rupture. However, if uroabdomen goes unrecognized, death can occur as early as 3 days. Occasionally, rupture secondary to obstruction may have a guarded prognosis if most of the bladder is necrotic.

BLADDER AND URETHRAL CALCULI

DEFINTIONS

When urine becomes supersaturated with dissolved salts, the salts may precipitate to form crystals (**crystalluria**). If the crystals are not excreted, they may aggregate into solid concretions known as *calculi*. **Urolithiasis** is a term that refers to having urinary calculi or uroliths in the kidney, ureter, bladder, or urethra. **Cystolithiasis** and **cystolithectomy** refer specifically to urinary bladder calculi and their removal, respectively. **Cystotomy** is a surgical incision into the urinary bladder, whereas **urethrotomy** is an incision into the urethra.

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TABLE 25.4 Drugs Used to Improve Urination

Drug	Mechanism of Action	Contraindications/Precautions
Phenoxybenzamine (Dibenzyline) *Dogs:* 0.25 mg/kg PO q8–12h[a] *Cats:* 0.5 mg/kg PO q12h (may cause hypotension); typically 1.25–2.5 mg/cat	Blocks the alpha-1 receptor on smooth muscle, causing relaxation; potent vasodilator	May cause prolonged hypotension in animals; use with caution in animals with cardiovascular compromise
Diazepam (Valium) *Dogs:* 2–5 mg/dog PO q8h *Cats:* 1–2.5 mg/cat PO q8h (duration of action is 1–2 h when given orally)	CNS depressant; functions as a muscle relaxant in the urinary tract	Idiopathic fatal hepatic necrosis reported in cats
Bethanechol (Urecholine)[b] *Dogs:* 5–15 mg/dog PO q8–12h[c] *Cats:* 1.25–5 mg/cat PO q8–12h[c]	Muscarinic, cholinergic agonist; used to increase contraction of urinary bladder	No obstructions must be present in the urinary or gastrointestinal tract, or the drug may cause severe pain or even rupture; should not be used in hyperthyroid animals; the drug may cause vomiting, ptyalism, and/or abdominal discomfort; can cause circulatory depression in sensitive animals
Cisapride (pharmacy compounded) *Dogs:* 0.1–0.5 mg/kg, PO q8–12h *Cats:* 2.5–5.0 mg/cat, PO q8–12h	Cholinergic agonist; used to treat megacolon and to increase the tone of the lower esophageal sphincter; might have some efficacy in treating bladder atony	Minimal side effects associated with use of this drug

[a]Can try giving 0.5 mg/kg once daily, but it is better to start with 0.25 mg/kg q12h to see if the drug will be effective.
[b]Be sure that no resistance to urinary or fecal outflow is present before using this drug.
[c]Because this is a powerful stimulant, it is best to start at a low dose, observe the effects, and slowly increase the dose over several days, if necessary. Higher doses may be tried but should be used with caution.
CNS, Central nervous system; *PO,* orally.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Most canine uroliths are found in the bladder or urethra. Struvite (magnesium ammonium phosphate) and calcium oxalate calculi are the most common canine uroliths, followed by urate, silicate, cystine, and mixed types. Over the past 20 years, a long-term increase in the proportion of canine urinary calculi that contain calcium oxalate has been noted, along with a long-term decrease in the proportion of canine urinary calculi that contain struvite.[10] A similar trend had previously been noted in cats.

UTIs with urease-producing bacteria are an important cause of struvite calculi in dogs. These bacteria split urea to ammonia and carbon dioxide. Hydrolysis of ammonia forms ammonium ions and hydroxyl ions, which alkalinize the urine and decrease struvite solubility. Bacterial cystitis also increases organic debris, which can serve as a nidus for crystallization. Female dogs tend to have more struvite-containing calculi than male dogs, most likely owing their propensity for UTIs. Feline struvite formation usually occurs without UTI.

Calcium oxalate calculi occur most commonly in dogs with transient, postprandial hypercalcemia and hypercalciuria. Many affected dogs have low-to-normal serum parathormone concentrations. Although rare, these calculi may also occur in dogs with defective tubular resorption of calcium, primary hyperparathyroidism, lymphoma, vitamin D intoxication, decreased urine concentrations of citrate, or increased dietary oxalate. Concurrent UTI is rare. Acidic urine favors calcium oxalate crystal formation. Dogs eating canned diets with a high amount of carbohydrate were found to be at increased risk for calcium oxalate urolith formation; dogs fed dry diets formulated to contain high concentrations of protein, calcium, phosphorus, magnesium, sodium, potassium, and chloride appeared to have fewer calcium oxalate calculi.

NOTE Struvite calculi in dogs are frequently associated with infection; be sure to culture the urine, bladder wall, and/or stone. Mucosal cultures may be positive in some patients when urine cultures are negative.

Urate calculi are usually composed of ammonium acid urate derived from metabolic degradation of endogenous purine ribonucleotides and dietary nucleic acids. Dalmatians have defective hepatic transport of uric acid, resulting in decreased production of allantoin and increased urinary excretion of uric acid. Dalmatians also have decreased proximal tubular resorption and distal tubular secretion of uric acid, making urate urolithiasis common in this breed. Dogs with hepatic insufficiency (e.g., congenital portosystemic shunts) may form ammonium acid urate stones as the result of increased renal excretion of ammonium urates. Secondary UTI may occur as a result of mucosal irritation.

Silicate uroliths are often jack shaped and probably are related to increased dietary intake of silicates, silicic acid, or magnesium silicate (Fig. 25.33). Male German shepherd dogs and English sheepdogs are at increased risk for formation of silica-containing urinary calculi. Cystine uroliths occur because of an inherited disorder of renal tubular transport. Cystine stones usually occur in acidic urine.

Although dissolution of some stones is possible, surgical removal is often necessary initially to allow a diagnosis of stone type. Appropriate medical management may help decrease the recurrence of canine uroliths (Table 25.5). Supersaturation of urine with salts appears to be the primary factor favoring calculi formation. Other factors (e.g., presence of a nidus on which the stone can form, decreased concentrations of urine crystallization inhibitors) also appear to contribute to stone formation.

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TABLE 25.5 Treatment and Prevention of Canine Urolithiasis

Urolith Type	Treatment Options	Prevention
Struvite	• Surgical removal (control infection first, if possible) • Dissolve stones feeding Hill's s/d diet • Voiding hydropropulsion if stones are small enough	• Feed Hill's c/d diet or Waltham Canine S/O Lower Urinary Support Diet • Monitor urine pH and urine sediment, **MUST PREVENT** urinary tract infection and eliminate any infections that occur as quickly as possible • Keep urine pH <6.5, BUN <10 mg/dL, and urine specific gravity <1.020
Calcium oxalate	• Surgical removal • Voiding hydropropulsion if stones are small enough	• Feed Waltham Canine S/O Lower Urinary Support Diet, Hill's u/d diet, or Hill's w/d diet; do not supplement vitamins C or D • Increase water consumption
Urate	• Surgical removal • Voiding hydropropulsion if stones are small enough • Dissolve stones using alkalinization with sodium bicarbonate or potassium citrate; feed Hill's u/d diet; administer allopurinol	• Feed Hill's u/d diet • Allopurinol if necessary (not for dogs with PSS) • Correct congenital PSS or lower blood ammonia concentrations
Silicate	• Surgical removal	• Prevent consumption of dirt
Cystine	• Surgical removal • Dissolve stones by feeding Hill's u/d diet and administering D-penicillamine or *N*-(2-mercaptopropionyl)-glycine (MPG)	• Feed Hill's u/d diet • Administer thiol-containing drugs if necessary

BUN, Blood urea nitrogen; *PSS,* portosystemic shunt.



FIG. 25.33 Silicate urolith from a 12-year-old cat with chronic hematuria.

NOTE It is necessary to remove and analyze stones to determine type; subsequent medical management is important to prevent recurrence.

DIAGNOSIS

Clinical Presentation

Signalment

Struvite calculi are more common in female than male dogs because females more commonly have UTI; however, urethral obstruction from stones is more common in males (Box 25.3). Uroliths may occur in dogs of any age, but they are most frequently observed in middle-aged dogs. Calculi in dogs younger than 1 year of age are often struvite secondary to UTI. Calcium oxalate uroliths are more common in male dogs, particularly miniature schnauzers, shih tzus, Pomeranians, Yorkshire terriers, and Maltese. Middle-aged to older dogs are most commonly affected. In cats, calcium oxalate uroliths now occur nearly as frequently as struvite uroliths. Approximately one-third of cats with calcium oxalate uroliths also have increased total serum calcium concentrations.

Urate uroliths most commonly occur in Dalmatians; however, a predisposition in English bulldogs has also been recognized.

BOX 25.3 Breed, Sex, and Age Predispositions for Urinary Calculi

Struvite
- Miniature schnauzers, bichon frises, cocker spaniels, shih tzus, miniature poodles, lhasa apsos
- Females more than males; middle-aged dogs
- Urinary tract infection with urease-producing bacteria (e.g., *Proteus, Staphylococcus*)

Calcium Oxalate
- Miniature schnauzers, shih tzus, Pomeranians, Yorkshire terriers, Maltese, lhasa apsos, bichon frises, Cairn terriers, miniature poodles
- Males, neutered males more than intact males, middle-aged to older dogs, obese dogs

Calcium Phosphate
- Yorkshire terriers

Urate
- Dalmatians and English bulldogs
- Dogs with congenital portosystemic shunts (Yorkshire terriers, miniature schnauzers, Pekingese)

Silicate
- German shepherds, English sheepdogs
- Males, middle-aged dogs

Cystine
- Dachshunds, basset hounds, English bulldogs, Newfoundlands, Chihuahuas, miniature pinschers, Welsh corgis, mastiffs, and Australian cattle dogs
- Males, middle-aged dogs

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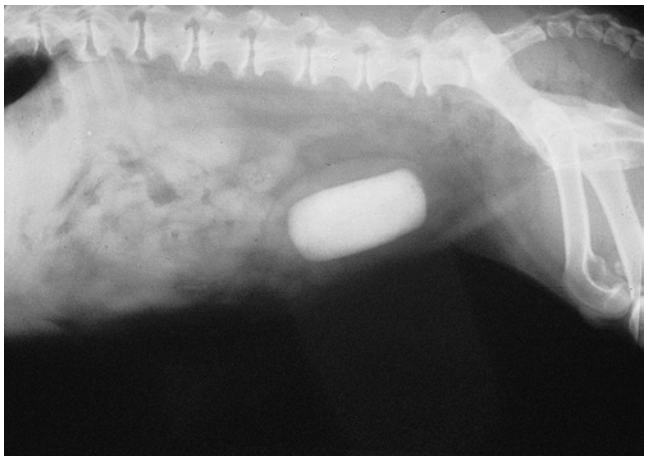


FIG. 25.34 A large radiopaque struvite calculus in the bladder of a dog with chronic cystitis.

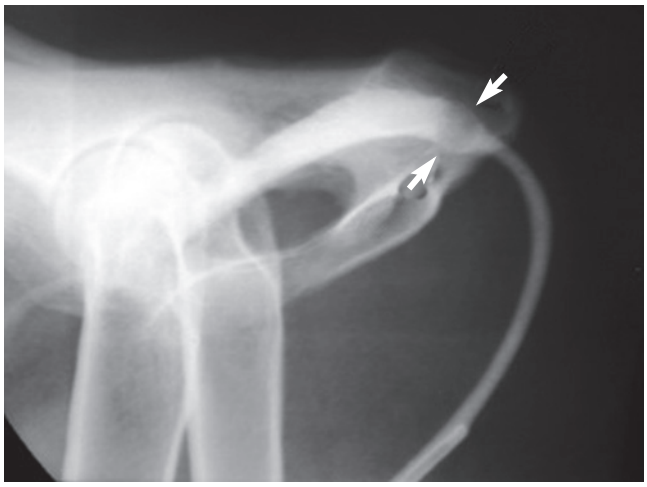


FIG. 25.35 A radiolucent stone *(arrows)* identified by retrograde positive contrast urethrography.

Other commonly reported breeds for urate urolithiasis are those predisposed to congenital portosystemic shunts (i.e., Yorkshire terriers, miniature schnauzers, Pekingese, lhasa apsos). Middle-aged, male German shepherd dogs and English sheepdogs seem to be at increased risk for silicate urolithiasis, although these stones occur in small-breed dogs as well. Cystine uroliths most frequently occur in middle-aged male Dachshunds. Other breeds that appear to be at increased risk for cystine urolithiasis include basset hounds, English bulldogs, Newfoundlands, Chihuahuas, miniature pinschers, and Welsh corgis.

History

Clinical signs of UTI (e.g., hematuria, pollakiuria, and stranguria) are common in dogs with cystic or urethral calculi. Small stones lodging in the urethra of male dogs may cause partial or complete obstruction. Bladder distention, abdominal pain, stranguria, perceived incontinence due to partial obstruction, and/or signs due to postrenal azotemia (e.g., anorexia, vomiting, and depression) may develop. Occasionally the bladder ruptures, causing uroabdomen (see p. 698).

Physical Examination Findings

The bladder wall is often thickened, and stones are occasionally palpable. Signs consistent with UTI may be noted. Abdominal pain, anorexia, vomiting, and/or depression may be noted if urinary tract obstruction occurs.

Diagnostic Imaging

Survey abdominal radiographs or ultrasonography is indicated in any animal with urolithiasis. In addition to defining the numbers and locations of bladder and urethral calculi, these procedures may detect calculi in the kidney and/or ureter. Calcium-containing uroliths (i.e., calcium phosphate and calcium oxalate) are the most radiopaque, whereas cystine and urate uroliths are the least radiopaque. Struvite calculi are normally radiopaque and are usually observed with survey radiography (Fig. 25.34). The size and number of stones are best evaluated on radiographic studies. Double-contrast cystography and/or retrograde urethrography may identify radiolucent stones in the bladder or urethra (Fig. 25.35); however, ultrasonography can detect calculi and can evaluate the kidneys and ureters for concurrent abnormalities.

NOTE Double-contrast cystography/urethrography is probably the most sensitive method for finding stones (even better than ultrasound).

Laboratory Findings

CBC, serum chemistry profile (including electrolytes), urinalysis, and urine culture should be performed. UTI is common, even when pyuria, hematuria, proteinuria, and bacteriuria are absent. Renal failure may be due to chronic pyelonephritis or obstructive uropathy (see p. 650). Findings suggestive of hepatic insufficiency (e.g., low BUN, hypocholesterolemia, hypoalbuminemia, increased serum bile acids) may be present in some animals with urate calculi.

NOTE Always identify and treat concurrent urinary tract infections.

DIFFERENTIAL DIAGNOSIS

Uroliths should be considered in any animal presenting with chronic UTI, hematuria, stranguria, pollakiuria, obstructive uropathy, or urinary incontinence. Other differentials include neoplasia and granulomatous inflammation.

MEDICAL MANAGEMENT

Urethral obstruction should be relieved and/or bladder decompression performed if necessary. Using a finger inserted into the rectum and massaging a urethral urolith toward the vagina may dislodge the urolith in female dogs. *Retrohydropropulsion* may be used to propel urethral stones back into the bladder in both male and female dogs (Fig. 25.36A). A catheter is placed in the urethra distal to the stone, and sterile saline or a combination of sterile saline and a small amount of sterile surgical lubricant (1:5) is forcibly infused while the urethra between the stone and the bladder is occluded by a finger in the rectum (or the vagina in females) (Fig. 25.36B). Once the urethra is dilated, digital pressure is removed in the hope of flushing the stone into the bladder (Fig. 25.36C). A urethral catheter is left in place until the animal can be taken to surgery for a cystotomy. Stones

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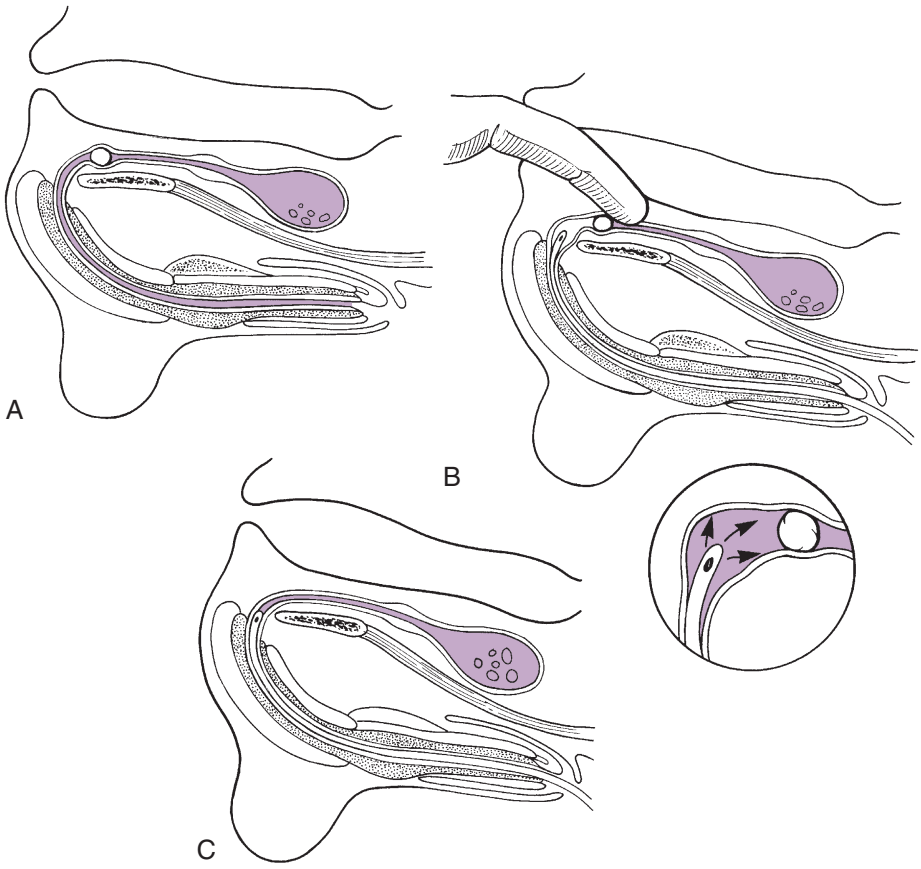


FIG. 25.36 (A) Retrohydropropulsion may be used to propel urethral stones back into the bladder. (B) Place a catheter in the urethra distal to the stone, and inject sterile saline while the urethra is occluded by a finger in the rectum (or the vagina in females). (C) Once the urethra is dilated, remove the finger, allowing the stone to be flushed into the bladder *(inset)*.

that cannot be hydropropulsed into the bladder are removed via urethrotomy (see p. 687).

Voiding urohydropropulsion may remove small cystoliths in male or female dogs. Under general anesthesia, a urethral catheter is placed and the bladder filled with sterile saline. The dog is then held upright for several minutes to allow stones to settle in the trigone and the proximal urethra. The bladder is then forcefully expressed. *Cystoscopic retrieval* using a flexible endoscope and basket can be performed for small stones in male and female dogs, but its use is dependent on the size of the animal and the availability of equipment. *Laser lithotripsy* via cystoscopy using a Ho:YAG laser has been reported to have successful urolith removal in 82% of dogs.[11] Cystoliths and urethroliths are fragmented using the laser and then fragments are removed by basket retrieval or voiding hydropropulsion.

NOTE Always try to flush urethral calculi into the bladder so that cystotomy (rather than urethrotomy) can be performed.

SURGICAL TREATMENT

Surgery should be considered if concurrent or predisposing anatomic abnormalities (e.g., urachal diverticula) are present, if medical dissolution is not possible or is inadvisable, if a bladder mucosal culture is required, or if the stones are large enough that attempting voiding hydropropulsion is likely to cause urethral obstruction. Although medical dissolution of struvite, urate, and cystine calculi is possible, surgical removal of calcium oxalate, calcium phosphate, and silicate stones is necessary. Disadvantages of medical dissolution may include cost, the need for frequent rechecks, possible urethral obstruction in males, and poor owner compliance with maintaining a suitable dietary regimen. Cystotomy (p. 685) should be performed preferentially over urethrotomy if stones can be flushed into the bladder preoperatively or intraoperatively. Cystotomy plus scrotal urethrostomy (p. 689) may be the most effective treatment in preventing recurrence of clinical signs in Dalmatians with urate calculi. Recurrence is common when a cystotomy alone is performed.

NOTE Calculi should be submitted for stone analysis (and usually culture) to guide postoperative management and to help prevent recurrence. One cannot diagnose the type of stone on the basis of crystals found in the urine (e.g., it is possible to have a core of oxalate with a struvite shell because of secondary UTI with struvite crystalluria).

Laparoscopically Assisted Cystoscopy

A rigid cystoscope can be used to aid removal of stones from the bladder and the urethra. A mini-laparotomy from a midline trocar site is performed to secure the cranial aspect of the bladder to the abdominal wall. A small cystotomy is performed and a rigid cystoscope (1.9–2.7 mm) is inserted into the bladder lumen. Calculi are visualized then are removed by retrieval forceps or basket retrieval devices.[12]

Preoperative Management

Postrenal azotemia and hyperkalemia should be treated before surgery (see p. 678). Fluid therapy should be initiated to promote diuresis. An ECG should be evaluated for arrhythmias. UTI should be controlled before surgery, and perioperative antibiotics should

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be considered if the animal is not already receiving antibiotics. However, prophylactic antibiotics may be withheld until after bladder mucosa has been excised for culture in animals with negative urine cultures.

NOTE If possible, eradicate concurrent urinary tract infection before surgery. Otherwise culture bladder mucosa and stones.

Anesthesia

If renal impairment is not present, many different anesthetic regimens may be used safely. If renal impairment is present, see p. 679 for suggested anesthetic protocols. Avoid mask or chamber induction if the patient is vomiting.

Surgical Anatomy

Refer to p. 684 for surgical anatomy of the bladder and urethra.

Positioning

The animal is placed in dorsal recumbency, and the abdomen is prepared for a ventral midline incision. The prepared area should extend from below the pubis proximally to the thorax.

SURGICAL TECHNICQUE

Bladder calculi are removed via cystotomy (see p. 685). *Perform a cystotomy and incise a small piece of bladder at the incision for culture and possible histologic examination (Video 25.1). Remove the bladder stones (Video 25.2) and carefully check the urethra for additional calculi. Also examine the bladder for urachal diverticula, and excise if necessary. In male dogs, place a catheter into the urethra from the penile orifice, and occlude the vesicourethral opening with a finger from within the bladder lumen. Have an assistant gently occlude the penile urethra around the catheter with fingers to minimize fluid leakage. Flush the catheter with sterile saline to maximally dilate the urethra (i.e., when additional saline cannot be flushed into the catheter). While fluid is still being flushed into the catheter, remove the finger at the vesicourethral opening. Repeat this procedure until it is certain that no stones remain in the urethral lumen.* Additionally, a catheter can be passed from the bladder out the urethra opening and the urethra flushed normograde to ensure that no stones are overlooked. *Submit the stones for mineral analysis and possibly for microbial culture.* Postoperative abdominal radiographs will confirm complete removal of radiodense calculi.

Stones lodged within the urethra causing obstruction that cannot be hydropropulsed into the bladder are removed via urethrotomy (see p. 687).

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Absorbable suture material (e.g., polydioxanone [PDS], polyglyconate [Maxon], polyglactin-910 [Vicryl], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], or polyglytone 6211 [Caprosyn]) is preferred for bladder and urethral surgery (see p. 696).

POSTOPERATIVE CARE AND ASSESSMENT

The animal should be closely monitored for urinary obstruction or leakage following surgery. Urine sediment and pH should be monitored regularly and UTI treated promptly. Therapy specific to the stone type should be implemented to help prevent stone recurrence (Box 25.4). D-Penicillamine may inhibit wound healing and should not be initiated earlier than 2 weeks after surgery. Readers are referred to other sources for specific recommendations regarding medical treatment and prevention of urolithiasis.

BOX 25.4 Drugs Used in the Treatment of Urinary Calculi

Allopurinol (Zyloprim)[a]

Dogs:
- To prevent reformation of urate stones: 7–10 mg/kg PO q8h and then decrease to q24h[b]
- To dissolve urate calculi: 15 mg/kg PO q12h

D-Penicillamine (Cuprimine)
- To dissolve cystine stones: 10–15 mg/kg PO q12h[c]

N-(2-Mercaptopropionyl)-Glycine (MPG) (Tiopronin)
- To dissolve cystine stones[c]:
  - 15 mg/kg PO q12h with a protein restricted, alkalinizing diet (U/D or UC)
  - 30–40 mg/kg PO q12h without changing diet—but will need to alkalinize urine with potassium citrate
- For prevention of cystine stones:
  - 10–15 mg/kg PO q12h with protein restricted diet
  - 30 mg/kg PO q12h without changing diet

[a]Use with caution in dogs with renal insufficiency. Also, the drug can cause xanthine uroliths if used with a non–purine-restricted diet.
[b]Dosage should be adjusted on the basis of uric acid excretion.
[c]Many side effects have been reported. See a medical text for more information.
PO, Orally.

COMPLICATIONS

Complications associated with cystotomy are uncommon; however, urine leakage is possible. The most common complication following cystotomy is failure to remove all the stones, which is reported to occur 20% of the time.[13] This complication may be avoided by thorough lavage of the urethra and performance of postoperative abdominal radiographs. The main complication of urethrotomy is hemorrhage, which may persist up to 7 days postoperatively. Urethral stricture is uncommon.

PROGNOSIS

The recurrence rate for calculi formation may be as high as 12% to 25%. Recurrence is more common in dogs with cystine and urate stones than in those with phosphate stones. Appropriate medical management (i.e., prevention of UTI) is necessary to decrease the recurrence of struvite calculi.

URETHRAL PROLAPSE

DEFINTIONS

Urethral prolapse is a protrusion of the urethral mucosa beyond the end of the penis.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Urethral prolapse is uncommon. It may occur after excessive sexual excitement or masturbation, or it may be associated with genitourinary infection.

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DIAGNOSIS

Clinical Presentation

Signalment

Young English bulldogs are most commonly affected, but it has also been reported in Boston terriers, Yorkshire terriers, and several other breeds. It is twice as common in intact males versus neutered males.[14]

History

The owner may notice a reddened protrusion at the tip of the penis and/or intermittent penile bleeding, which may worsen when the dog becomes excited or after urination. Prolapse may be intermittent, occurring only when the dog has an erection. Some affected dogs lick at the preputial orifice and may traumatize the exposed urethral mucosa.

NOTE Penile bleeding may be intermittent (e.g., during erection).

Physical Examination Findings

A small, reddened mass may be visible protruding from the tip of the penis when the penis is extruded from the preputial orifice (Fig. 25.37). Penile erection may cause the protrusion to enlarge. Necrosis of the prolapsed urethra may occur secondary to drying or self-inflicted trauma. The prepuce should be checked for balanoposthitis or neoplasia.

Laboratory Findings

Anemia may occur in dogs with intermittent or chronic bleeding. Urinalysis or urine culture should be performed to exclude UTI. Exclusion of a coagulopathy in dogs with intermittent prolapse may be indicated.



FIG. 25.37 Urethral prolapse in a dog. Note the small, reddened mass (urethra) protruding from the tip of the penis. (Courtesy H.P. Hobson, Texas A&M University.)

DIFFERENTIAL DIAGNOSIS

Urethral prolapse may be differentiated from other causes of preputial bleeding by extruding the penis and examining the urethral orifice. Urethritis, fracture of the os penis, urethral calculi, and urethral stricture may be associated with hematuria and/or preputial bleeding. Other possible causes of penile bleeding include preputial, penile, or urethral neoplasia and prostatic lesions.

MEDICAL MANAGEMENT

Concurrent infection of the genitourinary tract should be treated. If the urethral mucosa is not necrotic, the prolapse can occasionally be reduced by gently manipulating it with a sterile cotton swab or by placing a lubricated catheter into the urethral orifice. A purse-string suture of 5-0 or 6-0 suture material can be placed in the penis around the orifice and tightened to prevent the prolapse from recurring without obstructing urination. The suture should be removed after 5 days and the patient monitored for recurrence. Spontaneous recovery has not been reported.

SURGICAL TREATMENT

Surgical resection of the prolapsed urethra is usually the treatment of choice. If the prolapse can be reduced, placing several mattress sutures from the urethral lumen and tying them on the external penile surface may cause fibrosis and prevent recurrence (urethropexy). The sutures should be left in place for 2 to 3 weeks. Alternatively or concurrently, resection of the prolapsed mucosa is indicated. Bilateral orchiectomy should be performed, particularly in dogs that have prolapse associated with erection or sexual excitement.

Preoperative Management

The animal should be kept from traumatizing the urethra before surgery.

Anesthesia

Many different anesthetic regimens may be used safely if the animal is otherwise healthy.

Surgical Anatomy

Surgical anatomy of the urethra is discussed on p. 684.

Positioning

The animal is placed in dorsal or lateral recumbency and the penis extruded and gently cleaned with dilute chlorhexidine solution. Clipping of preputial hair is not necessary and may contribute to postoperative irritation.

SURGICAL TECHNIQUE

Prolapsed urethras may be treated by resecting the prolapsed tissue or by performing an urethropexy.

Resection

Place stay sutures in the urethral mucosa, and apply gentle traction to straighten the prolapsed tissue. Place one or two straight needles through the penile tissue (Fig. 25.38) or use stay sutures in the urethral mucosa distal to the proposed site of transection to prevent the urethra from retracting within the penis. Transect the urethra along its circumference, and suture it to the penis with 4-0 to 6-0

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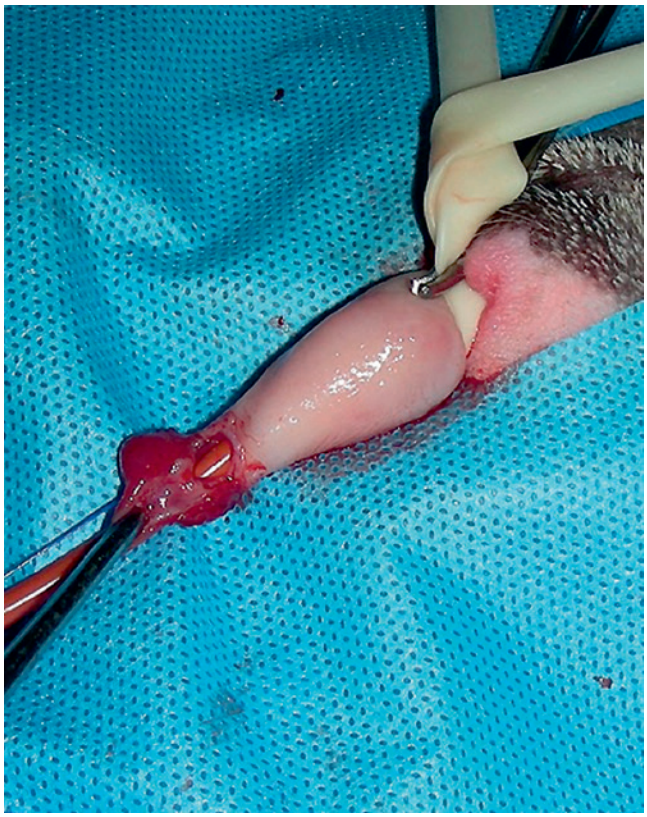


FIG. 25.38 To resect a urethral prolapse, place stay sutures in the urethral mucosa and apply gentle traction to straighten the prolapsed tissue. Use a Penrose drain or large umbilical tape (0.5 inch [1.2 cm]) to prevent retraction of the penis back into the prepuce.

monofilament absorbable or nonabsorbable sutures in a simple continuous pattern (Fig. 25.39).

Urethropexy

Manually extend the penis, and place a grooved director into the urethral orifice to reduce the prolapsed urethral mucosa. Pass the director to the distal aspect of the os penis. If this fails to achieve reduction of all urethral mucosa, have an assistant grasp the penis at its base and apply distal traction to invert the mucosa (Fig. 25.40A–B). Using 2-0 or 3-0 monofilament absorbable suture on a large-radius swaged-on taper-point needle, pass the suture full thickness through the penis from the external surface as far proximally on the penis as the needle curvature will allow. From the intraluminal surface, direct the needle distally out the urethral orifice (Fig. 25.40C). Use the grooved director as a receiving surface for the needle to prevent penetration of the opposite wall of the urethral lumen. Then pass the needle in reverse fashion from the urethral orifice to the external surface of the penis and exit 0.5 cm distal to the initial needle entry site (Fig. 25.40D). Tie the suture so that it creates a slight depression in the surrounding tissue (Fig. 25.40E). Place two to four equally spaced sutures. Pass an 8- or 10-Fr urinary catheter to ensure urethral patency. Do not remove the sutures.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Monofilament, absorbable suture material (e.g., polydioxanone [PDS], polyglyconate [Maxon], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn]) may be used.

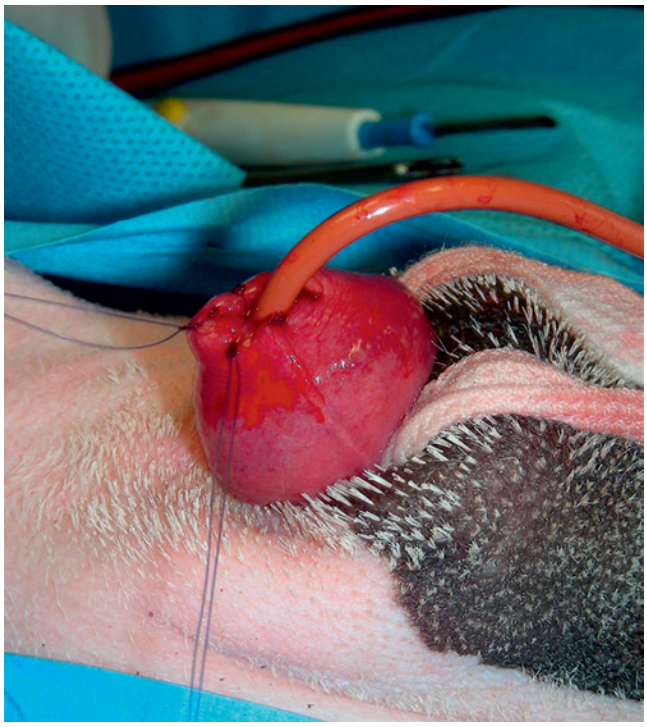


FIG. 25.39 After resecting the prolapsed urethra, suture the urethral mucosa to the penis with simple interrupted sutures.

POSTOPERATIVE CARE AND ASSESSMENT

An Elizabethan collar or sidebar should be used postoperatively to prevent the dog from licking. Tranquilizers may be helpful to prevent postoperative hemorrhage but should be used only in patients in which pain has been appropriately managed. Nonabsorbable sutures should be removed in 7 to 10 days.

COMPLICATIONS

Hemorrhage from the surgical site may occur for 7 to 14 days. The dog should be prevented from becoming excited during the early postoperative period.

PROGNOSIS

Without surgery, the prolapse will not spontaneously resolve. A 2014 study found a recurrence rate of 57%; the likelihood of recurrence was reduced when postoperative sedation was used (e.g., acepromazine or butorphanol).[14] Postoperative hemorrhage occurred in 39% of dogs; hemorrhage was less common when a simple continuous pattern was used instead of a simple interrupted.

BLADDER AND URETHRAL NEOPLASIA

DEFINTIONS

Transitional cell carcinomas (TCCs) are malignant tumors that arise from a transitional type of stratified epithelium and usually affect the urinary bladder. **Rhabdomyosarcomas** are uncommon, highly malignant tumors of striated muscle that may develop from pluripotent stem cells of the primitive urogenital ridge; they are remnants of the Müllerian or Wolffian ducts.

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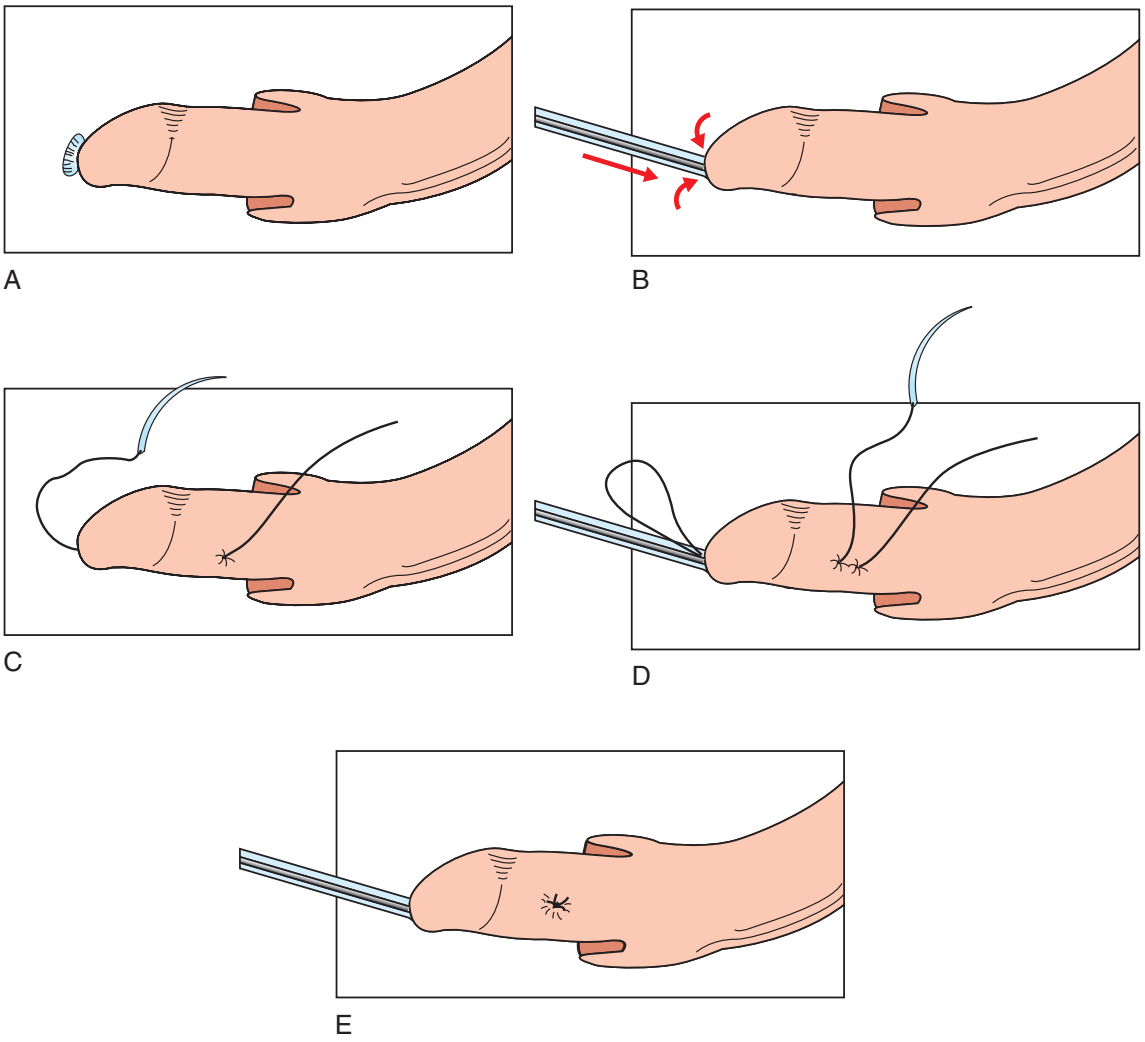


FIG. 25.40 (A) Urethropexy for urethral prolapse. (B) Pass a grooved director to the distal aspect of the os penis. (C) Using 2-0 or 3-0 monofilament absorbable suture on a large-radius swaged-on taper-point needle, pass the suture full thickness through the penis from the external surface, as far proximally on the penis as the needle curvature will allow. Direct the needle distally out the urethral orifice. Use the grooved director as a receiving surface for the needle to prevent penetration of the opposite wall of the urethral lumen. (D) Then pass the needle in reverse fashion from the urethral orifice to the external surface of the penis, and exit 0.5 cm distal to the initial needle entry site. (E) Tie the suture so that it creates a slight depression in the surrounding tissue. Place two to four equally spaced sutures. Pass an 8- or 10-Fr urinary catheter to ensure urethral patency.

GENERAL CONDITIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Bladder neoplasia occurs more frequently than neoplasia of the remainder of the urinary system in dogs. In cats, renal lymphosarcoma is more common than bladder neoplasia. It has been hypothesized that the prevailing variation in bladder tumors between dogs and cats occurs because of differences in the metabolism of tryptophan and its carcinogenic intermediary metabolites. Although dogs excrete aromatic amine metabolites of tryptophan in appreciable quantities into their urine, feline urine is almost devoid of them. Prolonged contact of the bladder mucosa with such carcinogenic substances may be important in the development of tumor. Cyclophosphamide may also cause bladder neoplasia in dogs. Most bladder tumors are malignant, and metastasis to the medial iliac lymph nodes and lungs is common. Local extension to the ureters and/or the urethra is also common.

TCCs are the most common tumors in canine and feline bladders; other malignant bladder tumors include squamous cell carcinoma, adenocarcinoma, fibrosarcoma, leiomyosarcoma, neurofibrosarcoma, rhabdomyosarcoma, and hemangiosarcoma. Fibroma, leiomyoma, hemangioma, rhabdomyoma, myxoma, and neurofibroma are benign bladder tumors. Inflammatory cystic polyps are occasionally found. Metastasis of other tumors to the bladder is uncommon, although extension of prostatic or urethral tumors may occur. Dogs with bladder tumors commonly have other concurrent, primary tumors elsewhere. Fibromas are

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benign mesenchymal tumors that may occur in the bladder. They may be incidental findings or may cause clinical signs similar to those of UTI. Frequently, they are pedunculated; single or multiple surgical excision is often curative. Single or multiple bladder papillomas may occur in older dogs and cause hematuria when ulcerated.

TCCs are the most common canine urethral neoplasms; urethral tumors are exceedingly rare in cats. Other urethral tumors include squamous cell carcinoma and adenocarcinoma. These tumors may be primary urethral masses, or they may be extensions of prostatic or bladder neoplasia. Malignant urethral tumors are frequently locally invasive and may metastasize to the medial iliac lymph nodes and lungs. Proliferative urethritis and granulomatous inflammation of the urethra in female dogs may cause clinical signs similar to those of urethral neoplasia (e.g., stranguria, hematuria, pollakiuria, vaginal discharge, and/or urinary obstruction). Neoplasia and granulomatous inflammation may be differentiated by cytologic evaluation of urethral aspirates or surgical biopsies. The cause of granulomatous urethritis is unknown. Affected dogs may respond favorably to immunosuppressive therapy (e.g., prednisone or prednisone plus cyclophosphamide) plus antibiotics.

NOTE Granulomatous inflammation may cause clinical signs similar to those of urethral tumors. These lesions must be differentiated.

DIAGNOSIS

Clinical Presentation

Signalment

Bladder tumors are more common in dogs than in cats. Older dogs weighing more than 10 kg are most commonly affected; however, botryoid rhabdomyosarcoma (a rare tumor) may occur in young large-breed dogs and young cats. Female dogs and male cats are at higher risk for developing bladder cancer. Shetland sheepdogs, beagles, collies, and various terrier breeds (especially Scottish terriers) seem to be predisposed to TCC. The average age of affected dogs and cats is approximately 10 years. Urethral tumors are more common in older female dogs.

History

Most dogs with bladder or urethral tumors are examined because of hematuria, pollakiuria, stranguria, and/or dysuria. Other signs include perceived incontinence due to partial obstruction, polyuria-polydipsia, lameness, and dyspnea. Lameness may be due to bone metastasis or hypertrophic osteopathy. The most common clinical sign in cats is intermittent or persistent hematuria. If the tumor causes urethral or bladder obstruction, signs of uremia (i.e., vomiting, anorexia, and depression) may occur. Rectal prolapse has rarely been associated with TCC of the feline bladder.

Physical Examination Findings

The most common physical examination findings include a urethral or caudal abdominal mass, prostatomegaly, bladder distention, abdominal pain, weakness, lymphadenopathy, cough or dyspnea, and/or lameness. Lameness may be associated with hypertrophic osteopathy paraneoplastic syndrome in dogs with TCC. Urethral and prostatic involvement is common in dogs with TCC of the bladder; therefore, in addition to careful abdominal palpation, rectal examination should be performed. Urethral masses in females may be palpated rectally or by digital examination of the vagina. Physical examination findings are normal in nearly one-third of dogs with bladder neoplasia.

Diagnostic Imaging

Survey abdominal radiographs are rarely diagnostic but may help exclude prostatic disease or urolithiasis. The medial iliac lymph nodes, pelvis, and vertebrae should be examined for enlargement or bony lysis due to possible metastasis. Diffuse thickening or calcification of the bladder wall is noted occasionally. Double contrast cystography and positive contrast urethrography and ultrasonography are the most useful tools for diagnosing urethral or bladder neoplasia (Fig. 25.41). Ultrasound is limited by the accessible acoustic window. Excretory urography may show hydroureter and/or hydronephrosis and irregular filling defects in the bladder. Double contrast cystography is most effective for delineating bladder wall and luminal masses. Retrograde urethrography (see p. 709) can be performed in dogs with suspected urethral neoplasia to determine the extent of the tumor and to check for evidence of trigonal involvement. Thoracic radiographs should be performed to identify pulmonary metastasis. Ultrasonography is useful to look for abdominal metastasis. Advanced imaging is always beneficial in determining the extent of local and metastatic disease. Although fine-needle aspirates of the mass may provide a presurgical diagnosis, they may cause seeding of the tumor along the needle tract.

Retrograde Cystoscopy

Depending on the patient's size and sex, rigid or flexible scopes can be used to visualize the lesion and facilitate biopsy. It is important in either case to be able to flush sterile saline ahead of the tip of the scope to facilitate visualization.

Laboratory Findings

CBC, serum biochemical profile, urinalysis, and urine culture should be performed in animals with bladder tumors. Hematologic



FIG. 25.41 Cystourethrogram in a dog with a large transitional cell carcinoma. Note the filling defect near the bladder trigone.

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and biochemical parameters are usually normal; however, elevations in serum creatinine and BUN may occur with partial obstruction of the lower urinary tract or ureter. Anemia is common in cats with bladder tumors. Hypereosinophilia has been reported in a cat with TCC of the bladder. Hematuria, pyuria, proteinuria, and bacteriuria are common. Although malignant cells may be found in the urine sediment of some dogs with bladder or urethral tumors, they are typically not detected in most cats with bladder neoplasia. Care should be taken to avoid confusing neoplastic and dysplastic cells; atypical transitional cells are common in animals with cystitis. In addition, prolonged exposure to urine may make interpretation of abnormal cells difficult. Emptying the bladder and performing cytologic evaluation of a saline wash may be helpful in some animals.

Transurethral Biopsy

Transurethral biopsy using a sterile urinary catheter is often diagnostic. *First, insert a finger into the rectum. Then, advance the largest urinary catheter that easily passes through the urethra until the tip is digitally felt to advance just to the point where the thickening or mass is palpated. At that point, apply negative pressure. If urine is obtained, empty the bladder. After the bladder is emptied, or if negative pressure is present to begin with, establish 8 to 12 mL of negative pressure with a syringe that has 10 mL of sterile saline in it, while pulling the catheter tip back out through the thickened area. Once the catheter tip is clearly out of the affected area, remove the catheter from the urethra, place the tip in a clot tube, and blow the tissue fragments in the tip out by forcing saline out through the catheter. Examine the fluid, and retrieve tissue fragments and use them to make squash cytology preparations. In the case of female dogs with severe obstruction of the distal urethra, the most that may be possible is to insert a small, stiff polypropylene catheter a few millimeters into the urethra (which is as far as is possible in many cases), apply suction, and then blow the tissue fragments out, as described previously.*

A veterinary version of rapid latex agglutination urine dipstick test (bladder tumor antigen test) has been screened for use in dogs with TCC of the lower urinary tract.[15] Test sensitivities were 88%, 87%, and 85% for all dogs with suspected and confirmed TCC, dogs with confirmed TCC at any site, and dogs with confirmed TCC of the urinary bladder, respectively. The test performed slightly better on centrifuged urine samples than on uncentrifuged urine samples.

DIFFERENTIAL DIAGNOSIS

Other causes of hematuria and/or bacteriuria (e.g., urolithiasis, prostatic disease, polypoid cystitis) should be excluded. Many cats with lower urinary tract neoplasia are treated presumptively for sterile cystitis with antibiotics, urinary acidifiers, and/or dietary changes for months before diagnosis. Nonneoplastic (granulomatous) infiltrative urethral disease should be differentiated from neoplasia by cytology and/or histopathology. Polypoid cystitis is an uncommon urinary bladder disease in dogs, characterized by inflammation, epithelial proliferation, and development of a polypoid mass or masses without histologic evidence of neoplasia. These lesions must be differentiated from neoplastic masses. A mass is not seen in some animals with polypoid cystitis, and diffuse thickening of the bladder is noted. Most affected dogs are females, which are typically presented for hematuria or recurrent UTIs. *Proteus* spp., *E. coli*, *Staphylococcus*, and *Enterococcus* are commonly isolated. Most of the masses are located cranioventrally in the bladder rather than at the bladder neck or trigone. Surgery with removal of all polyps is typically effective.

BOX 25.5 Medical Treatment of Canine Cystic Transitional Cell Carcinoma

Piroxicam (Feldene)
0.3 mg/kg PO q48h[a]

Omeprazole (Prilosec)
1–2 mg/kg PO q12h

[a]Start with every-other-day administration and observe response; every day administration may be associated with gastrointestinal ulceration. Do not use with any other nonsteroidal antiinflammatory drugs or with any steroids.
PO, Orally.

MEDICAL MANAGEMENT

If partial or complete urinary obstruction is present, the animal should be stabilized before surgery with fluids and cutaneous urinary diversion (urethral catheter or tube cystostomy). Electrolyte and acid-base abnormalities should be corrected, and concurrent UTI treated with appropriate antibiotics. An ECG should be evaluated for arrhythmias. Treatment of malignant bladder tumor with excision and/or adjuvant chemotherapy has also been reported. Piroxicam (Box 25.5) has been used to treat nonresectable TCC of the urinary bladder in dogs. Complete remission may occur in some dogs, whereas others have a subjectively improved quality of life despite the lack of tumor remission. The exact mechanism of piroxicam antitumor activity is unknown; however, cyclooxygenase (COX)-2 is highly expressed in TCC cells, and it has been suggested that this isoform may be involved in tumor cell growth. Thus inhibition of COX-2 may be the mechanism by which nonsteroidal antiinflammatory drugs exert their antineoplastic effects. Antiinflammatory activity might also be important. The most common side effect of piroxicam administration is gastrointestinal irritation (i.e., anorexia, melena, and/or vomiting). Concurrent use of misoprostol may help prevent gastric ulceration/erosion in selected cases (see Box 25.5).

SURGICAL TREATMENT

Surgical therapy is difficult because the most common site for urinary bladder neoplasia is the trigone. Although the ureters can be transected and implanted into the apex of the bladder following partial cystectomy, incontinence typically occurs if the trigone is removed. Similarly, implantation of the ureters at a distant site (i.e., the colon) following complete cystectomy typically causes pyelonephritis and/or incontinence (see p. 694). Tumor spread beyond the primary site is common. Transplantation of TCC to the subcutaneous tissue of the surgical incision has been reported in dogs; therefore the same instruments used for biopsy or resection of a bladder tumor should not be used on other tissues. Cystostomy catheter placement may also be considered (see p. 685). Surgical excision of neoplastic lesions may be curative if the tumor is benign (Fig. 25.42).

Resection of focal lesions of the urethra is possible with a transpubic surgical approach and urethral resection and anastomosis (see p. 686). Prepubic urethrostomy (see p. 692) with resection of neoplastic tissues may be performed if the distal

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FIG. 25.42 Leiomyoma of the bladder. Surgical excision of benign tumors or those located at the bladder apex may be curative.

urethra is involved. Urethral tumors that involve the entire length of the urethra or the bladder trigone are generally inoperable.

Palliative stenting of benign malignant urethral obstructions has been described and was effective in restoring luminal patency for a period of time.

Preoperative Management

See the discussion on medical management on p. 710.

Anesthesia

If renal impairment is not present, many different anesthetic regimens may be used safely. If renal impairment is present, see p. 650 for suggested anesthetic protocols.

Surgical Anatomy

Refer to p. 684 for the surgical anatomy of the bladder and the urethra.

Positioning

The animal is placed in dorsal recumbency, and the abdomen is prepared for a ventral midline incision. For bladder neoplasia, the incision should extend from above the umbilicus to the brim of the pelvis. With urethral neoplasia, the incision should be extended caudally to allow a pubic osteotomy to be performed.

SURGICAL TECHNIQUE

Examine the sublumbar lymph nodes, ureters, and other abdominal organs for evidence of tumor extension or metastasis. For bladder neoplasia, locate the entrance of the ureters into the trigone and excise the tumor, removing at least 1 cm of normal tissue. Be sure to avoid damaging the ureters. If a large portion of the bladder has been removed, place a urinary catheter and suture the bladder with a continuous appositional suture pattern.

If the bladder trigone is involved, consider palliative tube cystostomy (p. 685), ureterocolonic urinary diversion (p. 694), chemotherapy, or euthanasia.

For urethral neoplasia, check the trigone, ureters, sublumbar lymph nodes, and other abdominal tissue for evidence of neoplasia. Perform a pelvic osteotomy, and carefully examine the entire urethra. If the tumor does not involve the entire urethra or trigone, perform a urethral resection and anastomosis (see p. 686). If only the distal urethra is involved, and if neoplastic tissue can be resected, consider a prepubic urethrostomy (see p. 692).

In rare instances, a benign pedunculated urethral tumor may be removed through an urethrotomy incision (see p. 687).

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Absorbable suture material (e.g., polydioxanone [PDS], polyglyconate [Maxon], polyglactin-910 [Vicryl], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], polyglytone 6211 [Caprosyn]) is preferred for bladder and urethral surgery (see p. 696).

POSTOPERATIVE CARE AND ASSESSMENT

The animal should be observed for urinary leakage or obstruction following surgery. With ureterocolonic anastomosis, IV fluids should be continued for 24 to 72 hours to ensure diuresis. The animal should be encouraged to eat on the day following surgery. Kidneys should be monitored for function and postoperative infection. If neurologic dysfunction occurs, blood ammonia levels should be measured and the animal treated appropriately. After this procedure, the addition of 0.5 to 2 g of sodium bicarbonate to food twice daily may improve clinical signs associated with hyperchloremia and metabolic acidosis. Placement of Vaseline on the perineum may help prevent urine scalding. Following total cystectomy and ureter reimplantation into the vagina or prepuce, animals should be monitored for evidence of ureteral dehiscence and obstruction.

COMPLICATIONS

The most common complications of bladder and urethral surgery are urinary leakage and obstruction (see pp. 696 and 697). Pyelonephritis, renal failure, neurologic dysfunction, electrolyte abnormalities, metabolic acidosis, and diarrhea with subsequent perineal irritation are possible complications of total cystectomy with urinary diversion and ureterocolonic urinary diversion (see p. 694). UTI is a common complication of cystostomy tube placement.

PROGNOSIS

Because of the malignant nature of most lower urinary tract tumors, the prognosis is guarded. Reported median survival times do not exceed 1 year, regardless of treatment modality. With aggressive surgery, urethral tumors may have a better prognosis than bladder tumors. Chemotherapy may allow dogs with bladder tumors to survive for significantly longer periods than if they undergo surgery. Deracoxib and vinblastine have been shown to have antitumor activity against TCC in dogs.[16,17] For most owners, piroxicam as monotherapy or in conjunction with other agents may be the most appropriate medical therapy. The benefits of radiation therapy and photodynamic therapy in dogs are still being evaluated. Cystostomy tube placement in dogs with known or suspected TCC typically resolves stranguria, and most owners are satisfied with the procedure. UTIs are a common complication.

The results of ureterocolonic anastomosis in a large number of dogs have not been reported, but with improved techniques and prevention of the deleterious effects of pyelonephritis on renal function, long-term survival might be possible. In the future, newer treatment methods, such as photodynamic therapy, may improve the prognosis of animals with bladder tumors.

In a case series of 10 dogs that underwent total cystectomy with urinary diversion to the prepuce or vagina, median survival

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was 385 days, and they had fewer gastrointestinal and neurologic complications associated with ureterocolonic anastomosis.[4]

Median survival in cats with TCC of the urinary bladder was reported at 241 days; it was shown to be more prevalent in male cats, even though the condition itself is uncommon.[18]

URINARY INCONTINENCE

DEFINITIONS

Urinary incontinence is due to failure of voluntary control of the vesical and urethral sphincters, with constant or frequent involuntary passage of urine. Incontinence may be caused by neurogenic abnormalities or anatomic outflow obstruction (paradoxical or overflow incontinence), may be hormone-responsive (urethral sphincter mechanism incontinence), or may be due to inflammation (urge incontinence), congenital abnormalities (e.g., ectopic ureters, congenital urethral sphincter incontinence), or behavioral problems.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Urinary incontinence had been reported to occur in as high as 20% of spayed dogs, although a 2013 study found the prevalence of acquired urinary incontinence in spayed dogs to be 5.12%.[19]

No true bladder sphincter exists in the bitch, so continence is maintained by multiple interacting factors. Poor urethral tone, marked urethral hypoplasia, intrapelvic positioning of the bladder, ovariohysterectomy, obesity, and congenital abnormalities all have been implicated as potential causes of urinary sphincter mechanism incontinence in female dogs. Congenital urethral sphincter mechanism incontinence has also been described in dogs and cats. Some animals respond to estrogen supplementation or sympathomimetic drugs, particularly α-adrenergic stimulants (e.g., phenylpropanolamine, ephedrine) that increase urethral sphincter tone. Surgical alternatives to improve urethral resistance include colposuspension, cystourethropexy, periurethral injections of polytetrafluoroethylene or collagen, urethral slings, artificial urethral sphincters, and urethral lengthening procedures. Because these techniques are not uniformly successful, and because success has not been consistently well documented in a large number of animals with urinary incontinence, surgical treatment (other than for congenital abnormalities, such as ectopic ureters; see p. 663) is often reserved for animals that do not respond to medical management (see later discussion), have adverse reactions to medication, or when the owners decline to consider long-term drug therapy.

> **NOTE** Consider ectopic ureters (see p. 663) as a differential in any animal that develops urinary incontinence at a young age, including those that respond to medical therapy.

Estrogens probably exert their beneficial effect by improving smooth muscle contractility and sensitivity to α-adrenergic innervation, although the exact mechanism is unknown. Bladder neck position may affect continence; increases in intraabdominal pressure are transmitted to both the bladder and the proximal urethra in bitches with an intraabdominal bladder neck. However, dogs with more caudal (pelvic) bladders have this pressure transmitted to the bladder, but not the urethra. Experimentally, a rise in intraabdominal pressure leads to shortening of the functional urethral length, which might increase the adverse effect of bladder neck position in these dogs, thereby worsening incontinence.

DIAGNOSIS

Clinical Presentation

Signalment

Medium-sized and large-breed dogs, particularly Doberman pinschers, English sheepdogs, and springer spaniels, seem to be at increased risk. In a 2013 prevalence study, dogs greater than 15 kg were seven times more likely to develop urinary incontinence than dogs less than 15 kg.[17] Among small-breed dogs, miniature poodles may be at increased risk. Incontinence may be first noted at any age, depending on the cause.

History

Animals may have a lifelong history of urinary incontinence, or it may occur after ovariohysterectomy. Incontinence may be continuous, may be intermittent, or may occur only when the animal is excited or asleep. A history of "bed wetting" is probably one of the most important historical findings that suggest incontinence. The only reasons why dogs urinate on themselves while asleep are incontinence, stupor/coma, and weakness or pain that makes them unable and/or unwilling to get up when they have to urinate.

Physical Examination Findings

Physical examination findings are usually unremarkable. Occasionally the bladder is caudally displaced in the abdominal cavity. Signs of concurrent UTI (e.g., hematuria, dysuria, or stranguria) may be noted. In cats with urethral hypoplasia, vaginal aplasia is common with the uterine horns emptying into the caudal part of the dorsal wall of the bladder.

Diagnostic Imaging

Abdominal radiographs and ultrasound are helpful to rule out other causes of urinary clinical signs (stones, masses) or concurrent congenital abnormalities (e.g., hydronephrosis, hydroureter, renal aplasia). Ultrasound can sometimes find urine being ejected from ureteral orifices that are not at the expected location. Administration of furosemide during the examination may aid the ultrasonographer in finding ectopic ureters.

Excretory urography may locate the termination of the ureters into the urethra, thus diagnosing ectopic ureters; however, it is easy to have a nondiagnostic study. The vesicourethral junction may appear blunted and abnormally dilated, or the urethra may seem abnormally short. In cats with vaginal aplasia, radiographic evidence of a communication between the lumen of the uterus and the bladder may be noted.

Retrograde cystoscopy is sensitive and specific for diagnosing and defining ectopic ureters (Figs. 25.43 and 25.44). Most female dogs with ectopic ureters have vestibulovaginal abnormalities, which can make it difficult to find the urethral orifice. Ectopic ureters are usually easily identified as one advances up the urethra while infusing saline; however, some are located just at the junction of the urethra and the bladder, making them difficult to see. Observe suspicious folds or slits in the tissue for sporadic puffs of yellow urine to confirm visualization of a ureteral opening. Cystoscopy is also beneficial because it allows one to visualize the vestibule and genitourinary mucosa for other abnormalities.

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FIG. 25.43 Cystoscopic view of an ectopic ureter opening into the urethra, near the trigone.

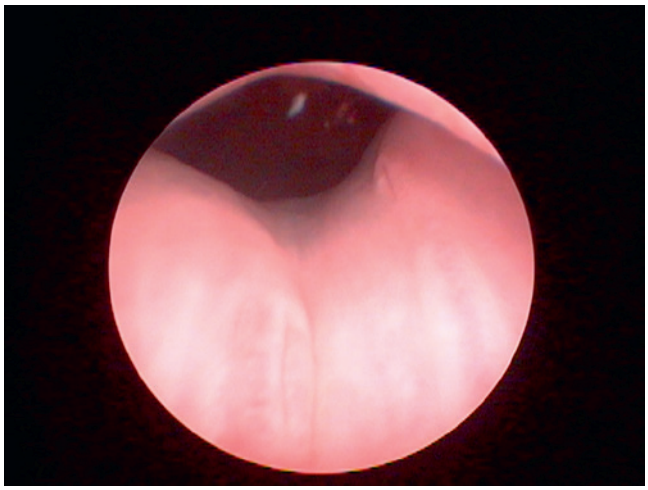


FIG. 25.44 Cystoscopic view of an ectopic ureter *(lower right corner)* opening into the distal urethra, near the urethral orifice.

Computed tomography (CT) excretory urography is the most sensitive method for diagnosis of ectopic ureters, and if available, should be considered the imaging modality of choice. Common abnormalities seen in dogs with intramural ectopic ureters include lack of a normal ureterovesicular junction, a urethral–ureteral orifice location, and lack of ureteral divergence.

Laboratory Findings

Other than findings consistent with upper or lower UTI in some animals, laboratory findings are unremarkable. Urine cultures should be performed in all animals with incontinence, even if the urinalysis is not suggestive of a UTI.

Other Diagnostics

Urodynamic testing, specifically urethral pressure profile, may be helpful in predicting incontinence following surgical correction of ectopic ureters. A urethral catheter that approximates the size of the urethral diameter is placed into the urinary bladder. Saline is instilled through the urinary catheter while resistance against saline flow is measured. As the catheter is withdrawn, a pressure tracing of the urethra is generated. However, owing to interference of results from inflammation (associated with UTI) or urethral spasm, as well as lack of availability of equipment and formal training, the practicality of this technique in dogs with suspected ectopic ureters or urethral sphincter mechanism incompetence is low.

DIFFERENTIAL DIAGNOSIS

Various causes of urinary incontinence must be differentiated. Urge incontinence secondary to cystic or urethral infection and/or inflammation should be excluded (e.g., response with use of appropriate antibiotics). Ectopic ureters should be detected and corrected surgically or endoscopically (see p. 663). Paradoxical incontinence (e.g., overflow caused by partial obstruction due to urethral calculi, neoplasia, or strictures) and neurogenic incontinence should be differentiated from urethral sphincter mechanism incompetence on the basis of imaging, neurologic examination, and/or catheterization. Urethral sphincter profilometry and cystometry can determine urethral sphincter tone and bladder emptying pressures, but these tests are seldom required.

MEDICAL MANAGEMENT

Dogs with suspected urethral sphincter mechanism incompetence should be treated with estrogens and/or sympathomimetic drugs initially, and concurrent UTIs should be treated. Diethylstilbestrol (DES), estriol, and/or α-adrenergic agonists (i.e., phenylpropanolamine or ephedrine; Box 25.6) may be used to increase urethral sphincter tone. If the animal responds to DES (usually within 5–7 days), the frequency of administration should be decreased to the lowest effective dosage (usually 1 mg given once weekly). DES is a relatively safe estrogen, seldom causing problems when used appropriately. Inappropriately high doses of DES may cause estrus-like signs, bone marrow toxicity, and/or alopecia; therefore frequent administration and/or dosages greater than 1 mg daily should be avoided and other drugs used instead. Estriol (Incurin) was approved by the United States Food and Drug Administration in 2011 for treatment of urinary incontinence in dogs, although it has been used worldwide since 2000. Dosing is once-a-day starting at 2 mg (regardless of body weight) and then titrated to the lowest effective dose. The manufacturer reports that 93% of dogs receiving Incurin were improved or continent by 6 weeks. Reports of adverse reactions are rare.

Use of α-adrenergic agonists with DES or estriol may allow lower dosages to be used. Phenylpropanolamine is used more frequently than ephedrine because it has fewer side effects (e.g., hyperexcitability, panting, and/or anorexia) and greater efficacy over time. A disadvantage of phenylpropanolamine is that it must be given two to three times per day (vs. daily with estriol or one to two times per week with DES).

Although DES and phenylpropanolamine can be used to treat males with hormone-responsive urinary incontinence, testosterone supplementation is sometimes more effective. This condition is uncommon, so other causes of incontinence should be thoroughly investigated. Repositol testosterone (cypionate; see Box 25.6) is

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BOX 25.6 Drugs Used in the Treatment of Urinary Incontinence

Phenylpropanolamine[a]
Dogs: 1–2 mg/kg PO q8–12h[b]
Cats: 1 mg/kg PO q12h

Ephedrine
Dogs: Start at 0.4 mg/kg, and gradually increase to 4 mg/kg PO q8–12h[c]
Cats: 2–4 mg/cat PO q8–12h

Diethylstilbestrol (DES)
Dogs: 0.1–1 mg/dog PO q24h (dose is proportional to the size of the dog) for 3–5 days, then same dose once every 5–7 days as needed, with maximum of 0.2 mg/kg/wk[d]

Estriol (Incurin)[e]
2 mg/dog PO q24h for 7 days, then decrease at weekly intervals until weekly dose of 0.5 mg per dog is achieved

Testosterone Cypionate (Depo-Testosterone)
Dogs: 1.1–2.2 mg/kg IM, once every 2–4 wk

Imipramine (Tofranil)
Dogs: 2–4 mg/kg PO q12–24h

[a]Proin (PRN Pharmacal) now has Food and Drug Administration approval for use in dogs.
[b]Best to start at low end of dose and gradually increase, if needed.
[c]Toxicity typically starts at 5 mg/kg, and death can readily occur at 10 mg/kg.
[d]If benefit is not noted after 5 days of daily therapy, then it is unlikely that further administration will benefit the patient, and other drugs should be used.
[e]May be combined with phenylpropanolamine.
IM, Intramuscular; *PO*, orally.

administered intramuscularly, but common side effects include aggression and prostatic hyperplasia, which usually resolve when the drug is discontinued. Testosterone cypionate should not be used more frequently than once every 4 weeks. Owing to its potential for abuse by humans, testosterone is a controlled substance.

SURGICAL TREATMENT

See p. 666 for surgical treatment of ectopic ureters.

Presently, no single surgical procedure will cure incontinence in all female dogs with urethral sphincter mechanism incontinence. Colposuspension has been performed on a large number of patients, and more than half have become continent after surgery. Cystourethropexy results in continence in most bitches immediately following surgery; however, incontinence recurs in a large number of treated dogs. Cystopexy may be more useful in dogs with perineal hernias than in those with incontinence. In animals with notable urethral hypoplasia, reconstruction of the bladder neck may eliminate or decrease incontinence (see later discussion). A sling procedure using a polyester ribbon passed through the obturator foramen, around the urethra, and fixed outside the pelvis resulted in less than 25% of dogs becoming continent. Other techniques include transobturator vaginal tape placement, and prostatopexy and deferentopexy for male dogs. More recently, placement of percutaneously adjustable hydraulic artificial urethral sphincter placement (AUS) has been described and used with success for establishing continence in several outcome studies.

Injection of polytetrafluoroethylene (Teflon) into the submucosa of the proximal urethra has been used in dogs, but rejection of the Teflon resulted in recurrence of incontinence in most dogs. Submucosal injection of collagen has been shown to have greater success in achieving continence, without inciting any inflammatory reaction or causing urinary retention. Return of incontinence may be caused by flattening of the collagen deposits and subsequent loss of effect.

NOTE Rule out other causes of incontinence and determine the efficacy of medical management before attempting surgical correction. UTI can cause medical therapy that otherwise would work to be ineffective.

Preoperative Management

Concurrent UTIs should be treated before surgery.

Anesthesia

If renal impairment is not present, many different anesthetic regimens may be used safely. If renal impairment is present, see p. 650 for suggested anesthetic protocols.

Surgical Anatomy

See p. 684.

Positioning

The animal is placed in dorsal recumbency, and the abdomen is prepared for a ventral midline incision. The prepared area should be sufficient to allow the incision to extend from the pubis proximally to the umbilicus.

SURGICAL TECHNIQUE

Although numerous techniques have been described to correct urethral sphincter mechanism incontinence, only colposuspension, cystourethropexy, collagen injection, AUS, and trigonal reconstruction are described here.

Colposuspension

Colposuspension is the surgery most commonly used to treat urethral sphincter mechanism incompetence in female dogs. This procedure involves placement of sutures from the cranial vagina to the prepubic tendon on either side of the proximal urethra, thereby positioning the proximal urethra within the abdomen and placing pressure on the urethra as it crosses the pubic brim.

Before performing surgery, place a large urethral catheter. Perform a caudal midline abdominal approach, and identify the vagina dorsal to the urethra. Use stay sutures or atraumatic forceps to manipulate the vagina cranially. Place two sutures of heavy gauge (0 to 2-0) nonabsorbable monofilament material through the vaginal wall on either side of the urethra to the prepubic tendon on either side of midline. Use the urethral catheter to prevent overtightening of the sutures, which may result in urethral obstruction. Close the abdomen routinely.

Cystourethropexy

The rationale behind cystourethropexy is similar to that for colposuspension because this procedure relocates the trigone to a more cranial intraabdominal position. Cystourethropexy involves permanently securing the urinary bladder in a more proximal direction, while the ventral wall of the proximal urethra is sutured to the prepubic tendon. By fixing the bladder and the proximal

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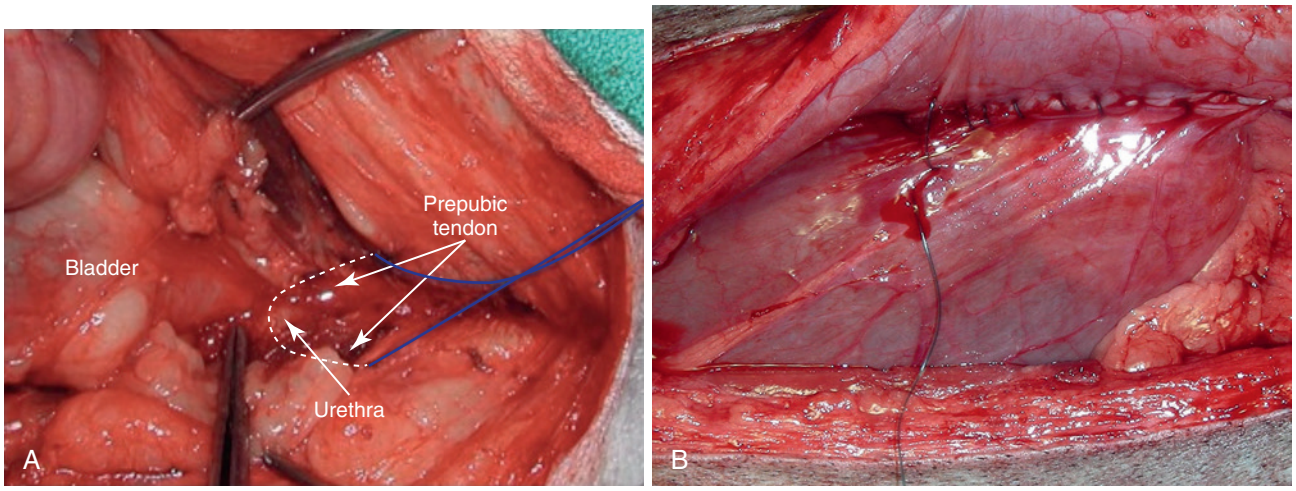


FIG. 25.45 Cystourethropexy. (A) For urethropexy, place two sutures between the prepubic tendon and the seromuscular layer of the ventral proximal urethral wall. (B) For cystopexy, place one or two rows of partial-thickness simple continuous suture lines between the ventral bladder wall and the body wall to either side of midline.

urethra within the abdominal cavity, the trigone and the proximal urethra are exposed to intraabdominal pressure that may improve the tone of the upper urethral sphincter.

To perform this surgery, pass the largest catheter possible into the urethra. Make a caudal abdominal midline approach and expose the pubic brim. Place a stay suture in the apex of the urinary bladder, facilitating cranial traction, and identify the proximal urethra. Pass a suture (0 to 2-0 nonabsorbable monofilament suture) from one prepubic tendon, transversely through the seromuscular layer of the ventral proximal urethra, and then through the other prepubic tendon (Fig. 25.45A). Before tying this suture, pass a second suture in an identical manner 3 to 5 mm cranial to the first. Tie both sutures gently to effectively close the caudal aspect of the abdominal wall.

Place a stay suture in the apex of the bladder to facilitate manipulation and positioning of the bladder. Position the bladder cranially in the abdomen without excessive tension. Use a No. 15 scalpel blade to gently scarify the ventral bladder wall for most of the length of the body. Create a scarified area of similar size on the ventral body wall just to the right or left of midline.

Do not incorporate the bladder wall in the abdominal wall closure. Place one or two rows of suture using a partial-thickness, simple continuous pattern (3-0 or 4-0 monofilament, absorbable material) to fix the bladder to the body wall (see Fig. 25.45B). After removal of the stay suture, close the abdominal wall, subcutaneous tissue, and skin routinely.

Endoscopic Collagen Injection

Position the dog in dorsal recumbency with the limbs extended cranially. Pass a cystoscope into the urethra via the external orifice. Inject collagen (approximately 2 mL/dog) at the 2, 6, and 10 o'clock positions (three injections) approximately 1.5 cm caudal to the neck of the bladder until the lumen of the urethra appears to be closed by the collagen deposits (Fig. 25.46). If bleeding hinders the view through the cystoscope, a laparotomy can be performed and the collagen injected under direct visualization, although this is seldom necessary.

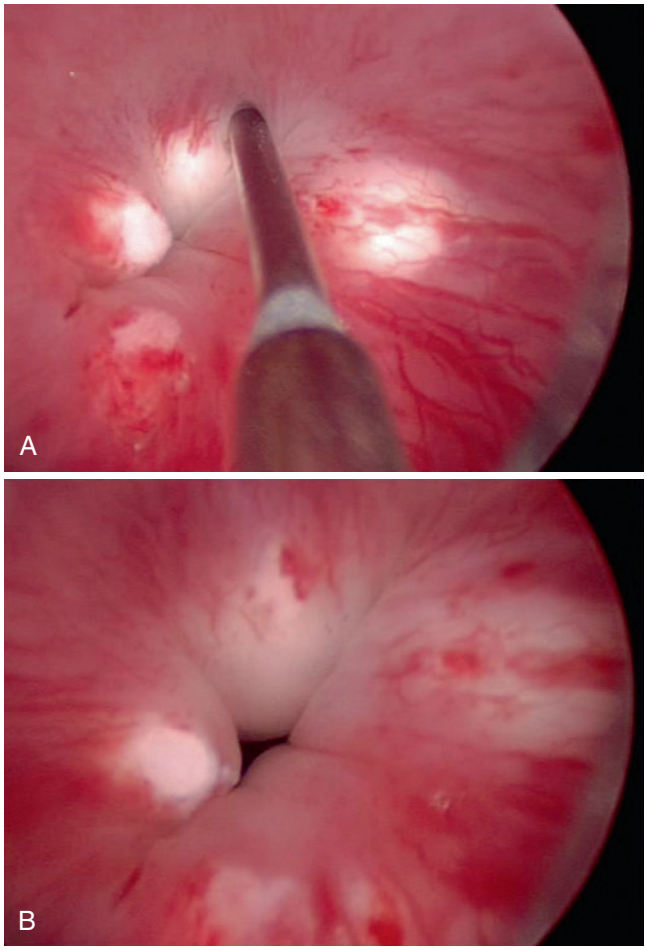


FIG. 25.46 (A) Cystoscopic image during initial collagen injection at the bladder trigone in a 1-year-old English bulldog that was still incontinent after surgical correction of bilateral ectopic ureters. (B) Appearance of the trigone after collagen injection.

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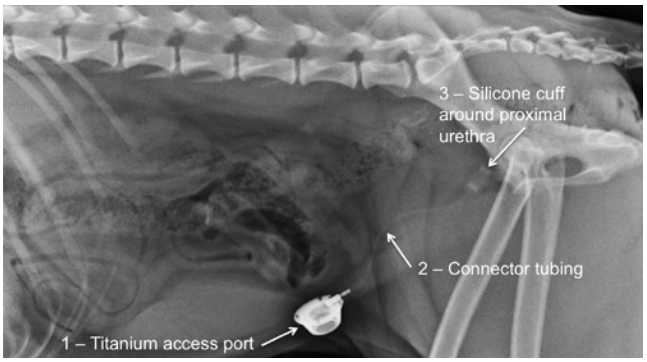


FIG. 25.47 Lateral abdominal radiograph of a 9-year-old large-breed dog illustrating the three components of an artificial urethral sphincter. *1,* Titanium access port with silicone hub; *2,* silcone catheter tubing connecting port to cuff; *3,* silicone cuff placed around the proximal urethra.

Artificial Urethral Sphincter Placement

AUSs are permanent, surgically-implanted devices that can be percutaneously adjusted to provide improved urinary continence in both male and female dogs and cats. The system consists of a silicone cuff and catheter connected to a titanium access port (Fig. 25.47). The cuff that is placed around the proximal urethra and the port is placed subcutaneously on the caudal abdomen or inner thigh. Using a Huber needle, the cuff is filled with variable amounts of saline from the access port to provide resistance to urine flow through the urethra. Frequently, the presence of the device itself around the proximal urethra will result in improved continence. The diameter of the urethral lumen is preoperatively estimated using ultrasound.

Make a caudal abdominal midline approach and expose the pubic brim. Place a stay suture in the apex of the urinary bladder, facilitating cranial traction, and identify the proximal urethra. Isolate a 2-cm segment of the proximal urethra from the periurethral fat using blunt dissection. Measure the urethral circumference using a piece of suture, umbilical tape, or a Penrose drain. Select an occlude catheter that is equal to or larger than the circumference of the urethra. Pass the catheter tubing through a stab incision in the abdominal wall and tunnel it lateral to the rectus abdominis in front of the cranial flank fold. Connect the tubing to the access port and secure the access port to the superficial fascia of the abdominal wall using monofilament, nonabsorbable suture. Prior to placing the cuff around the urethra, completely fill the system with saline, assess for leaks, and record the filling volume. Empty the system and place the cuff around the urethra, securing it in place with two 2-0 monofilament nonabsorbable sutures (e.g., polypropylene [Prolene] or polybutester [Novafil]) through the eyelets of the cuff. Close the abdominal wall, subcutaneous tissue, and skin routinely.

BLADDER FLAP RECONSTRUCTION OF A HYPOPLASTIC URETHRA

Perform a ventral cystotomy that extends into the proximal aspect of the hypoplastic urethra. Identify the ureteral openings. In addition to an allowance for suturing, make two stab incisions into the bladder wall caudal and lateral to the ureteral stoma, with the distance between the stab incisions representing the desired circumference of the new urethral tube (Fig. 25.48). A 3.5- or 5-Fr (cats) or 8-Fr (dogs) catheter should pass easily into the newly created urethral tube. *Use scissors to extend the incision toward the urethra, creating two full-thickness flaps. Reflect the flaps cranially (Fig. 25.49A). Suture the defect from the urethral end cranially to form the urethral tube (Fig. 25.49B) with a two-layer simple continuous suture pattern or a simple continuous suture pattern and a Cushing pattern. Use absorbable suture material (2-0 to 4-0). If the urethral lumen is compromised by placing two layers of sutures, suture the urethral tube with a single-layer appositional pattern, using care to ensure sufficient apposition of sutures such that urine leakage will not occur. Suture the flaps together.*

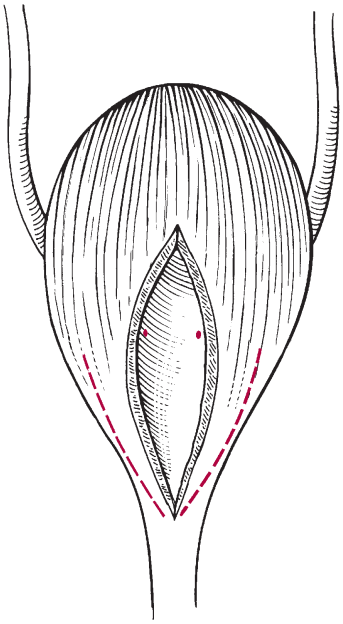


FIG. 25.48 For bladder flap reconstruction of a hypoplastic urethra, make two stab incisions into the bladder wall caudal and lateral to the ureteral stoma, then make two full-thickness flaps.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Absorbable suture materials (e.g., polydioxanone [PDS], polyglyconate [Maxon], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], or polyglytone 6211 [Caprosyn]) are preferred for bladder and urethral surgery (see p. 696). Bovine origin collagen may be used for the collagen injections (e.g., Zyplast).

POSTOPERATIVE CARE AND ASSESSMENT

The animal should be closely monitored for urinary obstruction or leakage following surgery. If urinary obstruction occurs, an indwelling urinary catheter should be placed and maintained for 3 to 5 days. Animals should be monitored for UTIs periodically after surgery.

COMPLICATIONS

If the urethral lumen is of insufficient width, or if swelling is excessive, urinary obstruction may occur. Other complications include return of incontinence and urinary leakage. Seroma formation is also possible with placement of an AUS.

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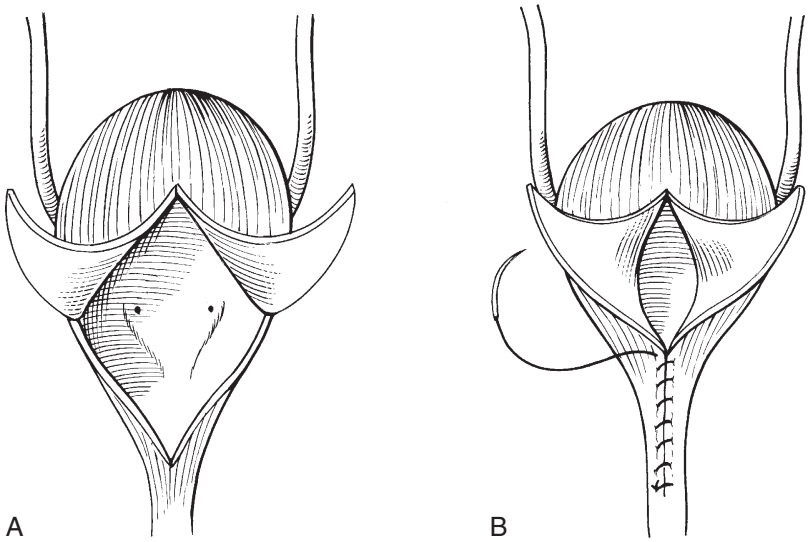


FIG. 25.49 (A) Use scissors to extend the incisions illustrated in Fig. 25.45 toward the urethra, creating two full-thickness flaps. (B) Reflect the flaps cranially and suture the defect from the urethral end cranially to form a urethral tube.

PROGNOSIS

Regardless of the surgical technique chosen, incontinence rates (complete or improved) range around 60% to 70% postoperatively. The most recent retrospective study in dogs receiving artificial urethral sphincters described markedly improved continence scores in 92% of dogs.[20] Across several studies, inflation of the cuff was not required in 33% to 45% of dogs to achieve continence.[20–22]

FELINE IDIOPATHIC CYSTITIS

DEFINITIONS

FIC is the currently accepted term to describe the idiopathic inflammatory process of the feline bladder and urethra that sometimes results in partial or complete urethral obstruction. *Feline interstitial cystitis* is essentially interchangeable with *feline idiopathic cystitis.* Other terminology in the past has included *feline urologic syndrome, feline lower urinary tract disease* (FLUTD), sterile cystitis, and *feline obstructive uropathy.* FLUTD is more accurately an umbrella term encompassing the clinical signs associated with several specific diseases of the feline lower urinary tract and should be avoided.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Cats with FIC may account for as many as 10% of all feline admissions to veterinary hospitals. Most cats with FIC have a neuroendocrine disease in which clinical signs may be initiated by stress and environmental factors.

NOTE Struvite crystals are not always associated with signs of FIC, and most cats with FIC do not have struvite crystals or calculi.

DIAGNOSIS

Clinical Presentation

Signalment

Overweight cats may be predisposed to FIC. Males and females are equally affected; however, male cats are more likely to be obstructed because of the small diameter of their urethras. Middle-aged cats are more commonly affected, and indoor cats may be at increased risk.

History

Unobstructed cats are usually brought in for evaluation of pollakiuria, stranguria, hematuria, and/or inappropriate urination. Cats with obstruction may appear uncomfortable or anxious and restless, may attempt to urinate frequently, may lick their genitalia, and may have abdominal pain. If obstruction has been present for longer than 36 to 48 hours, anorexia, dehydration, vomiting, collapse, stupor, hypothermia, and/or bradycardia may be noted (see p. 678).

Physical Examination Findings

If the cat is obstructed, the bladder will feel distended and firm (unless it has ruptured) and cannot be expressed. Abdominal palpation may elicit pain. Care should be taken when palpating the bladder of obstructed cats to prevent iatrogenic rupture. If the cat is not obstructed, the bladder will be empty, hard, and often painful when palpated.

Laboratory Findings

Laboratory findings may be normal or may show evidence of severe azotemia, metabolic acidosis, and/or hyperkalemia.

DIFFERENTIAL DIAGNOSIS

Other causes of urethral obstruction (e.g., stones, UTI, neoplasia, trauma) should be excluded on the basis of clinical signs, physical examination findings, and history.

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MEDICAL MANAGEMENT

For obstructed cats, fluid therapy should begin before laboratory data are returned. Fluids should be given intravenously to restore normal hydration, improve perfusion, and treat hyperkalemia (see p. 679). Physiologic saline solution, LRS, or Normosol-R should be used in case the cat is hyperkalemic. In all but the very sickest cats, this will be adequate to dilute hyperkalemia and reverse cardiotoxicity. If serum potassium is later found to be normal, a balanced electrolyte solution should be administered.

Obstruction should be promptly relieved by urethral catheterization or gentle penile massage, if possible. If the cat is severely depressed, minimal restraint may be necessary. In other cats, general anesthesia may be required (see later discussion). Sterile isotonic fluid should be used to flush plugs or calculi into the bladder. Nonmetal, smooth, well-lubricated catheters are preferred to minimize urethral trauma. If the catheter cannot be advanced, cystocentesis may be helpful. Cystocentesis may be indicated as an initial step of bladder decompression if the cat is in serious condition. If a normal stream is not present after catheterization, or if detrusor atony is present, an indwelling, soft urinary catheter may be sewn in place; however, this predisposes the cat to UTI. The cat should be stabilized before a perineal urethrostomy is considered. FIC is likely to be the cause of urethral obstruction in young adult male cats when insertion of a red rubber catheter into the bladder is easy at the time of presentation, and when a large amount of hemorrhagic urine is voided into the collection system. Some of these cats will void spontaneously when anesthetized for catheterization.

The routine use of antimicrobials in cats with FIC is not recommended. The use of antispasmodics, glycosaminoglycans, and antiinflammatories is widespread for these cases; however, evidence in support of the efficacy to prevent recurrence is limited. Intravesicular infusions with various agents (e.g., lidocaine, pentosan polysulfate sodium) have also been unrewarding, although a small study found 0% recurrence in cats receiving intravesicular glycosaminoglycans.[23]

SURGICAL TREATMENT

Perineal urethrostomy may be performed to prevent recurrence of obstruction in male cats who have repeated episodes of FIC, or to treat obstruction that cannot be eliminated by catheterization. It is also useful to treat strictures that occur following urethral obstruction and catheterization. With appropriate nonsurgical treatment of obstructed cats, this procedure can often be avoided. Postoperative bacterial UTIs after urethrostomy are possible owing to anatomic alterations of the urethral meatus, compromised intrinsic defense mechanisms, and underlying uropathy. Many cats have permanent loss of striated urethral sphincter function after this procedure, although incontinence is rare.

Preoperative Management

Electrolyte (i.e., hyperkalemia) and acid-base abnormalities should be corrected before anesthetic induction (see p. 678). Fluids should be given intravenously to restore normal hydration and to combat postobstruction diuresis. Cats who were initially severely uremic will often have a substantial postobstruction diuresis during which they require large volumes (i.e., sometimes greater than two to three times maintenance requirements) of IV fluids to prevent severe hypovolemia. Postobstruction diuresis occurs in 46% of cats following relief of obstruction.[24] Serum potassium concentrations must be monitored to prevent hypokalemia.

Anesthesia

An ECG should be monitored before, during, and after surgery for cardiac arrhythmias. If the cat has been adequately stabilized (i.e., hydration and potassium are normal), diazepam followed by propofol or alfaxalone may be used for induction; see Table 25.1). Thiobarbiturates are arrhythmogenic and therefore should be avoided or used cautiously in animals with preexisting arrhythmias. Isoflurane and sevoflurane in oxygen are the least cardiodepressant inhalation anesthetics and should be used for maintenance. Shocky, dehydrated, or hypovolemic patients often do not need premedications and should be induced with etomidate-benzodiazepine or very low dose propofol or alfaxalone (see Table 25.2). Opioids can be given intraoperatively to effect to prevent postoperative pain. Because ketamine is excreted via the kidneys in active form, it should be avoided or used very cautiously and at low doses in cats with urinary obstruction. Mask induction should not be used if the cat is vomiting.

Surgical Anatomy

For a description of the surgical anatomy of the urethra, refer to p. 684.

Positioning

The cat is placed in sternal recumbency with the perineal region elevated slightly. Ventilation may need to be assisted when the cat is positioned in this manner.

SURGICAL TECHNIQUE

Perineal urethrostomy is described on p. 690.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

A urinary catheter is placed in the urethra to help locate it during the operation. Monofilament absorbable (polydioxanone [PDS], polyglyconate [Maxon], poliglecaprone 25 [Monocryl], or glycomer 631 [Biosyn]) or nonabsorbable (polypropylene [Prolene] or nylon [Monosof]) suture is preferred. If nonabsorbable suture is used, the sutures should be removed 10 to 14 days after surgery. Tenotomy scissors and small, atraumatic forceps are useful.

POSTOPERATIVE CARE AND ASSESSMENT

Paper instead of gravel litter should be used until the wound is healed. Urinary cultures should be performed periodically to check for UTI. Indwelling catheters should not be used routinely following surgery because they promote stricture formation and/or UTI.

NOTE Stricture formation following perineal urethrostomy usually results from making the stoma too small, most commonly by not extending the incision into the distal pelvic (membranous) urethra at the level of the bulbourethral glands.

COMPLICATIONS

The most serious complication of perineal urethrostomy is stricture formation. Stricture formation in cats following perineal

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urethrostomy is generally a result of making the stoma too small owing to inadequate dissection to the bulbourethral glands, or it is a result of postoperative subcutaneous urine leakage and subsequent granulation tissue formation from inadequate mucosa-to-skin apposition (see p. 697). Urinary and fecal incontinence may occur if nerves are damaged during aggressive dissection around the pelvic urethra (see p. 697). Perineal urethrostomy may contribute to UTI postoperatively. Indwelling catheters may allow ascending bacterial infection and/or may cause fibrosis and stricture. Although incontinence is rare, some cats have permanent loss of striated urethral sphincter function after this procedure. Rectal prolapse has been reported following perineal urethrostomy in cats.

NOTE Evaluate cats with a perineal urethrostomy periodically for urinary tract infection.

PROGNOSIS

The mortality rate of obstructed cats can be as high as 20%. This high rate is often due to recurrence, failure to identify and address the underlying primary bladder disease, and financial constraints of owners reluctant to finance multiple or prolonged hospitalizations. Recurrence of obstruction has been historically reported to occur in 20% to 35% of cases, regardless of the cause of obstruction. In a 2013 retrospective study, there was an 11% recurrence by 24 hours and 24% recurrence by 30 days.[25] Risk factors associated with recurrence with that study include the use of a larger urethral catheter (5 Fr vs. 3.5 Fr) and the use of phenoxybenzamine instead of prazosin. In a 2013 prospective study, there was a 14.7% recurrence by 30 days, more commonly associated with older cats.[26]

Prognosis is generally good following perineal urethrostomy. A 2012 retrospective study reported a median survival time of 3.5 years with 47 of 86 cats still alive at the time of the study.[27] Recurrence of clinical signs of FIC was reported in 10.7% of cases.

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26

Surgery of the Reproductive and Genital Systems



GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Neuter refers to **ovariohysterectomy (OHE)** (surgical removal of the ovaries and uterus), **ovariectomy (OVE)** (surgical removal of the ovaries alone), or **orchiectomy** (surgical removal of the testicles). **Ovary-sparing surgery** involves complete removal of the uterus (**hysterectomy**) while leaving the ovaries intact. **Vasectomy** is ligation and transection of the vas deferens. **Castration** refers to removal of either the male or female sex organs, but it is most commonly used interchangeably for orchiectomy. **Mastectomy** is excision of one or more mammary glands or mammary tissue. **Episiotomy** is incision of the vulvar orifice to expose the vulva and vagina, whereas **episioplasty** or **vulvoplasty** is reconstruction of the vulva. **Prostatectomy** is removal of all or a portion of the prostate gland. **Hysterotomy** is a surgical incision into the uterus (e.g., cesarean section).

PREOPERATIVE MANAGEMENT

Reproductive surgery encompasses a variety of techniques designed to alter the animal's ability to reproduce, aid parturition, or treat or prevent disease of the reproductive organs (Box 26.1). The primary indication for reproductive tract surgery is to limit reproduction, but it may also be done to relieve dystocia, prevent or treat tumors influenced by reproductive hormones (e.g., mammary tumors, testicular tumors, perianal adenomas), control certain diseases of the reproductive tract (e.g., pyometra, metritis, prostatitis, tumor, prostatic abscessation), and help stabilize systemic disease (e.g., diabetes, epilepsy). Neutering is performed in some animals to prevent or alter behavioral abnormalities and to reconstruct traumatized, diseased, or malformed tissue. Diagnosis of reproductive tract disease is based on history, clinical signs, physical examination, diagnostic imaging (e.g., radiographs, ultrasound, computed tomography [CT], magnetic resonance imaging [MRI], bone scan), endoscopy, cytology, microbiology, hormonal assay, hematology, serum biochemistry profile, urinalysis, and other laboratory results.

History

History and clinical signs of animals needing reproductive surgery depend on the gender and disease. Most animals brought in for elective reproductive surgery (e.g., castration, OVE, OHE) are healthy. Asymptomatic animals with neoplasia may have a mass found incidentally by the owner. Those with genital tract infections may be severely ill and have fever, toxemia, incontinence, and/or obstruction. Prostatic disease is common in male dogs, especially intact male dogs, although clinical signs may be nonspecific (e.g., fever, malaise, vomiting, dehydration, caudal abdominal pain, gait abnormalities). Hematuria caused by reflux of blood from the prostatic urethra into the bladder and/or concurrent urinary tract infection may occur. Urethral discharge (preputial/urethral drip) is caused by passive release of blood, pus, or prostatic fluid into the prostatic urethra. Stranguria occurs when the prostate compresses or obstructs the urethra or there is inflammation caused by urinary tract infection. Urinary incontinence (see p. 712) may be caused by prostatic impingement on the pelvic nerves. Prostatic enlargement compressing the rectum can cause tenesmus or stools that are smaller than normal. Constipation may occur secondary to obstruction or pain during defecation.

Females

Physical Examination

Physical examination should include inspection and palpation of the abdomen, vulva, and mammary glands. Abdominal palpation may reveal an enlarged uterus, mass, visceral displacement, and/or pain. Abnormal vulvar skin folds, conformation, discharge, or enlargement may be noted. During estrus and proestrus, the vulva is swollen to two or three times its normal size, appears turgid, and has a hemorrhagic to straw-colored discharge. The swelling and turgidity diminish during estrus and diestrus. A vaginal examination is recommended when vaginal discharge or enlargement is detected. The vestibule and vagina should be visualized and digitally palpated. If the vagina is too small to allow vaginal examination, then a rectal examination may allow the clinician to palpate abnormalities otherwise inaccessible during physical examination. Mammary glands should be inspected for symmetry, texture, size, mobility, discharge, and the presence of masses.

Clinical Pathology

If masses are identified, cytology of aspirates or discharges may help differentiate inflammation from neoplasia. Vaginal, uterine, or mammary gland cultures are recommended if infection is suspected. Vaginal cytology should be consistent with the bitch's estrus cycle. The normal vaginal flora includes numerous aerobic and anaerobic bacteria (Box 26.2). Pure growth of bacteria from vaginal specimens can be normal unless accompanied by signs of reproductive tract disease. Persistent vaginal discharge is an indication for brucellosis testing.

Hormone Concentrations

Assessment of hormone levels is occasionally helpful in diagnosing female reproductive problems. In female dogs, estradiol levels are 10 pg/mL or less during late anestrus, 10 pg/mL or greater at onset of proestrus, and 50 to 100 pg/mL at late proestrus.

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ABSTRACT

Surgery of the reproductive tract involves a variety of techniques that affect an animal's ability to reproduce, aid a bitch or queen in dystocia, or treat certain specific diseases of the reproductive organs. Often these disease processes are influenced by reproductive hormones. Diagnosis of reproductive tract disease is based on, but not limited to, history, clinical signs, physical examination, laboratory results, diagnostic imaging, and hormonal assay.

KEYWORDS

ovariohysterectomy
ovariectomy
orchiectomy
vasectomy
castration
hysterectomy
hysterotomy
mastectomy
episiotomy
episioplasty
vulvoplasty
prostate
prostatectomy
pyometra
prolapse
hyperplasia
omentalization
marsupialization
phimosis
paraphimosis
priaprism
hypospadias
phallopexy

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BOX 26.1 Surgical Procedures of the Reproductive Tract

Ovariohysterectomy
Castration
Cesarean section
Cryptorchid castration
Mastectomy
Scrotal ablation
Episiotomy
Vasectomy
Episioplasty
Prostatic drainage
- Omentalization
- Marsupialization

Prostatectomy
Penile amputation
Preputial reconstruction
Phallopexy
Biopsy

BOX 26.2 Normal Vaginal Flora: Common Organisms

α- and β-hemolytic *Streptococcus* spp.
Staphylococcus spp.
Proteus spp.
Escherichia coli
Bacillus spp.
Bacteroides spp.
Pasteurella spp.
Anaerobic enterococci
Mycoplasma

TABLE 26.1 Methods to Determine Pregnancy Risk After Mating

Test	Result
Vaginal cytology within 48 h: • Saline-moistened swab in vagina for 1 min • Incubate swab in test tube with 0.5 mL saline for 10 min • Compress swab against tube and centrifuge at 2000 rpm for 10 min • Extend sediment on slide and diff-Quik stain	Detect spermatozoa
Progesterone levels	Low risk with levels <2 ng/mL High risk with levels >10 ng/mL

TABLE 26.2 Methods of Pregnancy Determination

Abdominal palpation	Perform 26–28 days, but before 30 days, after mating to detect uterine swellings approximately 3–5 cm in diameter
Transabdominal ultrasonography	Canine: • Detect embryonic vesicles 19–20 d after LH peak • Detect embryo and cardiac motion at day 23–25 after LH peak • Detect fetal movement and fetal heart rate by day 31–32 after LH peak Feline: • Detect gravid uterine enlargement 4–14 d postcoitus • Detect gestational sac 11–14 d postcoitus • Detect fetal pole 15–17 d postcoitus
Relaxin ELISA test	Positive 21 days after breeding
Abdominal radiography	• Detect enlarged canine uterus 31–38 d after LH peak • Detect canine fetal mineralization 45 d after LH peak • Detect enlarged feline uterus 25–35 d after LH peak • Detect general feline fetal mineralization 25–29 d before parturition or 25–35 d after LH peak; first evidence of mineralization is seen in the spinal column at 22–27 d, skull at 21–27 d, ribs at 20–25 d, scapula at 17–24 d, humerus at 20–24 d, femur at 19–23 d, radius at 15–22 d, tibia at 15–21 d, ulna at 5–21 d, pelvis at 8–20 d, fibula at 0–17 d, tail at 8–16 d, metatarsals and metacarpals at 3–14 d, phalanges at 0–11 d, calcaneus at 0–10 d, and teeth at 1–6 d before parturition

ELISA, Enzyme-linked immunosorbent assay; *LH,* luteinizing hormone.

Progesterone levels are 0.5 to 1 ng/mL during anestrus and proestrus, 2 to 5 ng/mL at luteinizing hormone (LH) surge; levels peak at 15 to 90 ng/mL 15 to 30 days later and slowly decline thereafter to 2 ng/mL or less in pregnant (mean, 63 days) or nonpregnant diestrus (50–100 days) bitches (Table 26.1). Progesterone greater than 2 ng/mL is necessary to maintain pregnancy. Progesterone must fall below 2 ng/mL to initiate parturition; a drop in progesterone levels to less than 1 to 2 ng/mL for 2 consecutive days will typically terminate pregnancy. Measuring LH helps distinguish between neutered and intact bitches or queens, ovarian remnant syndrome, and administration of exogenous sex steroids. Knowing the time of the LH surge (>1 ng/mL) is helpful in determining ideal breeding times and predicting parturition.

Diagnostic Imaging

Positive contrast vaginography may help evaluate vaginal anomalies, masses, or injuries if digital examination does not adequately define the problem. Positive contrast vaginography using a Foley catheter and a water-soluble iodinated contrast agent is easily performed. Endoscopy of the vagina is the best technique for direct visualization when performed properly. An otoscope or vaginal speculum allows visual inspection of the vestibule and caudal vagina; however, a longer endoscope is needed to evaluate the cranial vagina and cervix. Insufflation with air or water will distend mucosal folds and allow thorough evaluation.

Thoracic and abdominal radiographs and ultrasound, CT, or MRI images may be needed if reproductive tract tumors are suspected. Survey radiographs uncommonly identify the normal nongravid uterus. Radiographically, an enlarged gravid uterus can be detected within 31 to 38 days and fetal skeletal mineralization by 45 days after the LH peak (within a mean of 0.5 days of onset of estrus) (Table 26.2). In cats, it is difficult to know when the LH peak has occurred because behavioral estrus is variable

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(1–21 days); therefore fetal skeletal mineralization, which is first detected at 25 to 29 days before parturition, is used to predict when parturition will occur. Ovarian masses and follicular changes can be detected in the periovulatory period using ultrasonography. The normal, nongravid uterus can be identified using a high-frequency (>10 MHz) transducer. Using a fluid-distended urinary bladder as an acoustic window may reveal fluid-distended horns and a thickened uterine wall. Pregnancy and fetal viability can be detected ultrasonographically as early as 19 to 20 days after the LH surge in dogs or 15 to 17 days postcoitus in cats, but pregnancy diagnosis by ultrasound is uncomplicated at 30 days of gestation (see Table 26.2). Ultrasonographic signs of abortion or lack of fetal viability include changes in fetal anatomy, placental detachment, or fetal resorption; however, serial evaluations may be necessary. Ultrasonography can also detect uterine cysts, masses, or fluid; premature placental separation; and a thickened uterine wall.

Other Tests

Cystometrograms and urethral pressure profiles may occasionally help assess dogs with urinary incontinence (see Chapter 25) but are uncommonly performed. Histologic evaluation is often necessary to confirm the diagnosis of female reproductive disorders and allow a more accurate prognosis with most conditions.

Males

Physical Examination

Thorough abdominal and rectal palpation is needed to evaluate prostatic size, symmetry, texture, and mobility; and medial iliac lymph node size. Prostatic palpation sometimes elicits pain in animals with prostatitis. The scrotum should be examined for size, symmetry, thickening, masses, sensitivity, and scrotal adhesions. Testicles should be palpated for size, consistency, contour, symmetry, and sensitivity. The prepuce and penis are observed for signs of trauma, wounds, masses, irritation, and congenital abnormalities. The penis should be completely extruded from the prepuce for thorough examination.

Clinical Pathology

Cytology of prostatic fluid is one of the most informative tests to evaluate the male reproductive system. Prostatic fluid is best obtained by ejaculation, but a prostatic wash or fine-needle aspirate is sometimes acceptable. Prostatic massage or ejaculation fluid in normal dogs has few transitional cells, rare neutrophils, and varying numbers of erythrocytes. Fine-needle aspiration cytology is more likely to demonstrate neoplastic cells, increased neutrophils, bacteria, and other debris. Urine and prostatic fluid should be cultured to detect bacterial infections. Cytologic evaluation should be performed on all accessible masses. A biopsy is necessary to definitively diagnose prostatic, testicular, penile, preputial, or scrotal masses.

Hormone Concentrations

Measurement of serum hormone concentrations may aid in determining whether a male dog has been neutered or has a hormone-producing tumor; however, episodic release of gonadotropins makes interpretation of hormone assays difficult, and reference values vary between laboratories. In intact male dogs, serum testosterone concentrations range between 0.5 and 9 ng/mL, whereas serum estrogen concentrations are generally less than 15 pg/mL. Finding testosterone levels less than 100 pg/mL in dogs indicates that they have been neutered. In toms, testosterone secretion is episodic without diurnal rhythm. In normal toms, resting serum testosterone concentrations range from nondetectable to 1 to 6 ng/mL (81.5 nmol/L), and in castrated toms the concentrations are less than 0.5 ng/mL.

Diagnostic Imaging

Abdominal radiographs define prostatic size, shape, and location. Radiographically the normal prostate may be near the cranial brim of the pubis and should not displace the colon or bladder. Its contour should be smooth and symmetric. Abnormal prostates may be asymmetric, irregular, and/or displace adjacent viscera. Medial iliac lymph nodes, lumbar vertebrae, and the bony pelvis should be evaluated for evidence of metastases. A positive contrast cystourethrogram helps evaluate prostatic position in relation to the bladder, urethral size, mucosal contour, and prostatic reflux. Ultrasonography defines parenchymal homogeneity, contour, disease distribution, and urethral diameter. Cytology and biopsy specimens can be collected with ultrasound guidance. A bone scan and thoracic radiographs may help stage prostatic neoplasia. Occasionally, radiography may help evaluate os penis fractures and urethral extension of disease. Ultrasonography may be used to evaluate scrotal swelling and detect testicular abnormalities, including cryptorchidism, testicular torsion, and neoplasia. The normal testicular architecture is coarse but homogeneous, with the mediastinum testis represented as a central hyperechoic band. The epididymis is anechoic to hypoechoic, relative to the testicular parenchyma.

Other Tests

Evaluating urinary incontinence with a cystometrogram and urethral pressure profile is possible but uncommonly performed (see p. 713).

ANESTHESIA

Numerous anesthetic protocols may be used for elective surgery in healthy animals. As with any other surgery, a balanced technique is recommended to provide adequate anesthesia and analgesia. Suggested anesthetic protocols for healthy animals undergoing elective surgery are provided in Table 26.3. In addition, refer to Chapter 12 for a discussion of the variety of medications that may be used to anesthetize healthy animals (see Table 12.1 for anesthetic drugs, their doses, uses and precautions, and onset of action; and Fig. 12.1 for an anesthesia safety checklist).

Neutering has traditionally been recommended at 6 to 12 months of age. Early neutering (6–16 weeks) produces good results if precautions are taken to prevent hypoglycemia, hypothermia, and hemorrhage. Pediatric patients, less than 16 weeks of age, are metabolically different from adults in a variety of ways. As numerous systems are immature, administration of drugs requires special care and consideration. The cardiovascular system has limited reserve because of an inability to increase myocardial function, and cardiac output is rate dependent. The sympathetic nervous system is immature, making a puppy or kitten susceptible to bradycardia and hypotension. In addition, the circulating fluid volume has limited reserve, and dehydration is a risk with prolonged water restriction. Pediatric patients have relatively small lung volumes compared with adults, but a higher metabolic rate with subsequent higher oxygen requirements. Their body weight has a higher percentage of water, which alters the volume of distribution of many drugs. Hepatic metabolism

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is not mature until approximately 12 weeks of age. Additionally, renal tubular secretion may not mature until 8 weeks of age. The immature hepatic and renal metabolism along with hypoalbuminemia and a larger circulatory fluid volume make pediatric patients sensitive to anesthetic drugs. Drug actions may be more pronounced and prolonged. In neonatal patients less than 6 weeks of age, this is worsened by an increased permeability of the blood-brain barrier. Lastly, these young patients are at risk for hypoglycemia, hypothermia, and anemia.

Avoid administering acepromazine and nonsteroidal antiinflammatory drugs (NSAIDs) in animals less than 16 weeks of age. Although alpha-2 agonists are approved for use in dogs 12 weeks of age and older, lower doses should be considered in pediatric patients. Because of diazepam's active metabolites, midazolam is a much better choice for premedication. Ketamine may be used, but doses in the lower range are more appropriate because of its increased volume of distribution. Propofol, alfaxalone, sevoflurane, and isoflurane are excellent anesthetics for pediatrics. As with other age groups, opioids must be titrated to effect; however, lower doses are usually needed to achieve adequate analgesia. Buprenorphine is often a good choice because it has a prolonged duration of action and minimal side effects. Because these patients have immature sympathetic nervous systems, they should either be premedicated with an anticholinergic (e.g., atropine, glycopyrrolate) or it should be available in the operating room to treat bradycardia.

TABLE 26.3 Anesthetic Considerations in a Healthy Patient for Routine Surgery

Preoperative Considerations

Associated conditions	• Minimal; usually healthy
Bloodwork	• HCT • TP • In patients >5–7 y, consider electrolytes, liver enzymes, BUN, and Cr

Physical Exam — Normal

Premedications	• If patient is anxious, give:
	  • Diazepam (0.2 mg/kg IV), *or*
	  • Midazolam (0.2 mg/kg IV, IM), *plus*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM in dogs; 0.05–0.1 mg/kg IV, IM in cats), *or*
	  • Morphine[b] (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM), *or*
	  • Oxymorphone (0.1–0.2 mg/kg IV, IM), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM) if moderate pain anticipated

Intraoperative Considerations

Induction	• If premedicated, give:
	  • Propofol (2–4 mg/kg IV), *or*
	  • Ketamine (5 mg/kg IV with diazepam or midazolam as above), *or*
	  • Alfaxalone (2–3 mg/kg IV)
	• If not premedicated, give:
	  • Propofol (4–8 mg/kg) IV), *or*
	  • Alfaxalone (2–5 mg/kg IV), *or*
	  • Ketamine (5.5 mg/kg IV) with diazepam (0.28 mg/kg IV), *or*
	  • Dexmedetomidine[d] (33 µg/kg IM) plus butorphanol (0.66 mg/kg IM), plus ketamine (6.6 mg/kg IM); use half of all doses if given IV

Maintenance	• Isoflurane or sevoflurane, *plus*
	  • Fentanyl (2–10 µg/kg IV PRN in dogs; 1–4 µg/kg IV PRN in cats) for short-term pain relief, *plus* PRN
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Oxymorphone (0.1–0.2 mg/kg IV, IM), *or*
	  • Morphine[b] (0.1–1 mg/kg IV PRN in dogs; 0.05–0.2 mg/kg IV PRN in cats), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV PRN), *or*
	  • Dexmedetomidine[d] (low dose; 0.5–1 µg/kg IV), *or*
	  • Medetomidine[d] (low dose; 1–2 µg/kg IV), *plus*
	  • Ketamine (low dose; 0.5–1 mg/kg IV), *or*
	  • Ketamine CRI (0.5 mg/kg IV loading dose then 10 µg/kg/min IV)

Fluid needs	• 5–10 mL/kg/h plus 3× EBL • 10–20 mL/kg/h plus 3× EBL if open abdomen
Monitoring	• Blood pressure • HR • ECG • Respiratory rate • SpO_2 • Temperature • $EtCO_2$

Blocks	• If abdominal surgery, then epidural with:
	  • Morphine (0.1 mg/kg preservative free), *or*
	  • Buprenorphine (0.003–0.005 mg/kg diluted in saline)
	NOTE: can combine with bupivacaine (0.5 mg/kg) to increase duration

Continued

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TABLE 26.3 Anesthetic Considerations in a Healthy Patient for Routine Surgery—cont'd

Postoperative Considerations

Analgesia	• Fentanyl CRI (1–10 µg/kg IV loading dose, then 2–20 µg/kg/h IV), *or*
	• Hydromorphone CRI (0.025–0.1 mg/kg per hour IV in dogs), *or*
	• Morphine[b] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.05–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h in cats), *or*
	• Hydromorphone[a] (0.1–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats), *or*
	• Oxymorphone (0.1–0.2 mg/kg IV, IM), *or*
	• Buprenorphine[c] (5–20 µg/kg IV, IM, SC q4–8h), *plus*
	• +/– Ketamine CRI (2 µg/kg/min IV; if no previous loading dose, give 0.5 mg/kg IV prior to CRI), *plus*
	• In dogs:
	  • Carprofen (2.2 mg/kg q12h PO), *or*
	  • Deracoxib (3–4 mg/kg q24h for <7 days PO), *or*
	  • Meloxicam (0.1–0.2 mg/kg once SC, PO then 0.1 mg/kg PO q24h)
	• In cats:
	  • Meloxicam[e] 0.15 mg/kg SC once; approved for doses up to 0.3 mg/kg SC (see also Table 34.3), *or*
	  • Buprenorphine[c] (0.01–0.02 mg/kg OTM q6–12h)

Monitoring	• SpO_2 • Blood pressure • HR • Respiratory rate • Temperature
Bloodwork	HCT and TP if large blood loss
Estimated pain score	Depends on procedure

[a]Monitor for hyperthermia in cats.
[b]Give slowly to prevent histamine release.
[c]Buprenorphine is a better analgesic than morphine in cats.
[d]Use only in young animals.
[e]Black box warning added by the Food and Drug Administration in October 2010 identified cases of renal failure and death in cats with repeated uses of meloxicam. Meloxicam is approved for single use only in cats in the United States.
BUN, Blood urea nitrogen; *Cr,* creatinine; *CRI,* constant rate infusion; *EBL,* estimated blood loss; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *OTM,* oral transmucosal; *PO,* orally; *PRN,* as needed; *SC,* subcutaneous; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein.

Patients requiring cesarean section are often greater anesthetic risks because of hypovolemia, hypoglycemia, and hypocalcemia. The pregnant dog or cat has numerous metabolic changes, including increased circulating blood volume, cardiac output, heart rate, and oxygen consumption. Additionally, there are decreases in packed cell volume, lung volumes, and gastric emptying. The primary anesthetic goal is to adequately and safely anesthetize the mother without endangering the fetuses. Drugs that depress the mother also depress the fetus. Therefore minimal preoperative medications are used. The patient should be rehydrated if indicated. Preoxygenating is recommended in most pregnant patients for approximately 3 minutes. The patient is then rapidly induced and intubated. Propofol and alfaxalone are excellent induction agents associated with a rapid and smooth induction of anesthesia with no residual neonatal depression. Alfaxalone has been shown to be associated with improved neonatal viability in the first 60 minutes following delivery when compared with propofol.[1] Fentanyl may be given with induction and titrated to effect prior to the removal of puppies or kittens. Maintenance should be done with isoflurane or sevoflurane with oxygen. Nitrous oxide crosses the placental barrier, but lower levels are found in the fetus than in the mother. Nitrous oxide at 50% is less likely to cause neonatal hypoxemia, but if it is used, oxygen should be provided to the neonates upon delivery via a face mask or oxygen chamber. If time allows, a fentanyl epidural will assist in decreasing the mother's anesthetic requirement. This can be accomplished immediately after induction and before prepping of the abdomen. Before making the skin incision, a lidocaine line block may be used in cats (at reduced dosages) and dogs. It is recommended to minimize the medications given prior to the removal of the fetuses. Once this is completed, other opioids (e.g., hydromorphone, morphine, buprenorphine) and ketamine may be given. Have glycopyrrolate and naloxone available in the operating room, and administer glycopyrrolate if the mother becomes bradycardic due to uterine manipulation. Naloxone may be given at doses of one to two drops sublingually to the neonates if respiratory depression secondary to the fentanyl is suspected. Suggested anesthetic protocols for cesarean section are provided in Table 26.4. See pp. 735 to 738 for techniques for cesarean section and neonatal care.

ANTIBIOTICS

Perioperative antibiotics are not necessary for elective OVE, OHE, or castration. Antibiotic choice should be based on culture and susceptibility or on expected pathogens in patients with pyometra, metritis, or bacterial prostatitis. Until culture results are available, antibiotics used to treat pyometra should be efficacious against *Escherichia coli* because this is the most common pathogen. Aminoglycosides are nephrotoxic and should be avoided when possible because of the renal dysfunction seen in pyometra. Choice of prophylactic antibiotics for surgery involving tumors or trauma depends on the patient's condition and surgeon's preference (see Chapter 9).

Antibiotic selection for prostatic diseases should be based on culture results and expected blood-prostate barrier penetration. Antibiotics should be lipid-soluble (usually nonionized), non-protein bound, and have a high pKa (degree of drug ionization; high pKa = more basic). Those with a high degree of lipid solubility are best at crossing the blood-prostate barrier. Antibiotics with a high pKa are less ionized at physiologic conditions and concentrate in the prostate. Prostatic infections produce acidic prostatic fluid that helps trap antibiotics in the fluid. Basic antibiotics that concentrate in the prostate include erythromycin,

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clindamycin, and trimethoprim (Box 26.3). Enrofloxacin and doxycycline achieve high prostatic fluid concentrations and are effective against some resistant gram-negative urogenital pathogens.

SURGICAL ANATOMY

Female Reproductive Tract

The female reproductive tract includes the ovaries, oviduct, uterus, vagina, vulva, and mammary glands. The ovaries are located within a thin-walled peritoneal sac; the ovarian bursa is located just caudal to the pole of each kidney. The uterine tube or oviduct courses through the wall of the ovarian bursa. The right ovary lies further cranially than the left. The right ovary lies dorsal to the descending duodenum, and the left ovary lies dorsal to the descending colon and lateral to the spleen. Medial retraction of the mesoduodenum or mesocolon exposes the ovary on each side. Each ovary is attached by the proper ligament to the uterine horn and via the suspensory ligament to the transversalis fascia medial to the last one or two ribs. The ovarian pedicle

TABLE 26.4 Anesthetic Considerations in the Patient Undergoing a Cesarean Section

Preoperative Considerations

Associated conditions	• Increased HR, blood volume, oxygen consumption, and cardiac output • Decreased lung volume, gastric emptying, and HCT
Bloodwork	• HCT • TP • Glucose • Calcium • In patients >5–7 y, consider electrolytes, liver enzymes, BUN, and Cr
Physical exam	• Pregnant female, often in active labor

Premedications	• If patient is anxious, give:
	  • Fentanyl (2–10 µg/kg IV in dogs, 1–4 µg/kg IV in cats)
	• If patient is not tachycardic give:
	  • Glycopyrrolate (0.005–0.01 mg/kg IV)

Intraoperative Considerations

Induction	• Preoxygenate 3–5 min • Propofol (4–8 mg/kg IV), *or* • Alfaxalone (2–5 mg/kg IV), *or* • Etomidate (0.5–1.5 mg/kg IV); administer after giving midazolam (0.25 mg/kg IV) to prevent myoclonus

Maintenance	• Isoflurane or sevoflurane, *plus*
	• Fentanyl (2–10 µg/kg IV PRN in dogs, 1–4 µg/kg IV PRN in cats) for short-term pain relief
	• If muscle relaxation needed, give:
	  • Atracurium (0.2 mg/kg IV), *or*
	  • Vecuronium (0.01–0.02 mg/kg IV), *or*
	  • Cisatracurium (0.1 mg/kg IV)
	NOTE: Intermittent positive-pressure ventilation and train of four monitoring are needed when using muscle relaxants to monitor the depth of the block and to know when the block has worn off sufficiently to allow adequate spontaneous ventilation during recovery.
	NOTE: Muscle relaxants do not provide analgesia or anesthesia.
	• Glycopyrrolate (0.005–0.01 mg/kg IV) if bradycardia with uterine manipulation
	• After the removal of the neonates, give:
	  • Fentanyl (2–10 µg/kg IV PRN in dogs; 1–4 µg/kg IV PRN in cats) for short-term pain relief, *plus*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Oxymorphone (0.1–0.2 mg/kg IV, IM), *or*
	  • Morphine[b] (0.1–1 mg/kg IV PRN in dogs; 0.05–0.2 mg/kg IV PRN in cats), *plus*
	  • Ketamine (low dose) (0.5–1 mg/kg IV)
	  • Have naloxone available in the operating room; give 1–2 drops sublingual for reversal of opioid-induced respiratory depression

Fluid needs	• 10–20 mL/kg/h plus 3× EBL
Monitoring	• Blood pressure • HR • ECG • Respiratory rate • SpO_2 • Temperature • $EtCO_2$
Blocks	Epidural: • Morphine (0.075 mg/kg preservative free) *or* • Buprenorphine (0.002–0.004 mg/kg diluted in saline) Incisional: • Lidocaine (<5 mg/kg in dogs, 2–4 mg/kg in cats), *or* • Bupivacaine (<2 mg/kg)

Continued

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TABLE 26.4 Anesthetic Considerations in the Patient Undergoing a Cesarean Section—cont'd

Postoperative Considerations

Analgesia	• Fentanyl CRI (1–10 µg/kg IV loading dose, then 2–20 µg/kg/h IV), *or*
	• Hydromorphone[a] CRI (0.025–0.1 mg/kg/h IV in dogs), *or*
	• Morphine[b] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.05–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h in cats), *or*
	• Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats), *or*
	• Oxymorphone (0.1–0.2 mg/kg IV, IM), *or*
	• Buprenorphine[c] (5–20 µg/kg IV, IM, SC q4–8h) *plus*
	• In dogs:
	  • Carprofen (2.2 mg/kg PO), *or*
	  • Deracoxib (3–4 mg/kg PO), *or*
	  • Meloxicam (0.1–0.2 mg/kg SC, PO, once)
	• In cats:
	  • Meloxicam[d] 0.15 mg/kg SC once; approved for doses up to 0.3 mg/kg SC (see also Table 34.3), *or*
	  • Buprenorphine (0.01–0.02 mg/kg OTM)

Monitoring	• SpO_2 • Blood pressure • HR • Respiratory rate • Temperature
Bloodwork	HCT and TP if large blood loss
Estimated pain score	Moderately severe

[a]Monitor for hyperthermia in cats.
[b]Give slowly to prevent histamine release.
[c]Buprenorphine is a better analgesic than morphine in cats.
[d]Black box warning added by the Food and Drug Administration in October 2010 identified cases of renal failure and death in cats with repeated uses of meloxicam. Meloxicam is approved for single use only in cats in the United States.
BUN, Blood urea nitrogen; *Cr,* creatinine; *CRI,* constant rate infusion; *EBL,* estimated blood loss; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *OTM,* oral transmucosal; *PO,* orally; *PRN,* as needed; *SC,* subcutaneous; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein.

BOX 26.3 Antibiotics for Treatment of Reproductive Disorders

Cefazolin (Ancef, Kefzol)
22 mg/kg IV, IM q8h

Cefoxitin (Mefoxin)
Dogs: 30 mg/kg IV q6–8h
Cats: 22–33 mg/kg IV, IM q8h

Amoxicillin Plus Clavulanate (Clavamox)
Dogs: 12.5–25 mg/kg PO q12h
Cats: 62.5 mg/cat PO q12h

Ampicillin Plus Sulbactam (Unasyn)
50 mg/kg IV q8h

Ampicillin
22 mg/kg IV, IM, or SC q6–8h

Erythromycin
10–20 mg/kg PO q8–12h

Clindamycin (Antirobe)
Dogs: 11–33 mg/kg PO q12h, *or* 11 mg/kg IV (dilute and give slowly when administering IV)
Cats: 11–33 mg/kg PO, or SC q12–24h

Doxycycline (Vibramycin)
5 mg/kg PO q12h

Enrofloxacin (Baytril)[a]
Dogs: 7–20 mg/kg PO, IM, SC, or IV, q24h (dilute and give slowly over 30 minutes if given IV)
Cats: 5 mg/kg PO q24h

[a]Doses greater than 5 mg/kg per day may cause blindness in cats.
IM, Intramuscular; *IV,* intravenous; *PO,* orally; *SC,* subcutaneous.

(mesovarium) includes the suspensory ligament with its artery and vein, ovarian artery and vein, and variable amounts of fat and connective tissue. Canine ovarian pedicles contain more fat than feline ovarian pedicles, making it more difficult to visualize the vasculature. The ovarian vessels take a tortuous path within the pedicle. Ovarian arteries originate from the aorta. The left ovarian vein drains into the left renal vein; the right vein drains into the caudal vena cava. The suspensory ligament is a tough, whitish band of tissue that diverges as it travels from the ovary to attach to the last two ribs. The broad ligament (mesometrium) is the peritoneal fold that suspends the uterus. The round ligament travels in the free edge of the broad ligament from the ovary through the inguinal canal with the vaginal process. The uterus has a short body and long narrow horns. The uterine arteries and veins supply blood to the uterus. The cervix is the constricted caudal part of the uterus and is thicker than the uterine body and vagina. It is oriented in a nearly vertical position with the uterine opening dorsally. The vagina is long and connects with the vaginal vestibule at the urethral entrance. The clitoris is broad, flat, vascular, infiltrated with fat, and lies on the floor of the vestibule near the vulva. The clitoral fossa is a depression on the floor of the vestibule that is sometimes mistaken for the urethral orifice. The vulva is the external opening of the genital tract. The vulvar lips are thick and form a pointed commissure. The constrictor vulvae and constrictor vestibule muscles encircle the vulva and vestibule. See p. 748 for a description of the anatomy of the mammary glands.

NOTE If the broad ligament contains an excessive amount of fat, the vessels within it may need to be ligated.

Male Reproductive Tract

The major components of the male genital tract are the testicles, penis, and prostate. The prostate gland completely surrounds

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the neck of the bladder and beginning of the urethra. In dogs younger than 4 years, the prostate is usually located in the pelvic cavity at the brim of the pubis. The prostate begins to enlarge at puberty, becoming intraabdominal in location. It varies greatly in size at maturity. The prostate is encapsulated by fibromuscular tissue and is bilobate with a prominent middorsal sulcus. The dorsal sulcus continues into the prostatic parenchyma as the median septum. The ventrolateral surfaces of the prostate are covered by a fat pad. The parenchyma is lobulated with tubuloalveolar glands that empty through small ducts (12–20 ducts) into the prostatic urethra. The ductus deferens enters the craniodorsal surface of the prostate and courses caudoventrally to enter the urethra at the colliculus seminalis. The blood and nerve supply (pelvic and hypogastric nerves) are located in the lateral pedicles (peritoneal reflection), entering the prostate at the 10 o'clock and 2 o'clock positions when viewed in a transverse plane. The prostatic arteries originate from the urogenital artery (branch of internal iliac artery) and supply branches to the ductus deferens, urethra, urinary bladder, ureters, and rectum. The hypogastric (sympathetic) and pelvic (parasympathetic) nerves follow the vasculature and are essential for micturition and continence (Fig. 26.1). The pudendal nerve sends branches along the ventral surface of the urethra extending to the bladder neck. The pudendal nerve innervates the skeletal muscle of the external urethral sphincter. The iliac lymph nodes drain the prostate. In cats, bulbourethral glands are found caudal to the prostate at the ischial arch.

NOTE Scottish terriers generally have larger prostates than other similarly sized breeds.

The penis has a root, body, and glans. The root of the penis is formed by the right and left crura, which originate from the ischiatic tuberosity. Each crus is composed of corpus cavernosum penis surrounded by tunica albuginea. The two corpora extend side by side, separated by a median septum, along the length of the penile body to the os penis in the glans penis. The distal end of the penis or glans penis is covered by the prepuce, a mucosa-lined fold of integument. The distal end of the dog's penis is directed cranially and located ventral to the abdominal wall. The distal end of the cat's penis is directed caudal and ventral in the perineum. The glans of the feline penis is covered with caudally directed cornified spines, which are more prominent in intact males and regress within 6 weeks of castration. The feline os penis is very small, whereas in dogs it is a long, grooved, rough bone. The urethra travels through the ventral groove in the os penis and penis. The corpus spongiosum surrounds the urethra. The ischiocavernosus muscle arises from the ischiatic tuberosity and inserts on the crus. The retractor penis muscles originate from the ventral surface of the sacrum or the first two caudal vertebrae and extend distally on the ventral surface of the penis to insert at the level of the glans. The retractor and external anal sphincter muscles share muscle fibers. The bulbospongiosus muscle bulges between the ischiocavernosus muscles ventral to the external anal sphincter.

NOTE Surgery of the penis is often associated with significant hemorrhage because of the vascular nature of the cavernosus tissue.

The scrotum is located between the inguinal region and anus. In dogs, scrotal skin is thin and sparsely haired. The feline scrotum is more dorsal and densely haired than the canine scrotum. The scrotum is a membranous pouch with a midline septum that houses the testes, epididymis, and distal spermatic cords. The testis, epididymis, ductus deferens, and associated vessels and nerves are covered by visceral and parietal vaginal tunic and spermatic fascia. The testes are relatively small and ovoid. The epididymis is large, convoluted, and attached to the lateral side of the testis. The head of the epididymis communicates with the testis, and the caudal extremity or tail is continuous with the ductus deferens. The tail is attached to the testis by the proper ligament of the testis. The ligament of the tail of the epididymis attaches the epididymis to the vaginal tunic and the spermatic fascia. The ductus deferens loops around the ureter as it travels from the inguinal ring, enters the dorsal prostate, and terminates in the prostatic urethra. The ureter is dorsal to the ductus deferens. The spermatic cord begins at the inguinal ring where the testicular artery, testicular veins (pampiniform plexus), lymphatics, testicular autonomic nerve plexus, ductus deferens and its artery and vein, smooth muscle, and visceral layer of the vaginal tunic come together. The cremaster muscle travels along the external surface of the parietal tunic. The cremaster is a thin, flat extension of the internal abdominal oblique muscle.

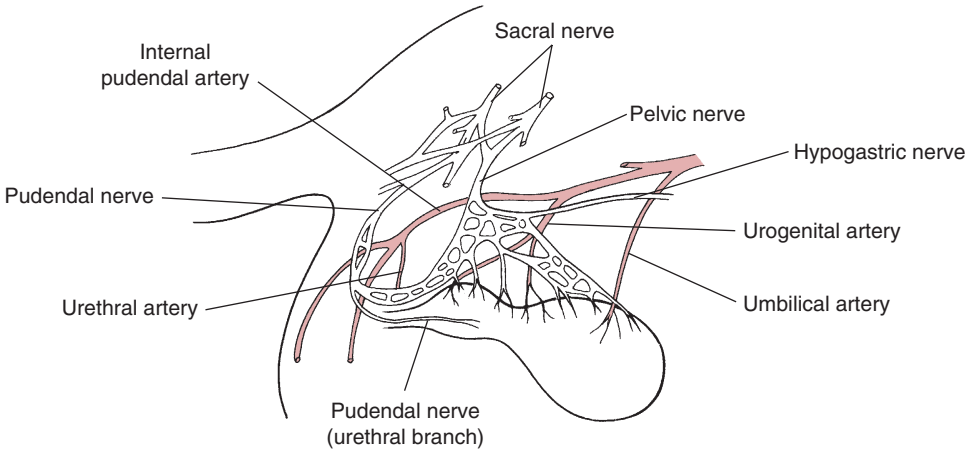


FIG. 26.1 Innervation to the prostate and bladder.

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NOTE The epididymis may be able to be palpated in dogs with epididymitis.

SURGICAL TECHNIQUE

Before elective surgeries, food should be withheld from adults for 12 to 18 hours and from pediatric patients for 4 to 6 hours. The ventral abdomen should be clipped and aseptically prepared for any procedure requiring celiotomy. The urinary bladder should be expressed if the patient has not voided immediately before induction. In dogs, the prescrotal area should be clipped and prepared for aseptic surgery; however, trauma to the scrotum (e.g., from clippers, antiseptic soaps, or solutions) must be avoided. Canine scrotal skin is sensitive and swells with minimal trauma or irritation. In cats, hair can be plucked or pulled from the scrotum. The prepuce or vestibule should be flushed with dilute antiseptic solutions before procedures involving these areas. For some procedures that involve the perineum, prostate, or penis, placement of a urethral catheter helps identify the urethra.

NOTE Numerous techniques are used for neutering dogs and cats; however, the goals are the same—to remove the ovaries, with or without the uterine horns and body, or testes, with secure ligature placement.

Pediatric tissues are more fragile than adult tissues and must be handled gently. In young animals, 3-0 to 5-0 ligatures should be used. Early neutering delays growth plate closure by an average of 8 to 9 weeks, resulting in increased bone length in male and female dogs and cats. Infantile vulva and mammary glands or penis, prepuce, and os penis persist following early neutering. Bitches are at a greater risk for development of urinary incontinence if OHE is performed before 3 months of age (Box 26.4). Large-breed dogs neutered before 6 months of age are at increased risk for development of excessive tibial plateau angle. However, early neutering affects weight gain, daily food consumption, and activity level to the same degree as neutering after puberty.

The traditional method of surgical sterilization of healthy female dogs, particularly in the United States, has been OHE; however, OVE, commonly practiced in European countries, is gaining more acceptance in the United States. Evaluation of the scientific literature found no significant difference in postoperative complications between OHE and OVE, including the development of urethral sphincter mechanism incompetence, endometritis, or pyometra. In addition, uterine tumors are uncommon, with most being benign (85%–90%) in nature. A 2011 prospective study found no difference in total surgical time, pain scores, or wound scores between dogs undergoing OHE versus OVE.[2] However, for novice surgeons, OHE is technically more complicated and time consuming, whereas OVE can be performed quicker and through a smaller abdominal incision (or by laparoscopy) with less traction on the genital tract. Therefore both OHE and OVE are considered appropriate procedures for neutering healthy female dogs.

BOX 26.4 Early-Age Gonadectomy Expectations

- Early gonadectomy is safe in dogs and cats >7 weeks of age
- Female dogs are at greater risk of urinary incontinence if ovariohysterectomy is performed before 3 months of age
- Growth plate closure is delayed by 8 to 9 weeks, resulting in increased long bone length
- Increased risk for excessive tibial plateau angle in large-breed dogs
- Penis, prepuce, and vulva may appear small and infantile if neutered at 6 to 8 weeks
- Not associated with increased obesity, amount of daily food consumption, activity level, lower urinary tract disease, long bone fractures, arthritis, immune suppression, or small urethra
- Associated with lower morbidity and quicker anesthetic recovery

Ovariohysterectomy/Ovariectomy

The most common reason to perform OHE or OVE is to prevent estrus and unwanted offspring. Alternative methods of inhibiting reproduction are listed in Box 26.5. Other reasons for OHE include prevention of mammary tumors or congenital anomalies; prevention and treatment of pyometra, metritis, neoplasia (e.g., ovarian, uterine, or vaginal), cysts (Figs. 26.2 and 26.3), trauma, uterine torsion, uterine prolapse, subinvolution of placental sites, vaginal prolapse, and vaginal hyperplasia; and control of some endocrine abnormalities (e.g., diabetes and epilepsy) and dermatoses (e.g., generalized *Demodex*). Many technical variations of OHE have been described, including flank and laparoscopic

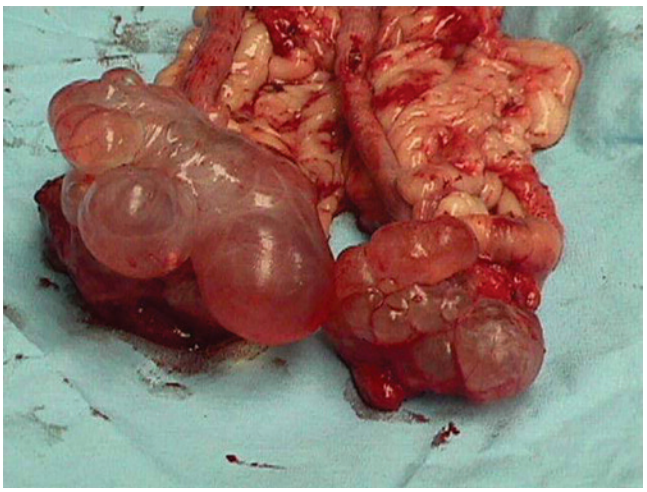


FIG. 26.2 Ovarian cysts identified during an elective ovariohysterectomy.

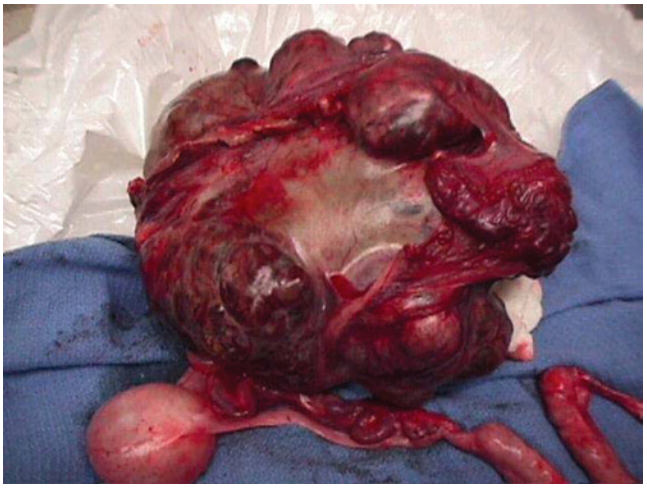


FIG. 26.3 Appearance of a large ovarian cystadenocarcinoma.

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BOX 26.5 Options for Pregnancy Prevention or Termination

Surgical Prevention or Termination

- Ovariohysterectomy
- Ovariectomy (prevention only)
- Castration (prevention only)
- Vasectomy (prevention only)

Medical Prevention

Megestrol Acetate

Dogs: 0.55 mg/kg PO q24h for 32 d for dogs in anestrous; 2.2 mg/kg PO q24h for 8 d for dogs in proestrus

Medical Termination

Prostaglandin $F_{2\alpha}$ (Lutalyse)

Dogs: 0.1 mg/kg SC, q8h for 2 d, then 0.2 mg/kg SC q8h until abortion is complete; may take >9 d[a]

Cats: 2 mg/cat IM q24h for 5 d, starting at least 30 d after breeding

Cloprostenol (Estrumate)

Dogs: 1–2 µg/kg SC, q24h for 5–7 d, starting at least 30 d after breeding or 1 µg/kg SC q48h for 3 doses combined with cabergoline (5 µg/kg PO) q24h for 9 d[b,c]

Cabergoline (Dostinex)

Dogs: 1.65 µg/kg SC, q48h for 5 d (efficacy depends on timing with LH surge)

Bromocriptine (Parlodel)

Dogs: 0.1 mg/kg/d PO for 6 d beginning on day 35 of gestation or 0.03 mg/kg PO q12h for 4 d after day 30 of gestation[d]

Mifepristone (Mifeprex)

Dogs: 2.5 mg/kg PO daily for 4–5 d beginning on day 32 of gestation

Aglepristone (Alizine, Alizin)

Dogs: 10 mg/kg SC, given twice q24h or 0.15 mg/kg SC given twice q24h

Cats: 15 mg/kg SC, q24h for 2 d, starting at day 33

Estrogen Compounds[e]

[a]Recommended techniques. Ideally begin drugs on day 25 after the LH peak or 20 to 28 d after first mating. Monitor abortion by ultrasound or by measuring plasma progesterone level. Progesterone levels <1–2 ng/mL for 2 consecutive days indicate pregnancy termination.
[b]Side effects may include excessive salivation, vomiting, defecation, urination, and papillary dilation followed by constriction. Consider hospitalization during administration.
[c]Estrumate (cloprostenol 250 µg/mL) must be diluted in saline at a ratio of 1:9 (1 mL cloprostenol to 9 mL saline). Do not reuse solution.
[d]This drug may have severe side effects and is not recommended.
[e]Not recommended due to dangerous side effects of medullary aplasia, metritis, pyometra, ovarian cysts, estrus-like behavior, and irreversible infertility.
IM, Intramuscular; *LH,* luteinizing hormone; *PO,* orally; *SC,* subcutaneous.
Modified from Wiebe VJ, Howard JP. Pharmacologic advances in canine and feline reproduction. *Top Companion Anim Med.* 2009;24:71–99.

approaches and the use of stapling equipment, ultrasonic scalpel, vessel-sealing devices, transfixation ligatures, or Miller's knots (Fig. 26.4). Only one technique for OHE is described here.

NOTE In dogs, make the incision immediately caudal to the umbilicus to facilitate ligation of the ovarian pedicles. Make the incision more caudal in cats to facilitate ligation of the uterine body.

Clip and surgically prepare the ventral abdomen from the xiphoid to the pubis. Identify the umbilicus and visually divide the caudal abdomen into thirds. In dogs, make the incision just caudal to the umbilicus in the cranial third of the caudal abdomen. More caudal incisions make it difficult to exteriorize canine ovaries. *In deep-chested dogs or in those with an enlarged uterus, extend the incision cranially or caudally to allow exteriorization of the tract without*

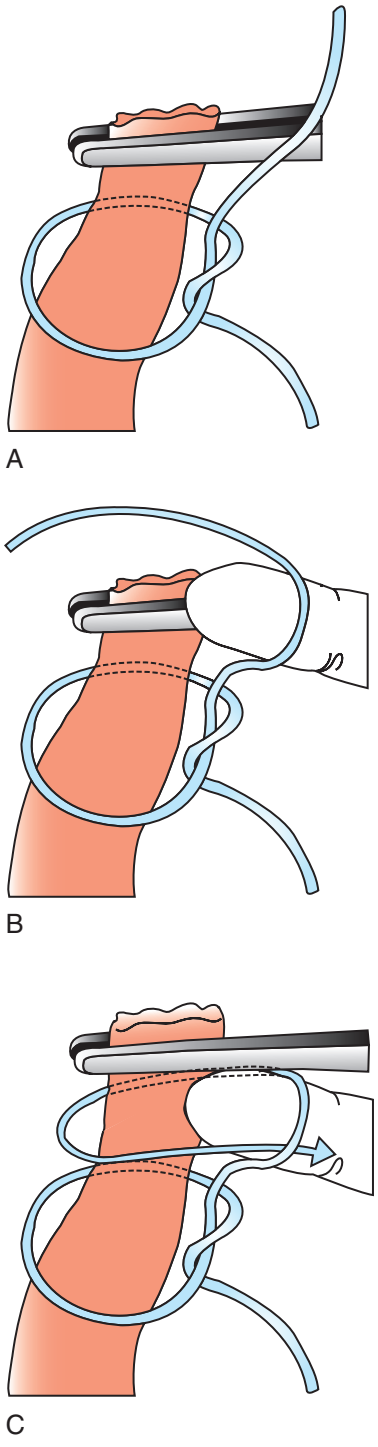


FIG. 26.4 A Miller's knot or clove hitch is preferred by some surgeons to occlude the pedicles. (A) Place a ligature on the pedicle and secure with a half hitch. Have one end of the suture relatively short and the other long. (B) Position your finger between the long end of the suture and the ligature (palm up). Grab the long end of the suture with a needle holder and bring it around the pedicle a second time. (C) Insert the end through the space reserved by your finger. Add two square knots to finish the ligature.

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excessive traction. In prepubertal puppies, making the incision in the middle third of the caudal abdomen facilitates uterine body ligation. *In cats, the body of the uterus is more caudal and difficult to exteriorize; therefore make the incision in the middle third of the caudal abdomen. Make a 4- to 8-cm incision through skin and subcutaneous tissue to expose the linea alba. Grasp the linea alba or ventral rectus sheath, tent it outward, and make a stab incision into the abdominal cavity. Extend the linea incision cranial and caudal to the stab incision with Mayo scissors. Elevate the left abdominal wall by grasping the linea or external rectus sheath with thumb forceps. Slide the OVE hook (e.g., Covault or Snook) with the hook against the abdominal wall, 2 to 3 cm caudal to the kidney (Fig. 26.5A). Turn the hook medially to ensnare the uterine horn, broad ligament, or round ligament, and gently elevate it from the abdomen. Anatomically confirm the identification of the uterine horn by following it to either the uterine bifurcation or ovary. If the uterine horn cannot be located with the hook, retroflex the bladder through the incision and locate the uterine body and horns between the colon and bladder. With caudal and medial traction on the uterine horn, identify the suspensory ligament by palpation at the taut fibrous band at the proximal edge of the ovarian pedicle (Fig. 26.5B). Stretch or break the suspensory ligament near the kidney without tearing the ovarian vessels to allow exteriorization of the ovary. To achieve this, use the index finger to apply caudolateral traction on the suspensory ligament while maintaining caudomedial traction on the uterine horn (Fig. 26.5C).*

Make a hole in the broad ligament caudal to the ovarian pedicle. Place one, two, or three Mosquito, Crile, or Rochester-Carmalt forceps across the ovarian pedicle proximal (deep) to the ovary (Fig. 26.5D). The proximal (deep) clamp serves as a groove for the ligature, the middle clamp holds the pedicle for ligation, and the distal clamp prevents backflow of blood after transection. When using two clamps, the ovarian pedicle clamp serves both to hold the pedicle and to make a groove for the ligature. When using a single clamp, it is placed just proximal to the ovary to prevent backflow bleeding.

Place an encircling or figure-eight ligature proximal to (below) the ovarian pedicle clamps (Fig. 26.5E). Choose an absorbable suture material for ligatures (i.e., 2-0 to 4-0 polydioxanone [PDS], polyglyconate [Maxon], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], or polyglactin 910 [Vicryl]). For figure-eight ligatures, begin by directing the blunt end of the needle through the middle of the pedicle, loop the suture around one side of the pedicle, then redirect the needle through the original hole from the same direction and loop the ligature around the other half of the pedicle. Securely tie the ligature. Remove one clamp or "flash" a single clamp while tightening the ligature to allow pedicle compression. Place a second circumferential ligature proximal to (below) the first to control hemorrhage that may occur from puncturing a vessel as the needle is passed through the pedicle. Some surgeons prefer to place the circumferential ligature or Miller's knot (see Fig. 26.4) before the transfixing ligature to eliminate hemorrhage if a vessel is punctured during transfixation.

Place a Mosquito hemostat on the suspensory ligament near the ovary (Fig. 26.5F). Transect the ovarian pedicle between the two remaining clamps, or proximal to the most distal clamp. Remove the clamp from the ovarian pedicle and observe for hemorrhage. Replace the clamp and religate the pedicle if hemorrhage is noted. Perform the identical procedure on the other side.

For OVE, place one or two encircling ligatures of absorbable suture material just caudal to the proper ligament at the tip of the uterine horn. Transect the mesovarium and proper ligament and remove the ovary. Open the ovarian bursa and examine the ovary to be certain that it has been removed in its entirety.

For OHE, trace the uterine horn to the uterine body. Grasp the other uterine horn, and follow it to the opposite ovary. Place clamps and ligatures as just described. Make a window in the broad ligament adjacent to the uterine body and uterine artery and vein. Place a Carmalt across the broad ligament on each side and transect (Fig. 26.5G). Apply a ligature around the broad ligament if the patient is in estrus or pregnant, or if the broad ligament is heavily infiltrated with vessels or fat. Apply cranial traction on the uterus, and ligate the uterine body cranial to the cervix.

Place a figure-eight suture (0 to 3-0 absorbable suture) through the body using the point of the needle and encircling the uterine vessels on each side. Place a circumferential ligature, modified Miller's knot, or strangle knot nearer the cervix (Fig. 26.5H). Place a Carmalt across the uterine body cranial to the ligatures. Grasp the uterine wall with forceps or Mosquito hemostats cranial to the ligatures. Transect the uterine body, and observe for hemorrhage. Religate if hemorrhage is observed. Some surgeons place one to three Carmalt forceps across the uterine body before ligation. In cats, clamps may cut rather than crush a friable or engorged uterus and cause transection before ligature placement. An alternative to ligatures is to use an ultrasonic scalpel, vascular sealer, or staples. *Replace the uterine stump into the abdomen before releasing the hemostats or forceps. Close the abdominal wall in three layers (fascia/linea alba, subcutaneous tissue, and skin).*

Laparoscopic-Assisted Ovariohysterectomy or Ovariectomy

Multiple variations of minimally invasive techniques for surgical sterilization have been described, including intracorporeal versus laparoscopic-assisted OHE, OHE versus OVE, and differences in the number of portals utilized (one, two, or three). A three-port technique for laparoscopic-assisted OHE in addition to a two-port and single-port techniques for laparoscopic OVE are described here.

With the patient in dorsal recumbency and following creation of pneumoperitoneum, place the laparoscope port just caudal to the umbilicus. Place two operating ports, one 3 to 5 cm cranial to the umbilicus and one 3 to 5 cm cranial to the pubis. Slightly tilt the animal to one side and use a blunt probe or Babcock or Kelly laparoscopic forceps in the caudal operating port to retract the intestines medially and locate the ovary. Grasp the proper ligament of the ovary with forceps and elevate the ovary away from the abdominal viscera and body wall. Transect the suspensory ligament (Video 26.1), the broad ligament of the uterus, and the ovarian vascular pedicle (Videos 26.2 and 26.3) using a bipolar vessel-sealing device (e.g., LigaSure) placed through the cranial instrument port. Observe for any bleeding from the ovarian pedicle. Remove the forceps from the proper ligament and tilt the animal the opposite direction. Perform the identical procedure on the remaining ovary, except leave the forceps attached to the proper ligament. Extend the incision of the caudal port, and remove the forceps and cannula as a unit to facilitate exteriorization of the ovaries and uterus. Complete the OHE by ligation of the uterine vessels and transection of the uterine body as described previously. Remove the remaining instrumentation, ensure complete evacuation of the pneumoperitoneum, and close the incisions routinely.

Alternatively, place the laparoscope port just caudal to the umbilicus and a single instrument port midway between the umbilicus and pubis. Tilt the animal to one side and use a blunt probe or Babcock or Kelly laparoscopic forceps in the instrument

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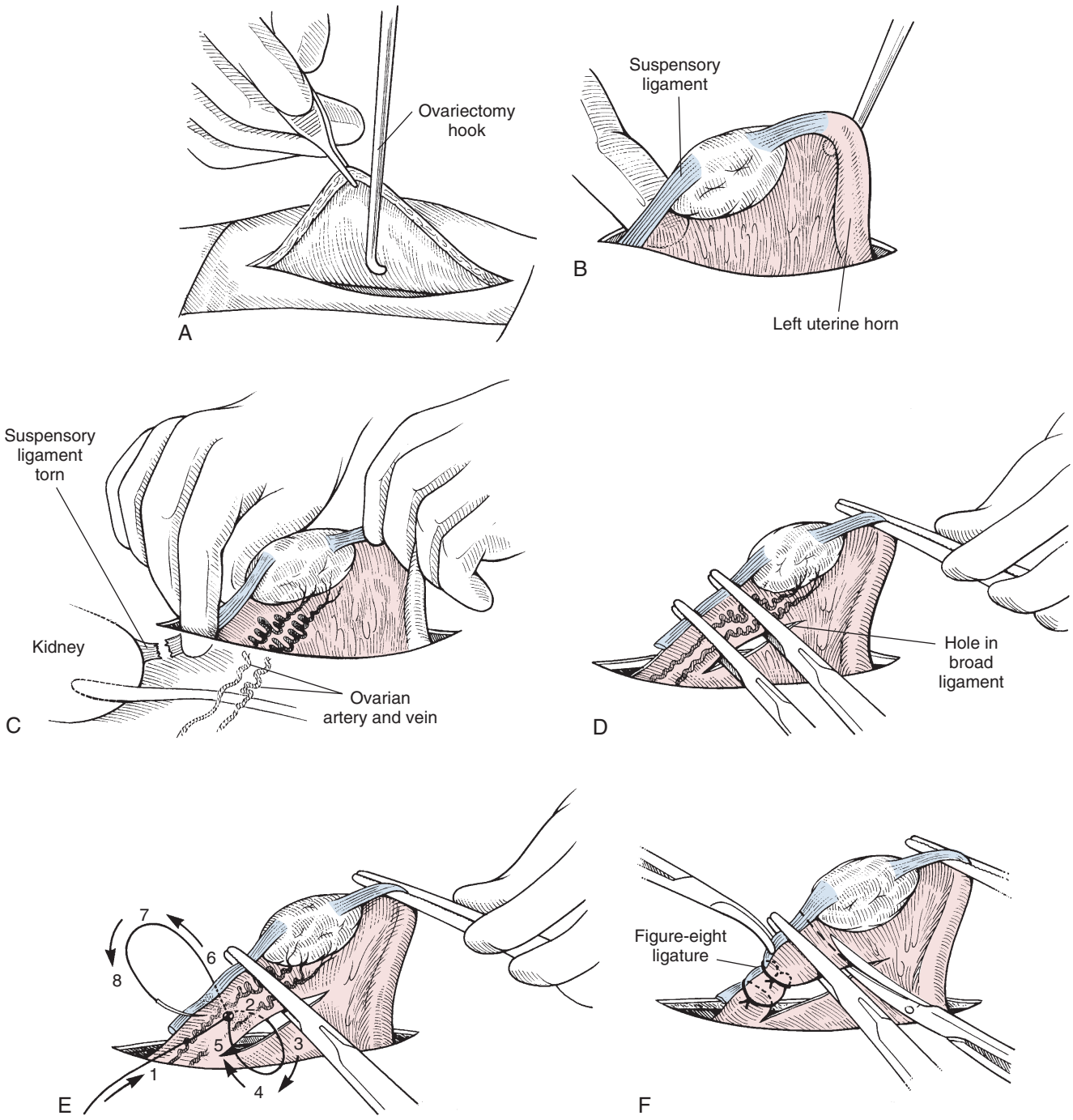


FIG. 26.5 (A) For ovariohysterectomy, elevate the abdominal wall with thumb forceps and slide the ovariectomy hook against the abdominal wall, 2–3 cm caudal to the kidney. (B) Exteriorize the uterine horn with the hook and identify the suspensory ligament at the cranial edge of the ovarian pedicle. (C) Stretch or tear the suspensory ligament to allow exteriorization of the ovary using the index finger to apply caudolateral traction on the suspensory ligament while maintaining caudomedial traction on the uterine horn. (D) Place two Carmalt forceps across the ovarian pedicle proximal to the ovary and one across the proper ligament (or place three forceps proximal to the ovary). Remove the most proximal clamp and place a figure-eight ligature at this site. (E) Direct the blunt end of the needle through the middle of the pedicle (*1* to *2*), loop the suture around one side of the pedicle (*3* to *4*), then redirect the needle through the original hole from the same direction (*5* to *6*), and loop the ligature around the other half of the pedicle (*7* to *8*). Securely tie the ligature (*1* and *8*). (F) Place a circumferential ligature proximal to the first ligature, then place a hemostat on the suspensory ligament near the ovary. Transect the ovarian pedicle distal to the clamp across the ovarian pedicle.

Continued

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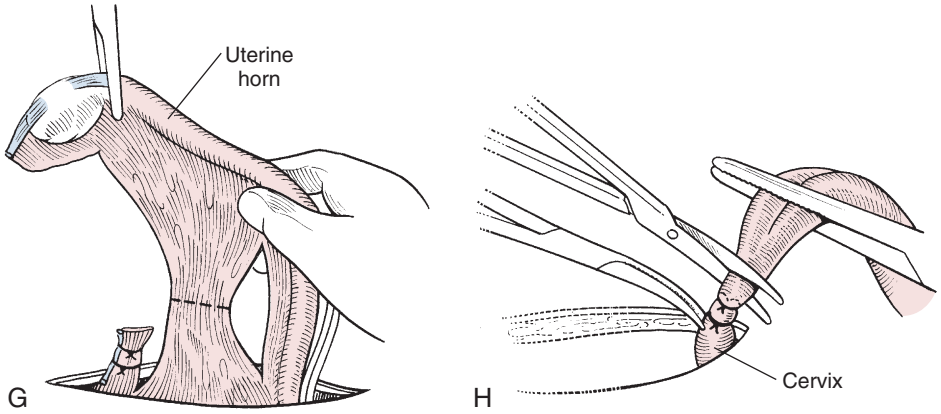


FIG. 26.5, cont'd (G) Separate the broad ligament from the uterine horn. Clamp and ligate the broad ligament *(dashed line)* if it appears vascular. (H) To ligate the uterus, place a figure-eight suture through the uterine body near the cervix. Place a second circumferential ligature closer to the cervix, place a Carmalt forceps distal to the ligatures, and transect between the Carmalt forceps and ligatures. Inspect the uterine stump for hemorrhage (use a Mosquito hemostat attached to the uterine wall to prevent retraction of the uterus into the abdomen).

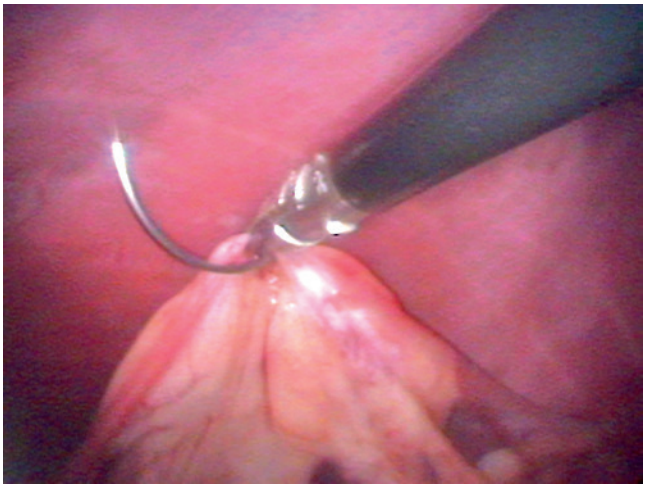


FIG. 26.6 Laparoscopic-assisted ovariectomy. The ovary is temporarily secured to the body wall by a transabdominal needle through the proper ligament.

port to retract the intestines medially and locate the ovary. Grasp the proper ligament of the ovary with forceps and elevate the ovary away from the abdominal viscera but against the body wall. Introduce a large curved needle through the body wall near where the ovary is being held (Fig. 26.6). Direct the needle through the proper ligament under direct laparoscopic visualization and then back through the body wall. Anchor the needle with a hemostat to keep the ovary suspended away from the abdominal viscera. Remove the laparoscopic forceps from the proper ligament and replace this instrument with a bipolar vessel-sealing device through the instrument port. Transect the suspensory ligament, ovarian vascular pedicle, and proper ligament. Tilt the animal in the opposite direction and repeat the procedure on the remaining ovary. Remove each ovary from the abdominal cavity through the instrument port immediately following transection, or retain the first ovary against the body wall using the transabdominal needle until the entire procedure is complete to facilitate easier removal of both ovaries. Remove remaining instrumentation, complete evacuation of pneumoperitoneum, and close the incisions routinely.

A single-port technique for OVE can be performed using a single-incision laparoscopic surgery port or a laparoscope with a working channel.

ORCHIECTOMY

Castration reduces overpopulation by inhibiting male fertility and decreases male aggressiveness, roaming, and undesirable urination behavior. It helps prevent androgen-related diseases, including prostatic diseases, perianal adenomas, and perineal hernias. Other indications for castration include congenital abnormalities, testicular or epididymal abnormalities, scrotal neoplasia, trauma or abscesses, inguinal-scrotal herniorrhaphy, scrotal urethrostomy, epilepsy control, and control of endocrine abnormalities.

Canine Castration

Either a prescrotal or perineal approach may be used for castration. A prescrotal approach is most common and more easily performed. The testicles are more difficult to exteriorize with a perineal approach, but this may be selected to avoid repositioning and aseptically preparing a second surgical site when the patient is in a perineal position for another surgical procedure (e.g., perineal hernia repair). Scrotal incisions are sometimes used when castrating prepubertal puppies, and this technique is becoming more common in high-volume spay-neuter clinics due to its efficiency.

Open Prescrotal Castration

Position the patient in dorsal recumbency. Verify the presence of both testicles in the scrotum. Clip and aseptically prepare the caudal abdomen and medial thighs. Avoid irritating the scrotum with clippers or antiseptics. Drape the surgical area to exclude the scrotum from the field. Apply pressure on the scrotum to advance one testicle as far as possible into the prescrotal area. Incise skin and subcutaneous tissue along the median raphe over the displaced testicle (Fig. 26.7A). Continue the incision through spermatic fascia to exteriorize the testicle. Incise the parietal vaginal tunic over the

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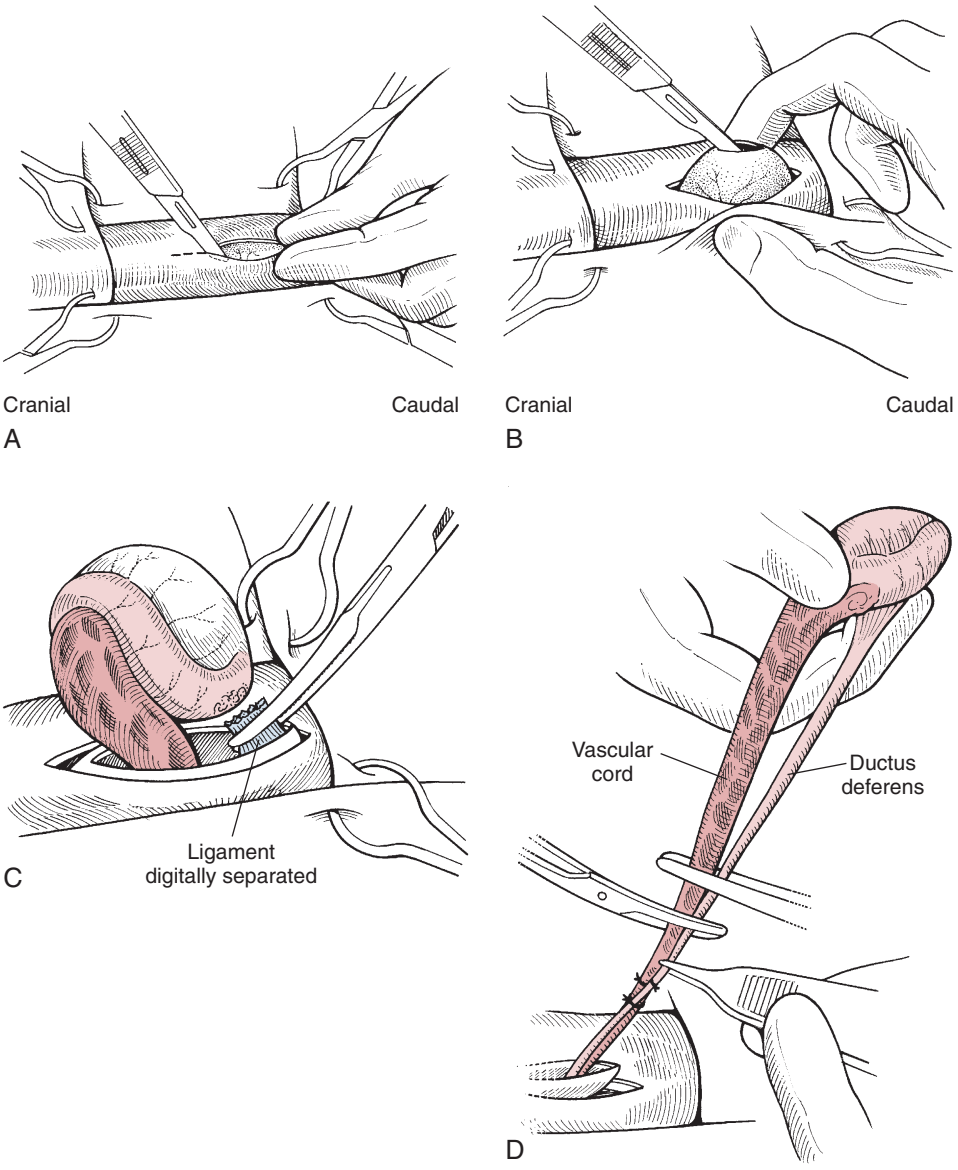


FIG. 26.7 (A) To perform an open canine castration, advance one testicle into the prescrotal area by applying pressure over the scrotum. Make an incision over the testicle. (B) Incise the spermatic fascia and parietal vaginal tunic. (C) Place a hemostat across the tunic where it attaches to the epididymis and digitally separate the ligament of the tail of the epididymis from the tunic. (D) Ligate the ductus deferens and vascular cord individually, and then encircle both with a proximal circumferential ligature. Apply a Carmalt forceps distal to the ligatures and transect between the clamp and ligatures.

testicle (Fig. 26.7B). Do not incise the tunica albuginea because this would expose the testicular parenchyma. Place a hemostat across the vaginal tunic where it attaches to the epididymis. Digitally separate the ligament of the tail of the epididymis from the tunic while applying traction with the hemostat on the tunic (Fig. 26.7C). Further exteriorize the testicle by applying caudal and outward traction.

Identify the structures of the spermatic cord. Individually ligate the vascular cord and ductus deferens, then place an encircling ligature around both. Many surgeons ligate the ductus deferens and pampiniform plexus together. *Use 2-0 or 3-0 absorbable suture (e.g., polyglactin 910 [Vicryl], polydioxanone [PDS], poliglecaprone 25 [Monocryl], polyglyconate [Maxon], or glycomer 631 [Biosyn]) for ligatures. Place a hemostat across the cord near the testicle. Grasp the ductus deferens with thumb forceps above the ligature and transect both the ductus deferens and vascular cord between the hemostat and ligatures (Fig. 26.7D). Inspect the cord for hemorrhage and replace the cord within the tunic. Encircle the cremaster muscle and tunic with a ligature. Advance the second testicle into the incision, incise the fascial covering, and remove the testicle as described. Appose the incised dense fascia on either side of the penis with interrupted or continuous sutures. Close subcutaneous tissue with a continuous pattern. Appose skin with an intradermal or an external simple interrupted suture pattern in the skin.*

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Closed Prescrotal Castration

Closed castration is performed similarly to the open technique just described, except that the parietal vaginal tunics are not incised.

Maximally exteriorize the spermatic cord by reflecting fat and fascia from the parietal tunic with a gauze sponge. Place traction on the testicle while the fibrous attachments between the spermatic cord tunic and scrotum are torn. Place a hemostat across the cord just distal to the testicle. Place an encircling ligature (e.g., 2-0 or 3-0 absorbable) around the entire spermatic cord and tunics. Pass the needle of a second ligature through the cremaster muscle, or between structures within the tunic, for a transfixation ligature proximal or distal to the first. Transect the cord between the most distal ligature and the hemostat, and inspect for hemorrhage. Advance the second testicle into the incision and remove as described previously. Close subcutaneous tissue with a continuous pattern. Appose skin with an intradermal or an external simple interrupted suture pattern in the skin.

Perineal Castration

Perineal castration is performed using the same techniques as for an open prescrotal castration. It is more difficult to displace the testicles into a caudal incision than into a prescrotal incision. An open technique must be used.

Make a midline skin and subcutaneous tissue incision dorsal to the scrotum in the perineum ventral to the anus. Advance one testicle to the incision and incise the spermatic fascia and tunic. Exteriorize the testicle and ligate the spermatic cord as described for an open or closed prescrotal castration.

Scrotal Ablation

Scrotal ablation is necessary for neoplastic scrotal diseases and for castration performed in conjunction with scrotal urethrostomy in dogs and perineal urethrostomy in cats. Other indications include severe scrotal trauma, abscesses, or ischemia. Scrotal ablation may improve the postcastration appearance of dogs if they have a pendulous scrotum. Surgical time is somewhat longer when scrotal ablation is performed.

Elevate the scrotum and testicles from the body wall. Make an elliptical skin incision at the base of the scrotum, being careful not to excise too much skin. Control hemorrhage with electrocoagulation, ligation, or pressure. Incise the vaginal tunics and remove the testicles as described for open castration, or alternatively perform a closed castration. Remove the scrotum after incising its median septum. Appose subcutaneous tissues with a simple continuous suture pattern (e.g., 3-0 absorbable suture). Appose skin edges with approximating interrupted sutures (e.g., 3-0 or 4-0 nonabsorbable suture) or using an intradermal pattern.

Feline Castration

Pluck hair from the scrotum rather than clipping (Fig. 26.8A). In kittens less than 16 to 20 weeks of age, plucking scrotal hair may be difficult. Use clippers to gently remove scrotal hair in these animals.

Position the cat in dorsal or lateral recumbency with the hind legs pulled cranially. Mobilize a testicle in the scrotum by applying pressure with the thumb and index finger at the base of the scrotum. Make a 0.5- to 1-cm incision over each testicle at the end of the scrotum from cranial to caudal (Fig. 26.8B). Incise the parietal vaginal tunic over the testicle and exteriorize the testicle. Digitally separate the attachment of the ligament of the tail of the epididymis to the vaginal tunic (Fig. 26.8C). Double ligate the spermatic cord with 3-0 absorbable suture or hemoclips, or remove the ductus deferens from the testicle and tie it with the vessels (discussed later). Alternatively, use a figure-eight knot (see p. 729). Transect the cord, inspect for bleeding, and replace it within the tunic. Excise the second testicle in a similar fashion. Resect any tags of tissue protruding from the scrotum. Allow the scrotal incision to heal by secondary intention.

Alternatively, to ligate the ductus deferens with the vessels, separate the ductus deferens from the testicle. Using the remainder of the spermatic cord (testicular vessels and testicle) as one strand and the ductus deferens as the other, tie two to three square knots (five to six throws) (Fig. 26.8D). Sever the vessels with attached testicle and ductus deferens distal to the knot. Inspect for hemorrhage.

For an overhand or figure-eight knot, the spermatic cord is tied on itself with the aid of a curved Mosquito hemostat. Preferably the parietal vaginal tunic is separated from the epididymis before tying the knot. *Place the hemostat on top of the cord (Fig. 26.9A). Wrap the distal (testicle) end of the cord over the hemostat once (Fig. 26.9B). Direct the wrapped hemostat ventral to the cord while holding the testicle in the opposite hand (Fig. 26.9C). Open the tips of the hemostat, and grasp the distal end of the cord (Fig. 26.9D). Transect the spermatic cord near the testicle and manipulate the severed end of the cord through the loops around the hemostat (Fig. 26.9E). Snug the knot, resect excess cord, inspect for bleeding, and replace the cord within the tunic before releasing (Fig. 26.9F).*

Cryptorchid Castration

Cryptorchidism is a congenital failure of the testicle(s) to descend into the scrotum. Testes are normally pulled into the scrotum soon after birth by fibrosis and contraction of the gubernaculum. Dogs and cats are considered to have cryptorchidism if there is no testicular descent by 2 months of age. One or both testicles may be in an abnormal position, although unilateral cryptorchidism is most common. Testicular agenesis (failure of testis development [one, monorchism; two, anorchism]) is rare. Cryptorchid testes are frequently small, soft, and proportionally misshapen. They may be in the inguinal area or abdominal cavity. Bilateral castration is recommended for cryptorchid animals because the condition is believed to be a sex-linked autosomal recessive trait in dogs. Retained canine testes are predisposed to neoplasia (seminomas and Sertoli cell tumors). If the testicle is in the inguinal region, it can often be palpated between the inguinal ring and scrotum once the animal is anesthetized; however, large inguinal fat pads may obscure testes in this area.

Advance unilateral, mobile inguinal testicles to the prescrotal incision and remove. Remove nonmobile testicles by making an incision over the inguinal ring. Dissect through subcutaneous fat, and mobilize and remove the testicle. Submit the testicles for histologic examination to verify removal of testicular tissue and to rule out neoplasia.

Nonpalpable testes must be located via exploratory laparotomy or laparoscopy. *Make a ventral midline incision from the umbilicus to the pubis or a paramedian incision adjacent to the prepuce when an exploratory laparotomy is performed. Find the testicle(s) by retroflexing the bladder, locating the ductus deferens dorsal to the neck of the bladder, and following the ductus deferens to the testicle. If the ductus deferens travels into the inguinal ring and the testicle cannot be manipulated into the abdomen, perform an inguinal incision. Avulse the ligament of the tail of the epididymis. Double ligate the testicular artery and vein, and ductus deferens separately. Transect and remove the testicle. Inspect for hemorrhage and close the abdomen in three layers.*

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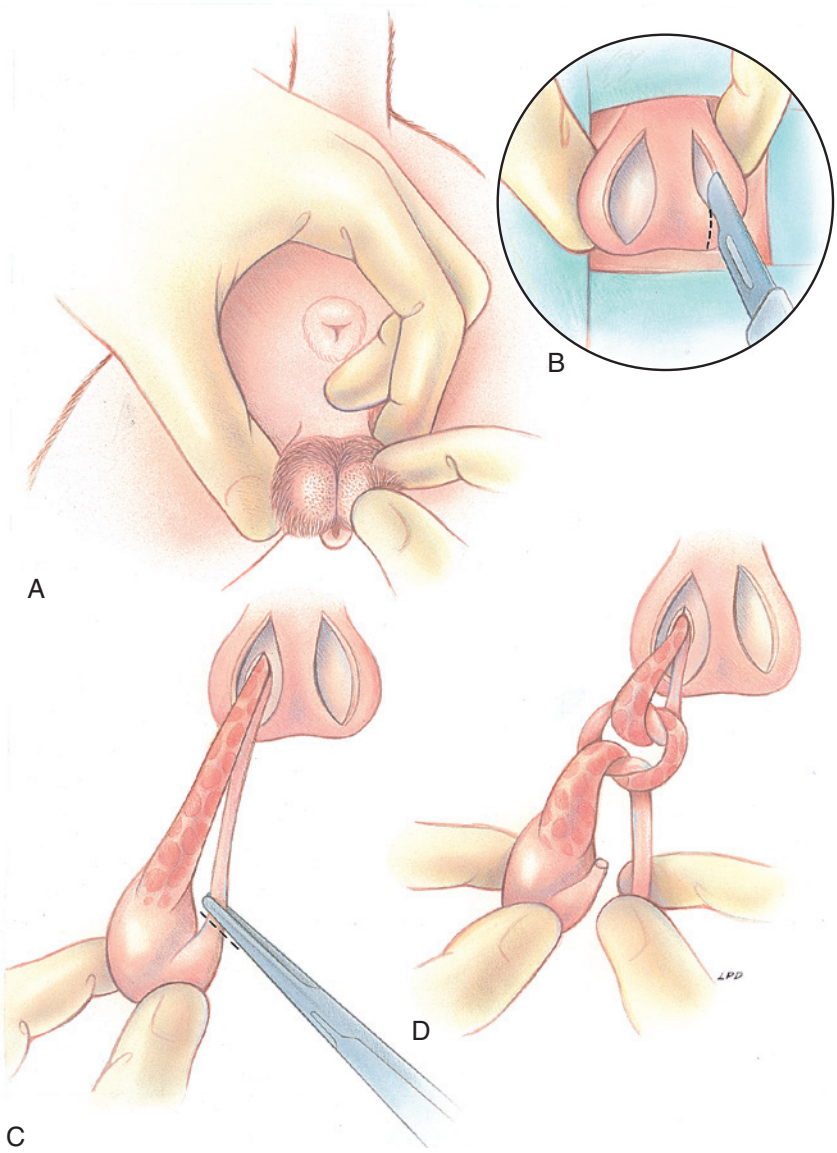


FIG. 26.8 (A) For feline castration, pluck hair from the scrotum and aseptically prepare the scrotum for surgery. (B), Make cranial to caudal skin incisions over each testicle. (C) Incise and separate the parietal tunic from the testicle, then transect the ductus deferens near the testicle. (D) Tie two to three square knots with the ductus deferens and the spermatic vessels.

NOTE Cryptorchid castration may also be performed laparoscopically or as a laparoscopic-assisted procedure (Fig. 26.10).

Vasectomy

Vasectomy inhibits male fertility while maintaining male behavioral patterns. Androgens continue to be produced because Leydig cells are not significantly altered. The technique is rarely recommended because roaming, aggression, and urine marking persist, whereas reduction of hormonally associated diseases does not occur. Most dogs become azoospermic by 1 week following vas occlusion; however, spermatozoa may persist in canine ejaculates for 3 weeks and feline ejaculates for 7 weeks after vasectomy. In dogs, flushing the ductus deferens at the time of vasectomy shortens the time to azoospermia. Vasectomized males should be evaluated after the procedure to document azoospermic ejaculates before contact with intact bitches. This technique should be discouraged as a means of population control.

Make a 1- to 2-cm incision over the spermatic cord between the scrotum and inguinal ring (Fig. 26.11A–B). Locate the spermatic cord, incise the vaginal tunic, and isolate the ductus deferens by blunt dissection (Fig. 26.11B). Double ligate the ductus deferens and resect a 0.5-cm section of ductus between ligatures (Fig. 26.11C). Repeat the procedure on the contralateral spermatic cord. Appose subcutaneous tissue and skin.

Cesarean Section

The goal of Cesarean section (C-section) or hysterotomy is to remove all fetuses from the gravid uterus as quickly as possible. The primary indications for C-section are actual or potential dystocia (i.e., oversized, malpositioned, or maldeveloped fetuses;

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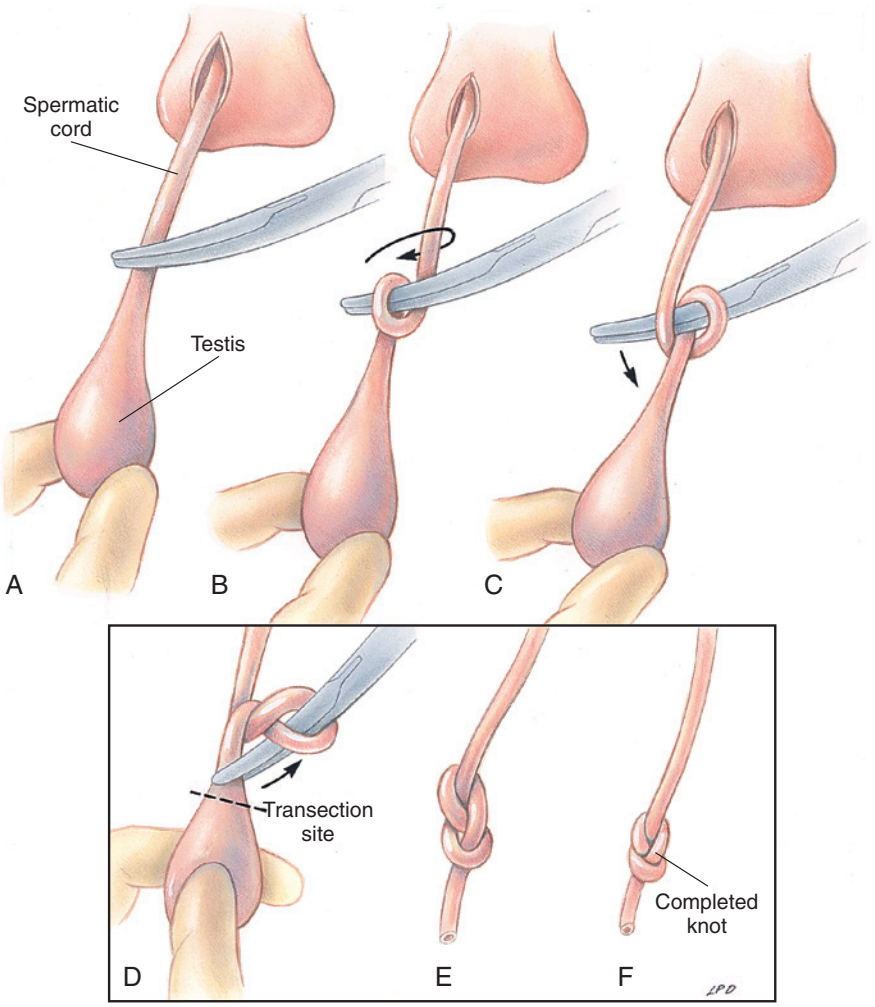


FIG. 26.9 An overhand technique may be used for feline castration. (A) Place a curved hemostat on top of the cord and wrap the spermatic cord over it. Direct the hemostat's tip (B) dorsally and then (C) ventrally around the cord opposite the testicle. (D) Grasp the cord near the testicle. (E) Transect the testicle and pull the end of the cord through the wrap. (F) Digitally snug the knot.

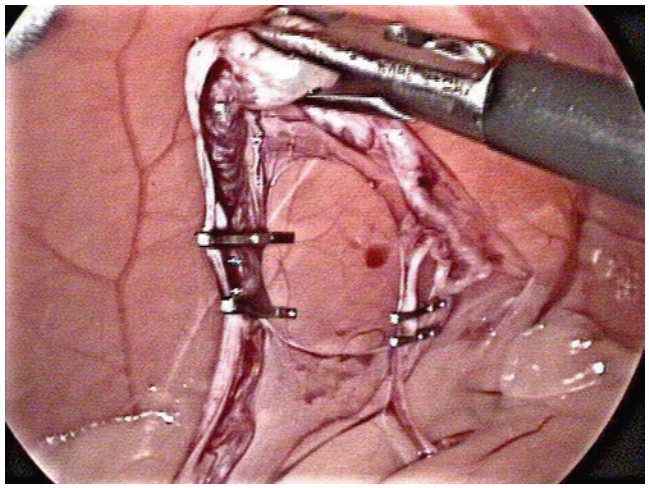


FIG. 26.10 This small, soft cryptorchid testicle is being removed laparoscopically. Note staples on the testicular vessels and ductus deferens.

small pelvic canal size; uterine inertia) or fetal putrefaction. Elective C-sections are often scheduled for brachycephalic breeds and other animals with a history of dystocia or those with pelvic fracture malunion. Breeds associated with high frequency of cesarean section include, but are not limited to, Boston terriers, English and French bulldogs, mastiffs, and Scottish terriers. C-sections for dystocia are more common in small dogs and brachycephalic breeds. Animals with dystocia often have fluid and electrolyte abnormalities that should be corrected before surgery. Although typically a postpartum problem, prepartum eclampsia causes hypocalcemia. Prophylactic antibiotics (e.g., cefazolin, 22 mg/kg intravenous [IV]) should be given if fetal death or uterine infection is suspected. Anesthetize these animals carefully (see p. 725 and Table 26.4); fetal depression and decreased viability are directly proportional to the degree of maternal depression.

OHE can be safely performed in conjunction with a C-section if the patient receives adequate fluid therapy. The C-section may be performed as described and followed by OHE, or an en bloc resection can be performed. En bloc OHE is performed before

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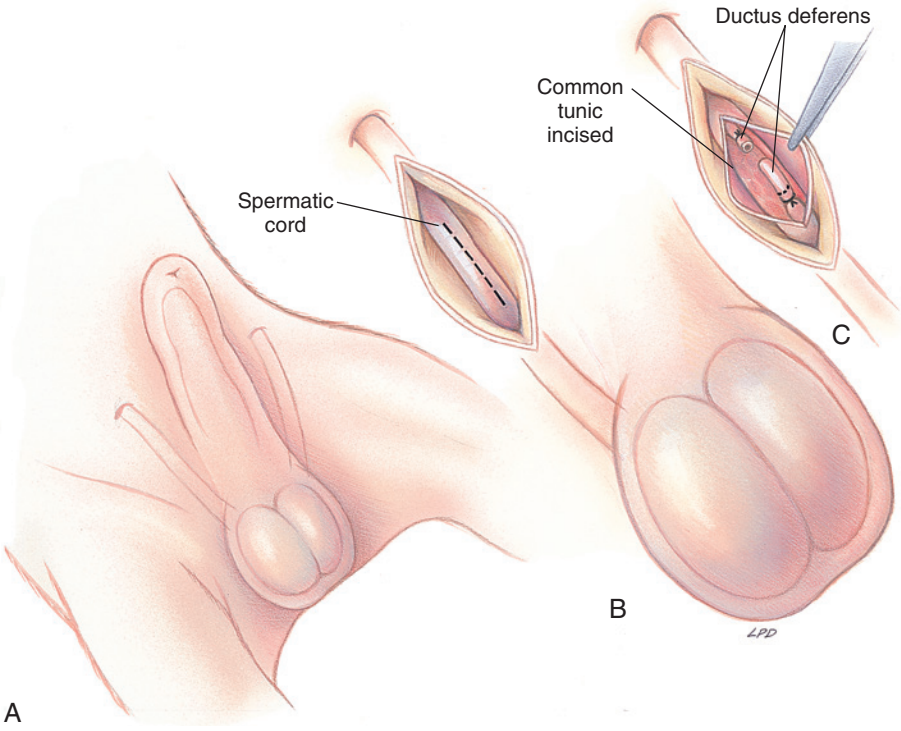


FIG. 26.11 Vasectomy. (A) The ductus deferens is located lateral to the prepuce and penis between the inguinal rings and scrotum. (B) Incise skin over the spermatic cord between the inguinal ring and scrotum. The *dashed line* indicates vaginal tunic incision site. (C) Incise the vaginal tunic and isolate the ductus deferens. Ligate the ductus deferens and remove a small segment.

hysterotomy (uterine incision) and removal of the neonates. Neonatal survival with en bloc resection is similar to that for other techniques for managing dystocia; however, this procedure is not recommended if the fetuses are distressed, bradycardic, or hypoxic. Changes in blood pressure and hematocrit are minimal following en bloc OHE, and mothering and lactation are normal. En bloc removal of the gravid uterus may be elective or necessary due to fetal death or questionable uterine integrity or health. Advantages of en bloc OHE of the gravid uterus include minimal anesthetic time, minimal potential for abdominal contamination, and population control without a second surgery. The disadvantages of this technique are that a second team is required to retrieve and then resuscitate the neonates, and that the uterus should be removed in less than 60 seconds to minimize the hypoxic event to the neonates.

Cesarean Section Without Ovariohysterectomy

Clip and perform a preliminary abdominal prep before anesthetic induction to minimize time from induction to delivery. Preoxygenate the bitch or queen if possible before induction. Anesthetize the animal using a general or regional protocol that is appropriate for the bitch or queen and minimizes neonatal depression (see the discussion on anesthesia on p. 725 and Table 26.4). Position the patient in dorsal recumbency. A reverse Trendelberg position can be used (head above the tail 15–30 degrees) to take pressure off the diaphragm and to minimize contamination to the abdominal cavity. Apply a final aseptic scrub to the ventral abdomen. Make a ventral midline incision from just cranial to the umbilicus to near the pubis. Elevate the external rectus sheath before making a stab incision through the linea alba to prevent inadvertent laceration



FIG. 26.12 Gravid horns should be carefully exteriorized from the abdomen to avoid tearing the uterine wall or vessels.

of the uterus. Exteriorize the gravid uterine horns by carefully lifting rather than pulling them out of the abdomen (Fig. 26.12) as uterine vessels are easily avulsed and the uterine wall readily tears. Isolate the uterus from the remainder of the abdomen with sterile towels or laparotomy pads. Tent and then incise the ventral

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uterine body to prevent lacerating the neonate. Extend the incision with Metzenbaum scissors in either a longitudinal direction along the ventral aspect of the uterine body or a U-shaped incision from one uterine horn to the other. The incision should be long enough to prevent tearing during extraction of the fetus.

Empty each horn by gently squeezing (milking) cranial to each fetus to move it toward the incision, then grasping and gently pulling it from the uterus (Fig. 26.13). Rupture the amniotic sac and clamp the umbilical cord with two curved Mosquito forceps and cut in between as each neonate is presented. Avoid contaminating the abdomen and surgical field with amniotic fluids. Aseptically pass each neonate to an assistant (see p. 738 for neonatal care). At term, the placenta is often expelled with the neonate; however, if the placenta has not separated, gently pull it from the endometrium. Do not forcibly separate the placenta from the uterine wall, or severe hemorrhage may occur. Palpate the pelvic canal and remove any fetus from this location. Uterine contraction usually begins when the fetuses are removed. *Administer oxytocin or ergonovine maleate (Box 26.6) if contraction has not occurred. Give oxytocin and compress the uterine walls if endometrial hemorrhage is severe. Lavage the external uterus to remove debris. Close the uterine incision with 3-0 or 4-0 absorbable suture using an appositional pattern in a single layer simple continuous pattern, a double layer appositional closure (mucosa and submucosa followed by muscularis and serosa), or an appositional closure followed by a second layer inverting pattern (Cushing or Lembert). Lavage the surgical site and replace contaminated towels, sponges, instruments, and gloves. Inspect for uterine vessel avulsion and control hemorrhage. Lavage the abdomen if contamination or spillage of uterine contents has occurred. Cover the uterine incision with omentum. Appose the abdominal wall in three layers (rectus fascia, subcutaneous tissue, and skin). Use an intradermal skin closure to eliminate suture ends that may irritate neonates. Clean all antiseptics, blood, and debris from the ventral abdomen and mammae.*



FIG. 26.13 Gently "milk" puppies toward the uterine incision by squeezing cranial to them.

BOX 26.6 Dose of Oxytocin to Initiate Uterine Contraction

Oxytocin

Dogs: 2 U/kg up to 20 U IM

Cats: 2–3 U IM or IV with a maximum of 3 U/cat (repeat up to 3 times every 30–60 min) 20 min after giving 1–2 mL of 10% calcium gluconate; if this fails then give 2 mL of 50% dextrose IV only and repeat oxytocin a third and last time

IM, Intramuscular; *IV,* intravenous.

For C-section with OHE, the fetuses are removed individually as described earlier and then a routine OHE is performed as described on p. 728.

En Bloc Resection

Perform en bloc OHE of the gravid uterus by first exteriorizing and isolating the ovarian pedicles and separating the broad ligament from the uterus to the point of the cervix. Manipulate fetuses in the vagina or cervix into the uterine body. Then double or triple clamp the ovarian pedicles and uterus just cranial to the cervix. Quickly transect between clamps and remove the ovaries and uterus. Give the uterus to a team of assistants to open and resuscitate the neonates. The time from clamping the uterus to removal of the neonates should be less than 60 seconds. *Double ligate ovarian and uterine pedicles. Inspect for hemorrhage and close the abdomen.*

Neonatal Care

See Fig. 26.14.

Gently suction the nares and nasopharynx. Provide oxygen by flow-by or small oxygen masks. Briskly rub and dry each neonate to stimulate the respiratory drive. If necessary, antagonize opioids by placing a drop of naloxone under the tongue. Stimulate respiration using a 25-gauge, ⅝-inch (1.5 cm) long hypodermic needle; place it 2 to 4 mm deep at the midline of the most dorsal aspect of the area between the upper lip and nose and rotate until bone is contacted. The use of doxapram for neonatal resuscitation is controversial as it is known to increase oxygen demand; increasing oxygen demand in a patient that is hypoxemic or bradycardic is contraindicated.

Ligate, transect, and disinfect the umbilical cord. Inspect each neonate for congenital or developmental anomalies (i.e., cleft palate, limb deformity, hernia, imperforate anus). Place neonates in a warm environment (32°C [90°F]) until their mother is able to care for them. Allow nursing as soon as the mother is extubated to ensure colostrum intake. Closely observe the mother and her behavior toward the neonates during the first few hours; some mothers will reject or kill their neonates. Discharge the bitch or queen and neonates from the hospital as soon as possible to reduce stress and exposure to potential pathogens.

Mastectomy

Mastectomy or removal of the mammary gland(s) is usually performed to remove tumors. One gland (simple mastectomy), several glands (regional mastectomy), or an entire chain (complete unilateral mastectomy) may be excised and the defect closed. Simultaneous removal of both mammary chains (complete bilateral mastectomy) causes significant suture line tension and should be avoided if possible. Staged procedures are advised to facilitate defect closure and reduce patient discomfort when bilateral mastectomy is necessary. OHE during the same anesthesia is performed before mastectomy to prevent seeding the abdomen with tumor cells. If the tumor crosses the midline, however, it may be excised first. Clean instruments and gloves should be used for the OHE. The technique for mastectomy is described on p. 749.

Episiotomy

An episiotomy is an incision of the vulvar orifice to allow access to the vestibule and vagina. It is indicated to surgically explore

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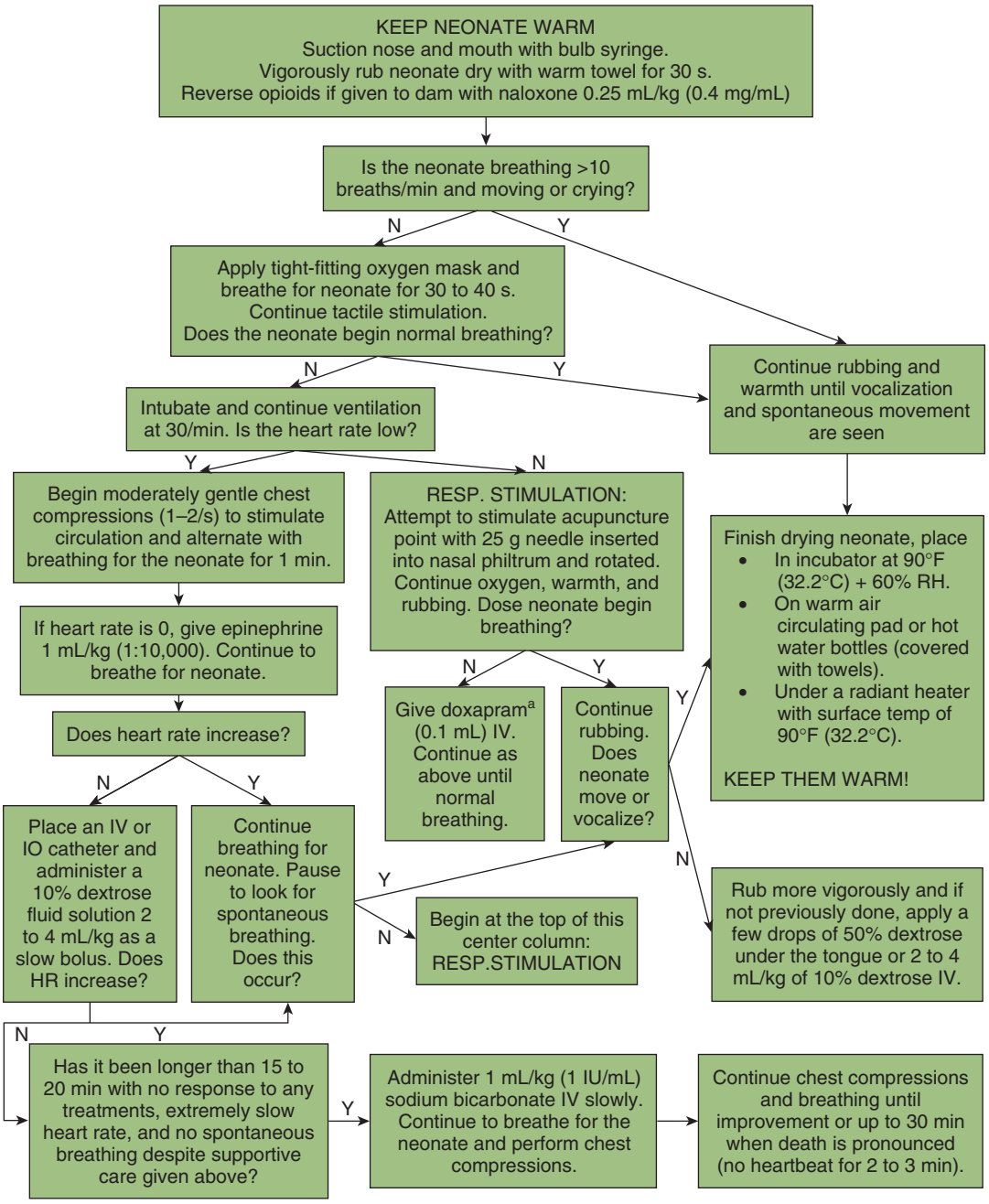


FIG. 26.14 Neonatal resuscitation flowchart. [a]See note on doxapram on p. 738 *HR,* Heart rate; *IO,* intraosseous; *IV,* intravenous; *Resp,* respiratory; *RH,* relative humidity.

the vagina, excise vaginal masses (Fig. 26.15), repair lacerations, modify congenital defects or strictures, expose the urethral papilla, and facilitate manual fetal extraction.

With the animal in a perineal position, place a noncrushing clamp (e.g., Doyen) with one shaft in the vagina on each side of the perineal midline (Fig. 26.16A). Make a midline skin incision through the dorsal commissure of the vulvar lips to just distal to the external anal sphincter muscle with a scalpel blade. Continue the incision through the muscle and vaginal wall with Mayo scissors (Fig. 26.16A). Control hemorrhage with hemostats, electrocoagulation, and ligatures. Place two or three horizontal mattress stay sutures full-thickness through the skin and vaginal mucosa on each side of the incision to facilitate retraction and hemostasis. Then remove the Doyen clamps and position a self-retaining retractor (e.g., Gelpi) to improve exposure, if necessary. Evaluate the vagina and vestibule, and perform any needed procedures. Close the episiotomy incision in three layers. Preplace an interrupted suture to realign and reappose the dorsal vulvar commissure. First, reappose the vaginal mucosa with simple interrupted or continuous sutures (e.g., 3-0 or 4-0 polydioxanone [PDS] or polyglyconate [Maxon]), tying the knots in the lumen (Fig. 26.16B). Then reappose muscles and subcutaneous tissue in a continuous pattern (Fig. 26.16C). Lastly, reappose skin with interrupted appositional sutures (e.g., 3-0 or 4-0 nylon or polypropylene). Place an Elizabethan collar,

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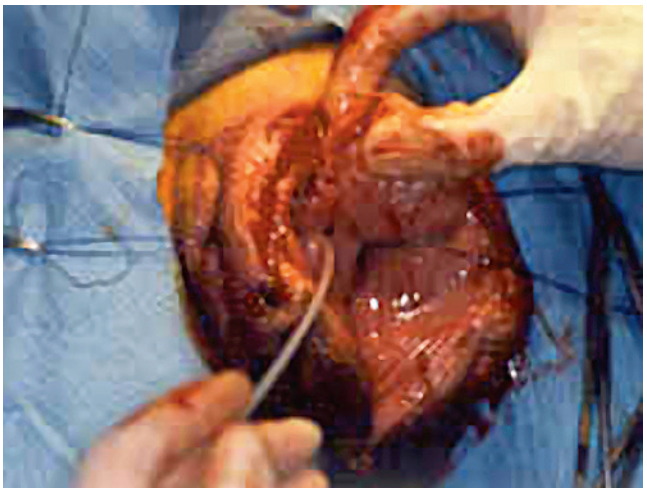


FIG. 26.15 Vaginal leiomyoma.

bucket, or sidebars after surgery to prevent self-trauma. To reduce inflammation and edema, apply cold compresses immediately after surgery and for 2 to 3 days, then warm compresses for 2 to 3 days.

Episioplasty

Episioplasty (vulvoplasty) is a reconstructive procedure most commonly performed to excise excess skin folds around the vulva, which cause perivulvar dermatitis and recurrent urinary tract infections (see p. 684). Skin fold pyoderma should be treated medically before surgical reconstruction.

Place the animal in a perineal (sternal) position with an appropriate amount of padding underneath the rear limbs. For large- or giant-breed dogs, support the limbs to avoid excessive pressure on the femoral nerves (Fig. 26.17). Place a purse-string suture in the anal orifice to minimize contamination of the surgical site. Assess the amount of skin to be excised by elevating the skin fold and evaluating expected tension (Fig. 26.18A). Beginning near the ventral vulvar commissure, make a crescent-shaped incision

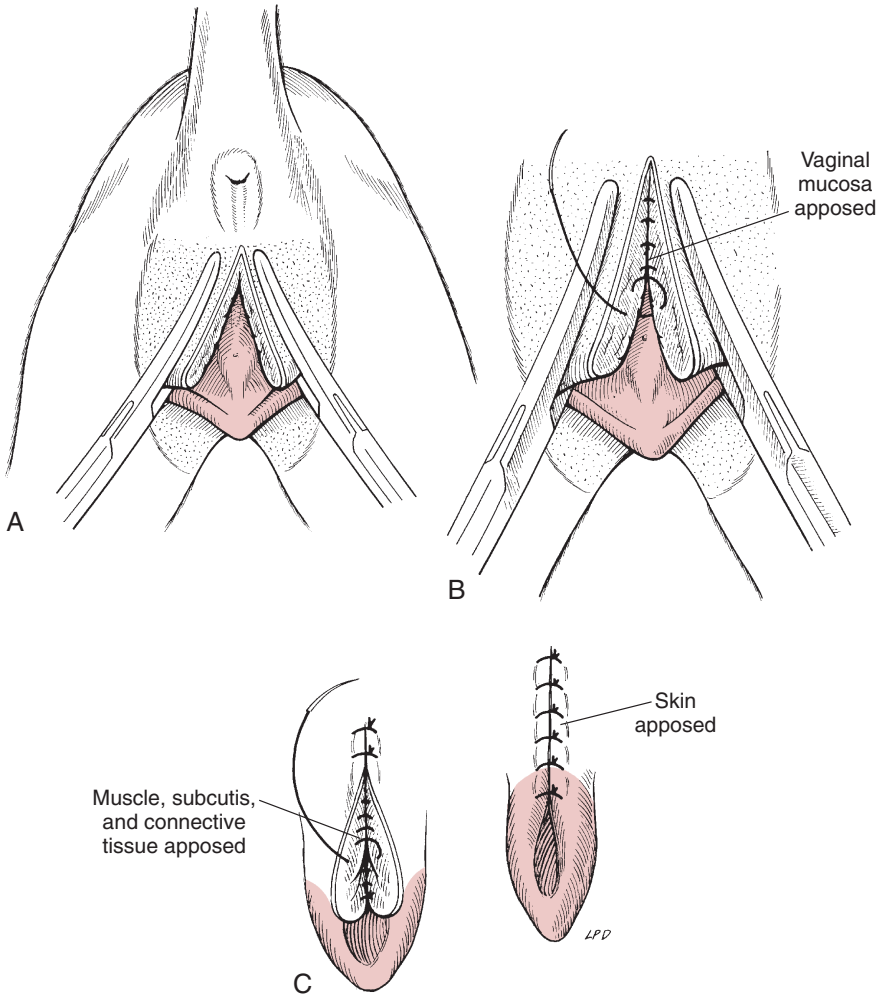


FIG. 26.16 (A) For episiotomy, place noncrushing forceps on each side of the proposed incision site. Make a full-thickness incision from the dorsal vulvar commissure to near the external anal sphincter muscle. Explore the vagina and vestibule. (B) Appose vaginal mucosa with a simple continuous suture pattern. (C) Appose muscle, subcutis, and connective tissue with a second layer of sutures and skin with a third layer of appositional sutures.

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FIG. 26.17 A 1-year-old Great Dane positioned in a perineal (sternal) position with limbs supported.

encircling the vulva at the proposed lateral and dorsal borders of the resection. Make a second crescent-shaped incision medial and parallel to the first to outline the ellipse of skin to be removed (Fig. 26.18B). Excise the outlined segment of skin and excess subcutaneous tissue (Fig. 26.18C). Place interrupted sutures at the 3 o'clock, 9 o'clock, and 12 o'clock positions to assess the effectiveness of the resection (Fig. 26.18D). Resect more skin along the outer margin if the vulva is still recessed or if skin folds persist. Bring the skin edges into approximation by first apposing the subcutaneous tissues using interrupted sutures with buried knots (e.g., 3-0 or 4-0 monofilament absorbable). Place the first sutures at the 12 o'clock, 3 o'clock, and 9 o'clock positions to symmetrically align the edges. Appose skin edges with simple interrupted sutures (e.g., 3-0 or 4-0 nylon, polybutester [Novafil], or polypropylene [Prolene]; Fig. 26.18E). Alternatively, use intradermal patterns to appose skin edges to avoid external sutures that may be irritating to the animal.

Place an Elizabethan collar or bucket over the head to prevent licking and chewing at the surgical site. Continue antibiotics if necessary to control the pyoderma.

Testicular Biopsy

Testicular biopsy may be performed in valuable breeding animals to help determine the cause of infertility or reduced fertility.

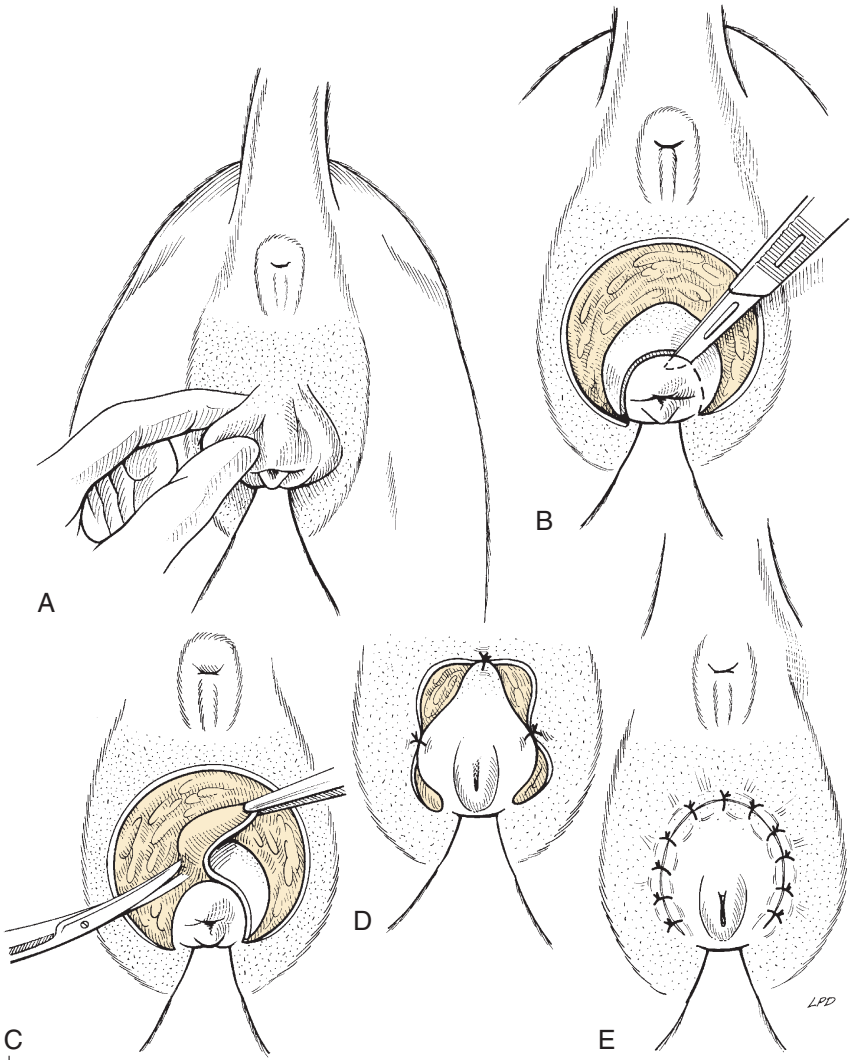


FIG. 26.18 (A) Episioplasty is performed after assessing the amount of skin to be excised. (B) Make two parallel crescent-shaped incisions encircling the vulva and (C) excise the outlined segment of skin and underlying subcutaneous tissues. (D) Appose skin edges by placing the initial sutures at the 12 o'clock, 3 o'clock, and 9 o'clock positions. (E) Appose remaining skin with simple interrupted sutures.

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Biopsies are obtained using a biopsy needle directed through the scrotal skin or by wedge resection. Ultrasound-guided percutaneous biopsy is warranted when a nonpalpable mass is identified deep within the parenchyma. A wedge of tissue is collected by making a prescrotal incision and then incising the spermatic fascia and tunics. Large blood vessels should be avoided to minimize hemorrhage.

Make a 1-cm incision through the tunica albuginea of one testicle with a No. 11 or 15 scalpel blade. Excise a wedge of testicular parenchyma with the blade. Appose the tunica albuginea with 4-0 to 6-0 monofilament absorbable suture (i.e., polydioxanone [PDS], poliglecaprone 25 [Monocryl], polyglyconate [Maxon], or glycomer 631 [Biosyn]). Appose skin edges with intradermal (subcuticular) or simple interrupted sutures. Better preservation of architectural detail is obtained by placing the sample in Bouin's, Zenker's, or Stevie's fixative rather than formalin.

Prostatic Biopsy

Prostatic biopsy is needed to definitively diagnose some prostatic diseases. Percutaneous techniques are preferred because they are less invasive, less expensive, and cause less morbidity. However, operative techniques allow collection of larger samples from more specific sites. The prostatic urethra must not be damaged, and specimens should be submitted for both histology and microbiologic culture. Percutaneous biopsies are performed using Tru-Cut, Biopty, or Franklin-Silverman biopsy needles. They may be guided by palpation (blind) or with the aid of ultrasonography. The latter is preferred because it facilitates guiding the needle to abnormal areas. Biopsy should not be performed if abscesses or cysts are suspected.

Ultrasound-Guided Biopsy

Position the patient in dorsal or lateral recumbency and ultrasonographically evaluate the prostate. Aseptically prepare the abdominal wall in the area in which the biopsy needle will be inserted. Nick the skin (3- to 5-mm incision) with a scalpel blade at the needle insertion site. Identify the desired biopsy site with ultrasound and visualize needle placement into the prostate. The needle must be parallel to the ultrasound beam to be visualized. *Collect two to three biopsies with a Biopty needle/instrument. Observe the prostate for hemorrhage or fluid leakage with ultrasound.*

Palpation-Guided Biopsy

Place the patient in a perineal position with the tail fixed over the back. Aseptically prepare the perineum around the anus. Mobilize and reposition the prostate in a more caudal position by having an assistant apply gentle dorsal and caudal pressure on the caudal abdomen. Make a nick incision (3–5 mm) slightly lateral to the midline, midway between the anus and ischial tuberosity. Confirm the location of the prostate by rectal examination. Insert the needle through the soft tissue ventral to the rectum. Guide the needle to the prostate digitally via rectal palpation. Penetrate the capsule at the caudal margin of the prostate with the needle in the closed position, then fully insert the inner cannula into the prostatic parenchyma. Quickly advance the outer cannula over the stationary inner cannula, or fire the trigger when using an automatic instrument to cut the specimen. Remove the needle from the prostate in the closed position. Evaluate the specimen size and collect additional samples if necessary.

Transrectal Fine-Needle Aspiration

With the tip of a gloved digit covering the tip of the needle (the finger should be on the bevel of the needle to help prevent puncturing your finger), carefully insert the digit into the rectum until the prostate is felt at the tip of the finger. Carefully slide the needle down the finger, through the rectal wall, and into the prostate. Perform a fine-needle aspiration as with any other organ. There are devices that are designed to help do this, but they are not necessary.

Open Biopsy

Collect prostatic biopsies during exploratory laparotomy with a biopsy needle or wedge excision. *Via a caudal midline abdominal incision, retract the urinary bladder cranially using stay sutures. Isolate the prostate from the remainder of the abdomen with sterile laparotomy pads. Palpate the prostate and select a biopsy site. Dissect periprostatic fat from the desired site. Excise a wedge of prostatic tissue using a No. 11 scalpel blade. Appose edges of the defect by placing cruciate or simple continuous monofilament absorbable sutures (e.g., 3-0 or 4-0 polydioxanone [PDS], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], or polyglyconate [Maxon]) in the prostatic capsule. Lavage the surgical site(s) and replace periprostatic fat. Close the abdomen in three layers.*

Prostatectomy

Total Prostatectomy

AP Total prostatectomy can be used for patients with tumors that have not metastasized; it is rarely performed for severe trauma or chronic prostatic disease that has been nonresponsive to other treatments. The procedure is infrequently performed because urinary incontinence commonly results, and metastatic rates of prostatic tumors at the time of diagnosis are very high.

Expose the prostate through a caudal ventral midline celiotomy and pubic osteotomy (see pp. 516 and 1215). Place a urethral catheter. Retract the urinary bladder cranially with stay sutures. Dissect the lateral pedicles and periprostatic fat directly from the capsule without damaging the dorsal plexus of vessels and nerves (Fig. 26.19A). Control hemostasis by ligation and electrocoagulation. Ligate and divide the prostatic vessels and ductus deferens as close to the prostate as possible. Dissect the prostate from the urinary bladder and extrapelvic urethra. Transect the urethra on both ends as close to the prostate as possible (Fig. 26.19B). Avoid the trigone and neck of the bladder. Remove the prostate. Advance the urethral catheter into the urinary bladder. Approximate the urethral ends with simple interrupted sutures using 4-0 to 6-0 synthetic monofilament absorbable suture (i.e., polydioxanone [PDS], polyglyconate [Maxon], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn]) on a taper point swaged-on needle. Place the first two sutures at the 12 o'clock and 6 o'clock positions, leaving the ends long to aid rotation of the urethra during suturing (Fig. 26.19C). Place the dorsal suture first. Space sutures approximately 2 mm apart and 1.5 to 2 mm from the edge. Place a cystostomy tube (see p. 685) or transurethral Foley catheter to divert urine for 5 to 7 days. Take a biopsy of a medial iliac or sublumbar lymph node, as well as the liver, to evaluate for metastasis. Replace contaminated instruments and gloves. Lavage the surgical site and abdomen. Place omentum around the anastomosis. Wire the pubic segment into place. Perform a three-layer abdominal wall closure. Place an Elizabethan collar, bucket, or sidebars after surgery to prevent displacement of the catheter and surgical site trauma.

Subtotal Prostatectomy

Subtotal prostatectomy is rarely indicated in valuable breeding dogs for benign prostatic hyperplasia (BPH) in lieu of castration,

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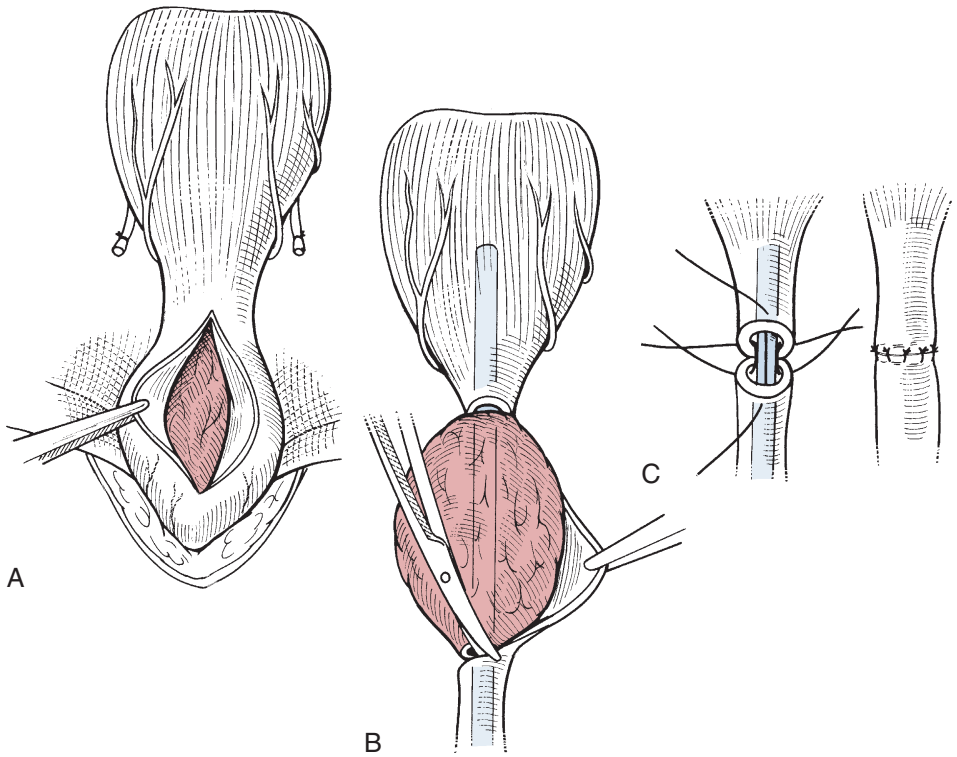


FIG. 26.19 (A) To perform total prostatectomy, separate the fat, fascia, vessels, and nerves from the prostate by dissecting directly against the capsule. (B) Dissect the cranial and caudal edges of the prostate from the urethra and transect the urethra as close to the prostate as possible. (C) Stent the urethra with a catheter and appose the ends with approximating sutures.

or in stable dogs with abscessation or cysts in lieu of drainage procedures. Intracapsular subtotal prostatectomy using electrocautery or Nd:YAG laser has been performed in a small number of dogs with prostatic cancer as a reasonable palliative option with a markedly decreased incidence of urinary incontinence when compared with total prostatectomy. The prostate is approached and exposed as for total prostatectomy (see previous discussion). Alternatively, laparoscopic, transrectal, or transurethral approaches have been described. A urinary catheter should be placed to aid urethral identification. Up to 85% of the prostatic parenchyma can be removed with urinary continence maintained. Submit excised tissue for histologic evaluation.

Subtotal Prostatectomy With Capsulectomy

Isolate and ligate or cauterize all vessels as they enter the prostatic capsule. Temporary occlusion of the aorta just cranial to its bifurcation into the external iliac arteries is sometimes recommended. *Excise the prostate within 5 mm of the urethra using scissors, an electrosurgical unit, an ultrasonic aspirator, or laser (Fig. 26.20A). Place a cystostomy tube if the urethral catheter is to be removed (see p. 685). Assess hemostasis and lavage the surgical site. Surround the prostatic urethra with omentum or prostatic fat. Close the abdomen routinely.*

Intracapsular Subtotal Prostatectomy

Incise the ventral median septum. Continue the incision through the parenchyma into the ventral urethra. Using an electrocautery or radiofrequency unit, resect all parenchyma except a 2- to 3-mm shell attached to the capsule (see Fig. 26.20B). Resect all the urethra except a 3- to 5-mm dorsal strip. Lavage the prostatic shell and close the capsule over a urethral catheter positioned in the urinary bladder. Use an approximating pattern for the first layer and an inverting pattern for the second layer of closure (e.g., 3-0 or 4-0 polydioxanone [PDS], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], or polyglyconate [Maxon]). Maintain the catheter for 10 days. Alternatively, use a laser or an ultrasonic surgical aspirator to remove parenchyma and preserve the urethra.

HEALING OF THE REPRODUCTIVE AND GENITAL SYSTEMS

Reproductive organs heal like other visceral tissue. Incisions into testicular parenchyma may cause an immunologic response and subsequent sperm granuloma. Scarring of the uterus may inhibit placentation. To decrease uterine adhesions, omentum can be placed over incisions. Failure of postpartum uterine involution may be caused by excessive collagen breakdown as a result of uterine collagenase activity. Optimal healing requires a good blood supply, accurate mucosal apposition, and minimal surgical trauma. Systemic factors (e.g., hypovolemia, shock, hypoproteinemia, debilitation, and infection) may delay healing and increase the risk of dehiscence.

SUTURE MATERIALS/SPECIAL INSTRUMENTS

Instruments needed for reproductive surgery include an OHE hook (e.g., Covault, Snook), retractors (e.g., Balfour [abdominal procedures], Gelpi or Weitlaner [perineal procedures], Finochietto [pelvic procedures], vaginal speculum or otoscope [vaginal procedures]), scissors (e.g., Metzenbaum, Mayo), forceps (e.g.,

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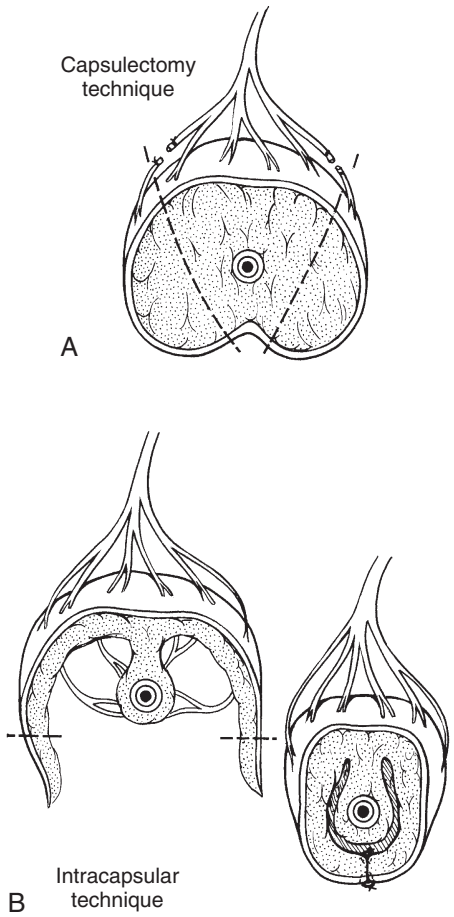


FIG. 26.20 Subtotal prostatectomy may be performed using (A) capsulectomy or (B) intracapsular techniques.

BOX 26.7 Suture Recommendations for Reproductive Surgery

- Select a monofilament, synthetic, absorbable suture for visceral closure (polydioxanone [PDS], polyglyconate [Maxon], poliglecaprone 25 [Monocryl], or glycomer 631 [Biosyn])
- Select an absorbable suture for ligatures (polydioxanone, polyglyconate, polyglactin 910 [Vicryl], poliglecaprone 25, or glycomer 631)

Doyen, Carmalt, and curved Mosquito), biopsy instruments (Biopty-cut biopsy needle with a Bard Biopsy Instrument), and drains. Orthopedic instruments are needed if a pelvic osteotomy is performed. An electrosurgical or radiofrequency unit, ultrasonic aspirator, laser unit, ultrasonic scalpel, vascular sealer, and laparoscopic equipment and instruments are sometimes beneficial.

Monofilament absorbable and nonabsorbable sutures are recommended for most reproductive tract procedures (i.e., 2-0 to 6-0 polydioxanone [PDS], polyglyconate [Maxon], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], polybutester [Novafil], polypropylene [Prolene], or nylon) (Box 26.7). Stapling equipment (transverse, ligate and divide, and skin staplers) is sometimes used.

POSTOPERATIVE CARE AND ASSESSMENT

Animals undergoing reproductive tract surgery should be monitored postoperatively for pain, hemorrhage, and infection. Postoperative analgesics are indicated (see Chapter 13 and Tables 13.2 and 13.3 for dosages of opioids). The incision site should be assessed twice daily for redness, swelling, or discharge. Activity should be limited to leash walks until sutures are removed (generally 10–14 days). Water is usually offered 4 to 6 hours after surgery, unless vomiting occurs. If the animal does not vomit, food can be given 6 to 12 hours postoperatively.

Nonelective procedures are often performed on sick animals with fluid, electrolyte, and acid-base abnormalities; these abnormalities require monitoring and continued treatment in the postoperative period. Therapeutic antibiotics should be continued in patients with preoperative infections. Surgical sites should be protected by using an Elizabethan collar, bucket, sidebars, or bandage to prevent self-trauma. Stool softeners may be administered after prostatic or perineal surgery to minimize discomfort during defecation (Box 26.8). Apply cold compresses two to three times daily for 2 or 3 days; then apply warm compresses for 2 or 3 additional days to minimize hemorrhage and swelling after perineal surgery.

BOX 26.8 Stool Softeners

Dioctyl Sodium Sulfosuccinate or Docusate Sodium (Colace)

Dogs: 50–200 mg PO q8–12h
Cats: 50 mg PO q12–24h

Lactulose (Chronulac)

Dogs: Start with 1 mL/4.5 kg PO q8h and adjust so that stools are soft and formed, but not watery
Cats: Start with 5 mL/cat PO q8h and adjust to effect

Psyllium (Metamucil)

Dogs: Start with 1 tsp/5–10 kg q12h in food and adjust until desired effect is obtained
Cats: Start with 1 tsp/cat q12h and adjust until desired effect is obtained

PO, Orally.

BOX 26.9 Common Errors in Reproductive Surgery

- Failure to support septic or debilitated patients with fluids, antibiotics, or nutrition
- Failure to completely resect tumors
- Failure to obtain a histologic diagnosis
- Failure to prevent self-mutilation of the surgical site

COMPLICATIONS

Most complications associated with reproductive surgery can be prevented by using good surgical technique (i.e., gentle tissue handling, good hemostasis, and aseptic technique) (Boxes 26.9 and 26.10). OHE is difficult in larger dogs and is associated with more complications. Hemorrhage primarily occurs from the ovarian pedicles, uterine vessels, or uterine wall when ligatures are improperly placed; it rarely occurs from vessels that accompany the suspensory ligament or those within the broad ligament. Excessive hemorrhage may occur when OHE is performed during estrus. Ureter ligation or trauma may occur when ligating a dropped or hemorrhaging ovarian pedicle when exposure of the caudal renal pole is inadequate. The ureter may also be ligated if the urinary bladder is distended and the trigone and ureterovesical junction are displaced cranially. Hydronephrosis necessitating ureteronephrectomy results unless the offending ligature is

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BOX 26.10 Potential Complications After Reproductive Surgery

Ovariohysterectomy
- Pain, hemorrhage, infection, dehiscence, urinary incontinence, ovarian remnant, ureteral ligation, fistula, adhesions

Castration
- Hemorrhage, scrotal hematoma, scrotal bruising, infection, dehiscence, urinary incontinence, behavior change, eunuchoid syndrome

Vasectomy
- Granuloma, scrotal swelling, incisional problems

Cesarean Section
- Hemorrhage, retained fetus, adhesions, uterine scarring, incisional problems, shock, hypothermia, hypocalcemia, agalactia, metritis, vomiting, anorexia

Mastectomy
- Pain, inflammation, hemorrhage, seroma formation, infection, ischemic necrosis, self-trauma, dehiscence, limb edema, tumor recurrence

Episiotomy
- Pain, swelling, inflammation, hemorrhage, infection, dehiscence, self-trauma

Episioplasty
- Inflammation, swelling, infection, dehiscence, recurrent perivulvar dermatitis

Testicular Biopsy
- Hemorrhage, infection, local hyperthermia, scarring, adhesions, immune-mediated orchitis, testicular atrophy, reduced sperm count (temporary)

Prostatic Biopsy
- Hemorrhage, hematuria, urine leakage, infection, tumor transplantation

Prostatectomy
- Hemorrhage, infection, incisional problems, urinary catheter displacement, urine leakage, dysuria, urethral stricture, urinary incontinence, tumor spread

promptly removed. Estrus may recur if ovarian tissue remains in the abdominal cavity. If this occurs, abdominal exploration during estrus may help identify the ovarian tissue. Fistulous tracts and granulomas may form if nonabsorbable multifilament suture material is used for ligations. These fistulae are usually located in the flank but also occur along the medial thigh or inguinal region. They intermittently discharge blood-tinged fluid or mucopurulent exudate. Discharge may diminish during antibiotic therapy but recurs when antibiotics are discontinued. These fistulae will not resolve until the suture material is removed. Caution must be exercised during dissection because there may be adhesions to the vena cava and other vital structures.

Post-OHE urinary incontinence is uncommon (prevalence is approximately 5%), but when it occurs, it may occur soon after surgery or in geriatric bitches. Previous studies have found a higher incidence of urinary incontinence in females neutered before 3 months of age (approximately 13%), yet a more recent study found no significant difference in the age at the time of OHE between incontinent and continent groups.[3] However, dogs with a body weight greater than 15 kg were seven times more likely to develop acquired urinary incontinence compared with dogs less than 15 kg. Causes of urinary incontinence include low estrogen levels, uterine stump adhesions or granulomas to the urinary bladder, and vaginoureteral fistulae. Despite the belief that OHE causes obesity, properly fed and exercised neutered animals should not gain excessive weight. Juvenile behavior and external genitalia may persist in animals neutered at a very young age (6–12 weeks). Hypothermia, hypoglycemia, blood loss, and tissue handling problems occur more commonly with prepubertal OHE. Other possible complications of OHE are uncommon but include self-trauma, incisional swelling, seroma, infection, delayed healing, dehiscence, trauma to intestines or spleen, cervical pyometra, endocrine alopecia, colonic obstruction, behavioral change, and *eunuchoid syndrome* (having the characteristics of a castrated male; secondary sex characteristics do not develop). One study of 1880 dogs undergoing OHE documented an overall complication rate of 7.5%; increasing patient weight and duration of anesthesia time were found to be risk factors for development of complications.[4]

Serious complications following properly performed castrations are rare but may include incisional problems (e.g., swelling, seroma formation, cellulitis, infection, self-trauma, dehiscence), hemorrhage, scrotal hematoma, scrotal bruising, abscess, granuloma, urinary incontinence, endocrine alopecia, behavioral changes, eunuchoid syndrome, and constriction of the colon. Trauma to the penis and urethra may occur during dissection, especially with scrotal ablation. Although unlikely, an unwanted pregnancy may occur if a recently castrated male copulates with a female in estrus because spermatozoa persist in the ductus deferens up to 21 days in dogs and 49 days in cats. Inadvertent prostatectomy has occurred during cryptorchidectomy.

Complications of C-section, with or without OHE, include hemorrhage, hypovolemia, hypothermia, hypocalcemia, anorexia, anemia, agalactia, metritis, and vomiting. Severe hemorrhage may necessitate OHE. Calcium levels should be monitored if eclampsia is suspected; affected dogs usually have ionized calcium levels less than or equal to 6 mg/dL. Eclampsia more commonly occurs in small-breed dogs with large litters. Eclampsia may occur anytime during the first postpartum month, and not all affected animals have typical signs (e.g., muscle tremors, tetany, and convulsions). An odorless, dark red-brown to serous uterine discharge or lochia is expected for 4 to 6 weeks postpartum. Incisional complications, such as swelling, infection, seroma, self-trauma, or dehiscence may occur. Uterine scarring may prevent future placentation and adhesions may interfere with uterine motility. Neonate mortality is most commonly associated with emergency surgery, large litters, brachycephalic bitches, and poor anesthetic choices.

Although rare, complications can occur after mastectomy (see p. 749), episiotomy, episioplasty, testicular biopsy, and prostatic biopsy (see Box 26.10). Dehiscence after episioplasty may occur if skin resection causes excessive suture line tension; however, perivulvar dermatitis will persist or recur if inadequate skin is excised.

Early postoperative complications of prostatectomy include hemorrhage, urine leakage, infection, and urethral catheter displacement; later complications include dehiscence, urethral stricture, and urinary incontinence. Urinary incontinence is expected in more than 85% of dogs after prostatectomy unless the prostate is of normal size and dissection does not traumatize trigonal innervation or vascularity.

Incontinence caused by diminished urethral sphincter tone may be treated with alpha-adrenergic agonists (phenylpropanolamine, imipramine, or ephedrine) that increase urethral

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BOX 26.11 Treatment of Urinary Incontinence

Phenylpropanolamine[a]
Dogs: 1–2 mg/kg PO q8–12h[b]
Cats: 1 mg/kg PO q12h

Ephedrine
Dogs: Start at 0.4 mg/kg, and gradually increase to 4 mg/kg PO q8–12h[c]
Cats: 2–4 mg/cat PO q8–12h

Diethylstilbestrol
Dogs: 0.1–1 mg/dog PO q24h (dose is proportional to the size of the dog) for 3–5 d, then (if urinary incontinence is controlled) administer the same dose once every 3–7 d as needed, with a maximum of 0.2 mg/kg/wk[d]

Estriol (Incurin)[e]
2 mg/dog PO q24h for 7 d, then decrease at weekly intervals until weekly dose of 0.5 mg/dog is achieved

Testosterone Cypionate (Depo-Testosterone)
Dogs: 1.1–2.2 mg/kg IM once every 2–4 wk

Imipramine (Tofranil)
Dogs: 2–4 mg/kg PO q12–24h *or* 5–15 mg/dog PO q12h

[a]Proin now has Food and Drug Administration approval for use in dogs.
[b]Best to start at low end of dose and gradually increase, if needed.
[c]Toxicity typically starts at 5 mg/kg and death can readily occur at 10 mg/kg.
[d]If benefit is not noted after 5 days of daily therapy, then it is unlikely that further administration will benefit the patient, and other drugs should be used.
[e]May be combined with phenylpropanolamine.
IM, Intramuscular; *PO,* orally.

sphincter tone or with diethylstilbestrol (preferred), estriol, or repositol testosterone (rarely used) (Box 26.11). Phenylpropanolamine is used more frequently than ephedrine because it has fewer side effects (e.g., hyperexcitability, panting, anorexia) and greater efficacy over time (see also p. 713).

SPECIAL AGE CONSIDERATIONS

Elective neutering is most advantageous when the animal is less than 1 year old. Undesirable behaviors are usually not learned by this age, and some tumors (e.g., mammary adenocarcinoma) can be inhibited. Pediatric tissues are more fragile than adult tissues and must be handled gently. Long bone growth increases slightly following prepubertal gonadectomy. Development of excessive tibial plateau angle may occur in large-breed dogs neutered before 6 months of age. Tumors, pyometra, and prostatic infections are more common in geriatric patients.

Surgery of the Female Reproductive Tract

SPECIFIC DISEASES

MAMMARY NEOPLASIA

DEFINITIONS

Lumpectomy is removal of a mass or part of a mamma; **simple mastectomy** is excision of an entire gland, and **regional mastectomy** is excision of the involved gland and adjacent glands. **Unilateral mastectomy** is the removal of all mammary glands, subcutaneous tissue, and associated lymphatics on one side of the midline, whereas **bilateral mastectomy** is the simultaneous removal of both mammary chains.

BOX 26.12 Canine Mammary Masses

Benign mixed tumors
Carcinomas
- Solid carcinomas
- Tubular adenocarcinomas
- Papillary adenocarcinomas
- Anaplastic carcinomas

Hyperplasia
- Adenomas
- Malignant mixed tumors
- Sarcomas
- Myoepitheliomas

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Mammary tumors are uncommon in male dogs, but the most common tumor of female dogs. They are less common in cats but still account for nearly one-third of all feline tumors. Approximately 35% to 50% of canine mammary tumors and 90% of feline mammary tumors are malignant. Canine mammary tumor types are listed in Box 26.12. Malignant mammary tumors spread via lymphatics and blood vessels to regional lymph nodes and lungs. Other less common metastatic sites include the adrenal glands, kidneys, heart, liver, bone, brain, and skin.

The cause of mammary gland neoplasia is unknown; however, many are hormone-dependent, and most can be prevented if OHE is performed before 1 year of age. The risk of mammary tumors for dogs spayed before their first estrus is 0.05%. This risk increases to 8% after one estrus cycle and 26% after the second estrus. Cats ovariectomized before 6 months of age have a 91% reduction of risk of mammary carcinoma development compared with sexually intact cats; those ovariectomized between 6 and 12 months have an 86% reduction of risk. In general, sexually intact dogs and cats have seven times the risk of developing mammary tumors compared with neutered animals. Estrogen and/or progesterone receptors are found in 50% of malignant and 70% of benign canine mammary tumors. Dogs with tumors containing estrogen or progesterone receptors live longer than those without. Progesterone receptors are found in some feline mammary tumors. Progesterone administration may be associated with the development of malignant mammary tumors in cats and benign tumors in dogs. Dogs with benign mammary tumors have more than a threefold risk of developing malignant mammary tumors.

In dogs, benign tumors are usually classified as benign mixed tumors (fibroadenomas), adenomas, or benign mesenchymal tumors (Fig. 26.21). Most canine malignant mammary tumors are carcinomas (see Box 26.12); however, sarcomas (<5%) and carcinosarcomas (malignant mixed tumors) occur. Sarcomas metastasize more readily than carcinomas. Some "malignant" mammary tumors do not recur or spread after surgery. Papillary or tubular carcinomas have a better prognosis than solid or anaplastic carcinomas. Inflammatory carcinomas are poorly differentiated carcinomas with extensive mononuclear and polymorphonuclear cellular infiltrates. It may be difficult to

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FIG. 26.21 A large mammary tumor in a 13-year-old female dachshund.

BOX 26.13 Mammary Tumors: Key Points

- Most mammary tumors are prevented by ovariohysterectomy in the first year of life.
- Male mammary tumors are rare but behave similar to those in females.
- All mammary tumors should be evaluated when first identified.
- Tumors in dogs are benign or malignant; 90% of those in cats are malignant.
- Expectations are primarily dependent on histologic type.
- Each mammary mass may be a different tumor, therefore remove them all.
- Provided tumor-free margins are obtained, the tumor excision technique does not affect survival in dogs; perform a chain mastectomy in cats.
- Do not excise inflammatory carcinomas; the prognosis is too poor.
- Adjuvant therapy is not routinely recommended for malignant tumors.

differentiate mastitis from inflammatory carcinoma on physical examination or with cytology. These tumors grow rapidly, invading cutaneous lymphatics and causing marked edema, inflammation, and pain. Dogs are likely to be anorectic and weak and experience weight loss. The tumors are poorly demarcated, firm, often ulcerated, and may involve both mammary chains. Some areas of involvement appear rashlike. Extensive lymphedema of the limbs may occur secondary to lymphatic occlusion or infiltration. Disseminated intravascular coagulation and thoracic metastasis are common with inflammatory carcinoma, and this tumor has a poor prognosis.

Approximately 90% of feline mammary tumors are adenocarcinomas; however, other types of carcinomas and sarcomas occasionally occur (see Box 26.12). Feline mammary tumors grow rapidly and metastasize to local lymph nodes and lungs early in the course of disease. Feline mammary tumors are not as well circumscribed as their canine counterparts; they are firm and often ulcerated. Mammary carcinomas in male cats behave similarly to those of females but are less common (Box 26.13). Feline mammary tumors must be differentiated from lobular hyperplasia and fibroepithelial hyperplasia. Hyperplasia is often associated with exogenous progesterone administration, but both benign and malignant tumors have been reported in male and female cats given supplemental progesterone. A unilateral mastectomy is recommended to remove feline mammary tumors because local recurrence is common with less radical procedures. Cats with malignant mammary tumors generally survive less than 1 year.

DIAGNOSIS

Clinical Presentation

Signalment

Mammary tumors are common in female dogs and cats. The greatest frequency of mammary tumors is found in poodles, Boston terriers, fox terriers, Airedale terriers, dachshunds, Great Pyrenees, Samoyeds, keeshonden, and sporting breeds (pointers, retrievers, setters, and spaniels). Almost all feline mammary tumors (99%) occur in intact females. Most mammary tumors occur in middle-aged or older animals; they are rare in young animals. The incidence of mammary tumors increases greatly after 6 years of age. Dogs develop mammary tumors at a median age of 10 to 11 years, whereas feline carcinomas occur most often between 8 and 12 years of age. Younger dogs are more likely to have benign tumors than older dogs, and they are rare in dogs less than 5 years of age.

History

Many mammary tumors are discovered during routine physical examination. Animals may be brought in because of a lump and/or abnormal discharge from the mammae. A delay of several months is common before a veterinarian evaluates the animal. Occasionally an animal with advanced disease is brought in because of dyspnea or lameness secondary to pulmonary or bone metastasis, respectively.

Physical Examination Findings

Mammary masses may be of various sizes (2–3 mm to 8 cm), but malignant tumors are often significantly larger than benign tumors. The most common site for canine mammary tumors is the caudal mammary glands, but 66% of dogs present with more than one tumor. Multiple masses may be found in one or both mammary chains. Most masses are easily movable, but occasionally they are fixed to the underlying muscle or fascia. Masses may be sessile or pedunculated, solid or cystic, ulcerated or covered with skin and hair. Inflammatory carcinoma or mastitis should be suspected if the glands are diffusely swollen with poor demarcation between normal and abnormal tissue. Inflammatory carcinomas are often ulcerated. Axillary or inguinal lymph node enlargement may be palpable, and sublumbar lymph node enlargement may be detected on rectal examination. Lameness or limb edema suggests metastasis. Weakness, anorexia, weight loss, and/or pain in the mammary region and limbs are common with inflammatory carcinoma.

Diagnostic Imaging

Thoracic radiographs (three views) should be evaluated for pulmonary metastasis. Thoracic metastasis occurs in 25% to 50% of dogs with malignant mammary tumors by the time of diagnosis. Pleural fluid may occur in cats with metastatic pulmonary disease. Abdominal radiographs should be evaluated for iliac lymph node enlargement with caudal tumors. Abdominal ultrasonography may detect abdominal metastasis. CT and MRI imaging may facilitate evaluation of invasive tumors and metastasis.

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Laboratory Findings

Minimum database (complete blood count, biochemistry profile, urinalysis) results are nonspecific for mammary neoplasia, but important in identifying concurrent geriatric problems or paraneoplastic syndromes. Aspiration or exfoliative cytology helps distinguish inflammatory, benign, and malignant masses. Detection of neoplastic cells in lymph node aspirates helps stage the disease. Pleural fluid should be evaluated cytologically. Bone scans help confirm bone metastasis. Definitive diagnosis is dependent on histopathology of biopsied or excised tissue. Each mass should be evaluated histologically because different tumor types may occur in the same individual. Immunohistochemical analysis of histologic specimens can provide useful prognostic information.

DIFFERENTIAL DIAGNOSIS

Mammary hypertrophy, mastitis, granulomas, duct ectasia, skin tumors, or foreign bodies (e.g., BB pellet or shot) are differential diagnoses. Mammary hypertrophy from endogenous or exogenous progesterone stimulation commonly occurs in young intact female cats 2 to 4 weeks after estrus (when progesterone concentrations are elevated). Hypertrophy can usually be ruled out based on history and cytologic findings. Mastitis occurs after estrus, parturition, or false pregnancy; the swelling is usually more localized than with inflammatory carcinoma.

MEDICAL MANAGEMENT

Reports on the efficacy of treatment modalities other than surgery are lacking. Chemotherapy may be beneficial in controlling some malignant tumors. Postoperative adjunctive chemotherapy has not been shown to improve outcomes or survival times in dogs or cats. In general, chemotherapy, radiation therapy, and hormonal therapy are not routinely recommended as an adjunct to surgery.

SURGICAL TREATMENT

Excision is the treatment of choice for all mammary tumors except inflammatory carcinomas. Excision allows histologic diagnosis and can be curative, improve quality of life, or modify disease progression. Inflammatory carcinomas are extremely aggressive, and surgery is of no value in controlling or palliating the disease. Selection of a surgical technique for removing the tumor and variable amounts of mammary tissue depends on tumor size, location and consistency, patient status, and surgeon preference. Survival is not influenced by technique unless incomplete resection is performed. However, local recurrence is decreased in cats when unilateral mastectomy is performed rather than lumpectomy. A combination of different techniques may be selected if an animal has several masses in both chains. All tumors should be excised because each mass may be a different tumor type. If complete excision is impossible with a single surgery, a second procedure should be delayed 3 to 4 weeks to allow healing and relaxation of stretched skin. OHE may be performed when the mammary tumor is removed. OHE should be done before mastectomy to prevent seeding the abdominal cavity with tumor cells. Although OHE is unlikely to prevent further development of mammary tumors, it will prevent uterine disease (e.g., pyometra and metritis) and eliminate female hormonal influence on existing tumors. However, in one study, dogs with grade 2 mammary carcinoma, estrogen receptor (ER)-positive tumors, or perioperative increased serum estradiol (E2) concentration had a decreased hazard of relapse when undergoing OHE at the time of mammary tumor removal compared with dogs not receiving OHE.[5]

Lumpectomy or *partial mammectomy* is the excision of a mass and a surrounding margin of grossly normal mammary tissue (≥1 cm). It is used when the mass is small (<5 mm), encapsulated, noninvasive, and at the periphery of the gland. Milk and lymph leakage from incised mammary tissue into the wound may cause postoperative inflammation and discomfort. *Simple mastectomy* is the excision of the entire gland containing the tumor. It is used when the tumor involves the central area of the gland or the majority of the gland. Removal of the entire gland may be easier than incising mammary tissue and prevents postoperative problems with milk and lymph leakage. *Regional mastectomy* involves excision of the involved and adjacent glands. This technique is selected when multiple tumors occur in adjacent glands in the chain or when the mass occurs between two glands. It is sometimes technically easier to remove the confluent caudal abdominal and inguinal glands than either gland alone. *Unilateral mastectomy* is performed when numerous tumors occur throughout the chain or in cats with a single mass because local recurrence is common. A unilateral mastectomy may take less time and be less traumatic than multiple lumpectomies or mastectomies. *Bilateral mastectomy* can be performed when numerous masses occur in both chains; however, skin closure can be extremely difficult or impossible. Therefore it is not recommended. Instead, staged unilateral mastectomies are preferred.

> **NOTE** Separate mammary masses on the same dog may be of different histologic types; therefore excise all masses and submit them for histologic examination. Be sure to mark them so that you can determine which mass originated from which site when the biopsy report returns.

Preoperative Management

A complete workup to determine the stage of the disease and identify other problems that may alter the prognosis is important. Ulcerated, infected masses should be treated with warm compresses and antibiotics for several days before surgery to reduce inflammation and allow the gross tumor margins to be more accurately assessed. Preoperative antibiotics are necessary only in severely debilitated patients or those with evidence of infection. If renal disease (e.g., secondary to hypercalcemia or malignancy) is present, preoperative fluids should be administered. The entire ventral abdomen and caudal thorax should be clipped. Each mammary chain should be carefully palpated and the location of each mass mapped. Additional masses are frequently identified once the hair has been removed.

Anesthesia

A variety of anesthetic protocols can be used in animals with mammary masses (see Table 26.3). General anesthetic is usually less stressful to the patient than local anesthetic, even when small lumps are resected. Consider administering an opioid epidural preoperatively and postoperatively if a large area of tissue is removed.

Surgical Anatomy

Dogs usually have five pairs of mammary glands; cats have four pairs. Mammary glands are compound, tubuloalveolar, apocrine

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BOX 26.14 Major Blood Vessels Supplying the Mammary Glands of Dogs and Cats

Mammary Glands 1 and 2
Ventral and lateral branches of the intercostal, internal thoracic, and lateral thoracic vessels

Mammary Glands 2 and 3
Cranial superficial epigastric vessels

Mammary Glands 4 and 5
Caudal superficial epigastric vessels

glands. The caudal superficial epigastric arteries and veins supply the caudal glands (Box 26.14). The caudal superficial epigastric artery arises from the external pudendal artery near the superficial inguinal lymph node. Branches of the cranial and caudal superficial epigastric arteries anastomose. The cranial thoracic mammae are supplied by the fourth, fifth, and sixth ventral and lateral cutaneous vessels and nerves (from intercostals) and branches of the lateral thoracic vessels (from axillary artery). The caudal thoracic mammae are supplied by the sixth and seventh cutaneous nerves, and vessels and branches of the cranial superficial epigastric vessels. The cranial superficial epigastric vessels supply the cranial abdominal mamma and skin over the rectus abdominis muscle. The axillary lymph node drains the three cranial glands, and the inguinal lymph node drains the two caudal glands. However, there are lymphatic connections between glands and across the midline.

Positioning

Position the patient in dorsal recumbency with the thoracic limbs fixed cranially and the pelvic limbs fixed caudally in a relaxed position. The entire ventral abdomen, caudal thorax, and inguinal areas should be clipped and prepared for aseptic surgery.

SURGICAL TECHNIQUE

Make an elliptical incision around the involved mammary gland(s), a minimum of 1 cm from the tumor (Fig. 26.22A). Continue the incision through subcutaneous tissue to the fascia of the external abdominal wall. Avoid incising mammary tissue; however, this is often impossible because mammary tissue may be confluent between adjacent glands. The midline separation between mammary chains is distinct.

Control superficial hemorrhage with electrocoagulation, hemostats, or ligation. Perform an en bloc excision by elevating one edge of the incision and dissecting subcutaneous tissue from the pectoral and rectus fascia using a smooth gliding motion of the scissors (Fig. 26.22B). Use traction on the elevated skin segment to facilitate dissection.

Abdominal and inguinal glands are loosely attached by fat and connective tissue and easily separated from rectus fascia. Thoracic glands adhere to the underlying pectoral muscles with little intervening fat or connective tissue.

Resect the inguinal fat pad and lymph node(s) with the inguinal mammary gland. The axillary lymph node is not included with en bloc resection of the thoracic glands. *Excise fascia if the tumor has invaded subcutaneous tissue.* Some neoplastic lesions will invade the abdominal musculature, and excision must include a portion of the abdominal wall.

Continue gliding scissor dissection until major vessels (i.e., cranial superficial epigastrics and caudal superficial epigastrics) to the gland are encountered. Isolate and ligate these vessels (Fig. 26.22C). Ligate the cranial superficial epigastric vessel where it penetrates the rectus abdominis between the caudal thoracic and cranial abdominal (third) mammary glands (see Box 26.14). Ligate the caudal superficial epigastric vessel adjacent to the inguinal fat pad near the inguinal ring (see Box 26.14). Ligate branches supplying the first and second thoracic mammary glands as they are encountered penetrating the pectoral muscles. Lavage the wound and evaluate for abnormal tissue. Undermine the wound edges and advance skin toward the center of the defect with walking sutures (see Fig. 15.22). If dead space is extensive, place a closed suction or Penrose drain to help prevent fluid accumulation. Appose skin edges with a subcutaneous or subcuticular suture pattern (Fig. 26.22E). Use 3-0 or 4-0 monofilament absorbable suture (polydioxanone [PDS], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], or polyglyconate [Maxon]) on a swaged-on taper point needle in either an interrupted or continuous pattern.

Skin apposition is most difficult in the thoracic region because the skin is less mobile and the ribs make the area less compressible than the abdomen.

Use an axillary or flank fold skin flap to close the defect if tension is excessive (see pp. 218–220). Use appositional monofilament absorbable skin sutures (e.g., 3-0 or 4-0 nylon, polybutester [Novafil], or polypropylene [Prolene]) or staples. Place a padded circumferential bandage to compress dead space, mobilize tissue, and support the wound.

POSTOPERATIVE CARE AND ASSESSMENT

Analgesics (see Chapter 13 and Tables 13.2 and 13.3 for dosages of opioids) and supportive care should be given as needed. An abdominal bandage should support the wound, compress dead space, and absorb fluid. Bandages are changed daily for the first 2 to 3 days, or as necessary to keep them dry. The wound should be inspected for inflammation, swelling, drainage, seroma, dehiscence, and necrosis. Any drains should be removed when drainage diminishes to a minimal amount (usually within 3–5 days). Bandages and sutures are generally removed 5 to 7 days and 7 to 10 days postoperatively, respectively. Patients with malignant tumors should be reevaluated for local recurrence and metastasis every 3 to 4 months.

COMPLICATIONS

Complications include pain, inflammation, hemorrhage, seroma formation, infection, ischemic necrosis, self-trauma, dehiscence, hind limb edema, and tumor recurrence. In dogs, local recurrence occurs within 2 years and varies from 20% to 73%.

PROGNOSIS

Significant prognostic factors in dogs are provided in Box 26.15. Significant prognostic factors in cats are tumor size, extent of surgery, and histologic grading. Risk of metastatic disease increases and survival time decreases with increasing primary tumor size. The prognosis for dogs with benign tumors is good with surgery. The prognosis for dogs with malignant tumors is variable and depends on several factors, including tumor type, tumor stage, tumor size, OHE status, and the presence of metastasis. Most dogs with malignant tumors without obvious metastasis at the

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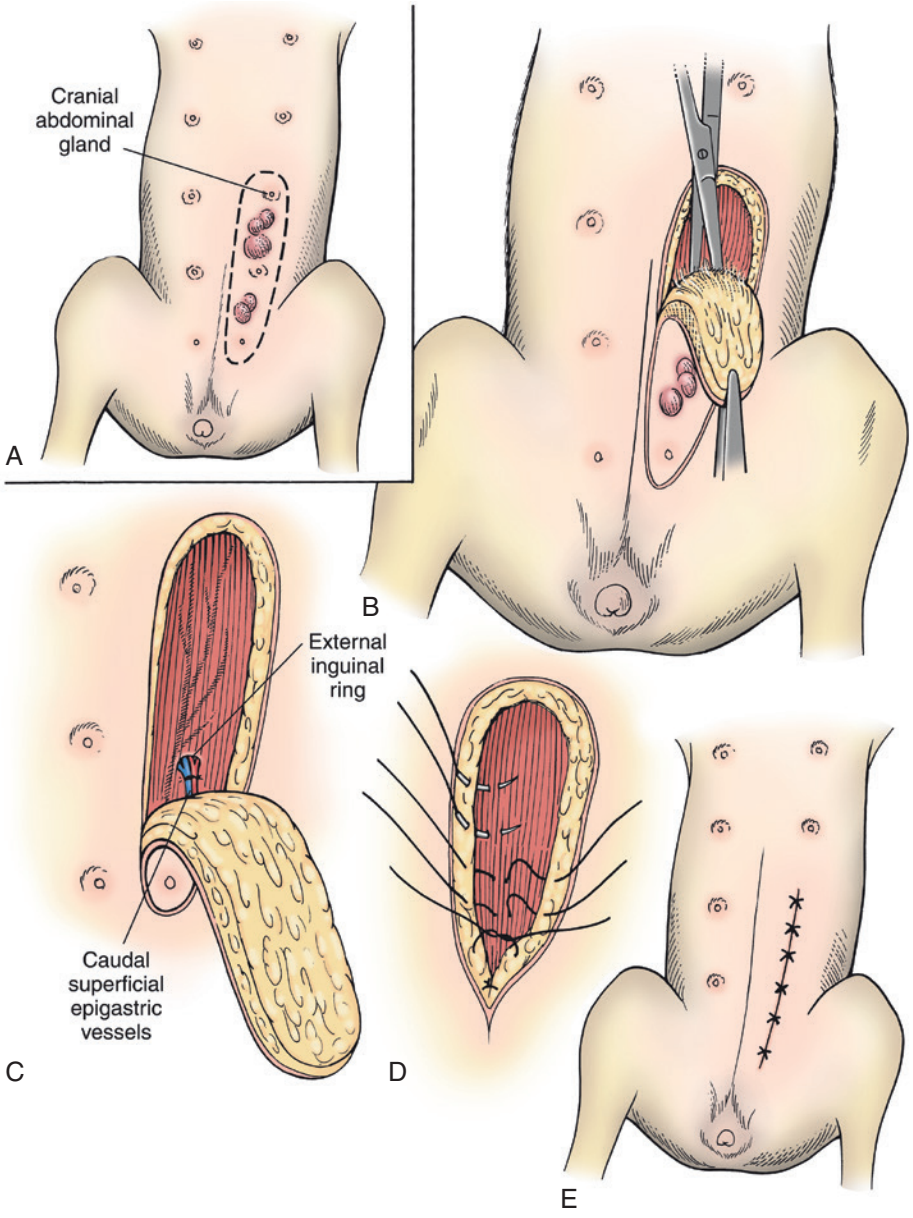


FIG. 26.22 (A) For caudal mastectomy, make an elliptical incision around the glands to be excised. (B) Incise subcutaneous tissue to expose the abdominal fascia. Elevate the cranial edge of the segment and separate subcutaneous tissue from the fascia by sliding sharp scissors along the abdominal fascia. (C) Ligate and divide the caudal superficial epigastric vessels near the inguinal ring. (D) Advance skin edges to the center of the defect with walking sutures and subcuticular sutures. (E) Appose skin edges with appositional sutures.

BOX 26.15 Significant Prognostic Factors for Mammary Tumors in Dogs

- Histologic type and immunohistochemical characteristics
- Degree of invasion
- Degree of nuclear differentiation and DNA ploidy
- Evidence of lymphoid cellular reactivity
- Tumor size
- Lymph node involvement
- Hormone receptor activity
- Presence of ulceration
- Fixation
- S-phase rate

time of surgery die or are euthanized for tumor-related problems within 1 to 2 years. Those with metastatic disease at the time of diagnosis have a shorter median survival (5 months vs. 28 months). Dogs with tumors less than 3 cm have a better prognosis (35% recurrence at 2 years; 22 months survival) than tumors greater than 3 cm in diameter (80% recurrence at 2 years; 14 months survival).

In cats, tumors less than 2 cm have less local recurrence than those greater than 2 to 3 cm. Cats with mammary carcinoma greater than 3 cm have a median survival of 6 months, whereas those with tumors less than 2 cm have a median survival of approximately 3 years. Queens with tumors less than or equal

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to 8 cm^3 in volume have the longest disease-free interval and median survival times (4.5 years after surgery). Queens with local disease rather than vascular or lymphatic tumor invasion had longer survival (22 months vs. 13 months, respectively). Likewise, a low mitotic index had a better prognosis than a high one (22 months vs. 12 months, respectively), and a low AgNOR index was more favorable than a high index (22 months vs. 14 months, respectively). The presence of multiple tumors does not affect the prognosis in dogs but may decrease survival in cats.

Adenocarcinomas confined to the duct epithelium may have a good prognosis after surgery. The prognosis worsens when neoplastic cells extend beyond the duct system and is poorest when neoplastic cells are found in blood or lymphatic vessels. Poorly differentiated adenocarcinomas have a 90% recurrence rate 2 years after surgery. The recurrence rate for moderately differentiated versus well-differentiated tumors is 68% versus 24%, respectively, 2 years after surgery. Median survival time for dogs with anaplastic carcinomas is 2.5 months compared with 21 months for adenocarcinoma, 16 months for solid carcinoma, and 14 months for other malignant tumors. Mammary gland sarcoma and inflammatory carcinoma have a very poor prognosis.

OHE at the time of tumor removal improved survival in some studies, but in others it did not affect survival or recurrence. A 2016 study found dogs with grade 2 mammary carcinoma, ER-positive tumors, or perioperative increased serum E2 concentration had a decreased hazard of relapse when undergoing OHE at the time of mammary tumor removal compared with dogs not receiving OHE.[5] Treatment modalities other than surgery may slow tumor progression, but few data are available to accurately predict their effectiveness.

UTERINE NEOPLASIA

DEFINITIONS

Leiomyoma and **leiomyosarcoma** are benign and malignant smooth muscle tumors, respectively, that may occur in the uterus. **Uterine adenocarcinomas** are malignant tumors of the uterine glands.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Uterine neoplasia is rare in the dog, with a reported incidence of 0.4% of all canine tumors, and it is even less common in cats (0.29%). Most tumors are incidental findings at necropsy or during abdominal exploration. Uterine tumors are more common in cats than other reproductive tract tumors. Tumors that may occur in the uterus are listed in Box 26.16. They may develop in the remnant of the uterine body following OHE and may have concurrent mammary tumors. Concurrent pathologic conditions may include cystic ovaries, cystic endometrial hyperplasia (CEH), and pyometra, suggesting a common hormonal influence.

Most uterine tumors in dogs (85%–90%) are leiomyomas that arise from the myometrium (Fig. 26.23). Leiomyomas are benign and generally noninvasive and slow growing. They may protrude into the uterine lumen on a stalk or cause the wall to bulge externally. German shepherd dogs have a syndrome characterized by multiple uterine leiomyomas, bilateral renal cystadenocarcinomas, and nodular dermatofibrosis. The most common malignant tumors of bitches and queens are leiomyosarcoma and endometrial adenocarcinoma, respectively. Leiomyosarcomas are grossly difficult to distinguish from leiomyomas. They are invasive tumors, usually slow to metastasize. Adenocarcinomas cause the endometrium to become thickened and nodular. The tumor may be solid or cystic, sessile, or polypoid, and may obliterate the uterine lumen. Multicentric adenocarcinoma has been reported. Metastasis is usually present at the time of diagnosis and may involve cerebrum, eyes, ovaries, adrenal glands, thyroid glands, lungs, liver, kidneys, bladder, intestines, pancreas, pericardium, myocardium, diaphragm, or regional lymph nodes.

BOX 26.16 Uterine Tumors

Leiomyoma
Fibroma
Leiomyosarcoma
Adenoma
Adenocarcinoma
Fibrosarcoma
Lipoma

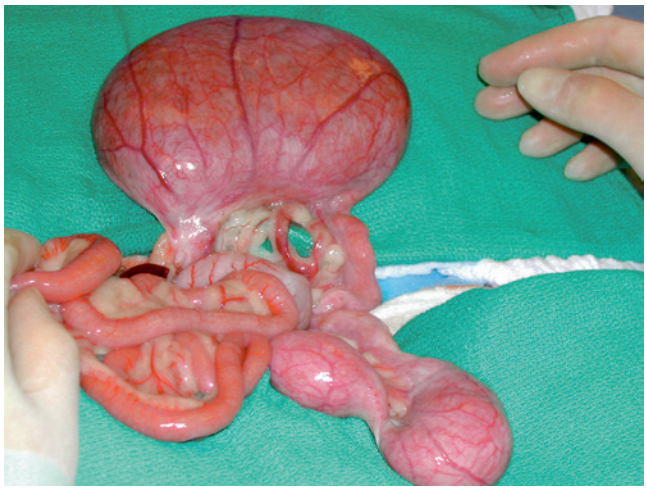


FIG. 26.23 Intraoperative image of a uterine leiomyoma and pyometra in a 6-year-old Yorkshire terrier.

DIAGNOSIS

Clinical Presentation

Signalment

No breed predilections have been reported. Most affected animals are middle aged or older.

History

Most uterine tumors cause no clinical signs unless they are large and compress the gastrointestinal or urinary tracts. Animals may have abnormal estrus cycles and/or a mucoid or hemorrhagic vaginal discharge because of tumor irritation and vascular erosion. Uterine tumors may obstruct the cervix and cause pyometra; therefore presenting clinical signs may include a purulent vaginal discharge, pyrexia, anorexia, vomiting, polydipsia, and/or polyuria. Tumor growth may compress the colon, bladder, or urethra,

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causing straining or obstruction. Other signs may include abdominal distention, dysuria, hematuria, dyspnea, and/or loss of consciousness.

Physical Examination Findings

Physical examination is often normal, although large masses may be palpated. A hemorrhagic vaginal discharge may be noted. Some uterine masses are palpable during rectal examination. Enlarged, asymmetric sublumbar lymph nodes may be palpated if the tumor has metastasized. Digital vaginal examination is usually normal. Animals with pyometra may be depressed, febrile, and pain sensitive on abdominal palpation, and they may have a purulent vaginal discharge (see p. 755).

Diagnostic Imaging

Radiography and ultrasonography may show a mass in the uterine area. The echogenicity of uterine masses is variable. Ultrasound-guided biopsies may provide information regarding tumor type. Abdominal radiographs, CT, and MRI images should be evaluated for evidence of lymph node enlargement or visceral metastasis, and thoracic radiographs (three views) should be evaluated for metastasis.

Vaginoscopy

Vaginoscopy may reveal abnormal discharge. Neoplastic cells may be identified from specimens obtained by endoscopic transcervical uterine cannulation if the tumor invades or involves the endometrium.

Laboratory Findings

Hematologic and serum biochemical profile results are nonspecific. The patient may be anemic if a chronic hemorrhagic discharge or paraneoplastic syndrome is present. Neoplastic cells are rarely identified on vaginal cytology. Definitive diagnosis requires histopathology.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for uterine masses include intestinal foreign bodies, tumor or fungal lesions, urinary tract masses, or lymph node enlargement secondary to neoplasia or inflammation. Differential diagnoses for vaginal discharge include estrus, parturition, abortion, placentitis, normal lochia, vaginitis, metritis, pyometra, placental subinvolution, mucometra, uterine torsion, or trauma.

MEDICAL MANAGEMENT

The effectiveness of chemotherapy and radiation therapy on uterine masses is unknown.

SURGICAL TREATMENT

OHE is the treatment of choice for uterine tumors.

Preoperative Management

Hydration, electrolyte, and acid-base abnormalities should be corrected before surgery. Patients with elevated blood urea nitrogen or creatinine concentrations should be given diuretics before surgery. If pyometra is present, antibiotic therapy should be initiated (see Box 26.3). Mature patients should fast for 12 to 18 hours before anesthetic induction.

Anesthesia

A variety of anesthetic protocols can be used in animals with uterine tumors if they are not debilitated and do not have concurrent pyometra. Anesthetic recommendations for animals with pyometra are provided in Table 26.5.

Surgical Anatomy

Surgical anatomy of the reproductive tract is discussed on p. 725.

Positioning

Patients are positioned in dorsal recumbency for a ventral midline celiotomy. The entire ventral abdomen and caudal thorax should be clipped and prepared for aseptic surgery.

SURGICAL TECHNIQUE

Perform a ventral midline celiotomy. Explore the abdomen for evidence of metastasis or other abnormalities. Take a biopsy or excise abnormal structures. Perform an OHE (see p. 728), removing the cervix if it is within 1 to 2 cm of the tumor. Culture the uterus if metritis or pyometra is suspected.

POSTOPERATIVE CARE AND ASSESSMENT

Fluid therapy should be continued if the patient was dehydrated or azotemic, and postoperative analgesics (see Chapter 13) should be given as needed (see Tables 13.2 and 13.3 for dosages of analgesics). Antibiotics are unnecessary unless a uterine infection was identified. Thoracic and abdominal radiographs should be evaluated periodically (e.g., 1–2 months, 6 months) if a malignant tumor was present. Complications of OHE are discussed on p. 744. The tumor may recur locally or metastasize.

PROGNOSIS

The prognosis for asymptomatic benign tumors without surgery is good unless the mass enlarges and impinges on gastrointestinal or urinary tracts. The prognosis following OHE is excellent for benign tumors and good for malignant tumors without evidence of metastasis or local infiltration. The prognosis for uterine adenocarcinomas is guarded because of its propensity to metastasize before diagnosis. The effectiveness of other treatment modalities for uterine tumors is unknown.

PYOMETRA

DEFINITIONS

Pyometra is an accumulation of purulent material within the uterus. Pyometra is sometimes referred to as *cystic endometrial hyperplasia-pyometra complex*. Uterine distention with sterile fluid is referred to as **hydrometra** (watery secretions), **mucometra** (mucoid secretions), or **hematometra** (bloody secretions). A **stump pyometra** is accumulation of purulent material in the vestige of the uterus that remains after OHE.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Pyometra is a potentially life-threatening condition associated with CEH. CEH and pyometra both develop during diestrus.

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TABLE 26.5 Anesthetic Considerations in the Septic Patient With Pyometra

Preoperative Considerations

Associated conditions	• Dehydration • Electrolyte abnormalities • Hypotension • Abnormal blood glucose • Anemia • Dysrhythmias • Often older patients with comorbidities • Adrenal suppression may be present in the critically ill patient
Bloodwork	• HCT • Electrolytes • BUN • Cr • TP • Lactate • Blood glucose, often serial glucose checks • Urinalysis
Physical exam	• May be dehydrated, tachycardic or bradycardic, hypotensive, vomiting, or hypothermic • Painful abdomen may be present • +/– Vaginal discharge
Other diagnostics	• Blood pressure • ECG • SpO_2 • Radiographs • +/– Ultrasound

Premedications	• Rehydrate for 4–6 h if possible; if closed pyometra, may have to give more rapid boluses to expedite time to surgery
	• Correct electrolyte and glucose abnormalities
	• Avoid sedatives in depressed patients
	• Avoid alpha-2 agonists and acepromazine
	• If patient is anxious, give:
	  • Midazolam (0.1–0.2 mg/kg IV, IM), *or*
	  • Diazepam (0.1–0.2 mg/kg IV)
	• If patient is not depressed, then give:
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM in dogs; 0.05–0.1 mg/kg IV, IM in cats), *or*
	  • Oxymorphone (0.1–0.2 mg/kg IV, IM), *or*
	  • Morphine[b] (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM) *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM)

Intraoperative Considerations

Induction	Preoxygenate 3–5 minutes
	• If dehydrated, give:
	  • Etomidate (0.5–1.5 mg/kg IV); (administer after giving a benzodiazepine), when possible avoid its use in critically ill patients, *or*
	  • Propofol (1–4 mg/kg IV) slowly, *or*
	  • Alfaxalone (2–3 mg/kg IV)
	• If hydrated, give:
	  • Propofol (2–6 mg/kg IV), *or*
	  • Alfaxalone (2–5 mg/kg IV)

Maintenance	• Isoflurane or sevoflurane, *plus*
	  • Fentanyl (2–10 µg/kg IV PRN in dogs; 1–4 µg/kg IV PRN in cats) for short-term pain relief, *plus*
	  • Fentanyl CRI (1–5 µg/kg IV loading dose, then 2–30 µg/kg/h IV), *or*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV PRN), *plus*
	  • Ketamine (low dose) (0.5–1 mg/kg IV), *or*
	  • Ketamine CRI (0.5 mg/kg IV loading dose, then 10 µg/kg/min IV)
	• For hypotension (to keep MAP 60–80 mm Hg) give phenylephrine, ephedrine, norepinephrine, or dopamine as needed

Fluid needs	• 10–20 mL/kg/h plus 3× EBL; higher rates of fluids are necessary if dehydration not corrected preoperatively • Consider colloids if persistent hypotension
Monitoring	• Blood pressure • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature • U/O

Continued

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TABLE 26.5 Anesthetic Considerations in the Septic Patient With Pyometra—cont'd

Blocks	Epidural: • Morphine (0.1 mg/kg preservative free) *or* • Buprenorphine (0.003–0.005 mg/kg diluted in saline) NOTE: Avoid local anesthetics for spinals and epidurals in hypotensive patients. Incisional: • Lidocaine (<5 mg/kg in dogs, 2–4 mg/kg in cats), *or* • Bupivacaine (<2 mg/kg)
Postoperative Considerations	
Analgesia	• Fentanyl CRI (1–10 µg/kg IV loading dose, then 2–20 µg/kg/h IV), *or* • Morphine[b] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.05–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h in cats) if no hypotension, *or* • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM, SC q4–8h or 0.01–0.02 mg/kg OTM q6–12h in cats), *or* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats), *or* • Oxymorphone (0.1–0.2 mg/kg IV), *or* • Hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs), *plus* • +/– Ketamine CRI (2 µg/kg/min IV. If no previous loading dose, give 0.5 mg/kg IV prior to CRI) NOTE: Avoid NSAIDs
Monitoring	• SpO_2 • Blood pressure • HR • Respiratory rate • Temperature • U/O • ECG if electrolyte abnormalities
Bloodwork	• HCT if significant blood loss • Repeat abnormal preoperative bloodwork • Serial blood glucose checks if necessary
Estimated pain score	Moderate to severe

[a]Monitor for hyperthermia in cats.
[b]Give slowly to prevent histamine release.
[c]Buprenorphine is a better analgesic than morphine in cats.
BUN, Blood urea nitrogen; *Cr,* creatinine; *CRI,* constant rate infusion; *EBL,* estimated blood loss; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *MAP,* mean arterial pressure; *NSAID,* nonsteroidal antiinflammatory drug; *OTM,* oral transmucosal; *PRN,* as needed; *SC,* subcutaneous; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein; *U/O,* urine output.

Occasionally the diagnosis is delayed and unrecognized until anestrus. In dogs, the diestrual period of a normal, nongravid bitch lasts approximately 70 days. The uterus is influenced by progesterone produced by ovarian corpora lutea. Progesterone stimulates the growth and secretory activity of the endometrial glands and reduces myometrial activity. CEH is an abnormal uterine response that develops during diestrus (luteal phase of cycle) when there is high or prolonged ovarian production of progesterone or exogenously administered progesterone. Excessive progesterone influence or an exaggerated progesterone response causes the uterine glandular tissue to become cystic, edematous, thickened, and infiltrated by lymphocytes and plasma cells. Fluid accumulates in endometrial glands and the uterine lumen with CEH. Uterine drainage is hindered by progesterone inhibition of myometrial contractility. This abnormal uterine environment allows bacterial colonization to cause pyometra. Administration of estrogen increases the risk of pyometra during diestrus. The risk of an intact bitch developing pyometra before 10 years of age is nearly 25%.

Estrogen increases the number of uterine progesterone receptors, which may explain the increased incidence of pyometra after estrogens are administered to prevent pregnancy. Uterine tumors sometimes obstruct the outflow of uterine secretions and may contribute to development of pyometra. Feline pyometra is less frequent than canine pyometra because development of luteal tissue requires copulation or artificially induced ovulation; however, cats treated with progestins for skin disease have an increased incidence of pyometra.

BOX 26.17 Organisms Most Commonly Cultured From Dogs With Pyometra

Escherichia coli
Staphylococcus aureus[a]
Streptococcus spp.[a]
Pseudomonas spp.[a]
Proteus spp.[a]
Pasteurella spp.
Klebsiella spp.
Haemophilus spp.
Serratia spp.
Moraxella spp.

[a]Also found as normal vaginal flora.

Infection causes the morbidity and mortality associated with pyometra. Leukocyte response to bacteria is inhibited in a progesterone-primed uterus. *E. coli* is the most common organism identified in canine and feline pyometra, but mixed infection is also common. *E. coli* has an affinity for the endometrium and myometrium. Bacterial invasion is thought to be opportunistic because the most commonly isolated organisms are also normal vaginal flora (see Boxes 26.2 and 26.17). Bacterial virulence is associated with the serotype, presence of K antigen, and cytotoxin necrotizing factor. Ascension of fecal flora into the uterus is the primary route of infection based on biochemical fingerprinting of bacteria. Other bacterial sources include the urinary tract and transient bacteremia. Vaginal discharge occurs if the cervix is

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BOX 26.18 Potential Abnormalities in Animals With Pyometra

Hypoglycemia
Renal dysfunction
Hepatic dysfunction
Anemia
Cardiac arrhythmias
Coagulation abnormalities

BOX 26.19 Criteria for Diagnosis of Systemic Inflammatory Response Syndrome[a]

Heart rate: >160 beats/min
Temperature: >103.5°F (40°C) or <100°F (38°C)
Respiration: >20 breaths/min or partial pressure of CO_2 <32 mm Hg
White blood cell count: >12,000/µL, <4000/µL, or >10% band neutrophils

[a]Diagnosis is made if two of the four criteria are present.

patent or "open." A closed cervix prevents discharge of infected fluid and causes more serious disease. Animals may become dehydrated and toxic. Septicemia and endotoxemia can develop if pyometra is untreated. Compression or overdistention of the uterus may allow infected uterine contents to leak out of the oviducts and cause peritonitis. Torsion of the distended uterus may also occur.

Concurrent abnormalities in animals with pyometra may include hypoglycemia, renal and hepatic dysfunction, anemia, and/or cardiac abnormalities (Box 26.18). Pyometra is often associated with systemic inflammatory response syndrome (SIRS) caused by production and release of inflammatory mediators with systemic effects (Box 26.19). Hypoglycemia is common in canine pyometra. Sepsis and SIRS deplete glycogen stores, increase peripheral glucose use, and decrease gluconeogenesis. Transient hyperglycemia occasionally occurs because of excessive catecholamine and glucagon release. Progesterone-induced growth hormone production may cause persistent hyperglycemia and glucosuria. Judicious insulin treatment may be required in patients with persistent hyperglycemia (i.e., greater than 300 mg/dL) after appropriate medical and surgical treatment.

Patients with pyometra may have prerenal azotemia, primary glomerular disease, reduced tubular concentrating ability, tubular interstitial disease, reduced glomerular filtration, and/or concurrent renal disease unrelated to the pyometra. Prerenal azotemia is due to poor perfusion, dehydration, and shock. Primary glomerular disease occurs secondary to immune-complex glomerulonephritis. Bacterial antigens also interfere with renal tubular concentrating ability. Once the bacterial antigen is removed, these changes typically resolve and normal renal function returns. Reduced tubular concentrating ability is from inhibition of antidiuretic hormone at the level of the renal tubule by bacterial endotoxins, obligatory solute load from decreased glomerular filtration rate, and other unknown factors. Normal tubular concentrating ability usually returns 2 to 8 weeks after OHE. Hepatocellular injury may be secondary to intrahepatic cholestasis and retention of bile pigments, toxicity from sepsis and endotoxemia, and/or poor perfusion.

Anemia may be caused by chronic inflammation suppressing erythropoiesis, loss of red cells into the uterine lumen, hemodilution, or surgical blood loss. Nonregenerative anemia should spontaneously resolve a few weeks after OHE. Coagulation deficits infrequently occur secondary to concurrent metabolic imbalances. Cardiac arrhythmias result from toxic effects of pyometra, shock, acidosis, and electrolyte imbalance.

DIAGNOSIS

Clinical Presentation

Signalment

Pyometra affects intact dogs more commonly than cats. Some breeds thought to be predisposed to pyometra include rottweilers, golden retrievers, collies, Saint Bernards, chow chows, miniature schnauzers, Irish terriers, Leonbergers, Airedale terriers, Cavalier King Charles spaniels, Bernese mountain dogs, and English cocker spaniels; however, other studies find no breed predisposition. Domestic shorthair and Siamese cats are affected more commonly than other breeds. Pyometra generally occurs in older (0.75–14 years; median, 7.9 years) intact bitches and queens; however, it may occur in younger animals that have been given exogenous estrogen (dogs) or progestins (cats). Nulliparous bitches are at moderately greater risk for pyometra than are primiparous and multiparous bitches, but this finding is not consistent across at-risk breeds.

History

Pyometra usually occurs several weeks (1–4 weeks in cats, 4–8 weeks in dogs [mean, 5.7 weeks]) after estrus, or following mismating injections or exogenous administration of estrogens or progestins. The animal may have a purulent or bloody vaginal discharge. Others have obvious abdominal distention, fever, partial to complete anorexia, lethargy, polyuria, polydipsia, vomiting, diarrhea, and/or weight loss. Animals with closed pyometra more commonly have vomiting and diarrhea and are generally very ill at presentation.

Physical Examination Findings

A purulent blood-tinged vaginal discharge may occur if the cervix is open. Uterine enlargement may be detected on abdominal palpation. Dehydration is frequent. Animals with endotoxemia or septicemia may be in shock, hypothermic, and/or moribund. Fever is infrequent.

Diagnostic Imaging

A fluid-filled uterus should be detected on abdominal radiographs (Fig. 26.24) and/or ultrasonography (Fig. 26.25). The enlarged uterus is located in the caudal abdomen and may displace intestines cranially and dorsally. Open pyometra or uterine rupture may cause enough drainage so that the uterus is not radiographically detected. Displacing the intestines with a wooden spoon or abdominal bandage may improve uterine visualization but should be performed with caution because a significantly distended uterus may rupture. Signs of uterine rupture and peritonitis (i.e., poor visceral detail) should be noted.

Abdominal ultrasound is the preferred method for imaging as it provides more detailed information about the uterine tissues and fluid within the lumen. In addition, uterine blood flow velocities can be used to help differentiate between CEH and CEH with pyometra.[6]

It is important to rule out pregnancy. Radiographic confirmation of pyometra may not be possible until 41 to 43 days after ovulation. Radiographically, fetal calcification can be identified after approximately 45 days of gestation. Ultrasonography can

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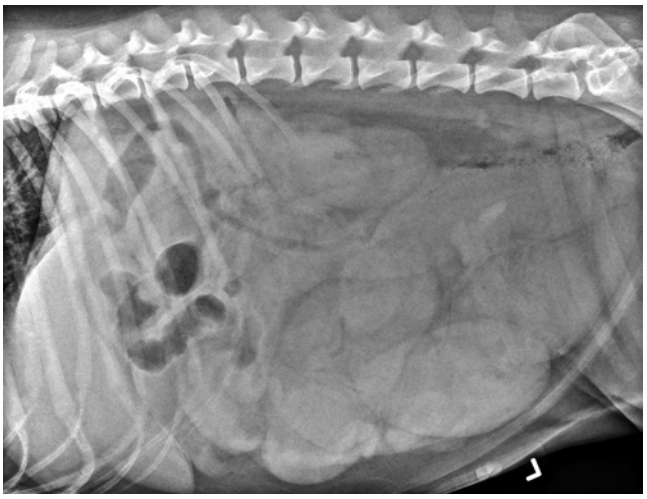


FIG. 26.24 Lateral abdominal radiograph of a dog with pyometra. Note the enlarged uterus in the caudoventral abdomen displacing viscera cranially and dorsally.

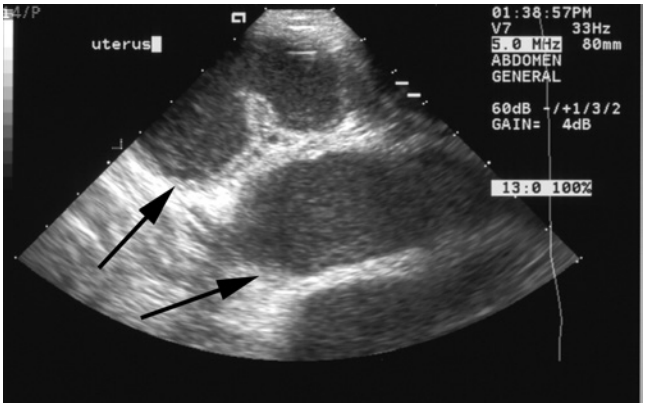


FIG. 26.25 Parasagittal ultrasound image of the caudal abdomen of a dog with pyometra. Note the enlarged, fluid-filled uterus *(arrows),* which is convoluted. The fluid within the lumen of the uterus is mildly echogenic.

identify fetal structures (see Table 26.2), assess fetal viability, identify uterine fluid, and determine uterine wall thickness and irregularities.

Laboratory Findings

Metabolic clinicopathologic abnormalities may occur. The most common hemogram findings are neutrophilia with a left shift, monocytosis, and white blood cell toxicity. White blood cell numbers usually exceed 30,000/µL with closed pyometras and may be as high as 100,000 to 200,000/µL. However, normal numbers of white blood cells are often seen with open pyometras. Leukopenia may indicate overwhelming infection and septicemia or uterine sequestration of neutrophils, and has been shown to be associated with an 18-fold increase in the likelihood of peritonitis in dogs with pyometra.[7] The high percentage of bands in most pyometras helps differentiate them from CEH with mucometra. Mild normocytic, normochromic, nonregenerative anemia or nonregenerative, microcytic, hypochromic anemia may occur. Clotting abnormalities and disseminated intravascular coagulation may occur in severely affected patients.

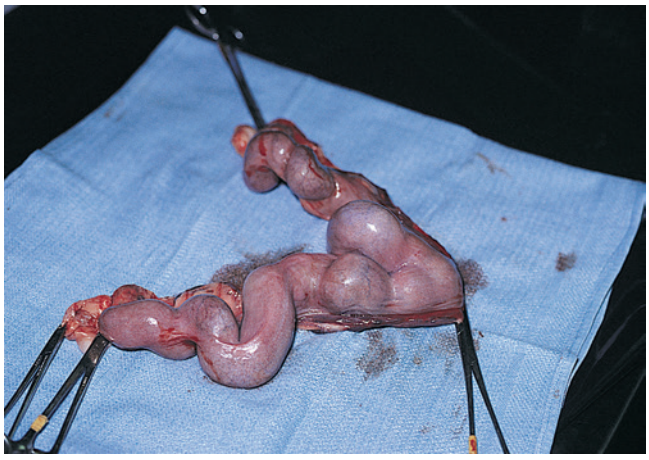


FIG. 26.26 An enlarged, friable uterus in an animal with pyometra. Compare this with the enlarged uterus in an animal with mucometra in Fig. 26.27. The two cannot be differentiated radiographically.

NOTE Do not rule out pyometra in animals with leukopenia or normal white cell counts. Sequestration of neutrophils in the enlarged uterus may cause neutropenia despite severe infection.

Common biochemical abnormalities include hyperproteinemia, hyperglobulinemia, and azotemia. Hyponatremia and hyperkalemia may occur with severe vomiting or diarrhea, mimicking hypoadrenocorticism. Less common abnormalities include hyperbilirubinemia and increased alanine aminotransferase and alkaline phosphatase activities (secondary to toxemia-induced hepatocellular damage and cholestasis). Hyperglycemia or hypoglycemia may be associated with concurrent diabetes or sepsis. Although C-reactive protein elevations help differentiate pyometra from CEH with mucometra, the test has not been readily available. However, a commercially available automated method for measuring canine C-reactive protein has recently been validated.[8] Blood lactate levels do not appear to correlate with the severity of clinical signs. Urinalysis may reveal isosthenuria, proteinuria, and/or bacteriuria. To prevent uterine puncture and abdominal contamination, cystocentesis should not be performed if pyometra is suspected. Open pyometras (and some closed pyometras) have a septic vaginal exudate (i.e., neutrophils, many degenerate, with bacteria). Aerobic and anaerobic bacterial culture and susceptibility are essential for selection of appropriate antibiotics.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include mucometra, hydrometra, hematometra, hydrocolpos, pyovagina, pregnancy, metritis, placentitis, uterine torsion, and peritonitis (Figs. 26.26 and 26.27). Because of its high percentage of band neutrophils, elevated C-reactive protein, elevated alkaline phosphatase, and evidence of clinical illness, pyometra is more likely than mucometra.

MEDICAL MANAGEMENT

Medical evacuation of the uterus with prostaglandin therapy (prostaglandin $F_{2\alpha}$ [$PGF_{2\alpha}$]) is inappropriate for critically ill patients because evacuation is neither immediate nor complete.

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FIG. 26.27 Intraoperative appearance of the uterus in an animal with mucometra.

BOX 26.20 Medical Therapy for Pyometra[a]

Antibiotics for 2–3 weeks (see Box 26.21)

Prostaglandin $F_{2\alpha}$
0.1 mg/kg SC once on day 1, 0.2 mg/kg SC once on day 2, 0.25 mg/kg SC once daily on days 3–7, *or*
0.15 mg/kg administered intravaginally, raise hindquarters for 3–5 min, *or*

Aglepristone (Alizine, Alizin)
10 mg/kg SC on days 0, 2, 5, 8, *or*
10 mg/kg SC on days 1, 2, and 7

With Cloprostenol (Estrumate)
1 µg/kg SC on days 3 and 7, *or*

Cloprostenol (Estrumate)
1 µg/kg SC q24h for 7–14 d or every third day

With Cabergoline (Galastop)
5 µg/kg PO q24h for 7–10 d

[a]Dosages and efficacy of treatment are not definitively established. Breeding should be attempted during the next estrus.
SC, Subcutaneous.

Multiple protocols have been described to medically address pyometra. Medical therapy with antibiotics for 2 to 3 weeks and with $PGF2_{\alpha}$ or preferably aglepristone (antiprogestin) alone or combined with cloprostenol (synthetic prostaglandin), or combination of cabergoline (dopamine agonist) with cloprostenol (Box 26.20) are safe and effective methods for metabolically stable, valuable, breeding animals. Most recently, a modified protocol of aglepristone administration (administration at 0, 2, 5, and 8 days) had 100% success rate in 47 dogs, with no recurrence in a 24-month follow-up period.[9] Medical therapy is most appropriate for animals with an open pyometra. More than one series of prostaglandin injections may be necessary. Owners must be informed that $PGF_{2\alpha}$ therapy is not approved for use in dogs and cats, and serious complications (e.g., uterine rupture or leakage of intraluminal contents into the abdomen and sepsis) are possible. Short-term side effects (30–60 minutes) include panting, salivation, emesis, defecation, urination, mydriasis, nesting, tenesmus, lordosis, vocalization, and intensive grooming. High doses of prostaglandin may cause ataxia, collapse, hypovolemic shock, respiratory distress, or death. $PGF_{2\alpha}$ therapy may cause reduced fertility. Vulvar discharge increases and clinical signs begin to improve within 24 to 48 hours of initial aglepristone injection. Including an antilipopolysaccharide to reduce endotoxins may be beneficial. Mating should occur during the next estrus cycle. Expect pyometra to recur in 20% of animals during subsequent estrus cycles.

BOX 26.21 Selected Antibiotics of Use in Animals With Pyometra

Cefazolin (Ancef, Kefzol)
22 mg/kg IV, SC, IM q8h

Cefoxitin (Mefoxin)
Dogs: 30 mg/kg IV q6–8h
Cats: 22–33 mg/kg IV, IM q8h

Amoxicillin Plus Clavulanate (Clavamox)
Dogs: 12.5–25 mg/kg PO q12h
Cats: 62.5 mg/cat PO q12h

Ampicillin Plus Sulbactam (Unasyn)
50 mg/kg IV q8h

Ampicillin
22 mg/kg IV, IM, SC q6–8h

Enrofloxacin (Baytril)
Dogs: 7–20 mg/kg PO, IV q24h (dilute and give slowly over 30 min if given IV)
Cats: 5 mg/kg PO q24h

IM, Intramuscular; *IV,* intravenous; *PO,* orally, *SC,* subcutaneous.

SURGICAL TREATMENT

Treatment (OHE) should not be delayed more than is absolutely necessary. Morbidity and mortality are associated with concurrent metabolic abnormalities and organ dysfunction (see previous discussion). Surgical drainage of the uterus without OHE is not recommended but has been successful in a few cases. The corpus lutea are removed and each horn lavaged and suctioned. Indwelling drains are placed through the cervix to allow daily lavage with diluted antiseptics. Laparoscopic-assisted OHE for pyometra has been shown to be feasible in selected cases.[10,11]

Preoperative Management

Surgery should not be delayed more than a few hours while medical therapy (i.e., fluid therapy) is instituted, especially in patients with closed pyometra. Urine output, glucose, and arrhythmias should be monitored preoperatively. Hydration, electrolyte, and acid-base imbalances should be corrected before surgery, if possible (the prognosis is improved when azotemia is corrected before surgery). A broad-spectrum antibiotic effective against *E. coli* (e.g., cefazolin, cefoxitin, enrofloxacin, ampicillin plus sulbactam, and ticarcillin plus clavulanate; Box 26.21) should be given IV while awaiting antibiotic susceptibility results. Aminoglycosides are nephrotoxic and not recommended because of the prevalence of renal dysfunction with pyometra. Fluid input and urine output should be monitored to help assess renal

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BOX 26.22 Diuretic Therapy

Mannitol[a]
0.25–1 g/kg IV over 15 min

Furosemide (Lasix)
2–4 mg/kg IV, PO, SC q8–12h or as CRI (load with 0.66 mg/kg bolus, then give 0.66 mg/kg/h for 4–5 h; alternatively, can estimate the IV or PO dose to be given over the course of the next 24 h and then give that amount as a CRI over the next 24 h). Be sure patient is volume replenished before administering.

[a]This is a potentially dangerous drug because it is not metabolized if it is not excreted (consult an internist or medical text before using).
CRI, Constant-rate infusion; *IV,* intravenous; *PO,* orally; *SC,* subcutaneous.

function. Diuretics (e.g., furosemide [Box 26.22], 2–4 mg/kg IV, intramuscular [IM], or subcutaneous or 20% dextrose IV) may be administered in volume-overloaded patients with reduced urine production. Administration of antiarrhythmics may occasionally be necessary.

Anesthesia

Anesthetic protocols vary greatly depending on patient status. Animals that are systemically ill need to be closely monitored during anesthesia (see Table 26.5). For a discussion of anesthetic recommendations in septic patients, see pp. 515 and 532. Anesthetic management of stable animals undergoing abdominal surgery is found in Table 19.1.

Positioning

Position the patient in dorsal recumbency for a ventral midline celiotomy. The entire ventral abdomen should be clipped and prepared for aseptic surgery.

Surgical Anatomy

Surgical anatomy of the reproductive tract is described on p. 725.

SURGICAL TECHNIQUE

Expose the abdomen through a ventral midline incision beginning 2 to 3 cm caudal to the xiphoid and extending to the pubis. Explore the abdomen and locate the distended uterus. Observe for evidence of peritonitis (e.g., serosal inflammation, increased abdominal fluid, petechiation). Obtain abdominal fluid for culture, evacuate the urinary bladder by cystocentesis, and collect a urine specimen for culture and analysis if not previously submitted. Carefully exteriorize the uterus without applying pressure or excessive traction. A fluid-filled uterus is often friable; therefore lift rather than pull the uterus out of the abdomen.

NOTE Do not use a spay hook to locate and exteriorize the uterus because it may tear. Do not correct uterine torsion because this will release bacteria and toxins.

Isolate the uterus from the abdomen with laparotomy pads or sterile towels. Place clamps and ligatures as previously described for OHE except that the cervix may be resected in addition to ovaries, uterine horns, and uterine body. Ligate the pedicles and enlarged vessels within the broad ligament with absorbable monofilament suture material (2-0 or 3-0 polydioxanone [PDS], polyglyconate [Maxon], poliglecaprone 25 [Monocryl], or glycomer 631 [Biosyn]), and transect at the junction of the cervix and vagina. Alternatively, use a bipolar vessel-sealing device on ovarian pedicles and broad ligament (Fig. 26.28). Thoroughly lavage the vaginal stump, but do not oversew the cut edge. Culture the contents of the uterus without contaminating the surgical field. Remove laparotomy pads and replace contaminated instruments, gloves, and drapes. Lavage the abdomen and close the incision routinely unless peritonitis is present (see p. 534). Submit the tract for pathologic evaluation.

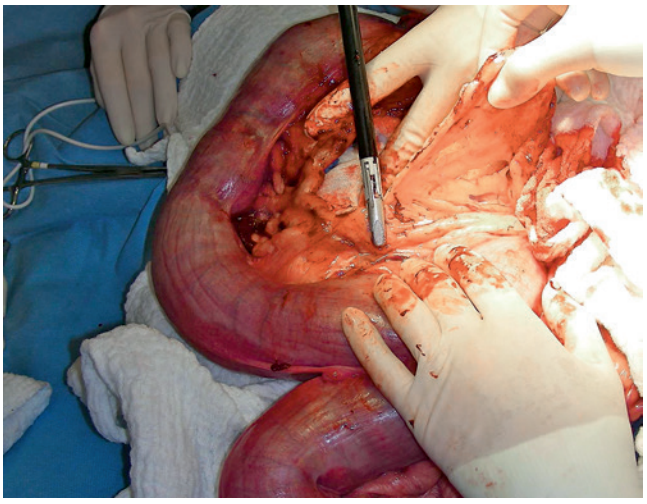


FIG. 26.28 Intraoperative image of an enlarged uterus in a 7-year-old golden retriever with pyometra. Ovarian pedicles and vessels within the broad ligament were ligated using a bipolar vessel-sealing device (LigaSure; Medtronic).

POSTOPERATIVE CARE AND ASSESSMENT

Give analgesics as necessary (see Chapter 13; for dosages of opioids, see Tables 13.2 and 13.3). These patients should be monitored closely for 24 to 48 hours for sepsis and shock, dehydration, and electrolyte/acid-base imbalances. Severe hypoproteinemia or anemia may require plasma or blood transfusions, respectively. Fluid therapy should be continued postoperatively until the animal is eating and drinking normally. Antibiotic therapy based on culture and sensitivity results should be continued for 10 to 14 days. Diuretics (see Box 26.22) may be given postoperatively if urine production is reduced. Evidence of abdominal discomfort, elevated temperature, or pain suggests peritonitis.

COMPLICATIONS

Complications associated with elective OHE may also occur following OHE for pyometra (see p. 744). Septicemia, endotoxemia, peritonitis (due to ruptured uterus), and cervical or stump pyometra may occur. Stump pyometra may be associated with residual ovarian tissue. In these cases, the remaining stump should be excised and residual ovarian tissue removed. Other complications include urinary tract infection, surgical wound infection, cardiac arrhythmias, multiple organ injury, uveitis, and thromboembolic disease.

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PROGNOSIS

Death usually occurs without surgical or medical therapy. Total mortality has been reported to be 10%, including patients that were euthanized; in this same study, there was a 1% mortality rate in dogs that went to surgery, lower than historical mortality rates of 5% to 8%.[7] A few animals spontaneously recover following corpus luteum regression and uterine drainage, but pyometra recurrence at subsequent diestrus is common. Pyometra persists or recurs after medical therapy in approximately 20% of dogs. However, 40% to 74% of bitches and 81% of queens produce at least one normal litter after prostaglandin therapy.

Prognosis following surgery is good if abdominal contamination is avoided, shock and sepsis are controlled, and renal damage reversed by fluid therapy and bacterial antigen elimination. Death may occur when metabolic abnormalities are severe and unresponsive to appropriate therapy.

VAGINAL PROLAPSE/HYPERPLASIA/TUMOR

DEFINITION

Vaginal prolapse/hyperplasia occurs during estrus or proestrus as a result of edematous enlargement of vaginal tissue. **Vaginal prolapse** involves the 360-degree protrusion of mucosa, whereas **vaginal hyperplasia** may originate from a stalk of mucosa on the floor of the vagina, both usually cranial to the urethral papilla. *Vaginal hypertrophy, vaginal edema, vaginal fold prolapse, estral eversion,* and *estral hypertrophy* have also been used to describe this condition.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Vaginal hyperplasia/hypertrophy occurs uncommonly, usually during proestrus and estrus. The mucosa is not truly hyperplastic but enlarges because of edema. Normal estrogenic stimulation causes vaginal mucosa to become hyperemic, edematous, and keratinized. These normal effects are accentuated with vaginal prolapse/hyperplasia, causing edematous mucosa to evert during proestrus and estrus and occasionally at the end of diestrus or near parturition. Prolapse may occur with hyperestrogenism or weakness of vaginal connective tissue. The amount of edema and eversion is extremely variable. Severe edema causes vaginal tissue to protrude from the vulva. Although the protruding mass may be large, the origin of the mass is usually small (approximately 1 cm) and located on the vaginal floor cranial to the urethral orifice. The width of the mass varies from stalklike to involving the entire vaginal floor. Prolapsing tissue promotes straining, which further increases the amount of prolapsed tissue. The edematous tissue mechanically obstructs and interferes with normal breeding. Tissue protruding from between the vulvar lips is often traumatized by abrasion, licking, or drying. Trauma results in ulceration and bleeding. The mass may compress surrounding structures, causing stranguria, hematuria, or tenesmus. Prepartum bitches may concurrently prolapse their urinary bladder. Although edema resolves spontaneously when the follicular phase of the cycle and ovarian production of estrogen have elapsed, prolapse may recur with each succeeding estrus cycle.

NOTE Vaginal prolapse/hyperplasia appears to be familial. Affected animals should not be bred.

DIAGNOSIS

Clinical Presentation

Signalment

Although rare, vaginal prolapse/hyperplasia is most common in large-breed dogs. It most commonly occurs in young bitches (≤2 years) during one of their first three estrus cycles. Vaginal prolapse/hyperplasia is extremely rare in cats.

History

Protrusion of a mass from the vulva, vulvar discharge, or bleeding is typical. Bitches refuse to allow intromission during breeding or have signs referable to fecal or urinary difficulties. The history should indicate if the animal is in estrus, proestrus, or gravid (rarely). Other signs of vaginal disease include frequent perineal licking, pollakiuria, dysuria, and perineal enlargement and swelling.

Physical Examination Findings

A mass may be seen protruding between the vulvar lips, or the perineum may bulge. Acute prolapse and nonprotruding prolapses are characterized by a glistening, edematous, pale pink mucosal surface. Chronic prolapses appear leathery (dry and dull), corrugated, and sometimes ulcerated or fissured. The mass should be examined carefully to determine origin, size at the base, locations of the vaginal lumen and urethral opening, and the extent of tissue damage. Vaginal palpation should identify a mass arising from the ventral vaginal floor. Vaginal areas other than those just cranial to the urethral orifice should feel normal. Vaginal cancers can cause severe pain, making vaginal examination impossible without chemical restraint.

Diagnostic Imaging

Radiographs are unnecessary unless neoplasia or visceral herniation is suspected. Concurrent herniation of the urinary bladder or intestines into the prolapsed tissue may require positive contrast studies for confirmation.

Laboratory Findings

Vaginal cytology should confirm estrogen stimulation (i.e., red blood cells in the absence of cornified vaginal epithelial cells). Aspiration cytology helps differentiate prolapse from neoplasia.

DIFFERENTIAL DIAGNOSIS

Uterine prolapse (see p. 761) and vaginal tumors are the most difficult to differentiate from vaginal prolapse/hyperplasia (Fig. 26.29; see also Fig. 26.15). The most common types of vulvar-vaginal tumors are fibroleiomyoma, lipoma, leiomyosarcoma, squamous cell carcinoma, and transmissible venereal tumor (TVT). Most vulvar-vaginal tumors occur in old (≥10 years), intact females. Benign vulvar-vaginal tumors are most common and respond to local excision and OHE. Fibroleiomyoma is the most common benign tumor. Fibroleiomyomas originate around the urethral papilla and are usually pedunculated, smooth, firm, and pale. The most common malignant tumors are TVTs. These tumors tend to be broad-based, irregular, friable, and bleed easily. Malignant vulvar-vaginal tumors are often locally invasive and metastasize early to local lymph nodes. Nonneoplastic differentials include vaginal cysts, hydrocolpos, and septa or congenital malformations.

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FIG. 26.29 (A) Uterine prolapse and tearing following parturition. (B) Close-up of prolapse showing contamination and tearing.

MEDICAL MANAGEMENT

If protrusion is not circumferential, vaginal prolapse will spontaneously resolve when estrogen influence diminishes. Estrus may be shortened by administering gonadotropin releasing hormone (50 µg/40 lb [18 kg]) or human chorionic gonadotropin (500–1000 IU, IM) to induce ovulation. Animals with vaginal prolapse/hyperplasia should not be used for breeding because the disease has a familial predisposition. Artificial insemination may be considered when a valuable bitch will not allow intromission and the owners insist on breeding. TVTs can be treated with vincristine (0.025 mg/kg up to 1 mg or 0.5 mg/m^2 IV, weekly for 3–6 weeks) or combination chemotherapy. TVTs also respond to local excision, radiation therapy, and immunotherapy. TVTs sometimes regress spontaneously.

SURGICAL TREATMENT

OHE is recommended to prevent recurrence and injury to the everted mucosa. Large, protruding masses may require manual reduction via an episiotomy and vulvar sutures to prevent reprolapse until the edematous tissue shrinks. Resection of the protruding tissue without OHE is not recommended because the procedure is associated with significant hemorrhage and does not prevent recurrence during subsequent estrus cycles. Resection of protruding tissue is recommended when the tissue is severely damaged or necrotic. Reduction or resection without OHE may require hysteropexy, cystopexy, and/or colopexy to prevent recurrent prolapse and herniation, respectively. OHE and mass excision or biopsy is recommended for all vaginal tumors except TVTs. Many vaginal tumors are under hormonal influence and regress after OHE.

Preoperative Management

Protruding mucosa should be lavaged with warm saline or water to remove debris and necrotic tissue. An antibiotic or antibiotic/steroid ointment can be applied to the exposed tissue and the mass replaced within the vagina or vestibule, if possible. An Elizabethan collar, bucket, or sidebars are indicated to prevent self-trauma before surgery.

Anesthesia

Anesthetic recommendations for stable animals undergoing reproductive surgery are in Table 26.3.

Surgical Anatomy

Anatomy of the male and female reproductive system is described on pp. 725 and 726, respectively. Vaginal lymphatic drainage is to the internal iliac lymph nodes.

Positioning

The patient is positioned in dorsal recumbency for OHE. The entire ventral abdomen and perineum should be clipped and prepared for aseptic surgery. Episiotomy requires that they be repositioned in a perineal position (i.e., ventral recumbency, pelvic limbs over the edge of a padded table, and tail fixed dorsally over the back [see Fig. 26.17]).

SURGICAL TECHNIQUE

Perform an OHE (see p. 728), and take a biopsy of the mass to rule out neoplasia. Perform an episiotomy if necessary to allow biopsy. Replace the protruding mass into the vagina or vestibule. Lavage, lubricate, and reduce the prolapsed tissue by digital manipulation. Maintain reduction by placing two to three large horizontal mattress sutures (e.g., 2-0 nylon or polypropylene) between the vulvar lips.

If resection of necrotic or severely traumatized tissue is necessary, position the patient in a perineal position and perform an episiotomy to expose the mass. Place and maintain a urethral catheter during the procedure. In stages, incise the base of the edematous tissue. Control hemorrhage with pressure, ligatures, or electrocoagulation. Appose adjacent mucosal edges with interrupted or continuous approximating sutures (e.g., 3-0 or 4-0 polydioxanone [PDS], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], or polyglyconate [Maxon]). Edema should resolve within 5 to 7 days of OHE.

POSTOPERATIVE CARE AND ASSESSMENT

Patients should be supported postoperatively with fluids and analgesics (see Chapter 13; for dosages of opioids, see Tables 13.2 and 13.3), as needed. Cold compresses should be applied immediately after episiotomy for 2 to 3 days, followed by warm compresses for 2 to 3 days to reduce inflammation and swelling. Self-trauma resulting from perineal discomfort associated with

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episiotomy or vulvar sutures may cause dehiscence; an Elizabethan collar, bucket, or sidebars should be used postoperatively. The vagina should be palpated 5 to 7 days after mass reduction or OHE, and vulvar sutures removed if tissue eversion has regressed with minimal threat of reprotrusion. Hemorrhage may occur following amputation of the protruding edematous tissue, but it is self-limiting if good surgical technique was employed.

PROGNOSIS

The prognosis is excellent following OHE; otherwise recurrence during subsequent estrus and difficult conception are common. Edema will resolve when estrogen levels diminish at the end of estrus. Offspring may be predisposed to the condition.

UTERINE PROLAPSE

DEFINITIONS

Uterine prolapse *(uterine eversion)* is an eversion and protrusion of a portion of the uterus through the cervix into the vagina during or near parturition.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Uterine prolapse is rare. It is similar to estrus-associated vaginal prolapse/hyperplasia (see p. 759); however, uterine prolapse is associated with parturition and involves the entire vaginal circumference. The cervix must be dilated for uterine prolapse to occur. One or both uterine horns may prolapse and reside in the cranial vagina or be everted through the vulva. Uterine prolapse usually occurs with prolonged labor. The everted tissue is doughnut-shaped and discolored from venous congestion, trauma, and debris. Uterine prolapse may tear the broad ligament and uterine artery. Hemorrhage may lead to hypovolemic shock unless controlled quickly.

DIAGNOSIS

Clinical Presentation

Signalment

The condition is rare but may occur near or at parturition. There is no recognized age predisposition. Although rare, the condition occurs more commonly in cats than in dogs.

History

Uterine prolapse is associated with excessive straining during parturition. A mucosal mass is generally noticed protruding from the vulva. Vague signs of abdominal distress and tenesmus may be noted. Signs of hemorrhagic shock may occur if the ovarian or uterine vessels have ruptured. Other signs may include restlessness, abnormal posture, pain, perineal bulging, licking, and dysuria.

Physical Examination Findings

Uterine prolapse is diagnosed on physical examination by digital examination of the vagina or visual observation (see Fig. 26.29). Perineal bulging may occur. Everted mucosa may protrude through the vulva or be digitally palpated in the vagina. A fornix will be identified by inserting a probe or finger along the protruding mass if it is a vaginal mass or prolapse, but not if it is a uterine prolapse. The animal may be stable or show signs of hemorrhagic shock (e.g., pale mucous membranes, tachycardia, and weak pulses).

Diagnostic Imaging

A gravid uterus or postpartum uterus may be identified on radiographs or ultrasound. Vaginoscopy may be used to confirm the diagnosis.

Laboratory Findings

Specific laboratory abnormalities are not seen. Anemia may be present if the uterine artery has ruptured.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include vaginal prolapse/hyperplasia (see p. 759), vaginal tumor (see p. 759), and uterine torsion.

MEDICAL MANAGEMENT

Medical treatment is rarely successful. Shock should be treated with fluids (plus or minus glucocorrticoids) and acid-base and electrolyte imbalances corrected. The protruding mass should be lavaged with warm saline and gently massaged to reduce edema. Lavaging with hypertonic dextrose solution may reduce swelling. The mass should be lubricated with a water-soluble gel and manually replaced by using external pressure and flushing sterile fluid under pressure into the uterine horn. After replacement, administration of oxytocin (5–10 U) promotes uterine involution and, together with closure of the cervix, helps prevent recurrence.

SURGICAL TREATMENT

The goals of treatment are to replace the uterus (see previous discussion under Medical Management) and prevent infection. Treatment options include manual reduction, manual reduction with immediate OHE, reduction during celiotomy, and amputation of the mass. OHE should be performed if tissue is devitalized or irreducible or if broad ligament vessels have ruptured. Laparotomy may be necessary to facilitate manual reduction by placing cranial traction on the broad ligament or uterus. Occasionally, uterine amputation is necessary to allow reduction. Everted uterine tissue may be amputated similarly to that described for vaginal prolapse/hyperplasia (see p. 760); however, the uterine arteries must be ligated. After uterine amputation, an OHE should be performed. Vaginopexy may be performed during cesarean section or celiotomy, or when the patient is stable.

> **NOTE** Catheterize the urethra during uterine amputation to prevent traumatizing it or the urethral papilla.

Preoperative Management

Patients in shock should have surgery as soon as they are stabilized. Shock should be treated with fluids (plus or minus glucocorrticoids) and acid-base and electrolyte imbalances corrected. Prophylactic antibiotics are indicated when the prolapse is contaminated or traumatized. Hair should be clipped from the abdomen, perineum, and the areas prepared for aseptic surgery. Viability of the prolapsed tissue should be assessed, and if the tissue appears healthy, the mass should be lavaged and replaced. Use techniques described in the Medical Management section.

Anesthesia

Anesthetic recommendations for animals undergoing reproductive surgery are provided on p. 722. Animals in shock require special care during induction and anesthesia. Anesthetic protocols for

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debilitated and shocky patients are provided in Table 26.5. Epidural anesthesia (see Table 13.5) may facilitate prolapse reduction and reduce postoperative straining. Local anesthetics should not be used for epidurals unless volume depletion has been corrected.

Positioning

Manual reduction may be accomplished with the patient in ventral, dorsal, or lateral recumbency. A perineal position is recommended for episiotomy and dorsal recumbency for celiotomy.

Surgical Anatomy

Surgical anatomy of the reproductive tract is provided on pp. 725 and 726.

SURGICAL TECHNIQUE

Reduce acute prolapses manually. Lavage the protruding tissue with warm saline or water and diluted antiseptic. Hypertonic agents (e.g., sugar) may reduce edema and facilitate reduction. *Gently compress the mass to reduce edema while attempting to reduce the prolapse. If necessary, perform an episiotomy to assist reduction. Insert a urethral catheter. Place horizontal mattress sutures between the vulvar lips to maintain reduction and prevent recurrence. If necessary, perform celiotomy to facilitate reduction by cranial uterine traction, ensure proper alignment of the uterine horns, and assess integrity of the vasculature.*

POSTOPERATIVE CARE AND ASSESSMENT

Shock, dehydration, and blood loss should be treated and analgesics given as necessary (see Chapter 13; for dosages of opioids, see Tables 13.2 and 13.3). Urination should be monitored because swelling and pain may cause urethral obstruction. If dysuria or anuria is anticipated, a urinary catheter should be placed. Antibiotics should be continued postoperatively if the uterus appears moderately to severely traumatized and OHE is not performed. Complications include hemorrhage, shock, dehydration, infection, necrosis, urethral obstruction, recurrence, and death.

PROGNOSIS

Complete uterine prolapse will not regress spontaneously. Survival following successful manual reduction of uterine prolapses is common, but infertility and dystocia may occur with subsequent breeding. The prognosis following OHE is excellent if shock and hemorrhage are treated appropriately.

Surgery of the Male Reproductive Tract

SPECIFIC DISEASES

PROSTATIC HYPERPLASIA

DEFINITIONS

Prostatic hyperplasia is a benign enlargement of the prostate. Increased numbers of prostatic cells occur secondary to androgenic hormone stimulation.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

BPH is the most common canine prostatic disorder; age related, it occurs in 95% of male dogs by 9 years of age. Potential causes of BPH include an abnormal ratio of androgens to estrogens, increased number of androgen receptors, and increased tissue sensitivity to androgens. The primary androgen promoting hyperplasia is dihydrotestosterone, which is irreversibly converted from testosterone. Conversion occurs in the epithelial cells by the action of 5-alpha reductase. Dihydrotestosterone enhances growth in both prostatic stromal and glandular components. Testosterone declines with age, but estrogen levels remain the same and induce nuclear dihydrotestosterone receptors, which may increase the sensitivity of the prostate to dihydrotestosterone. Other hormones, including estrogen, prolactin, and growth hormone, are believed to be involved. BPH may be a normal aging change unassociated with clinical signs; however, substantial enlargement may cause constipation, tenesmus, altered stool shape, and/or dysuria. Prostatic enlargement rarely causes urinary obstruction; canine glands expand outward rather than inward as in humans. Pressure on the pelvic diaphragm may contribute to the development of a perineal hernia.

Hyperplasia may be glandular or complex. Glandular hyperplasia affects dogs as young as 1 year of age and peaks at 5 to 6 years. There is a uniform proliferation of secretory structures with glandular hyperplasia, and gland consistency is normal. Complex hyperplasia is seen in dogs as young as 2 years of age, but predominantly occurs between 8 and 9 years. Cystic dilated alveoli are present with heterogeneous epithelial cells varying from normal to nonfunctional cuboidal cells. Acini are filled with eosinophilic material, and plasma cells and lymphocytes are present in the hyperplastic stroma.

DIAGNOSIS

Clinical Presentation

Signalment

Sexually intact male dogs are affected. doberman pinschers, Scottish terriers, Bouvier des Flandres, rottweilers, Bernese mountain dogs, and German pointers may be predisposed to BPH. The disease is found in most sexually intact male dogs over 6 years of age. Mean age at diagnosis is 8.6 years for most prostatic diseases.[12]

History

Dogs may have tenesmus, hematuria, and/or urethral bleeding. Owners may observe ribbon-like stools.

Physical Examination Findings

Rectal palpation reveals symmetric, nonpainful prostatic enlargement, which is soft and has a smooth contour. The median cleft is preserved. The prostate may be two to six times larger than normal.

Diagnostic Imaging

Radiographically the prostate appears symmetrically enlarged. The prostate is considered enlarged if it is greater than 70% of the distance between the sacral promontory and the pubis on lateral radiographs. The colon may be displaced dorsally and the urinary bladder cranially. Ultrasound is useful for evaluation of prostate size only when it is being used for serial comparisons

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in the same dog. Normal values are not available for ultrasound of the prostate. Ultrasonographically, hyperplasia is seen as diffuse, symmetric prostatic involvement, often with multiple, small cysts. Overall glandular echogenicity is normal to increased. Small areas of decreased echogenicity may be seen if cystic hyperplasia is present.

Typical features seem in CT images of BPH include symmetric prostatomegaly and heterogeneous prostatic parenchyma. Diagnosis of BPH should be considered when the ratios of prostatic dimensions (height, width, length) to the sixth lumbar vertebrae exceed 2.94, 3.38, and 3.05, respectively.[13]

NOTE Aspirates or core biopsies should be collected during ultrasonography to establish a tentative diagnosis of BPH.

Laboratory Findings

Ejaculate volume is reduced, but total sperm count and fertility are not affected. Ejaculate cytology reveals hemorrhage and mild inflammation without sepsis. Prostatic epithelial cells, erythrocytes, and a few leukocytes are identified. Urinalysis may identify blood. Definitive diagnosis of BPH requires histopathology, but typically a presumptive diagnosis is made based on historical information, physical examination findings, and ultrasonographic examination.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include prostatic squamous metaplasia, prostatic cysts (see p. 768), paraprostatic cysts (see p. 768), prostatitis (see p. 764), prostatic neoplasia (see p. 771), and prostatic abscesses (see p. 764) (Box 26.23 and Table 26.6). Prostatic aspiration or biopsy (see p. 742) is necessary to differentiate benign and malignant prostatic enlargement. Ultrasonography may differentiate parenchymal enlargement from cystic change.

MEDICAL MANAGEMENT

Estrogens, progestins, synthetic steroids, and antiestrogenic drugs have been used for their antiandrogenic effects, but relapses occur after therapy stops (Table 26.7). Finasteride is a synthetic steroid type II 5-alpha reductase inhibitor that significantly decreases dihydrotestosterone (approximately 58%) without affecting serum testosterone or semen quality, and reduces prostate diameter (approximately 20%) and volume (approximately 43%) (see

BOX 26.23 Prostatic Disease: Key Points

- Most dogs with prostatic disease present at approximately 8 years of age; those with tumors are usually ≥10 years old.
- Enlarged prostates can contribute to perineal hernia formation.
- Most geriatric intact male dogs have benign prostatic hyperplasia, but often it does not cause clinical signs.
- Hyperplastic prostates are enlarged, nonpainful, and symmetric.
- Castration is the best treatment for benign prostatic hyperplasia, but valuable breeding dogs may respond to finasteride.
- Prostatic cysts may be associated with benign prostatic hyperplasia. Cysts are either within the parenchyma or outside the parenchyma (paraprostatic).
- Cystic prostates palpate similar to those with hyperplasia unless there are paraprostatic cysts; these are enlarged, fluctuant, usually nonpainful, and asymmetric.
- Drain or excise prostatic cysts and castrate.
- Sertoli cell tumors predispose the prostate to squamous metaplasia, which can lead to prostatic cysts that can then abscess.
- *Escherichia coli* is the most common organism isolated from abscessed prostates.
- Abscessed prostates are enlarged, fluctuant, usually painful, and asymmetric.
- Abscessed prostates may concurrently be neoplastic.
- Drain, administer appropriate antibiotics, and castrate dogs with prostatic abscesses.
- Castrated male dogs presenting with prostatic disease are at high risk for neoplasia.
- Prostatic carcinomas are the most common tumor; most originate from ductal/urothelial tissue and are nonandrogen sensitive.
- Neoplastic prostates may be normal size, but they are often enlarged, asymmetric, nodular, firm, and fixed.
- Prostatic tumors are invasive and advanced at the time of diagnosis; therefore treatment is palliative. Survival time is longer if treated with NSAIDs (piroxicam, carprofen).
- Expect urinary incontinence if total prostatectomy is performed.

NSAIDs, Nonsteroidal antiinflammatory drugs.

TABLE 26.6 Clinical Signs of Prostatic Disease

	DIAGNOSIS			
Signs	**Hyperplasia**	**Infection/Abscess**	**Cyst**	**Neoplasia**
Prostatomegaly	+	+	+	±
Symmetric prostatic enlargement	+	±	±	±
Pain on prostatic palpation	–	±	–	±
Fluctuant prostate	–	±	±	±
Lymphadenomegaly	–	±	–	±
Ultrasound	Normal, or ↓ echogenicity	Hypoechoic to anechoic cavities	Anechoic cavities	Heterogeneous irregular urethra
Cytology	Hemorrhage	Hemorrhage, inflammation, bacteria	Hemorrhage	Atypical cells ± inflammation and bacteria
Peripheral leukocytosis	–	±	±	±
Pyuria	Rare	+	Rare	±
Systemic signs	±	±	±	±

+, Present; –, absent; ±, variable.

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TABLE 26.7 Drugs Used for Medical Management of Benign Prostatic Enlargement

Drug	Dosage	Expectations
Finasteride (Proscar)	0.1–0.5 mg/kg PO q24h for 2–3 mo[a] *or* 5.0 mg PO q24h for dogs weighing 5–50 kg[a]	Decreases dihydrotestosterone, prostatic diameter, and volume No adverse effects on semen or testosterone levels Safe
Diethylstilbestrol[b]	0.2–1 mg PO every 2–3 d for 3–4 wk	Potentially causes infertility, myelosuppression (anemia, thrombocytopenia, pancytopenia), and prostatic squamous metaplasia leading to abscessation; not recommended
Progestins:		
Medroxyprogesterone[b] acetate (Depo-Provera)	3 mg/kg (minimum dose of 50 mg) IM or SC; repeat dose in 4–6 wk if signs persist	53% have reduced prostate size
Cyproterone acetate	1.25–2.5 mg/kg PO q24h for 15 days	Monitor for weight gain, hypothyroidism, diabetes mellitus, mammary nodules; possible testes degeneration, and decreased serum testosterone and LH
Chlormadinone acetate[c]	1–3 mg/kg SC every mo	
Osaterone acetate[c]	2.0 mg/kg PO q24h for 7 d	
	0.25 mg/kg PO q24h for 7 d	

[a]The dose is uncertain; these are two dosages that have been suggested.
[b]Should not be used to treat dogs with prostatic disease.
[c]Limited data available regarding their use.
IM, Intramuscular; *LH,* luteinizing hormone; *PO,* orally; *SC,* subcutaneous.

Table 26.7); it is currently the drug of choice in dogs for which castration is not an option. Estrogen therapy reduces prostatic size but is not recommended because it causes infertility, squamous metaplasia, abscessation, and aplastic anemia. Medroxyprogesterone acetate alleviates signs of hyperplasia within 4 to 6 weeks in most (84%) dogs; however, they recur at an average of 13.6 months. Potential progestin side effects include increased appetite, weight gain, mammary neoplasia and dysplasia, and diabetes mellitus. Osaterone acetate and delmadinone acetate (antiandrogens) have been shown to improve clinical signs and induce clinical remission with minor adverse effects, with osaterone reducing prostatic gland volume quicker than delmadinone.[14]

SURGICAL TREATMENT

Asymptomatic animals do not require therapy. Castration is the best treatment for dogs with clinical disease. Castration permanently involutes the prostate within 3 to 12 weeks. Subtotal prostatectomy is a possible option for valuable breeding animals (see p. 742).

Preoperative Management

Constipation, tenesmus, and urine retention should be treated symptomatically. Stool softeners may facilitate defecation (see Box 26.8).

Anesthesia

Anesthetic recommendations for animals undergoing elective reproductive surgery are provided on p. 722 (see Table 26.3).

Surgical Anatomy

Surgical anatomy of the prostate is provided on p. 727.

Positioning

The animal is positioned in dorsal recumbency for prescrotal castration (see p. 732) and a perineal position for perineal castration (see p. 734). See p. 732 for recommendations for clipping and surgically prepping for castration. Position the animal in dorsal recumbency for subtotal prostatectomy (see p. 742).

SURGICAL TECHNIQUE

Castration is described on p. 732. Subtotal prostatectomy is described on p. 742.

POSTOPERATIVE CARE AND ASSESSMENT

Analgesics should be provided for pain (see Chapter 13; for dosages of opioids, see Tables 13.2 and 13.3), if necessary. Symptomatic treatment for constipation, tenesmus, and urine retention may be necessary until involution diminishes clinical signs. Prostatic involution can be evaluated ultrasonographically. A 50% reduction in prostatic size is expected within 3 weeks and 70% by 9 weeks after castration.

COMPLICATIONS

Complications of castration are found on p. 745. The most common complication of BPH is secondary bacterial infection causing prostatitis. Perineal hernias are less common complications. Drugs used for medical management are listed in Table 26.7.

PROGNOSIS

The prognosis following castration is excellent. Although symptomatic medical therapy alone may initially be helpful, clinical signs recur or worsen without castration.

PROSTATIC ABSCESSES

DEFINITIONS

Prostatic abscesses are localized accumulations of purulent material within the prostatic parenchyma. **Prostatitis** is an infection of the prostate gland, with or without abscess formation.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Prostatitis is common in dogs but rare in cats. Infection occurs when bacteria colonize the prostatic parenchyma. The prostatic

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BOX 26.24 Normal Prostatic Defense Mechanisms Against Infection

Local production of IgA and IgG prostatic antibacterial factor
Mechanical urethral flushing during urination
Urethral high-pressure zone
Urethral peristalsis
Surface characteristics of the urethral mucosa

IgA, Immunoglobulin A; *IgG,* immunoglobulin G.

epithelium creates a blood-prostate barrier because of its lipid bilayer. A high prostatic secretion concentration of zinc probably provides antibacterial activity and normal sperm function. Bacterial colonization of the prostate is reduced by normal defense mechanisms (Box 26.24). Ascending urethral infection is typical, but hematogenous infection is possible. Factors predisposing to infection include disruption of normal parenchymal architecture, urethral disease, urinary tract infections, altered urine flow, altered prostatic secretions, and reduced host immunity. Prostatic cystic hyperplasia, squamous metaplasia, and cysts increase risk of infection. Androgenic hormones are necessary for prostatic secretions; estrogenic hormones decrease secretory activity and may cause prostatic squamous metaplasia leading to cyst formation with subsequent abscessation. Microabscesses form and coalesce, causing large abscesses if not treated promptly. Prostatic enlargement may compress the colon (and rarely the urethra), causing obstruction. Abscess rupture may cause septicemia, peritonitis, and cardiovascular collapse. Pressure on the pelvic diaphragm associated with large prostatic abscesses or cysts may contribute to perineal hernia.

DIAGNOSIS

Clinical Presentation

Signalment

Abscesses primarily occur in older, sexually intact males with prostatitis, squamous metaplasia, or cysts. Although prostatic abscesses may occur in dogs as young as 2 years of age, most are older than 8 years. Feline prostatic infections are rare.

History

Dogs may have recurrent or nonresponsive urinary tract infections. Animals are usually brought in because of an acute onset of depression or lethargy, a tendency to strain when urinating or defecating, hematuria, vomiting, discomfort or pain, and polyuria or polydipsia (Box 26.25). Other clinical signs include fever, anorexia, diarrhea, and dehydration. Clinical signs result from compression of adjacent structures or infection.

Physical Examination Findings

Abscessed prostates are generally enlarged, painful, and asymmetric with fluctuant areas (Box 26.26; see also Table 26.6). Rectal palpation is often painful; caudal abdominal pain, lumbar pain, and/or pelvic limb stiffness may be seen. Peritonitis may cause abdominal distention. The scrotum and testicles should be palpated for masses, enlargement, or increased sensitivity. Some animals have perineal hernias, subcutaneous edema, and/or feminization. Depression, fever, anorexia, vomiting, diarrhea, and dehydration are associated with severe infections. Additionally, signs of tachycardia, pale or injected mucous membranes, delayed capillary refill, and/or weak pulses suggest sepsis and shock.

! BOX 26.25 Clinical Signs in Dogs With Prostatic Abscesses

Depression/lethargy
Straining to urinate or defecate
Hematuria
Vomiting
Discomfort or pain
Polyuria/polydipsia

BOX 26.26 Physical Examination Findings on Rectal Palpation in Dogs With Prostatic Abscesses

Prostatomegaly
Pain
Asymmetry with fluctuant areas

BOX 26.27 Radiographic Changes in Dogs With Prostatic Abscesses

Prostatomegaly
Indistinct prostatic borders

Diagnostic Imaging

Radiographic findings include prostatomegaly, indistinct borders, and occasional mineralization (Box 26.27). Prostatic emphysema is rare. Loss of abdominal visceral detail suggests peritonitis (see p. 528). Ultrasonographic evaluation may reveal alterations in echogenicity, and fluid-filled spaces with irregularly defined margins may be seen. Fluid within the lesion may have mixed echogenicity or a flocculent appearance.

NOTE Aspiration with ultrasound guidance usually establishes the diagnosis but should be performed with caution as thin-walled abscesses may rupture secondary to aspiration.

Laboratory Findings

Neutrophilic leukocytosis with a left shift, toxic neutrophils, and monocytosis may occur. Additional abnormalities may include elevated serum alkaline phosphatase and alanine transaminase activities, azotemia, hyperglobulinemia, hypoglycemia, and hypokalemia. Hematuria, pyuria, and bacteriuria are common. Prostatic wash or fine-needle aspiration cytology yields highly cellular smears with large numbers of neutrophils and smaller numbers of macrophages and epithelial cells. Squamous metaplasia, hyperplasia, or normal epithelial cells may be detected. The most commonly isolated bacteria are listed in Box 26.28. Occasionally, anaerobic organisms or *Mycoplasma* are isolated. Urine culture, in addition to prostatic fluid culture, is indicated because concurrent infections are common. Antimicrobial sensitivity should be determined for all pathogens. Histologic evaluation may show localized or diffuse inflammation; glandular lumens are typically filled with neutrophils, bacteria, and necrotic

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BOX 26.28 Most Commonly Isolated Bacterial Organisms in Animals With Prostatic Abscesses

Escherichia coli
Pseudomonas spp.
Staphylococcus spp.
Streptococcus spp.
Proteus spp.

debris. Fibrosis, atrophy, and stromal accumulations of lymphocytes and plasma cells are found with chronic prostatitis.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include prostatitis, prostatic cyst, paraprostatic cyst, prostatic neoplasm, prostatic hyperplasia, rectal mass, and intrapelvic masses (see Table 26.6 and Box 26.23).

MEDICAL MANAGEMENT

Prostatitis and small prostatic abscesses are treated with antibiotics, fluid therapy, and nutritional support. If the animal is in SIRS (see p. 530), fluid replacement therapy should be initiated as soon as possible. If hypokalemia (see Table 4.6) and hyponatremia are present, IV supplementation is required. Hypoglycemia is common in SIRS, and glucose supplementation may be needed (i.e., 2.5%–5% dextrose-containing fluids) or given as a slow IV bolus (e.g., 10% dextrose) if rapid replacement is necessary. Urine output should be monitored (normal urine output is more than 1–2 mL/kg per hour). Glucocorticoid administration in patients with SIRS is controversial. At one time, glucocorticoids were routinely administered; next, steroids were considered ineffective in these patients. Currently, there is thought that select patients not successfully resuscitated with fluids and pressors may respond to physiologic doses of glucocorticoids.

Broad-spectrum antibiotic therapy should be initiated as soon as the diagnosis is made. There are several combinations that are often effective (e.g., ampicillin plus sublactam and enrofloxacin, enrofloxacin and clindamycin [see p. 726]). A second-generation cephalosporin (e.g., cefoxitin sodium) may be used as a sole agent (see p. 531). If a resistant bacterial infection is present in an animal with renal compromise, piperacillin plus tazobactam may be considered (see p. 80) while waiting for culture/sensitivity. Initial antibiotic therapy should be altered based on results of susceptibility testing.

In select cases (dogs with mild to moderate clinical signs, with no evidence of peritonitis), ultrasound-guided percutaneous prostatic drainage can be safely performed. This procedure is associated with a good outcome; however, multiple drainages are required for resolution. Castration should also be performed, usually following the initial drainage.

After heavy sedation or during general anesthesia, insert a 22-gauge spinal needle with an attached extension set and 20-mL syringe into the fluid-filled cavity with ultrasound guidance (Fig. 26.30). If necessary, have an assistant displace the prostate cranially via rectal digital manipulations. Completely evacuate the fluid and submit samples for cytology, culture, and susceptibility evaluation. Following drainage, provide supportive care and monitor the animal closely for signs of leakage from the prostatic puncture or injury to adjacent structures for 24 to 48 hours. Two to four additional drainage procedures may be necessary.

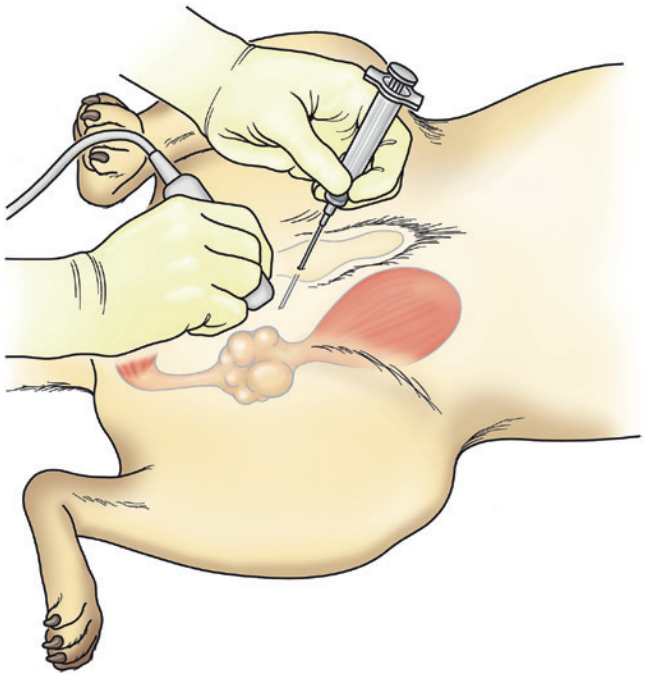


FIG. 26.30 Technique for ultrasound-guided percutaneous prostatic drainage.

SURGICAL TREATMENT

Acute bacterial prostatitis and prostatic abscesses are potentially life-threatening. Shock therapy must be initiated promptly (see previous discussion). Large abscesses should be drained and castration performed when the patient is stable. Castration may reduce the duration of infection. Prostatic biopsy (see p. 742) should be performed during drainage or resection. Subtotal prostatectomy (see p. 742) is indicated in stable patients for recurrent abscessation or cysts that have not responded to drainage procedures. Rarely, total prostatectomy (see p. 742) is performed for recurrent prostatic infections.

Preoperative Management

The animal should be stabilized before surgery (see the previous discussion). Place a urinary catheter before surgery to facilitate intraoperative identification of the urethra.

Anesthesia

Anesthetic recommendations for animals in shock are provided in Table 19.2. Anesthetic recommendations for stable animals undergoing abdominal surgery are provided in Table 19.1.

Surgical Anatomy

Surgical anatomy of the prostate is provided on p. 727.

Positioning

The ventral abdomen and medial thighs should be clipped and aseptically prepared for surgery and the prepuce flushed with 0.1% povidone-iodine or a 1:40 dilution of 2% chlorhexidine

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solution. The patient is positioned in dorsal recumbency for a midline celiotomy.

SURGICAL TECHNIQUE

Large abscesses or cysts should be drained. Choice of drainage procedures depends on the size and location of the abscess or cyst. Marsupialization (see p. 769) is an option if the abscess or cyst can be mobilized to the ventral abdominal wall and if the capsule can hold sutures. It is more commonly used for cysts than abscesses. Prostatic omentalization is currently the treatment of choice for abscess drainage and requires less postoperative care. Placement of multiple drains should be considered only if omentalization is not possible. Subtotal prostatectomy is an option if abscesses recur. Regardless of method of abscess drainage, perform castration before performing the abdominal exploration.

Omentalization

Place a urethral catheter. Expose the prostate through a ventral midline celiotomy from the umbilicus to the pubis. Extend the incision caudally, and perform a pubic osteotomy if necessary to adequately expose the prostate (see pp. 689 and 694). Place Balfour retractors to facilitate exposure. Explore the abdomen and isolate the bladder and prostate with laparotomy sponges. Place traction sutures through the bladder wall to retract the prostate cranially. Make stab incisions bilaterally in the lateral aspects of the prostate gland and remove the purulent material by suction. Explore and digitally break down any loculated abscesses within the parenchyma. Identify the prostatic urethra by palpation of the previously placed urethral catheter. Enlarge the stab incisions by resection of the lateral capsular tissue. Submit excised tissue for histopathologic examination. Introduce omentum through one capsulotomy wound with forceps introduced through the contralateral wound (Fig. 26.31A). Pass the omentum around the prostatic urethra, exit it through the same incision, and anchor it to itself with absorbable mattress suture (Fig. 26.31B). Place a closed-suction drain within the caudal abdominal cavity. Close the abdomen routinely. Alternatively, omentum can be inserted through both capsular incisions.

POSTOPERATIVE CARE AND ASSESSMENT

Analgesics should be given as necessary (see Chapter 13; for dosages of opioids, see Tables 13.2 and 13.3) and the patient monitored for sepsis, shock, and anemia. Fluid and nutritional support should be provided until the patient is stable and able to eat. Appropriate antibiotics should be given for 2 to 3 weeks. Ideally, urine or prostatic fluid culture should be performed 3 to 5 days after starting antibiotics and 2 to 3 days after antibiotics are discontinued. The abdomen should be bandaged to protect drains, and an Elizabethan collar or sidebars used to prevent self-trauma and drain removal. Recurring or persistent infection should be identified by culturing prostatic fluid and performing ultrasonography every 3 to 4 months for 1 year.

COMPLICATIONS

The most common short-term complications following omentalization are listed in Box 26.29. Subtotal prostatectomy may cause shock, urine leakage, and urinary incontinence. Death and urinary incontinence are more common following subtotal prostatectomy than after omentalization procedures.

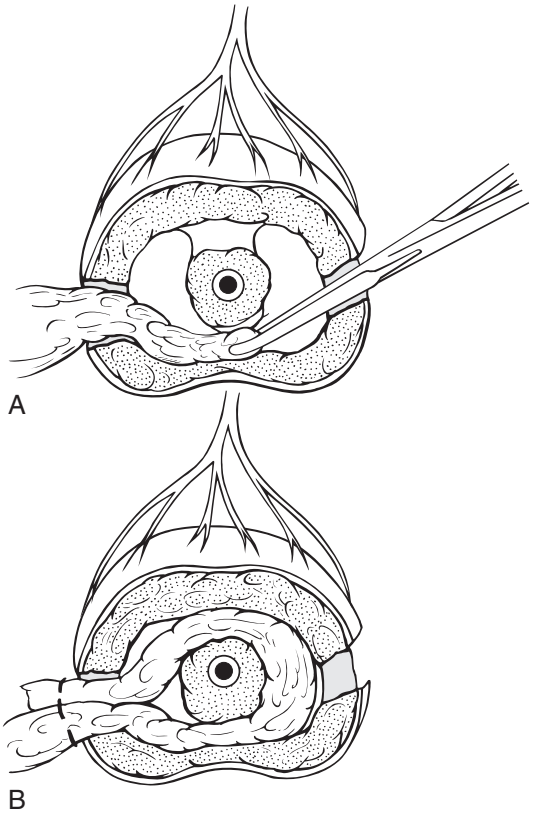


FIG. 26.31 Omentalization of a prostatic abscess. After making stab incisions bilaterally in the lateral aspects of the prostate gland—suctioning, exploring, and lavaging the abscess cavity—introduce omentum through one capsulotomy wound with forceps. (A) The omentum is introduced through the contralateral wound. (B) Then pass the omentum around the prostatic urethra, exit it through the entry incision, and anchor it to itself with absorbable mattress sutures.

BOX 26.29 Short-Term Complications After Prostatic Drainage

- Hypoproteinemia
- Shock
- Subcutaneous edema
- Hypokalemia
- Hypoglycemia
- Incisional infections
- Anemia
- Urine leakage
- Sepsis
- Urinary incontinence

Long-term complications following omentalization include recurrent prostatitis, abscesses, urinary tract infections, urinary incontinence, urethrocutaneous fistula formation, and paraprostatic cyst formation. Recurrence rates are approximately 18% for prostatic disease, 33% for urinary tract infection, and 46% for urinary incontinence.

PROGNOSIS

Antibiotics and supportive therapy may resolve small abscesses. Large, untreated abscesses will eventually cause septicemia,

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toxemia, and death. Immediate postoperative mortality may approach 25%; if the prostatic abscess has ruptured, mortality approaches 50%. Fair to excellent results are expected if the patient survives 2 weeks after surgery. Peritonitis may occur after abscess rupture or surgical contamination. Prognosis after omentalization appears good if sufficient omentum is placed within the prostate. Subtotal prostatectomy using an ultrasonic aspirator has a good prognosis because it provides long-term disease resolution.

PROSTATIC CYSTS

DEFINITIONS

A **prostatic cyst** is a nonseptic, fluid-filled cavity within or attached to the prostate. Included are **parenchymal cysts** associated with prostatic hyperplasia or **paraprostatic (periprostatic) cysts,** which are attached to the prostate and may communicate with it.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Parenchymal prostatic cysts occur within or have a physical communication with the prostatic parenchyma (Fig. 26.32). They are common in dogs and may be associated with BPH (see p. 762). Their etiology is unknown, but some are congenital. Sertoli cell tumors or exogenous estrogens may cause squamous metaplasia, which occludes ducts, causing secretory stasis with progressive acinar dilation. Cysts coalesce as they enlarge and are surrounded by dense collagen that can ossify. Small cysts often become confluent, forming larger cavities. Parenchymal cysts are typically found throughout the gland. Parenchymal prostatic cysts are lined by compressed epithelium (transitional, cuboidal, or squamous) and filled with secretory material and cellular debris.

Paraprostatic (periprostatic) cysts are rare compared with other types of prostatic disease. Paraprostatic cysts are adjacent and attached to the prostate but seldom communicate with the parenchyma. They may originate from the uterus masculinus, an embryonic structure derived from the Müllerian duct system and attached on the dorsal prostatic midline. These cysts are often large, extending into the perineal fossa or abdomen. They may displace and compromise adjacent viscera and their function. Paraprostatic cysts are usually filled with pale yellow to orange fluid; hemorrhage changes it to brownish red. Histologically the wall of a paraprostatic cyst resembles the wall of a parenchymal cyst (compressed epithelium and dense collagen). Some walls are calcified. Prostatic cysts may become infected and abscess. Pressure on the pelvic diaphragm may contribute to the development of a perineal hernia.

DIAGNOSIS

Clinical Presentation

Signalment

Prostatic cysts are most common in older, intact male, large-breed dogs.

History

Dogs are often asymptomatic until the cysts become large enough to cause rectal, bladder, or urethral obstruction. Presenting complaints include depression, inappetence, stranguria, dysuria, incontinence, tenesmus, or bloody penile discharge.

Physical Examination Findings

Clinical signs and examination findings resulting from prostatic cysts are similar to those caused by prostatic hyperplasia (see p. 763). However, paraprostatic cysts are asymmetric, fluctuant, and sometimes cause abdominal distention. The most common physical finding is a palpable abdominal mass. Sterile prostatic cysts are not painful. The scrotum and testicles should be palpated for concurrent masses, enlargement, or increased sensitivity, all of which indicate a Sertoli cell tumor. Perineal bulge or abdominal distention may occur with large cysts. The perineum is palpated to detect herniation.

Diagnostic Imaging

Prostatic cysts and paraprostatic cysts may be difficult to differentiate from the urinary bladder without a cystourethrogram. Prostate or cyst wall mineralization may be detected on survey radiographs. Ultrasonography helps detect and define cavitary changes (Fig. 26.33). Prostatic parenchymal cysts typically appear

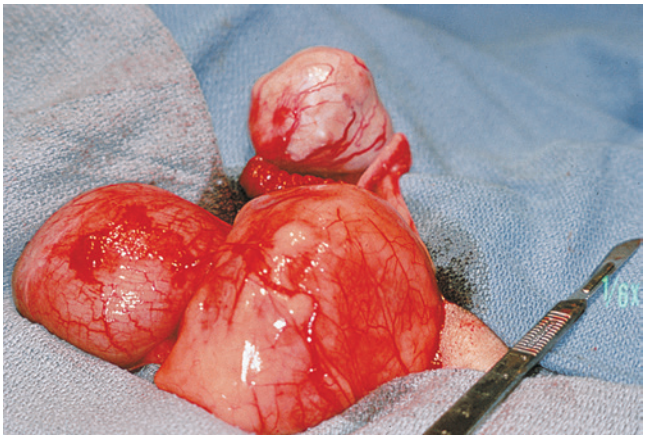


FIG. 26.32 Intraoperative appearance of a large prostatic cyst with squamous metaplasia. Note the enlarged, nodular cryptorchid testicle (dorsal) in which a Sertoli cell tumor was diagnosed histologically.

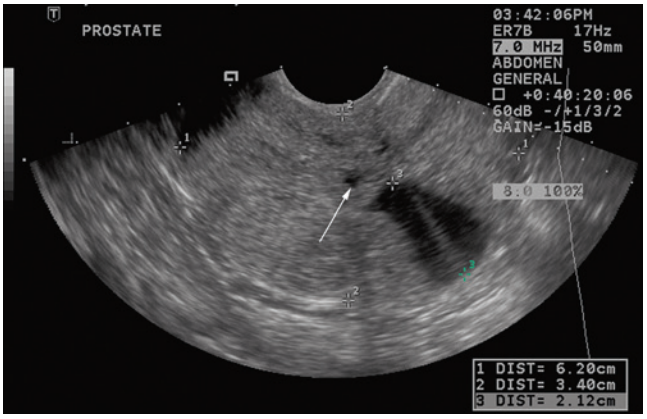


FIG. 26.33 Transrectal transverse ultrasound image of a dog's prostate, which is enlarged because of benign hypertrophy. Note the urethra in the center *(arrow)*. There is a 2-cm-diameter cyst within the left lobe of the prostate (anechoic region). This cyst is typical of prostatomegaly resulting from benign hypertrophy.

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BOX 26.30 Prostatic Tumor Types

Adenocarcinoma
Transitional cell carcinoma
Undifferentiated carcinoma
Squamous cell carcinoma
Leiomyosarcoma
Hemangiosarcoma

as anechoic cavitary lesions with more regularly defined margins than abscesses. However, this finding is not 100% specific, and cytology of the fluid is necessary for a definitive diagnosis. Paraprostatic cysts are usually large anechoic structures with internal septa. Some communicate with large anechoic cavities within the prostatic parenchyma.

NOTE Ultrasound-guided aspirates can be obtained; however, be careful to prevent iatrogenic peritonitis if infection is likely.

Laboratory Findings

Specific laboratory abnormalities are rare. Aspiration of a sterile, yellow to serosanguineous fluid with minimal inflammation suggests a paraprostatic cyst. Cytologic evaluation reveals prostatic epithelial cells and few leukocytes, but more erythrocytes and hemosiderophages than with prostatic hyperplasia. Numerous squamous epithelial cells suggest squamous metaplasia.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include prostatic abscess, squamous metaplasia, neoplasia (Box 26.30), or hyperplasia.

MEDICAL MANAGEMENT

Medical therapy includes treatment of constipation and urine retention. Stool softeners (see p. 744) may be given and the bladder drained by centesis or catheterization as needed. Percutaneous drainage by centesis is palliative but may cause abscessation (see p. 742).

SURGICAL TREATMENT

Treatment for small parenchymal cysts is castration. Dogs with large cysts should be castrated and the cyst drained, resected, or debulked. Incomplete resection may be necessary to prevent incontinence.

Preoperative Management

Animals with prostatic cysts are generally stable. Perioperative antibiotics are reasonable if marsupialization or prolonged surgery is anticipated.

Anesthesia

Anesthetic recommendations for animals undergoing reproductive surgery are provided in Table 26.3.

Positioning

The ventral abdomen, ventral perineum, and medial thighs are clipped and prepared for aseptic surgery. The dog is positioned in dorsal recumbency for a midline celiotomy.

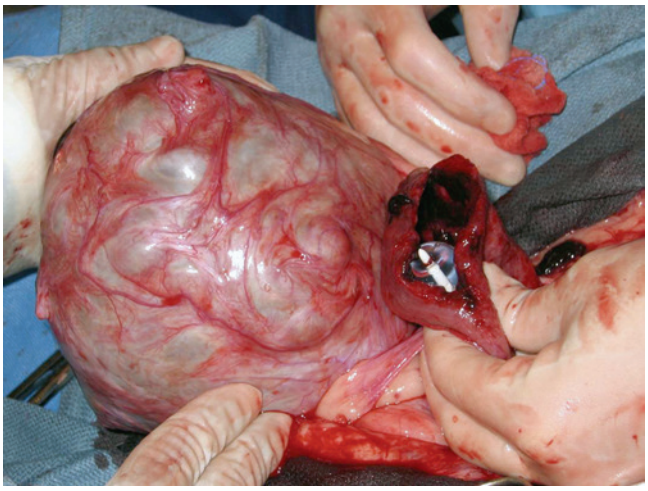


FIG. 26.34 This large paraprostatic cyst originated from the dorsolateral aspect of the prostate and caused urethral obstruction and rupture of the urinary bladder.

SURGICAL TECHNIQUE

Castration should be performed and large cysts resected or drained (Fig. 26.34). Cystic fluid should be cultured and a biopsy taken from the prostate (see p. 742). Nonresectable cysts may be drained by omentalization (Fig. 26.35), marsupialization or with multiple drains. Subtotal prostatectomy (see p. 742) may be appropriate for recurring cysts.

Omentalization

Omentalization of a large parenchymal prostatic cyst is performed similar to omentalization of an abscess. Omentalization of a paraprostatic cyst differs somewhat from omentalization of an abscess (see p. 767).

Expose the prostate through a caudal abdominal celiotomy. Identify the cyst, make a single stab incision into the cyst, and aspirate its contents. Take samples for culture, cytology, and histopathology. Dissect and resect the cyst wall without damaging innervation or vasculature to the prostate and urinary bladder (see Fig. 26.35A). Pack omentum into the cystic remnant and secure it with absorbable horizontal mattress sutures (see Fig. 26.35B). Lavage and close the abdomen routinely.

Marsupialization

Expose and isolate the prostate as described on p. 767 for omentalization. Make a second incision (5–8 cm) through the abdominal wall lateral to the prepuce over the abscess or cyst cavity (Fig. 26.36A). Excise 0.5 to 1 cm of abdominal muscle (Fig. 26.36B). Suture the capsule or the cyst wall to the external rectus fascia (Fig. 26.36C). Use continuous or interrupted 3-0 to 4-0 monofilament absorbable (polydioxanone [PDS], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], or polyglyconate [Maxon]) sutures. Facilitate suturing by having an assistant elevate the prostate toward the abdominal wall. Incise the abscess or cyst wall and suction the contents. Place a second layer of simple continuous or interrupted sutures (e.g., 3-0 or 4-0 nylon or polypropylene) between the skin edge and capsule or cyst edge (see Fig. 26.36D–E). Take a biopsy of the prostatic parenchyma. Digitally break down trabeculae and fibrous bands to create a confluent cavity. Lavage the cavity and surgical site, place omentum around the marsupialization, and close the abdomen in three layers.

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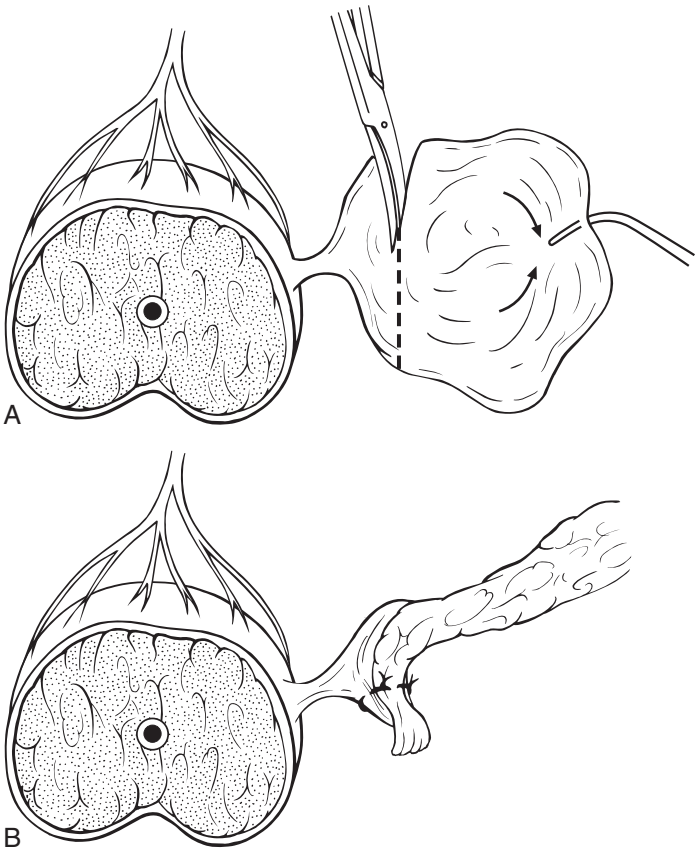


FIG. 26.35 Omentalization of a paraprostatic cyst. (A) After exposing the cyst, make a single stab incision into the cyst and aspirate its contents. Take samples for culture, cytology, and histopathology. Dissect and resect the cyst wall without damaging innervation or vasculature to the prostate and urinary bladder. (B) Pack omentum into the cystic remnant and secure it with absorbable horizontal mattress sutures.

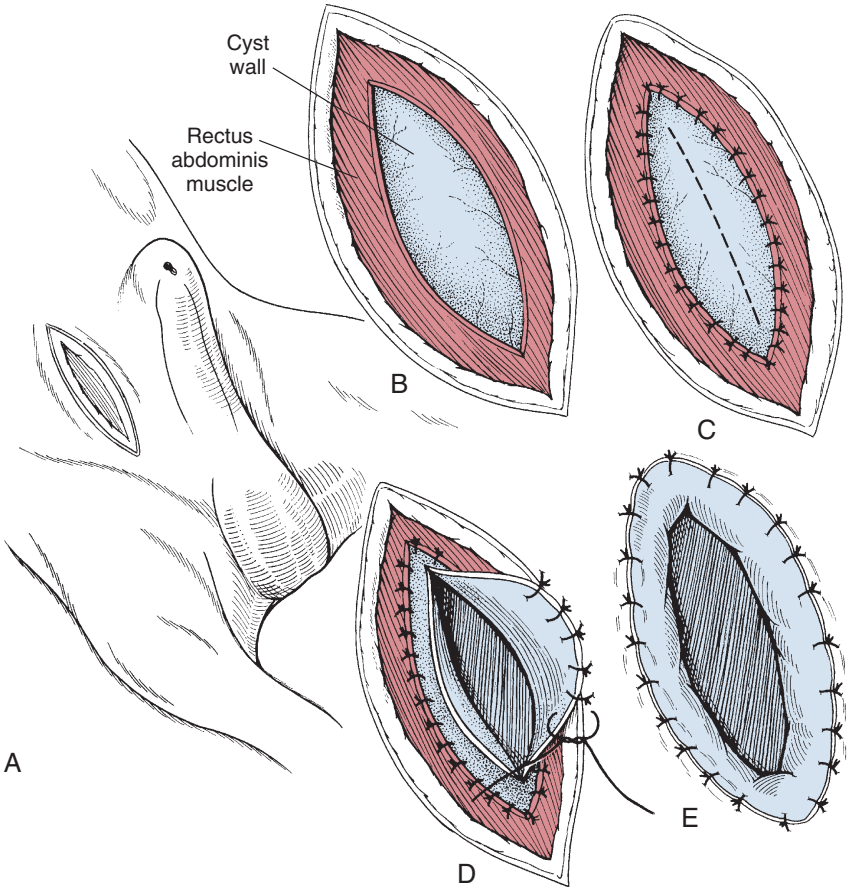


FIG. 26.36 (A) To marsupialize a prostatic cyst or abscess, make a longitudinal incision lateral to the prepuce over the enlarged mass. (B) Excise an ellipse of abdominal muscle. (C) Suture the prostatic capsule to the external rectus sheath. (D–E) Make an incision through the prostatic capsule and suture the edge of the capsule to skin.

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An alternative technique is to incise the ventrolateral aspect of the cyst or abscess wall and suction the cavity before suturing it to the rectus fascia. The capsule or cyst wall is then sutured 5 mm from the incised edge of the cavity to the rectus fascia. This variation has a higher risk of abdominal contamination.

POSTOPERATIVE CARE AND ASSESSMENT

Analgesics (see Chapter 13; for dosages of opioids, see Tables 13.2 and 13.3) and supportive care (e.g., fluids and electrolytes) should be given as necessary. Monitor closely for shock and infection. Medical therapy may manage urine retention, constipation, and discomfort. See p. 767 for postoperative care and potential complications following prostatic or cystic drain placement. Drains should be left in place for 1 to 3 weeks. Marsupialization may cause a permanent fistula or may close prematurely. Urine may be voided through the marsupialization for a few days if urethral erosion is present. Adhesions following omentalization are minimized if exposed parenchyma within the prostatic capsule is oversewn and covered with omentum.

PROGNOSIS

The prognosis is good to fair after castration and surgical drainage. Some prostatic and paraprostatic cysts recur and require repeated drainage, but this is rare if the dog is castrated. Recurrence following subtotal prostatectomy or omentalization is uncommon. Overzealous resection may cause detrusor atony, incontinence, or bladder ischemia. Infection of the cyst may occur following marsupialization.

PROSTATIC NEOPLASIA

DEFINITIONS

Prostatic tumors may originate from epithelial tissue (**carcinomas**), smooth muscle tissue (**leiomyosarcoma**), or vascular structures (**hemangiosarcoma**).

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Although prostatic neoplasia is the most common prostatic disease in neutered male dogs, it is still uncommon in dogs (0.2%–0.6%) and rare in cats. Castration is considered to be a risk factor for development of prostatic neoplasia, particularly prostatic transitional cell carcinoma (TCC). Testicular hormones are not believed to cause prostatic tumors, but adrenal and pituitary hormones may be influential. Most prostatic tumors originate from the prostate basal cell and less so from acinar epithelium. Types of prostatic tumors are listed in Box 26.30. Well-differentiated adenocarcinomas are found more frequently in sexually intact dogs, whereas histologic types with more anaplasia and TCC are more common in castrated dogs. In 68 prostatic biopsy samples in dogs with prostatic carcinoma, six distinct growth patterns were found: papillary, cribriform, solid, small acinar/ductal, signet ring, mucinous.[15] Cyclooxygenase (COX)-1 expression is present in 94.1% and COX-2 expression in 88.2% of dogs with prostatic carcinomas. Because COX-2 is not expressed in normal prostate glands, it may be involved in the pathogenesis of canine prostatic carcinoma. Prostatic carcinomas are locally invasive and metastasize early to regional lymph nodes (iliac, pelvic, and sublumbar), lung, and bone. They frequently invade bone, bladder, colon, and surrounding tissue by direct extension. Other metastatic sites include the liver, spleen, kidney, heart, adrenal glands, skeletal muscle, and subcutaneous tissue. Bone involvement may cause pain or pathologic fractures. Hypertrophic osteopathy has occasionally been associated with prostatic tumors. Prostatic enlargement causes compression and partial obstruction of the colon, rectum, and sometimes urethra. Pitting edema of the pelvic limbs may occur secondary to lymphatic invasion. Most tumors involve the trigone and urethra and have metastasized at the time of diagnosis. The behavior of feline prostatic tumors is unknown.

DIAGNOSIS

Clinical Presentation

Signalment

Prostatic neoplasia occurs in both intact and neutered males, but is more common in neutered males. Medium to large-breed dogs, such as doberman pinschers, Shetland sheepdogs, Scottish terriers, beagles, German shorthaired pointers, Airedale terriers, and Norwegian elkhounds are overrepresented. The average age of occurrence is 10 years. Prostatic tumors are very rare in cats.

History

Signs may include weight loss, pelvic limb lameness, weakness, or edema, tenesmus, dyschezia, urine retention or incontinence, stranguria, dysuria, polyuria, polydipsia, hematuria, and abdominal, pelvic, or lumbar pain. Prostatic neoplasia may cause substantial emaciation and debility. Metastasis may produce other signs (e.g., dyspnea). Castrated dogs presenting with prostatic disease are more likely to have neoplasia than other types of prostatic disease. The interval between castration and onset of disease is highly variable.

Physical Examination Findings

The animal may be debilitated and weak. Lymph node infiltration and lymphatic obstruction can cause pelvic limb edema. The prostate may be normal sized, but it is often asymmetrically enlarged. Pain, firmness, fixation, and nodular irregularity are characteristic of prostatic neoplasia. Medial iliac lymph node enlargement may be detected during rectal examination. Skeletal palpation sometimes elicits pain secondary to bone metastasis.

Diagnostic Imaging

Thoracic radiographs should be evaluated for metastasis. Abdominal and pelvic radiographs should be evaluated for prostatic size and mineralization, lymph node enlargement, colonic displacement, and osteolytic or proliferative vertebral or pelvic lesions. Retrograde urethrocystography may determine urethral size and mucosal smoothness, and prostatic symmetry. Most prostatic tumors involve the urethra and trigone of the urinary bladder. Ultrasonography may define the prostatic mass as cystic or solid and can be used to evaluate abdominal lymph nodes for metastasis. Finding prostatic mineralization in castrated dogs is highly suggestive of prostatic neoplasia; intact dogs are unlikely to have neoplasia if no mineralization is found. Prostatic aspiration and biopsy are facilitated by ultrasound guidance (see p. 742). Scintigraphy may be used to locate metastatic sites involving bone.

Laboratory Findings

Hematology, serum biochemistry profile, urinalysis, urine culture, and electrocardiography are indicated. Results are nonspecific

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BOX 26.31 Additional Cytologic Criteria of Malignancy

Variation in nuclear and nucleolar size
Variable and increased nuclear cytoplasmic ratio
Nuclear molding
Abnormal mitotic figures
Coarsely clumped chromatin

for prostatic neoplasia, although paraneoplastic syndromes and concurrent problems may be identified. Anemia, hematuria, or pyuria may be detected. Elevated concentrations of total acid phosphatase (21.4 ± 18.9 U/L) and prostatic acid phosphatase (11.9 ± 2.8 U/L) may indicate prostatic cancer.

Cytology or histopathology is needed for definitive diagnosis. Fine-needle aspiration of prostatic neoplasia may yield a moderately cellular sample with abnormal epithelial cells; however, this procedure should be used with caution as there is risk of dissemination of tumor cells along the needle tract in dogs with TCC. Neoplastic-appearing epithelial cells have large, prominent, multiple nuclei with multiple nucleoli and cytoplasmic vacuolation. Additional cytologic criteria for malignancy are provided in Box 26.31. Prostatic washes are less reliable in obtaining neoplastic cells. Transurethral biopsy may be diagnostic if the tumor has invaded the urethra.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include prostatic hyperplasia, abscesses, cysts, paraprostatic cysts, prostatitis, rectal masses, or pelvic masses. It can be difficult to differentiate cancer from prostatic hyperplasia associated with prostatitis (see Table 26.6).

MEDICAL MANAGEMENT

Chemotherapeutic protocols are not successful in the management of prostatic neoplasia. Radiation therapy can be used to decrease prostate size but does not improve survival times. COX inhibitors (e.g., piroxicam 0.3 mg/kg PO [orally] q24–48h or carprofen 2.2 mg/kg PO q12h) have been shown to improve survival in dogs with prostatic carcinomas, probably because of their binding to COX receptors. Concurrent administration of omeprazole (1–2 mg/kg PO q12h), pantoprazole (1 mg/kg IV q12–24h) or misoprostol (2–5 µg/kg PO q8–12h) may help ameliorate adverse effects associated with chronic use of NSAIDs. Proton pump inhibitors have fewer side effects than misoprostol and are probably as effective.

SURGICAL TREATMENT

Treatment is rarely successful. Treatment protocols combining surgery, chemotherapy, and radiation therapy are being investigated, but their efficacy is presently unknown.

Preoperative Management

Clip and aseptically prepare the ventral abdomen, ventral perineum, and medial thighs.

Anesthesia

Anesthetic recommendations for animals undergoing reproductive surgery are provided in Table 26.3.

Surgical Anatomy

Surgical anatomy of the male reproductive tract is provided on p. 726.

Positioning

The patient is positioned in dorsal recumbency for a midline celiotomy. The entire ventral abdomen and inguinal area should be clipped and prepared for aseptic surgery.

SURGICAL TECHNIQUE

Castration may temporarily slow tumor growth. Prostatectomy may be curative if the tumor is diagnosed early. Unfortunately, most tumors are advanced when diagnosed, making preservation of trigonal innervation impossible. Such dogs are often unacceptable pets after prostatectomy because of urinary incontinence. Palliation of clinical signs may occur following intracapsular subtotal prostatectomy, performed with electrocautery or Nd:YAG laser, with a markedly decreased incidence of urinary incontinence and longer survival time when compared with total prostatectomy. Transurethral resection of prostatic carcinoma can be successful in palliating clinical signs. Similarly, transurethral stents (balloon-expandable or self-expandable) can palliate clinical signs. Tube cystostomy can also be considered as a palliative option (see p. 685).

POSTOPERATIVE CARE AND ASSESSMENT

Analgesics (see Chapter 13; for dosages of opioids, see Tables 13.2 and 13.3) and fluids should be given as necessary. The bladder should be decompressed and urine diverted for 4 to 5 days with a urinary catheter or cystostomy tube after prostatectomy (see p. 742). Patients should be monitored for urine leakage, incontinence, or infection. Reevaluation at frequent intervals for local recurrence and metastasis is recommended. Complications and treatment of animals following prostatectomy are described on p. 744.

PROGNOSIS

The prognosis is poor because of metastasis, recurrence, and poor quality of life associated with urinary incontinence. Hormonal therapy has not been useful in dogs. Most untreated dogs are euthanized within 1 to 3 months because of progressive clinical signs.

TESTICULAR AND SCROTAL NEOPLASIA

DEFINITIONS

Sertoli cells are supporting elongated cells of seminiferous tubules that nourish spermatids. **Leydig cells** are interstitial tissue cells believed to be responsible for internal secretion of testosterone.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Scrotal tumors are most commonly mast cell tumors (MCTs; 54.6%). Mast cells are normal immune system cells and are important in inflammatory responses to tissue trauma. Cytoplasmic granules in mast cells contain heparin, histamine, platelet-activating factor, and eosinophilic chemotactic factor.

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The number and type of granules in MCTs depend on the degree of tumor differentiation. Well-differentiated MCTs contain more heparin, whereas undifferentiated tumors have more histamine. The cause of MCTs is unknown, although chronically inflamed areas have been reported to be at increased risk for tumor development. In dogs, 50% of MCTs are malignant, especially those in the preputial, inguinal, and perineal areas. Regional lymph nodes, spleen, liver, and bone marrow are common metastatic sites. MCTs have no distinctive appearance. They may be raised, hairless, ulcerated, erythematous, well-defined, or diffuse skin thickenings. Manipulation of MCTs may cause degranulation, erythema, and wheal formation. Steroid administration may cause degranulation. Gastroduodenal ulcers occur in up to 80% of dogs with MCTs because of histamine release. Ulcers may cause anorexia, vomiting, diarrhea, or melena (see p. 428). Heparin and proteolytic enzyme release may prolong coagulation and delay wound healing after resection.

Other reported scrotal tumors include melanocytoma, malignant melanoma, vascular hamartoma, hemangiosarcoma, hemangioma, and cutaneous histiocytomas.[16] Melanomas originate from melanocytes and melanoblasts, cells of neuroectodermal origin. Masses may be brown to black or occasionally nonpigmented. Melanomas are more common in dogs than in cats. Tumors originating in the skin tend to be benign. Local recurrence and distant metastasis are common with malignant melanomas. Lymph node metastasis usually occurs first, and then to the lungs.

The most common testicular neoplasms (e.g., Sertoli cell tumors, interstitial [Leydig] cell tumors, seminomas, and mixed germ cell–stromal tumors) occur with equal frequency. Other testicular tumors are rare (Box 26.32). Many old dogs have multiple tumors in one or both testicles. Tumors involving scrotal testes are usually benign, whereas those in cryptorchid testes may be malignant. Metastases are slow-growing but are occasionally detected in lumbar, deep inguinal, and external iliac lymph nodes. Visceral metastasis is rare. Extratesticular interstitial cell and Sertoli cell tumors have been reported in a small number of previously neutered dogs and cats. Testicular tumors in cats are very uncommon.

Testicular tumors interfere with testicular function, invading or compressing seminiferous tubules or producing excessive estrogen or testosterone. Interstitial cell tumor production of excess testosterone may contribute to perianal adenomas, perineal hernia, and BPH. Sertoli cell tumors producing excess estrogens may cause squamous metaplasia of the prostate, feminization, and/or myelotoxicity.

Sertoli cell tumors arise from sustentacular cells. Normal and neoplastic Sertoli cells produce estrogenic hormones. Sertoli cell tumors are usually solitary but may be multiple and bilateral. They are more common in cryptorchid than scrotal testes. Tumors are discrete, with expansile growth, compressing and destroying surrounding testicular tissue. Large tumors may cause distention or destruction of the tunic, and growth may extend along the spermatic cord. They are firm, multilobulated, and gray-white with areas of necrosis, hemorrhage, or cysts. Dogs with Sertoli cell tumors often have signs of hyperestrogenism (Box 26.33), especially those with large tumors. Signs regress with castration and tumor removal. Persistence or recurrence of clinical signs suggests estrogen-producing metastasis. Sertoli cell tumors have a higher rate of metastasis than other testicular tumors.

Interstitial (Leydig) cell tumors occur in scrotal testes as multiple or solitary forms and frequently coexist with Sertoli cell tumors. Most interstitial cell tumors are benign, soft, encapsulated, and rarely exceed 1 to 2 cm in diameter. Interstitial cell tumors may cause testicular enlargement but are difficult to palpate. On cut surfaces they are discrete, round, tan to yellow-orange masses with foci of hemorrhage or cystic spaces. Dogs with interstitial cell tumors may be infertile. These tumors produce androgens or contribute to androgenic hormone imbalance. Perineal hernia, perianal adenomas and hyperplasia, and prostatic disease have been associated with interstitial cell tumors.

Seminomas arise from testicular germ cells and occur commonly in cryptorchid and scrotal testicles. They are usually solitary but may be multiple, bilateral, and coexist with other tumor types. Seminomas can be large, replacing most testicular tissue. They are softer than Sertoli cell tumors with a glistening, pinkish gray-tan, multilobulated, unencapsulated cut surface. Signs of feminization rarely occur. They rarely metastasize.

DIAGNOSIS

Clinical Presentation

Signalment

Scrotal and testicular tumors are more common in dogs than in cats. They usually occur in dogs older than 10 years; however, tumors in cryptorchid animals may occur earlier. Cryptorchidism predisposes to Sertoli cell tumors and seminomas. Cryptorchid dogs are 13.6 times more likely to develop testicular tumors than

BOX 26.32 Scrotal and Testicular Tumors

Scrotal Tumors
Mast cell tumors
Melanomas

Testicular Tumors
Sertoli cell
Interstitial cell
Seminoma
Embryonal carcinoma
Lipoma
Fibroma
Hemangioma
Chondroma
Teratoma

! BOX 26.33 Signs of Hyperestrogenism

Bilateral symmetrical alopecia
Brittle hair
Poor hair regrowth
Thin skin
Hyperpigmentation
Nipple elongation
Mammary enlargement
Penile atrophy
Preputial swelling and sagging
Squatting micturition
Reduced libido
Male attraction
Testicular atrophy
Prostatic atrophy or cystic enlargement
Anemia
Thrombocytopenia or neutropenia

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normal dogs. Dogs with inguinal hernias are 4.6 times more likely. All breeds are susceptible to development of testicular tumors, although a particular predisposition in Maltese dogs has been reported. Dogs predisposed to scrotal MCTs include English bulldogs, English bull terriers, boxers, Boston terriers, and American pit bull terriers.[14]

History

Affected, asymptomatic animals may be brought in for evaluation of a mass seen or felt in the scrotal or inguinal areas or for endocrine abnormalities (e.g., changes in hair coat, infertility, lethargy, feminization [see Box 26.33], perianal tumors, or prostatic disease).

Physical Examination Findings

Scrotal skin should be examined for inflammation, nodules, masses, and ulceration. Scrotal skin should be of uniform thickness. Both testicles should be evaluated for symmetry, firmness, irregularity, scrotal adhesions, and sensitivity. Small or deep intraparenchymal testicular tumors are not detectable on palpation, but the testes may be firm and hard, or one testicle may be firm and the other atrophied. If the animal is cryptorchid, the inguinal area should be checked for a retained testicle and the abdomen for a mass. Sublumbar lymphadenomegaly and prostatomegaly may be detected by rectal examination. The abdomen should be palpated for splenomegaly, hepatomegaly, lymph node enlargement, and signs of metastasis (i.e., with MCTs). Sertoli cell tumors and seminomas may cause feminization (see Box 26.33).

Diagnostic Imaging

Intraabdominal testicles may be seen radiographically as caudal abdominal masses. Radiographs also help identify intraabdominal lymphadenomegaly and organomegaly. Ultrasound may delineate scrotal and testicular neoplasia, abscesses, ischemia, testicular torsion, and scrotal hernias. Testicular tumors have variable echogenicity on ultrasound examination.

Laboratory Findings

Hematology, serum biochemistry panel, and urinalysis are indicated in animals with scrotal or testicular tumors. Nonregenerative anemia, leukopenia, and thrombocytopenia may be associated with hyperestrogenism and myelotoxicosis. Fine-needle aspirate cytology of scrotal and testicular lesions helps identify neoplastic cells, fungal elements, abnormal sperm, bacteria, and inflammation. Fine-needle aspiration of the testicle is rarely performed, but it can help differentiate neoplasia from abscesses or granulomas. Cytology of the preputial mucosa may reveal cornification secondary to Sertoli cell tumor estrogen production. Fine-needle aspirate cytology is usually diagnostic for MCTs, but degranulated or poorly granular mast cells may be difficult for the novice cytologist. Buffy coat smears rarely reveal mastocytemia. Microcytic hypochromic anemia or a regenerative anemia plus hypoalbuminemia suggest gastrointestinal hemorrhage. More than 10 mast cells per 1000 nucleated cells in the bone marrow is abnormal. Tumor histopathology is necessary to grade the tumor and determine prognosis. Cytology from melanomas usually reveals round to spindle-shaped cells, frequently containing brown to black granules. It is easy to confuse amelanotic melanomas with fibrosarcoma on cytology.

Brucella canis infections may be diagnosed with serology or blood culture. Semen evaluation to determine fertility is rarely performed when neoplasia is diagnosed. Serum testosterone levels are sometimes elevated with interstitial cell tumors. Sertoli cell tumors and seminomas sometimes increase serum estradiol concentrations.

DIFFERENTIAL DIAGNOSIS

Other differentials for testicular masses include sperm granuloma, fibrosis, hematoma, spermatocele, varicocele, orchitis, and epididymitis. Other differential diagnoses for scrotal disease include dermatitis, self-trauma, chemical burn, and laceration. *B. canis* infection should be considered in animals having scrotal dermatitis, orchitis, reproductive failure, epididymitis, discospondylitis, or testicular atrophy. Testicular biopsy is discussed on p. 741.

NOTE Be sure to test for *Brucella canis* infection in dogs with unexplained scrotal or testicular disease.

MEDICAL MANAGEMENT

MCTs may respond to chemotherapy or radiation therapy. The efficacy of chemotherapy or radiation therapy for other scrotal or testicular tumors is unknown.

SURGICAL TREATMENT

Tumor excision gives the best chance for a good prognosis. Removal of both testicles is recommended for testicular neoplasia. Castration is described on p. 732. If the owner insists on preserving breeding potential, unilateral castration of the neoplastic testicle may be performed. The testicles should be submitted for histologic examination. Scrotal ablation (see p. 734) and castration are recommended to treat scrotal tumors and testicular tumors with scrotal adhesions. Even discrete MCTs may extend deep into surrounding tissue; therefore 3-cm margins on all sides are recommended.

Preoperative Management

Few patients with testicular tumors are debilitated, except those with myelosuppression, testicular torsion, or concurrent diseases (Fig. 26.37). Bone marrow suppression may necessitate blood transfusions and antibiotics. An antihistamine (e.g., diphenhydramine

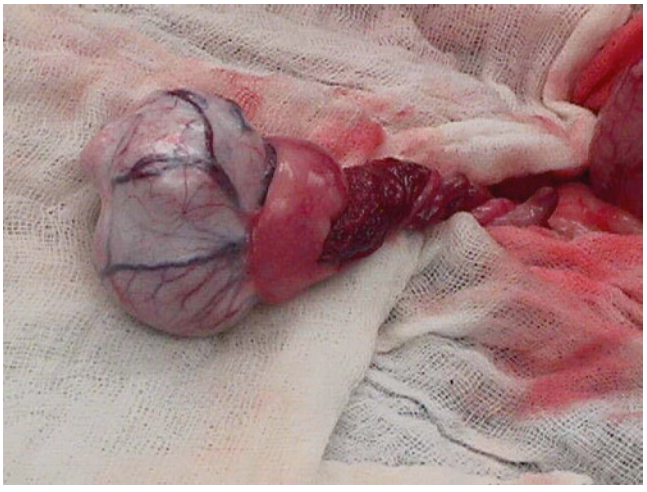


FIG. 26.37 Nodular, neoplastic cryptorchid testicle with testicular torsion. Note the twist in the spermatic cord.

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[Benadryl], 0.5–4 mg/kg IV, slowly) and proton pump inhibitor (e.g., omeprazole [Prilosec], 1–2 mg/kg PO, q12h or pantoprazole 1 mg/kg IV) should be preoperatively administered to patients with MCTs to protect against gastroduodenal ulceration (see Box 18.33). These drugs may be given IV immediately before surgery but should be given extremely slowly to avoid hypotension. Sucralfate suspension may be given for existing ulcers (see p. 429). Sucralfate should be given 1 hour after administration of other oral medications because it may interfere with their absorption.

Anesthesia

Anesthetic recommendations for animals undergoing reproductive surgery are provided in Table 26.3.

Positioning

Position the patient in dorsal recumbency for a prescrotal castration or an exploratory laparotomy. The entire ventral abdomen should be clipped and prepared for exploratory laparotomy; see p. 732 for surgical preparation recommendations for animals undergoing castration.

SURGICAL TECHNIQUE

Castration and scrotal ablation are described on pp. 732 to 734.

POSTOPERATIVE CARE AND ASSESSMENT

Analgesics (see Chapter 13; for dosages of opioids, see Tables 13.2 and 13.3) and supportive care should be given as necessary. Patients with MCTs should be continued on a proton pump inhibitor or protectant if gastrointestinal ulceration occurs. Adjunctive therapy for malignant tumors may prove beneficial. Patients with malignant tumors should be reevaluated every 3 to 4 months for recurrence or metastasis. Complications associated with paraneoplastic syndromes and metastasis may become evident or persist following castration.

PROGNOSIS

Surgery is curative for most testicular tumors. The prognosis for interstitial cell tumors, Sertoli cell tumors without metastasis or myelotoxicity, and seminomas without signs of hyperestrogenism is excellent. Myelotoxicity may be fatal despite appropriate therapy, but it usually improves within 2 to 3 weeks of tumor removal. Chemotherapy can be instituted if Sertoli cell tumors or seminomas have metastasized. Low-grade scrotal MCTs are less likely to recur or metastasize. Nonresectable or incompletely resected MCTs may respond to radiation therapy or chemotherapy. Efficacy of nonsurgical treatment modalities for other tumors is unknown.

HYPOSPADIAS

DEFINITION

Hypospadias is a developmental anomaly in males in which the urethra opens ventral and caudal to the normal orifice.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Hypospadias is rare, but many affected animals have other congenital or developmental anomalies. It occurs as a result of failure of the genital folds and genital swellings to fuse normally during fetal development. This causes abnormal development of the penile urethra, penis, prepuce, and/or scrotum. Hypospadias is accompanied by hypoplasia of the corpus cavernosum urethra. The urethra opens anywhere along its length at one or more locations. Hypospadias is classified based on the location of the urethral opening as glandular, penile, scrotal, perineal, or anal. The prepuce is similarly affected and ventrally incomplete. In some cases, the penis may be underdeveloped and abnormal (ventral or caudal deviation and blunt) and the scrotum may be divided. Urine may pool within the prepuce, causing irritation and infection of the penis and preputial lining (balanoposthitis).

NOTE Do not use animals with hypospadias for breeding.

DIAGNOSIS

Clinical Presentation

Signalment

Breed predisposition has not been documented, but a familial predisposition has been suggested in Boston terriers. The defect is present at birth.

History

Small defects and those occurring in the glans may not cause problems. Some patients with hypospadias of the glans and abnormal preputial development may be evaluated because of a chronically exposed penis. Larger and more caudal urethral openings cause urine pooling within the prepuce or dermatitis as a result of urine contact. A preputial discharge may occur. There may be a history of urinary incontinence or infection.

Physical Examination Findings

Skin irritation or preputial inflammation may be identified. The preputial opening may be incompletely formed and the scrotum divided. The penis should be completely extruded from the prepuce and examined. A fibrous band may be noted running from the glans to the urethral opening and deviating the penis (persistent frenulum) (Fig. 26.38). The urethral opening is identified on the ventral aspect of the penis along the normal urethral path.

Diagnostic Imaging

Radiographs are unnecessary for diagnosis, but they occasionally identify other congenital anomalies.

Laboratory Findings

Urine culture may be positive. Other laboratory results are nonspecific.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include pseudohermaphroditism, true hermaphroditism, urethral fistula or trauma, persistent penile frenulum, and penile hypoplasia.

MEDICAL MANAGEMENT

Urine scalding should be treated with frequent bathing and application of water-impermeable ointments near the urethral

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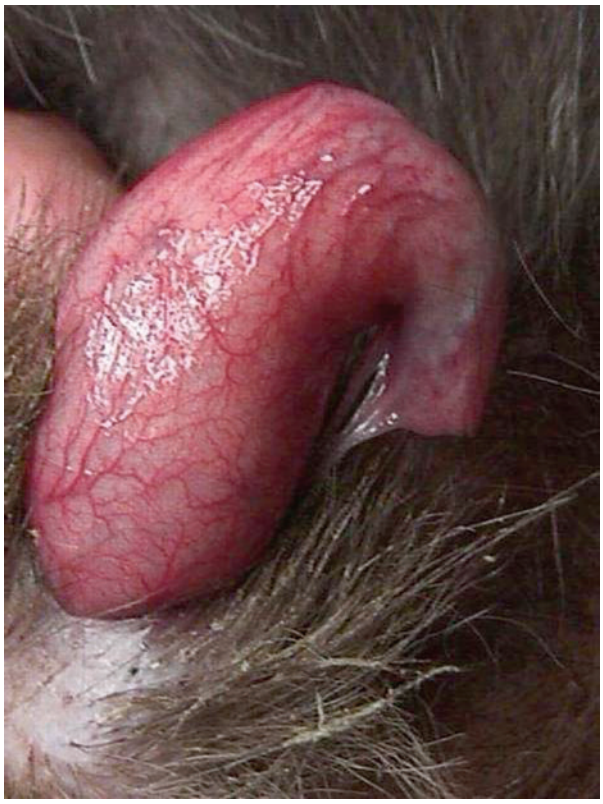


FIG. 26.38 A persistent frenulum is deviating the glans penis ventrally. Note the thin band of fibrous tissue.

opening. The penile mucosa should be kept moist with ointments. If urine pooling occurs, the prepuce should be flushed daily with physiologic saline solution.

SURGICAL TREATMENT

Abnormal urethral openings near the penile tip may not require surgery. In other cases, reconstruction (with or without penile amputation) is advised. Preputial reconstruction is needed in patients with an incompletely formed preputial orifice and hypospadias at the glans. Closing the preputial orifice to its normal extent prevents constant penile exposure. Small urethral defects can be repaired primarily. Excision of the external genitalia (with urethrostomy; see p. 688) is recommended for major developmental defects involving the urethra, prepuce, and penis. Penile amputation is also indicated for severe trauma and neoplasia. Neutering affected animals is recommended.

Preoperative Management

A preoperatively placed urethral catheter facilitates urethral identification.

Anesthesia

Elective surgery on pediatric patients should be delayed until they are approximately 8 weeks old. Recommendations for anesthetic management of pediatrics for reproductive surgery are provided on p. 722 in the general anesthesia section of this chapter.

Surgical Anatomy

Surgical anatomy of the penis is provided on p. 727.

Positioning

Dorsal recumbency is recommended unless the urethra opens in the perineal or anal regions, in which case a perineal position is preferred. The ventral abdomen, medial thighs, and ventral perineum should be clipped and prepared for aseptic surgery.

SURGICAL TECHNIQUE

Prepuce Reconstruction

Incise the mucocutaneous junction on the caudoventral aspect of the prepuce (Fig. 26.39). Separate mucosa from skin. Reappose the mucosa beginning at a more cranial location with simple interrupted monofilament absorbable sutures (e.g., 4-0 to 6-0 polydioxanone [PDS], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], or polyglyconate [Maxon]). Appose skin with a second layer of simple interrupted sutures (e.g., 3-0 or 4-0 nylon, polybutester [Novafil], or polypropylene [Prolene]). If this creates an orifice that is too small to allow penile extrusion, incise the dorsocranial aspect of the prepuce and suture the mucosa to the skin on each side with interrupted sutures (e.g., 4-0 nylon, polybutester, or polypropylene).

Urethral Reconstruction

Expose the defect by extrusion of the penis from the prepuce or by midline preputiotomy. Close small urethral defects by incising the margins of the defect and apposing the urethral edges over a urethral catheter. Use 4-0 to 6-0 monofilament absorbable suture (e.g., polydioxanone [PDS], polyglyconate [Maxon], poliglecaprone 25 [Monocryl], or glycomer 631 [Biosyn]) in a simple interrupted or continuous pattern. Close skin over the urethral repair with 3-0 or 4-0 nonabsorbable suture (e.g., nylon or polypropylene) using an appositional pattern.

Subtotal Penile Amputation

Make an elliptical incision around the prepuce, penis, and scrotum, preserving adequate skin for closure (Fig. 26.40A). Dissect the penis from the body wall from cranial to caudal (Fig. 26.40B). Ligate or cauterize preputial vessels. Perform a castration as with scrotal ablation (see p. 734). Locate and ligate the dorsal penile vessels just caudal to the desired amputation site. Perform a urethrostomy; scrotal urethrostomy (see p. 689) is preferred. Reflect or transect the retractor penis muscle. Make a midline urethral incision over the catheter. Place a circumferential ligature around the penis just caudal to the proposed amputation site and just cranial to the urethrostomy site. Amputate the penis in a wedge fashion (Fig. 26.40C). Appose the tunica albuginea to close the end of the penis with 3-0 or 4-0 absorbable monofilament suture (e.g., polydioxanone [PDS], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], or polyglyconate [Maxon]). Appose urethral mucosa to skin at the urethrostomy site with simple interrupted 4-0 to 6-0 monofilament absorbable or nonabsorbable sutures (e.g., polydioxanone, polyglyconate, poliglecaprone 25, glycomer 631, polybutester [Novafil], polypropylene [Prolene], or nylon) (Fig. 26.40D). Close subcutaneous tissue and skin cranial and caudal to the urethrostomy in two layers (Fig. 26.40E).

POSTOPERATIVE CARE AND ASSESSMENT

Analgesics should be given as necessary (see Chapter 13; for dosages of opioids, see Tables 13.2 and 13.3) and urination monitored by observing for a nonrestricted urine stream. Hemorrhage may occur from cavernous tissue for several days, especially during

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FIG. 26.39 Preputial reconstruction to narrow the orifice is accomplished by incising the mucocutaneous junction *(dashed line)* and reapposing the mucosa and skin in separate layers beginning at a more cranial location.

excitement or urination. Hemorrhage may be minimized by keeping the animal quiet and calm during the early postoperative period. An Elizabethan collar or sidebars should be used to prevent self-trauma. Hemorrhage, urine leakage, infection, seroma, and dehiscence are potential incisional complications. Urethral or preputial reconstruction may cause stricture formation. Urethral stricture may interfere with urine flow and produce obstruction. Preputial stricture may prevent extrusion of the penis.

PROGNOSIS

Hypospadias is not life-threatening; however, penile exposure and urine-induced dermatitis cause discomfort. Surgery usually salvages an animal as a pet, improves cosmesis, and reduces urine-induced dermatitis.

PHIMOSIS

DEFINITION

Phimosis is the inability of the penis to protrude from the prepuce or sheath.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Phimosis is rare. It is usually the result of a preputial opening that is too small or absent, and may be developmental or a result of trauma and preputial stenosis. It may also occur secondary to penile or preputial neoplasia or preputial cellulitis. Inability to extrude the penis causes preputial irritation and infection secondary to urine pooling within the prepuce. Balanoposthitis causes preputial discharge.

DIAGNOSIS

Clinical Presentation

Signalment

There is no known breed predisposition. Congenital phimosis is recognizable in neonates but may go undetected for months. Congenital stenosis has been reported in Bouvier des Flandres, German shepherd dogs, Labrador retrievers, golden retrievers, and mixed-breed dogs. Acquired phimosis may occur at any age.

History

Affected animals may retain urine, dribble urine, or be unable to copulate. Animals without a preputial opening cannot void urine appropriately and have preputial swelling. Acquired cases have a history of lacerations and scarring, sucking of a puppy's prepuce by littermates or licking by the bitch, or neoplasia.

Physical Examination Findings

Affected animals have a small or nonexistent preputial opening. There may be evidence of previous preputial trauma, and a purulent or hemorrhagic preputial discharge is common. The prepuce may be distended with urine, inflamed, and infected. Manual extrusion or palpation of the penis may reveal a mass preventing advancement of the penis.

Diagnostic Imaging

Imaging is unnecessary unless neoplasia is suspected.

Laboratory Findings

Results of laboratory tests are nonspecific. Preputial cytology may reveal inflammation and infection. Bacteria may be cultured from the prepuce.

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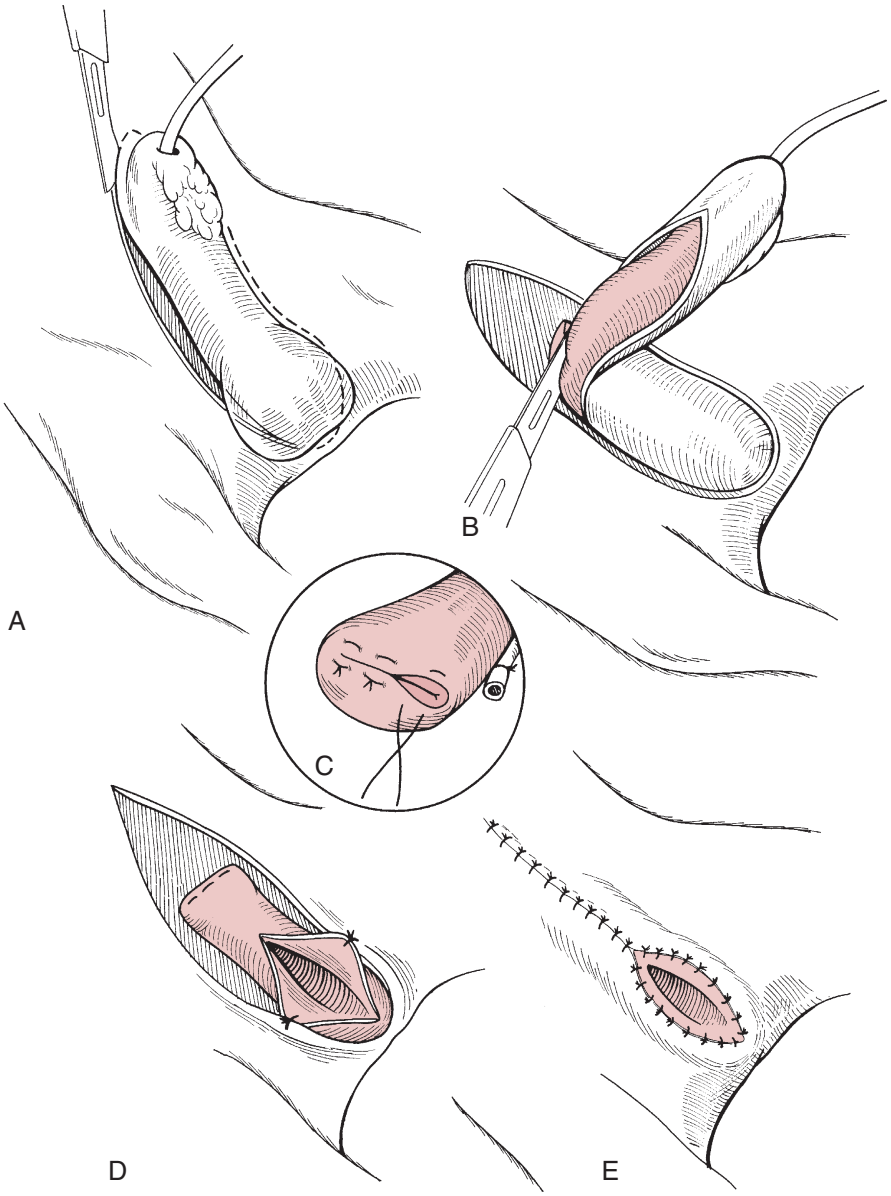


FIG. 26.40 Penile neoplasia, trauma, or congenital anomalies may be treated by subtotal penile amputation and scrotal urethrostomy. (A) Make an elliptical incision around the base of the scrotum and prepuce and remove the testicles. (B) Separate the penis from the external abdominal wall to just caudal to the os penis. (C) Amputate the distal penis and appose the tunica albuginea over the cavernous tissue. (D–E) Incise the urethra in the scrotal area and suture urethral mucosa to skin.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include penile hypoplasia, persistent frenulum, and hermaphroditism.

MEDICAL MANAGEMENT

Phimosis caused by an inflammatory or infectious disease may be relieved by warm compresses, antibiotic therapy, and urine diversion with a catheter. The prepuce should be lavaged daily with physiologic saline solution to reduce urine scalding.

SURGICAL TREATMENT

Phimosis caused by a developmental anomaly or stricture is managed by reconstruction of the preputial orifice. The goal of surgery is to enlarge the preputial orifice and allow unrestricted movement of the penis in and out of the prepuce.

NOTE Recommend neutering animals with small preputial openings.

Preoperative Management

The prepuce should be lavaged with a dilute antiseptic solution before surgery and a urinary catheter placed to divert urine.

Anesthesia

Anesthetic recommendations for animals undergoing reproductive surgery are provided in Table 26.3.

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Surgical Anatomy

Surgical anatomy of the male reproductive system is provided on p. 726.

Positioning

The patient is positioned in dorsal recumbency and the end of the prepuce clipped and prepared for aseptic surgery.

SURGICAL TECHNIQUE

Enlarge the preputial opening by making a full-thickness incision at the craniodorsal aspect of the prepuce. Determine the desired length and width of the preputial incision based on the severity of phimosis. Remove a small wedge (3–5 mm) of prepuce with the base at the mucocutaneous junction (Fig. 26.41A). Appose mucosa to the ipsilateral skin edge on each side with a simple interrupted suture pattern (e.g., 4-0 to 6-0 polydioxanone [PDS], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], or polyglyconate [Maxon]) (Fig. 26.41B–C). Extrude the penis completely to examine for other developmental defects, injuries, or masses.

Amputate the tip of the prepuce if stenosis is too long to be relieved by incision and if adequate preputial length can be maintained. Amputation may cause a shortened prepuce, allowing chronic penile protrusion and exposure. *Identify the site of resection and amputate the preputial tip (Fig. 26.42A). Circumferentially appose preputial mucosa to skin with a simple interrupted or continuous suture pattern (e.g., 4-0 to 6-0 polydioxanone, poliglecaprone 25, glycomer 631, or polyglyconate) (Fig. 26.42B–C). Neuter affected animals.*

POSTOPERATIVE CARE AND ASSESSMENT

Analgesics and supportive care should be given as needed. Warm compresses and antibiotics may be used to treat balanoposthitis. An Elizabethan collar, bucket, or sidebars should be used to prevent self-trauma. Phimosis may persist if the incision is not long enough. Persistent protrusion of the glans may occur if the ventrocaudal prepuce is incised. Self-trauma may cause dehiscence and stricture formation.

PROGNOSIS

Without surgery, balanoposthitis may become severe and cause discomfort. A second surgical procedure may be necessary after the animal matures.

PARAPHIMOSIS

DEFINITION

Paraphimosis is the inability to retract the penis into the sheath or prepuce. **Priapism** is persistent erection of the penis without sexual excitement. **Phallopexy** is a procedure to create a permanent adhesion between the penile shaft and preputial mucosa.

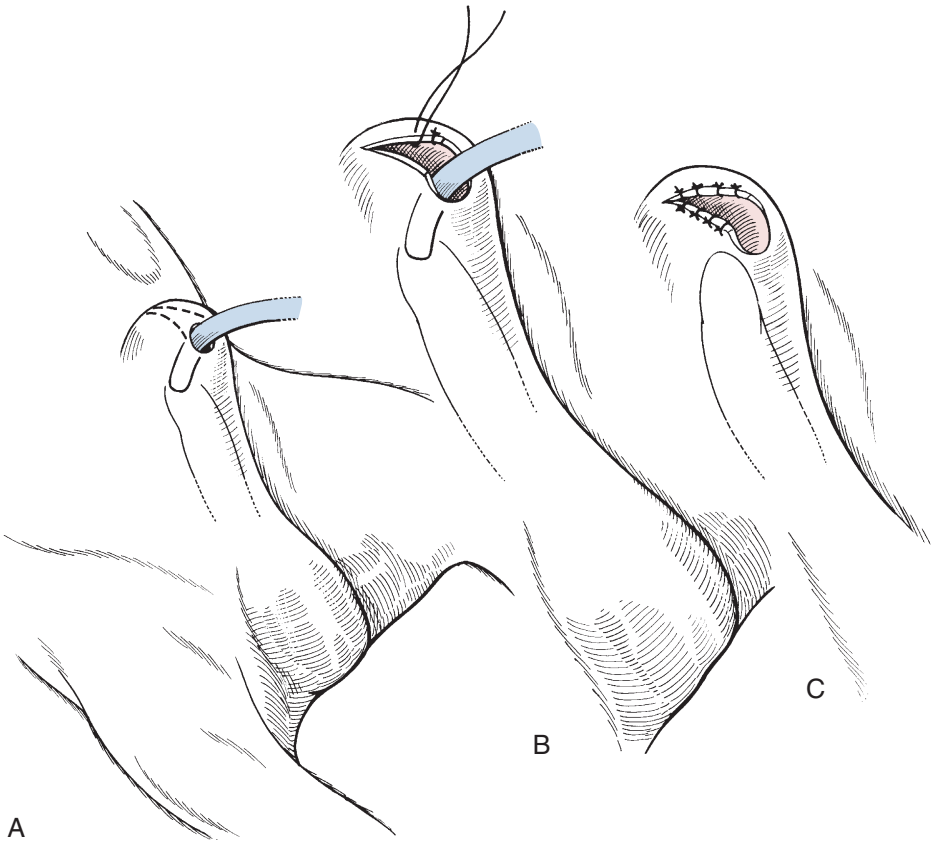


FIG. 26.41 (A) For phimosis, enlarge the preputial orifice by resecting a full-thickness wedge from the craniodorsal aspect *(dashed line)*. (B–C) Appose mucosa to the ipsilateral skin edge on each side.

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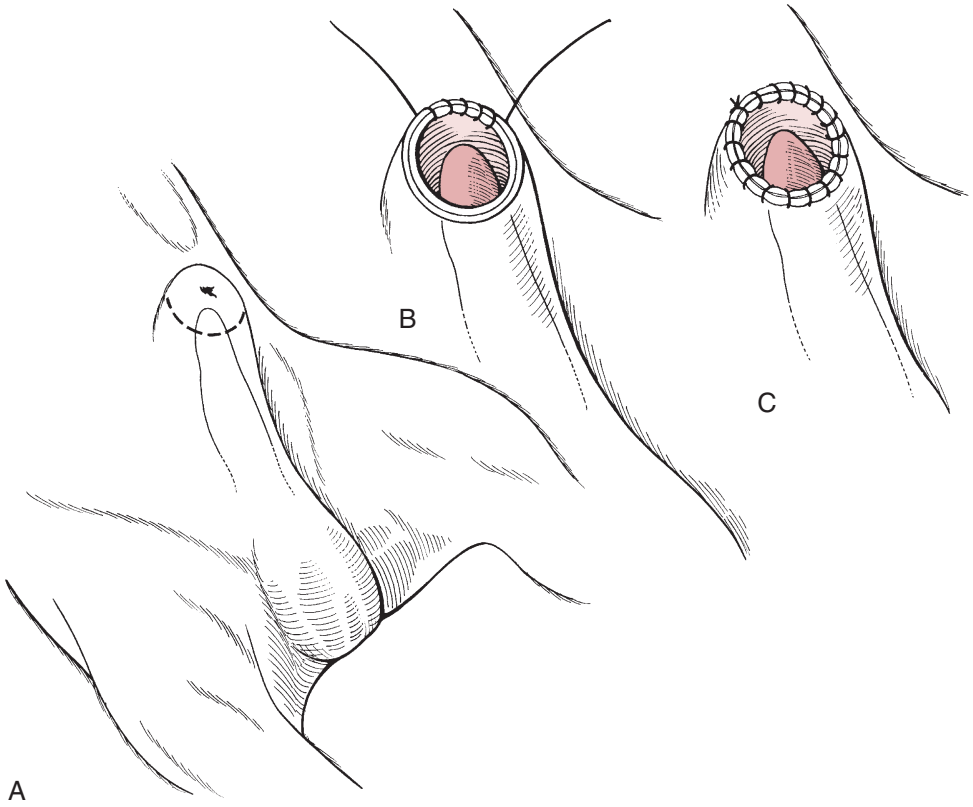


FIG. 26.42 (A) To enlarge the preputial orifice, resect the tip of the prepuce and (B–C) suture preputial mucosa to skin.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Paraphimosis may be associated with copulation, masturbation, trauma, penile hematoma, neoplasia, foreign bodies, pseudohermaphroditism, neurologic deficits, or constriction by preputial hairs (Fig. 26.43). The penis may be unable to retract within the prepuce because the edges of the prepuce roll inward or the preputial orifice is too small to accommodate the swollen or engorged penis. Initially the penis appears normal. However, when the penis cannot be retracted, it is easily traumatized and circulation is impaired. Impaired circulation causes the penis to become edematous, which further compromises circulation (Fig. 26.44). Vascular engorgement may progress to thrombosis of the corpus spongiosum and necrosis. A moderately compromised, chronically protruded penis will become dry, fissured, and cornified.

DIAGNOSIS

Clinical Presentation

Signalment

The condition occurs more often in dogs than in cats. Sexual hyperactivity preceding paraphimosis may be noted in young dogs.

History

Paraphimosis can result from priapism, masturbation, or excessive sexual activity. Canine paraphimosis occurs most commonly after an erection. It may occur in long-haired cats if the penis becomes entangled in hairs. It may also be associated with posterior paralysis or the inability of preputial muscles to pull the prepuce over the penis after an erection.

Physical Examination Findings

Paraphimosis is diagnosed by visual inspection. The exposed, swollen, edematous penis is painful (see Fig. 26.44). The traumatized penis may be fissured, lacerated, and/or bleeding. The animal may require sedation or anesthesia before penile examination. The severity of penile trauma and vascular compromise should be determined. The prepuce should be evaluated to determine whether it is too short or whether the orifice is too small or too large. The retracted penis should normally be covered by at least 1 cm of prepuce cranial to its termination.

Diagnostic Imaging

Images are unnecessary unless urethral trauma is suspected.

Laboratory Findings

Laboratory findings are nonspecific.

DIFFERENTIAL DIAGNOSIS

Paraphimosis must be differentiated from priapism, vascular thrombosis, chronic urethritis, stretching or weakness of the retractor penis muscles, and hypoplastic or surgically damaged preputial muscles. Mechanical, vascular, or neural causes should be suspected when the penis is easily reduced.

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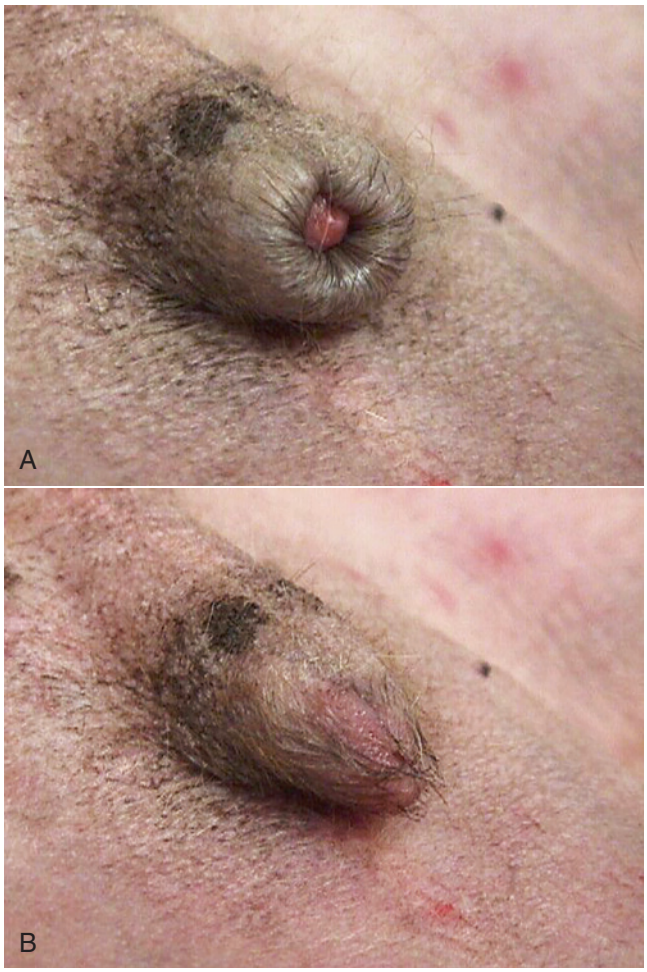


FIG. 26.43 Mild paraphimosis. (A) The tip of the glans is protruding from the prepuce and unable to retract as a result of inversion of the skin. (B) Reduction was accomplished by caudal traction on the prepuce to unroll the inverted skin.



FIG. 26.44 Penile trauma, inflammation, and edema secondary to paraphimosis.

MEDICAL MANAGEMENT

Initially, the prepuce should be pulled back until it unfolds and the preputial mucocutaneous junction is identified. This allows restoration of penile circulation and resolution of edema. The penis should be examined carefully for constricting foreign bodies and the protruded, edematous penis cleaned with warm physiologic saline solution or water. To reduce edema, gently massage the penis and apply a hypertonic or hygroscopic agent (e.g., granulated sugar or dextrose). Glucocorticoids and diuretics may reduce edema after the constriction is relieved. When swelling has decreased, the prepuce should be flushed with a mild antiseptic soap or lubricant, and the preputial edges dilated or retracted, thus allowing the penis to retract within the prepuce.

SURGICAL TREATMENT

Patients with acute paraphimosis are often managed conservatively. Others may require preputial reconstruction, phallopexy, or penile amputation. A preputiotomy may be necessary to allow retraction of the penis into the prepuce if conservative measures fail. If the prepuce is of adequate length and the orifice too large, narrow it (see Fig. 26.39). Enlarge the preputial opening if the prepuce is of adequate length and the orifice too small (see Figs. 26.41 and 26.42). When the prepuce is too short, it may be lengthened or the penis may be amputated (see Fig. 26.40). Preputial deficiencies of less than 1 to 2 cm may be corrected by cranial advancement of the prepuce. Partial amputation of the penis is indicated for severe trauma or abnormalities of the penis or prepuce, neoplasia, recurring urethral prolapse, and recurring paraphimosis. Partial penile amputation is applicable when the site of transection is cranial to the caudal end of the os penis. Castration is recommended to prevent recurrence of paraphimosis because of sexual activity. Phallopexy is indicated for cases of recurrent paraphimosis or as the initial surgical treatment.

NOTE Castration may prevent recurrent paraphimosis caused by sexual activity.

Preoperative Management

See the discussion on medical treatment presented earlier.

Anesthesia

Anesthetic recommendations for animals with reproductive disorders are provided in Table 26.3.

Surgical Anatomy

The prepuce covers the nonerect penis in dogs and cats. The dog's prepuce should normally extend approximately 1 cm beyond the end of the penis.

Positioning

Position the patient in dorsal recumbency. Clip the prepuce and surrounding skin and prepare them for aseptic surgery.

SURGICAL TECHNIQUE

Preputiotomy

Make a full-thickness dorsal or ventral linear incision in the prepuce. If the preputial orifice is of normal size, anatomically reappose

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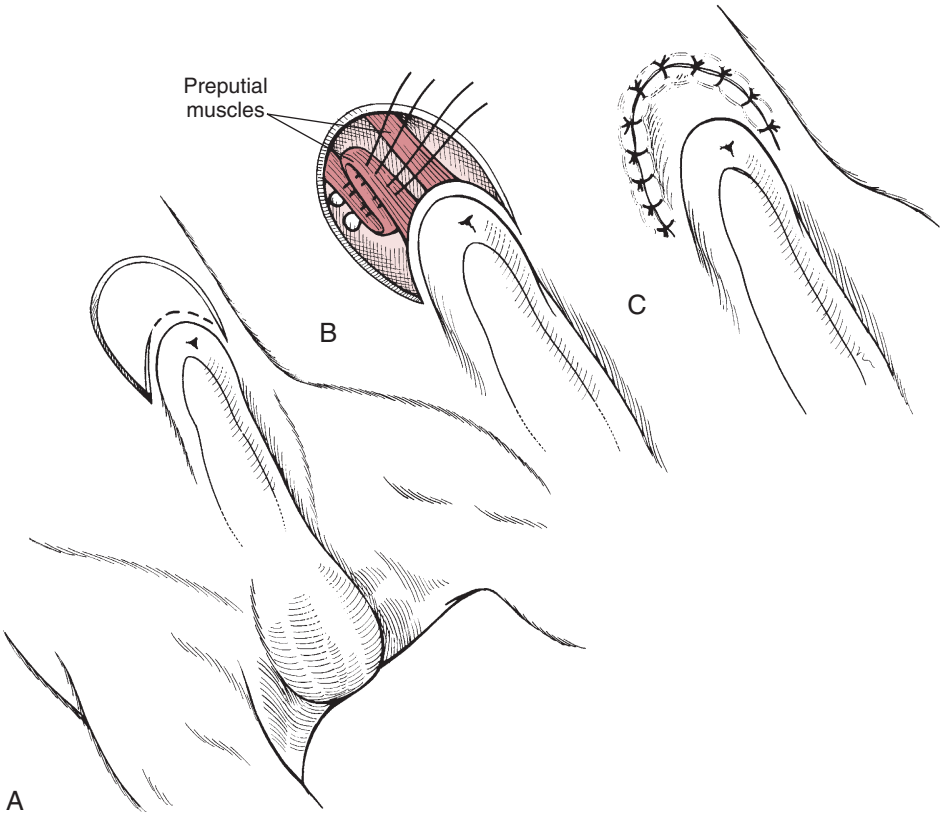


FIG. 26.45 (A) Lengthen the prepuce by removing a crescent-shaped piece of skin cranial to the prepuce, (B) shortening the preputial muscles, and (C) advancing the prepuce cranially.

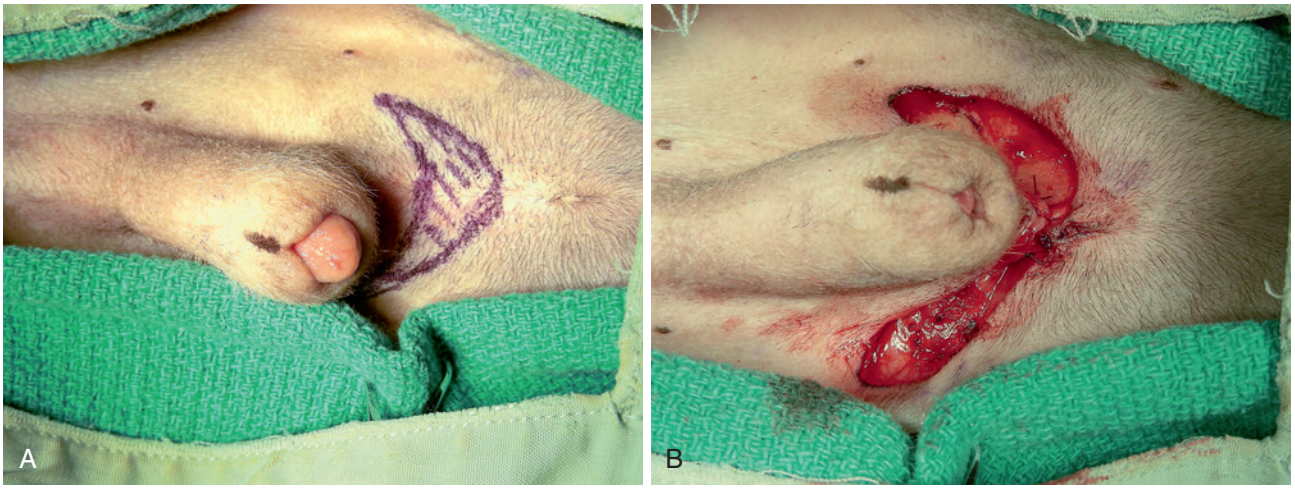


FIG. 26.46 Intraoperative image of preputial lengthening in a 7-year-old Chihuahua with mild paraphimosis. (A) Proposed incision. (B) Shortening of preputial muscles.

mucosa (e.g., 4-0 to 6-0 polydioxanone [PDS], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], or polyglyconate [Maxon] approximating sutures) and skin (e.g., 3-0 or 4-0 nylon, polybutester [Novafil], or polypropylene [Prolene] approximating sutures) in separate layers.

Preputial Lengthening

Lengthen or translocate the prepuce cranially by resecting a crescent-shaped piece of skin from the body wall just cranial to the prepuce (Fig. 26.45A and Fig. 26.46A). Preserve the preputial vessels. Identify the preputial muscles and shorten them by overlapping and suturing or by segmental excision and reapposition (Figs. 26.45B and 26.46B). Close subcutaneous tissue and skin in two layers to further advance the skin cranially (Fig. 26.45C).

Alternatively, the prepuce can be lengthened with a two-stage procedure in which oral mucosa is transplanted cranial to the prepuce and later rolled into a tube to cover the end of the penis.

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Partial Penile Amputation

Place a urethral catheter to facilitate orientation and prevent urethral trauma. Extrude the penis from the prepuce and maintain this position by snugly closing the preputial orifice around the penis with a towel clamp. Place a Penrose drain tourniquet caudal to the proposed amputation site. Make a lateral V-shaped incision through the tunica albuginea and cavernous tissue on each side of the urethra and os penis (Fig. 26.47A). Transect the os penis with bone cutters as far caudally as possible, being careful not to traumatize the urethra (Fig. 26.47B). Transect the urethra 1 to 2 cm cranial to the penile transection and spatulate the dorsal aspect. Identify and ligate the dorsal artery of the penis after loosening the tourniquet. Fold the spatulated urethra over the transected end of the penis (Fig. 26.47C). Appose urethral mucosa to the tunica albuginea; include some cavernous tissue with each bite (Fig. 26.47D). Use 4-0 to 6-0 monofilament absorbable suture (i.e., polydioxanone [PDS], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], or polyglyconate [Maxon]) with a swaged-on, taper point needle in a simple interrupted or continuous pattern. Shorten the prepuce if the new penile tip cannot be extruded from the prepuce; the prepuce should extend approximately 1 cm cranial to the retracted penis.

Resect an ellipse of prepuce approximately the same length as the amount of penis that was amputated (Fig. 26.47E). Make an elliptical, transverse, full-thickness incision in the midportion of the prepuce (beginning approximately 2 cm caudal to the cranial junction of the prepuce and body wall). Remove this ventral skin

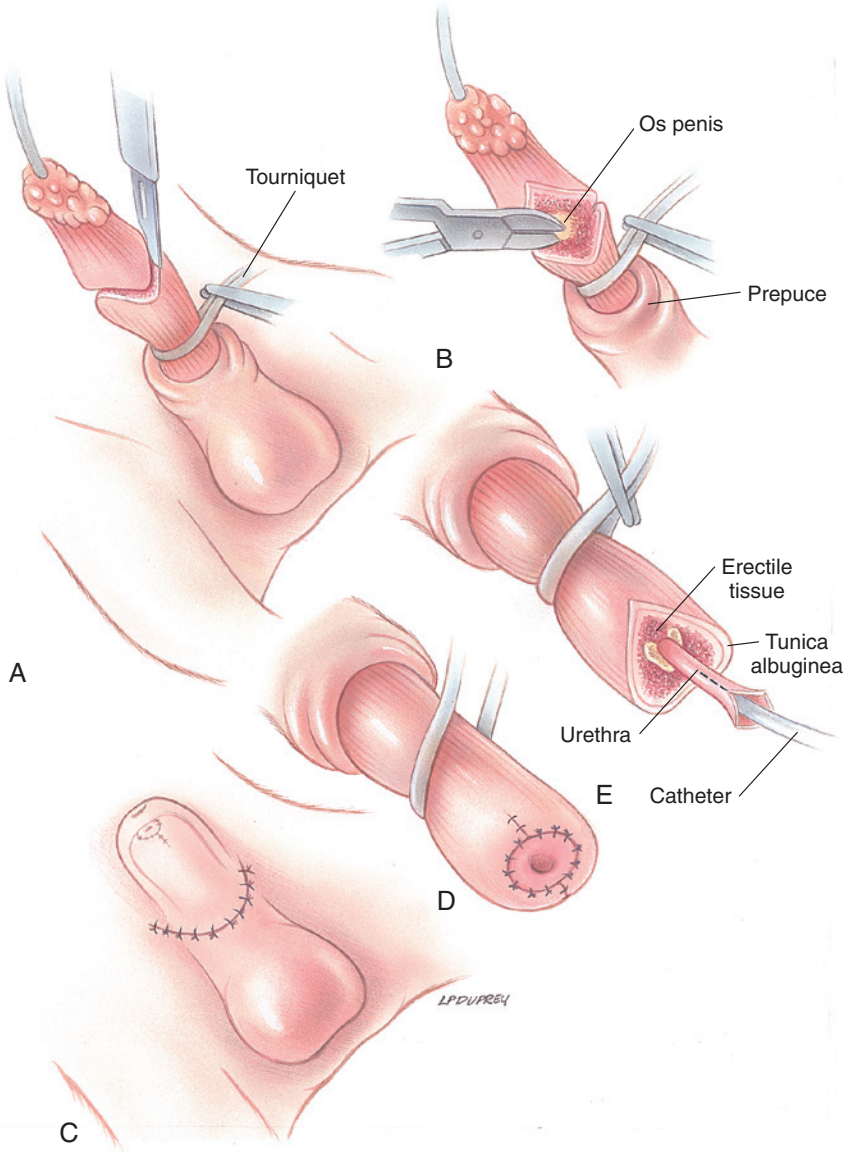


FIG. 26.47 (A) During partial penile amputation, retract the prepuce, place a tourniquet around the penis, and make a lateral V-shaped incision through the tunica albuginea and cavernous tissue. (B) Transect the os penis as far caudal as possible. (C) Transect the urethra 1 to 2 cm cranial to the penile transection and spatulate the end. (D) Appose urethral mucosa to the tunica albuginea. (E) Shorten the prepuce to allow extrusion of the distal penis by removing a full-thickness segment from the midsection.

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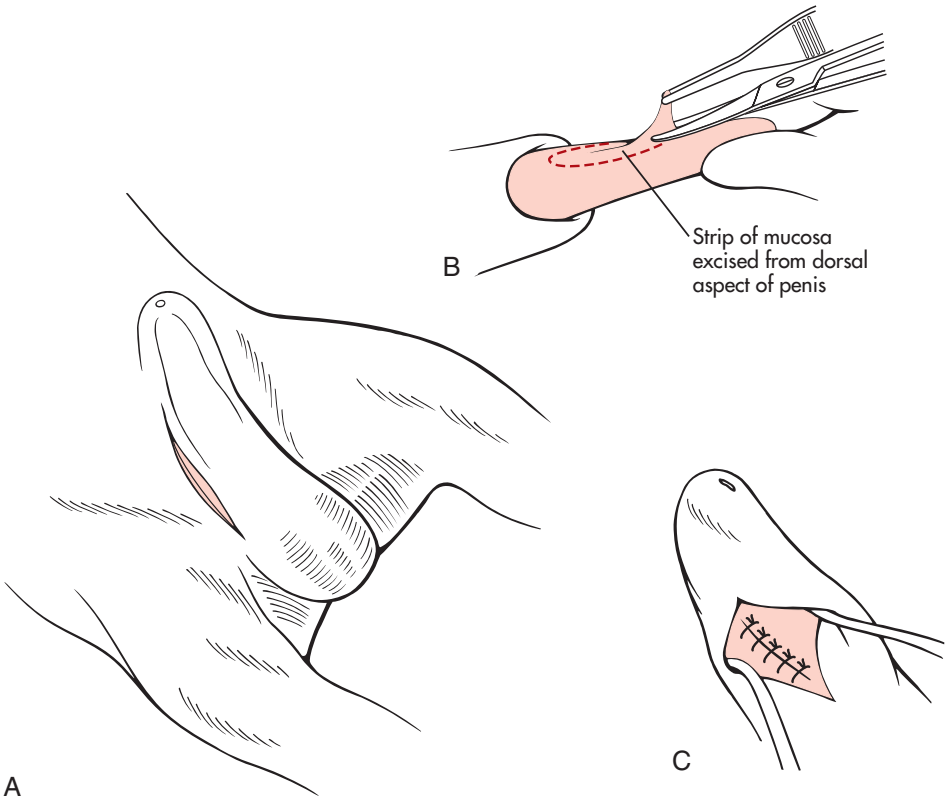


FIG. 26.48 Phallopexy. (A) Perform a preputiotomy, beginning 2.5 cm caudal to the preputial orifice, by making a full-thickness incision into the prepuce at the junction of the prepuce and body wall. Extend the incision caudally 2 to 3 cm. (B) After excising a strip of preputial mucosa from the dorsal midline of the preputial lumen, exteriorize the penile shaft through the preputial orifice and excise a strip of mucosa 0.5 cm wide and 1.5 cm long from the dorsal midline of the midportion of the glans penis beginning 2 cm caudal to the tip. (C) Replace the penis within the prepuce and appose the penile and preputial incisions with simple interrupted 3-0 or 4-0 monofilament absorbable sutures.

and mucosal segment, reflect the penis caudally, and resect a similar segment of dorsal preputial mucosa. Close the defect by first apposing the dorsal and then ventral preputial mucosa with 4-0 or 5-0 monofilament absorbable suture (e.g., polydioxanone, poliglecaprone 25, glycomer 631, or polyglyconate) in a simple interrupted or continuous pattern. Then appose skin with approximating 3-0 or 4-0 nonabsorbable sutures (e.g., nylon, polypropylene [Prolene], or polybutester [Novafil]).

Phallopexy

Phallopexy prevents complete exteriorization of the glans, thus eliminating the risk of recurrent paraphimosis and subsequent penile trauma. *Perform a preputiotomy beginning 2.5 cm caudal to the preputial orifice by making a full-thickness incision into the prepuce at the junction of the prepuce and body wall (Fig. 26.48A). Extend the incision caudally 2 to 3 cm. Retract the penis laterally. Beginning 3 cm caudal to the preputial opening, excise a strip of preputial mucosa 0.5 cm wide by 1.5 cm long from the dorsal midline of the preputial lumen. Then exteriorize the penile shaft through the preputial orifice and excise a strip of mucosa 0.5 cm wide and 1.5 cm long from the dorsal midline of the midportion of the glans penis, beginning 2 cm caudal to the tip (Fig. 26.48B). Care is taken to prevent incising the pars longa glandis. Replace the penis within the prepuce, and appose the penile and preputial incisions with simple interrupted 3-0 or 4-0 monofilament absorbable sutures. Place the most cranial sutures first, then retract the prepuce caudally to determine if the glans can be exteriorized. If exteriorization is possible, remove the sutures, extend the preputial mucosal excision caudally, and begin suturing at a more caudal location. Complete the pexy by placing six to eight sutures between the incised edges of the penile and preputial mucosa (Fig. 26.48C). Close the preputiotomy incision in layers: preputial mucosa, subcutaneous tissue, and skin. Perform a castration (see p. 732).*

POSTOPERATIVE CARE AND ASSESSMENT

Analgesics should be given as necessary; an Elizabethan collar, bucket, or sidebars should be used to prevent self-trauma. Avoid exposure that will provoke excitement or sexual stimulation. Hemorrhage usually occurs during urination or excitement for several days after penile amputation. Persistent licking and signs of discomfort are not seen after phallopexies have healed, and balanoposthitis has not been reported following this procedure.

COMPLICATIONS

Dehiscence, stricture, infection, hemorrhage, urine retention, and recurrence are potential complications. Ventral preputial

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incisions may lead to chronic exposure of the glans penis. Preputial advancements may relax postoperatively, allowing the distal penis to be reexposed.

PROGNOSIS

The prognosis with manual reduction or reconstruction plus castration is good; however, recurrence is common if the animal is not castrated. Prognosis following phallopexy is good.

PENILE AND PREPUTIAL TRAUMA AND NEOPLASIA

DEFINITIONS

A penile hematoma is a localized collection of blood that accumulates secondary to laceration or puncture of the cavernous tissue.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The prepuce and penis may be traumatized by animal bites, vehicular or other accidents, and human attacks (Fig. 26.49). Trauma may cause penile hematomas or os penis fracture. The hematoma swelling may cause the penis to protrude. Lacerations or punctures may bleed for days.

Neoplasms commonly found on skin occur on the prepuce (Box 26.34). Neoplasms of the penis and mucosal lining of the prepuce include TVTs, squamous cell carcinomas, hemangiosarcomas, and papillomas. TVTs are contagious and are spread by sexual contact or licking. They are wartlike, friable, and bleed easily (Fig. 26.50).



FIG. 26.49 Penile necrosis that occurred after trauma and necessitated partial penile amputation and preputial reconstruction.

BOX 26.34 Preputial Tumors

Benign

Hemangiomas
Papillomas
Histiocytomas

Malignant

Melanomas
Mast cell tumors
Hemangiosarcomas
Squamous cell carcinomas

DIAGNOSIS

Clinical Presentation

Signalment

Trauma and tumors are more common in intact males. Young animals are more commonly traumatized; old animals more commonly have tumors.

History

Signs of penile or preputial disease include serosanguineous, hemorrhagic, or purulent preputial discharge, inability or unwillingness to copulate, or pain. Some animals have phimosis (see p. 777) or paraphimosis (see p. 779). The urethra may be obstructed or lacerated, which causes dysuria, anuria, or urine extravasation. Many dogs are asymptomatic.

Physical Examination Findings

An injury or abnormal mass may be detected on physical examination. The prepuce may appear swollen, inflamed, nodular, lacerated, ischemic, and/or necrotic. There may be diffuse inguinal swelling because of the leakage of urine from the urethra into surrounding tissue. Abnormalities involving preputial skin are usually apparent; preputial mucosal lesions may be detected only by palpation. It may be impossible to exteriorize the penis for examination if there is a mass within the prepuce or on the penis. In other cases, paraphimosis is present because of injury causing inflammation, edema, and engorgement (or a mass prevents penile retraction). Penile deviation may occur secondary to traumatic fractures of the os penis. Rectal palpation may reveal medial iliac lymphadenomegaly.

Diagnostic Imaging

Abdominal and thoracic radiographs are indicated to stage tumors. Survey radiographs may reveal os penis fractures. A urethrogram may help assess urethral involvement with penile trauma or tumors. Ultrasonography may be useful to provide information if the penis cannot be exteriorized.

Laboratory Findings

Laboratory results are nonspecific for penile trauma or neoplasia. Cytology of preputial discharges may show toxic neutrophils, excess bacteria, fungi, or foreign material, but these findings can be found in normal animals. Cytology of preputial or penile



FIG. 26.50 Penile transmissible venereal tumors.

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masses may help identify tumor type. TVTs have large round cells with numerous mitotic figures.

DIFFERENTIAL DIAGNOSIS

Hematomas, abscesses, granulomas, and fungal infections may cause similar lesions.

MEDICAL MANAGEMENT

Some injuries heal spontaneously. Penile hematomas should be allowed to resolve spontaneously unless they cause persistent paraphimosis. TVTs are treated with vincristine (0.5 mg/m^2 IV or 0.025 mg/kg up to 1 mg IV) weekly for 3 to 6 weeks. Alternatively, radiation therapy is effective against TVTs resistant to chemotherapy and at metastatic sites.

SURGICAL TREATMENT

TVTs respond well to chemotherapy or radiation therapy; however, other tumors should be resected. Partial or complete penile amputation is necessary for severely traumatized, necrotic, or neoplastic lesions (see pp. 776 and 783). Hematomas causing persistent paraphimosis can be surgically exposed and evacuated; however, they may recur. Os penis fractures with minimal displacement require no surgery. Displaced fractures may be splinted with an indwelling polypropylene urethral catheter spanning the os penis and sutured to the tip of the urethra. More comminuted fractures may be stabilized with small plates, or the penis may be amputated.

Preoperative Management

The prepuce and penis should be lavaged with dilute antiseptic solutions. Antibiotics should be given if the penile or preputial tissue is severely damaged or necrotic.

Anesthesia

Anesthetic management of animals undergoing reproductive surgery is provided in Table 26.3.

Surgical Anatomy

Surgical anatomy of the male reproductive tract is provided on p. 726.

Positioning

Position the patient in dorsal recumbency. The ventral abdomen, ventral perineum, and medial thighs should be clipped and prepared for aseptic surgery.

SURGICAL TECHNIQUE

Preputial Lacerations

Debride, lavage, and appose preputial lacerations. Close full-thickness injuries in two layers, first apposing the preputial mucosa (e.g., 4-0 to 6-0 polydioxanone [PDS], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], or polyglyconate [Maxon]), subcutaneous tissue (3-0 or 4-0 monofilament absorbable), and then skin (e.g., 3-0 or 4-0 nylon, polybutester [Novafil], or polypropylene [Prolene]) with approximating sutures. Hematomas that cause persistent paraphimosis can be surgically exposed and evacuated. *Incise the tunica albuginea over the hematoma. Remove blood clots and fibrin. Lavage the cavity and snugly appose the tunica albuginea. Take care to maintain an adequate preputial orifice diameter and length when reconstructing preputial lacerations.*

Penile Lacerations or Punctures

Suture the tunica albuginea to close penile lacerations or punctures, and minimize hemorrhage during excitement or urination. Use 4-0 to 6-0 monofilament absorbable (e.g., polyglyconate [Maxon], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], or polydioxanone [PDS]), simple interrupted sutures on a swaged-on, taper point needle. Bleeding from small penile punctures or lacerations during penile engorgement is minimized by suturing the tunica albuginea.

POSTOPERATIVE CARE AND ASSESSMENT

Analgesics and antibiotics should be given as needed and the animal monitored for hemorrhage or urine leakage. Apply cold compresses following treatment of trauma or hematomas to reduce hemorrhage. An Elizabethan collar, bucket, or sidebars are used to prevent self-trauma. Reevaluation for tumor recurrence or metastasis should be performed every 3 to 4 months for 1 year. Hemorrhage, seroma, infection, urine leakage, dehiscence, stricture, recurrence, and metastasis are potential complications. Urethral obstruction may occur because of callus formation following os penis fractures.

COMPLICATIONS

Some injuries heal by secondary intention without complications; however, nonsutured preputial lacerations may fistulate. In other cases, persistent hemorrhage, urine extravasation, infection, and stricture may cause morbidity.

PROGNOSIS

The prognosis is good following appropriate surgical treatment for most injuries. The prognosis following tumor excision depends on the biological behavior of the tumor and the tumor stage at presentation.

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27

Surgery of the Cardiovascular System



GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Cardiac surgery includes procedures performed on the pericardium, cardiac ventricles, atria, venae cavae, aorta, and main pulmonary artery. **Closed cardiac procedures** (i.e., those that do not require opening major cardiac structures) are most commonly performed; however, some conditions require **open cardiac surgery** (i.e., a major cardiac structure must be opened to accomplish the repair). Open cardiac surgery necessitates that circulation be arrested during the procedure by inflow occlusion or cardiopulmonary bypass. **Venous inflow occlusion** provides brief circulatory arrest, allowing short procedures (<4 minutes) to be performed. Longer open cardiac procedures require establishing an extracorporeal circulation by **cardiopulmonary bypass** to maintain organ perfusion during surgery.

PREOPERATIVE MANAGEMENT

Animals requiring cardiac surgery often have prior cardiovascular compromise that should be corrected or controlled medically when possible before anesthetic induction (Box 27.1). Congestive heart failure (CHF), particularly pulmonary edema, should be managed with diuretics (e.g., furosemide) and angiotensin-converting enzyme (ACE) inhibitors (e.g., enalapril, benazepril, lisinopril) and an inodilator (pimobendan) before surgery. Cardiac arrhythmias should be recognized and treated (see also the discussion under Postoperative Care and Assessment). Ventricular tachycardia should be suppressed before surgery with class I antiarrhythmic drugs (i.e., lidocaine and procainamide). Other antiarrhythmic drugs to consider include sotalol and amiodarone. Lidocaine is effective for management of ventricular tachyarrhythmias during and immediately after surgery. Supraventricular tachycardia may require β-adrenergic blockers (e.g., esmolol, propranolol, atenolol) or calcium channel–blocking drugs (e.g., diltiazem) before surgery. Atrial fibrillation should be controlled before surgery with a β-blocker or a calcium channel blocker with or without digoxin to lower the ventricular response rate to below 140 beats/min. Alternatively, amiodarone may be used to control the ventricular response rate, and in a small percentage of cases, to convert atrial fibrillation to normal sinus rhythm. Animals with bradyarrhythmias should undergo an atropine or glycopyrrolate response test before surgery. If bradycardia is not responsive to atropine or glycopyrrolate, temporary transvenous pacing or constant intravenous (IV) infusion of isoproterenol (see management of bradycardia on p. 828) may be required.

All animals should undergo a complete echocardiographic evaluation before cardiac surgery; an incomplete or inaccurate diagnosis can have devastating consequences. With the availability of Doppler echocardiography, cardiac catheterization is rarely necessary before cardiac surgery.

ANESTHESIA

Anesthesia of the patient with cardiac compromise has risks that vary depending on the cause of the underlying disease. For example, the anesthetic protocol that is safest for the patient with mitral regurgitation (MR) can be dangerous for the patient with aortic stenosis (AS). The pathophysiology of the patient's cardiac condition needs to be fully understood. Likewise, the practitioner needs to have a working knowledge of the pharmacology of the drugs used to manipulate heart rate and blood pressure. Although cardiac surgery is typically performed at veterinary teaching hospitals and referral institutions, veterinarians from a variety of practices are required to anesthetize the patient with heart disease. See p. 800 for a discussion of anesthesia in patients with MR and p. 810 for information on anesthesia in the patient with subaortic stenosis (Tables 27.1 to 27.3). See also the discussion of anesthesia for the patient with cardiac tamponade on p. 821.

Preanesthetic medication is appropriate for most animals undergoing cardiac surgery. Parenteral opioids (i.e., hydromorphone, oxymorphone, butorphanol, buprenorphine, and fentanyl) induce sedation with minimal cardiovascular effects; however, all opioids can produce respiratory depression and/or bradycardia. Alpha-2 agonists (e.g., dexmedetomidine) and acepromazine should be avoided in cardiac patients owing to significant alterations in hemodynamic parameters associated with their administration. Anticholinergics (i.e., atropine and glycopyrrolate) should be administered only as needed. Carefully consider whether an elevated heart rate will assist or hinder forward flow of blood in the individual patient. Benzodiazepines (e.g., diazepam 0.2 mg/kg, midazolam 0.2 mg/kg) have minimal cardiopulmonary effects and enhance sedation when given alone or combined with opioids. Some patients may have an unpredictable behavioral response (e.g., excitation, aggressiveness) to benzodiazepine administration; therefore they are often used in combination with an opioid.

Induction of anesthesia should be undertaken with caution in animals with cardiopulmonary compromise. Propofol produces rapid induction but a dose-dependent cardiac depression. The addition of fentanyl decreases propofol requirements in healthy dogs with less alteration in cardiovascular parameters. Alfaxalone induction, with or without the addition of fentanyl, has a less negative chronotropic effect than fentanyl.[1] An anesthetic study in sick dogs showed that induction of anesthesia with alfaxalone resulted in similar cardiorespiratory effects when compared with the fentanyl-diazepam-propofol combination and was found to

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ABSTRACT

Cardiac surgery requires a thorough knowledge of cardiovascular anatomy, physiology, instrumentation, and suture material. Conditions requiring surgical intervention can be congenital (e.g., patent ductus arteriosus, pulmonic stenosis) or degenerative (e.g., mitral valve disease). Procedures can be performed on a closed, beating heart or on a open heart using either venous inflow occlusion or cardiopulmonary bypass. Some conditions can be addressed through interventional methods not requiring a thoracotomy approach. Regardless, medical management of any cardiovascular compromise should be optimized if possible prior to general anesthesia.

KEYWORDS

cardiac surgery
inflow occlusion
cardiopulmonary bypass
congestive heart failure
arrhythmias
mitral valve disease
patent ductus arteriosus
pulmonic stenosis
aortic stenosis
ventricular septal defect
atrial septal defect
tetralogy of Fallot
pericardial effusion
pericardial constriction
bradycardia
pacemaker

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BOX 27.1 Selected Drugs Used in the Management of Animals With Cardiac Disease

Diuretics

Furosemide (Lasix)

2–4 mg/kg PO, IV, SC q6–24h as needed, or can give in severe heart failure as a CRI (1–2 mg/kg/h)

Spironolactone

1–2 mg/kg PO q12–24h, not to exceed 4 mg/kg/d

Antihypertensives

Amlodipine (Norvasc)

0.25–0.75 mg/kg PO q24h

Benazepril (Lotensin)

Dogs: 0.25–0.5 mg/kg PO q12–24h
Cats: 0.25–0.5 mg/kg PO q24h

Diltiazem (Cardizem)

Dogs: 0.5–1.5 mg/kg PO q8h
Extended release (Dilacor) 2–4 mg/kg PO q12h
Cats: 1.75–2.4 mg/kg PO q8h of immediate-release formulation
Once-daily formulation (Dilacor, Cardizem CD) 10 mg/kg PO

Esmolol (Brevibloc)

0.05–0.1 mg/kg slow IV bolus q 3–5 min or 50–200 µg/kg/min CRI

Atenolol (Tenormin)

Dogs: 0.1–2.0 mg/kg PO q12–24h (gradually up-titrate the dose)
Cats: 0.5–5 mg/kg PO q12–24h

Antiarrhythmics

Intravenous Antiarrhythmic Drugs: Ventricular

Lidocaine

IV bolus (2 mg/kg increments up to total dose of 8 mg/kg) then IV drip at 50 µg/kg/min (500 mg in 500 mL of fluid, administered at maintenance rate [66 mL/kg/d]); CRI can be increased to 75–100 µg/kg/min if indicated clinically.

NOTE: If seizures occur, stop the drug and consider using another antiarrhythmic agent.

Procainamide (Pronestyl)

5–25 mg/kg slow IV bolus then 25–50 µg/kg/min as CRI

Amiodarone[a]

1 mg/kg IV repeated up to a total cumulative dose of 3 mg/kg; alternatively, 150 µg/kg/min as a CRI.

Intravenous Antiarrhythmic Drugs: Supraventricular

Diltiazem (Cardizem)

1–8 µg/kg/min CRI for acute SVT

Oral Antiarrhythmic Drugs: Ventricular

Amiodarone[a]

15–20 mg/kg PO q24h for 5–7 days, then 10 mg/kg PO q24h (may be able to reduce to q48h)

Mexiletine (Mexitil)

4–8 mg/kg PO q12h

Sotalol (Betapace)

1–3 mg/kg PO q8h

Oral Antiarrhythmic Drugs: Supraventricular

Extended-Release Diltiazem (Dilacor XR; Cardizem CD)

2–5 mg/kg PO q12h

Sotalol (Betapace)

1–3 mg/kg PO q12h

Amiodarone[a]

15–20 mg/kg PO q24h for 5–7 d, then 10 mg/kg PO q24h (may be able to reduce to q48h)

Digoxin (Lanoxin)

0.005–0.01 mg/kg PO q12h

Atenolol (Tenormin)

0.1–2.0 mg/kg PO q12h (gradually up-titrate the dose)

Inotropes

Pimobendan (Vetmedin)

0.25–0.3 mg/kg PO q12h (can increase to q8h in refractory cases)

Dobutamine

Dogs: 5–15 µg/kg/min IV CRI
Cats: 2.5–5 µg/kg/min IV CRI

Dopamine

5–15 µg/kg/min IV CRI

Amrinone (Inocor)

1–3 mg/kg IV over 5 min then 10–100 µg/kg/min IV CRI

Vasopressors

Ephedrine

0.03–0.1 mg/kg IV, intermittent bolus PRN (start at the low range and increase as needed)

Phenylephrine

0.1–1 µg/kg/min IV CRI
20–200 µg IV intermittent bolus PRN

Norepinephrine (Levophed)

0.05–2 µg/kg/min IV CRI for acute hypotension
0.1–2 µg/kg/min IV CRI for profound hypotension

Epinephrine

0.01–0.1 µg/kg/min IV CRI for hypotension
0.1–0.2 mg/kg IV for cardiac arrest

Vasodilators

Hydralazine (Apresoline)

0.5–3 mg/kg PO q12h
0.5–1 mg/kg IV

Nitroglycerin

1–5 µg/kg/min IV to effect

Nitroprusside

1–5 µg/kg/min IV to effect

[a]Solvents used in the commercial intravenous formulations can result in acute anaphylactic reactions.
CRI, Constant-rate infusion; *IM,* intramuscular; *IV,* intravenous; *PO,* orally; *PRN,* as needed; *SC,* subcutaneous; *SVT,* supraventricular tachycardia.

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TABLE 27.1 Cardiovascular Parameters for Patients With Mitral Regurgitation, Subaortic Stenosis, or Tamponade

Parameter	Mitral Regurgitation	Subaortic Stenosis	Cardiac Tamponade
LV preload	Normal to ↑	↑	↑
Heart rate	↑	Slow normal to ↓	↑
Rhythm	Maintain NSR	Maintain NSR	Maintain NSR
Contractility	Maintain	Maintain	Maintain
SVR	↓	Modest ↑	↑
PVR	Avoid ↓	Avoid ↓	Maintain

LV, Left ventricular; *NSR,* normal sinus rhythm; *PVR,* pulmonary vascular resistance; *SVR,* systemic vascular resistance.
Modified from Jaffe RA, Samuels SI. *Anesthesiologist's Manual of Surgical Procedures.* 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2009.

TABLE 27.2 Anesthetic Considerations in the Patient With Mitral Regurgitation[a]

Preoperative Considerations

Associated conditions	• Chronic volume overload • Hypertension • Tachydysrhythmias • Ventricular ectopy • Cardiac dysfunction relative to degree of mitral regurgitant fraction • Renal insufficiency (may be occult) • Hepatic congestion • Pulmonary congestion
Bloodwork	• HCT • Electrolytes • BUN • Cr • TP • Urinalysis • +/– PT/PTT[b]
Physical examination	• Often older patient with comorbidities • Holosystolic murmur at left cardiac apex • Dyspnea and orthopnea may be present • If crackles present indicating pulmonary edema and CHF, reconsider immediate need for anesthesia • +/– Hepatomegaly
Other diagnostics	• Blood pressure • ECG • SpO_2 • Echocardiography • Radiographs, thoracic ± abdominal

Premedications	• Keep animal calm
	• Avoid sedatives in dyspneic or depressed patients
	• If patient is anxious, give:
	  • Midazolam (0.1–0.2 mg/kg IV, IM) *or*
	  • Diazepam (0.1–0.2 mg/kg IV)
	• If patient is not depressed or dyspneic, give:
	  • Hydromorphone[c] (0.05–0.2 mg/kg IV in dogs; 0.05–0.1 mg/kg IV in cats), *or*
	  • Oxymorphone (0.05–0.2 mg/kg IV, IM), *or*
	  • Morphine[d] (0.1–0.2 mg/kg IV or 0.2–0.4 mg/kg IM), *or*
	  • Buprenorphine[e] (0.005–0.02 mg/kg IV, IM)
	• Avoid alpha-2 agonists and induction doses of ketamine; avoid acepromazine in moderate to severe MR.

Intraoperative Considerations

Induction	• If mild MR, give:
	  • Etomidate (0.5–1.5 mg/kg IV), give after administering a benzodiazepine to avoid myoclonus, *or*
	  • Propofol (1–4 mg/kg IV), *or*
	  • Alfaxalone (2–5 mg/kg IV)
	• If moderate or severe MR, give etomidate (0.5–1.5 mg/kg IV)

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TABLE 27.2 Anesthetic Considerations in the Patient With Mitral Regurgitation[a]—cont'd

Maintenance	• Isoflurane or sevoflurane, *plus*
	  • Fentanyl (2–10 µg/kg IV PRN in dogs and 1–4 µg/kg IV PRN in cats) for short-term pain relief, *plus* PRN
	  • Fentanyl CRI (1–5 µg/kg IV loading dose, then 2–30 µg/kg/h), *or*
	  • Hydromorphone[c] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Oxymorphone (0.05–0.2 mg/kg IV PRN), *or*
	  • Buprenorphine[e] (0.005–0.02 mg/kg IV PRN)
	• For hypotension (to keep MAP 60 mm Hg), give ephedrine, dobutamine, norepinephrine, vasopressin, or epinephrine as needed; phenylephrine only at low doses
	• For hypertension (to keep MAP 60 mm Hg), give nitroglycerin CRI (0.5–5 µg/kg/min) IV or nitroprusside CRI (1–5 µg/kg/min) IV or fentanyl boluses for pain
	• Keep heart rate in high normal range. For bradycardia, give:
	  • Glycopyrrolate (0.005–0.01 mg/kg IV), *or*
	  • Atropine (0.02–0.04 mg/kg IV)
	• Avoid nitrous oxide, hypoxemia, hypercarbia, and acidosis

Fluid needs	• 5–10 mL/kg/h if minimal EBL and minimal evaporative losses, or 10–20 mL/kg/h if open abdomen/thorax with higher evaporative losses, plus 3× EBL. Higher rates of fluids may be necessary to keep preload high. Have blood available in OR if EBL is high. • Recommend 2 large IV catheters if blood loss is expected
Monitoring	• Blood pressure • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature • U/O • +/Arterial line[b] • +/CVP[b] • +/PT/PTT[b] • Blood gases[b]

Blocks	• Epidural:
	  • Morphine (0.1 mg/kg preservative free), *or*
	  • Buprenorphine (0.003–0.005 mg/kg diluted in saline) if appropriate
	• Intercostal nerve blocks, if appropriate
	• Incisional block:
	  • Lidocaine (<5 mg/kg in dogs; 2–4 mg/kg in cats), *or*
	  • Bupivacaine (<2 mg/kg)

Postoperative Considerations

Analgesia	• Fentanyl CRI (1–10 µg/kg IV loading dose, then 2–20 µg/kg/h IV), *or*
	• Morphine[d] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.05–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h in cats) if not hypotensive, *or*
	• Buprenorphine[e] (0.005–0.02 mg/kg IV, IM q4–6h or 0.01–0.02 mg/kg OTM q6–12h in cats), *or*
	• Hydromorphone[c] (0.05–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats), *or*
	• Oxymorphone (0.05–0.2 mg/kg IV, IM q3–4h), *or*
	• Hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs) *plus* PRN
	• For sedation, give:
	  • Midazolam (0.1–0.2 mg/kg IV, IM PRN), *or*
	  • Diazepam (0.1–0.2 mg/kg IV PRN)
	• Avoid NSAIDs if cardiac compromise is enough to cause decreased renal and hepatic perfusion

Monitoring	• SpO_2 • Blood pressure • HR • Respiratory rate • Temperature • U/O • ECG • +/– Arterial line[b] • +/– CVP[b] • +/– Blood gases, PT, PTT[b]
Bloodwork	• HCT if significant blood loss • Repeat abnormal preoperative bloodwork • +/– Blood gases, PT, PTT[b]
Estimated pain score	Depends on type of surgery performed

[a]Patients with compromised cardiac output have longer drug onset times. Be patient and wait for adequate circulation time before repeating a dose.
[b]Should be considered in invasive abdominal/thoracic surgeries and other long procedures.
[c]Monitor for hyperthermia in cats.
[d]Give slowly to prevent histamine release.
[e]Buprenorphine is a better analgesic than morphine in cats.
BUN, Blood urea nitrogen; *CHF,* congestive heart failure; *Cr,* creatinine; *CRI,* constant-rate infusion; *CVP,* central venous pressure; *EBL,* estimated blood loss; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *MAP,* mean arterial pressure; *MR,* mitral regurgitation; *NSAID,* nonsteroidal antiinflammatory drug; *OR,* operating room; *OTM,* oral transmucosal; *PRN,* as needed; *PT,* prothrombin time; *PTT,* partial thromboplastin time; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein; *U/O,* urine output.

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TABLE 27.3 Anesthetic Considerations in the Patient With Aortic Stenosis or Subaortic Stenosis[a]

Preoperative Considerations

Associated conditions	• History of syncope • Left ventricular hypertrophy • Ventricular ectopy • Cardiac dysfunction relative to degree of stenosis • Renal insufficiency (may be occult) • Hepatic congestion • Pulmonary congestion • Platelet dysfunction
Bloodwork	• HCT • Electrolytes • BUN • Cr • TP • Urinalysis • +/– PT/PTT[b]
Physical examination	• Often younger patient • Systolic murmur at left cardiac base • Weakness with activity • If crackles present indicating pulmonary edema and CHF, reconsider immediate need for anesthesia • +/– Hepatomegaly • +/– Weak pulses
Other diagnostics	• Blood pressure • ECG • Echocardiography • Radiograph, thoracic, +/– abdominal • SpO_2

Premedications	• Keep animal calm
	• Avoid sedatives in dyspneic or depressed patients
	• If patient is excitable, give:
	  • Midazolam (0.1–0.2 mg/kg IV, IM), *or*
	  • Diazepam (0.1–0.2 mg/kg IV)
	• Wait to give opioids
	• Avoid alpha-2 agonists, acepromazine, atropine, glycopyrrolate, and ketamine

Intraoperative Considerations

Induction	• Etomidate (0.5–1.5 mg/kg IV); give after administering a benzodiazepine to avoid myoclonus

Maintenance	• Isoflurane or sevoflurane at one-half to 1 MAC *plus*
	  • Fentanyl (2–10 µg/kg IV PRN in dogs; 1–4 µg/kg IV PRN in cats) for short-term pain relief, *plus*
	  • Fentanyl CRI (1–5 µg/kg IV loading dose then 2–30 µg/kg per hour IV), *or*
	  • Hydromorphone[b] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Oxymorphone (0.05–0.2 mg/kg IV PRN), *or*
	  • Buprenorphine[d] (0.005–0.02 mg/kg IV PRN)
	• For hypotension (to keep MAP >60 mm Hg), give:
	  • Phenylephrine (20–200 µg boluses or 0.1–1 µg/kg/min IV CRI), *or*
	  • Norepinephrine (0.05–2 µg/kg/min IV CRI)
	• Keep heart rate in low-normal range. For tachycardia, give:
	  • Esmolol (0.05–0.25 µg/kg IV) boluses every 2–5 min to effect and/or CRI (50–200 µg/kg/min IV) to maintain normal to low-normal heart rate
	  • Fentanyl boluses for pain

Fluid needs	• Avoid nitrous oxide, hypoxemia, hypercarbia, and acidosis • 5–10 mL/kg/h if minimal EBL and minimal evaporative losses, or 10–20 mL/kg/h if open abdomen/thorax with higher evaporative losses, plus 3× EBL. Higher rates of fluids may be necessary to keep preload high. Have blood available in OR if EBL is high. • Recommend 2 large IV catheters if blood loss is expected[b]
Monitoring	• Blood pressure • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature • U/O • +/– Arterial line[b] • +/– CVP[b] • +/– PT/PTT[b] • Blood gases[b]

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TABLE 27.3 Anesthetic Considerations in the Patient With Aortic Stenosis or Subaortic Stenosis[a]—cont'd

Blocks	• Epidural:
	  • Morphine (0.1 mg/kg preservative free) *or*
	  • Buprenorphine (0.003–0.005 mg/kg diluted in saline) if appropriate
	• Local anesthetics for spinal or epidural are contraindicated
	• Intercostal nerve blocks if appropriate
	• Incisional block:
	  • Lidocaine (<5 mg/kg in dogs; 2–4 mg/kg in cats), *or*
	  • Bupivacaine (<2 mg/kg)

Postoperative Considerations	

Analgesia	• Fentanyl CRI (1–10 µg/kg IV loading dose, then 2–20 µg/kg/h IV), *or*
	• Hydromorphone[c] (0.05–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats), *or*
	• Oxymorphone (0.05–0.2 mg/kg IV, IM q3–4h), *or*
	• Hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs), *or*
	• Buprenorphine[d] (0.005–0.02 mg/kg IV, IM q4–8h or 0.01–0.02 mg/kg OTM q6–12h in cats), *plus* PRN
	• For sedation:
	  • Midazolam (0.1–0.2 mg/kg IV, IM PRN), *or*
	  • Diazepam (0.1–0.2 mg/kg IV PRN)
	• Avoid NSAIDs if cardiac compromise is enough to cause decreased renal and hepatic perfusion

Monitoring	• SpO_2 • Blood pressure • HR • Respiratory rate • Temperature • U/O • ± ECG[b] • ± Arterial line[b] • ± CVP[b] • ± Blood gases, PT, PTT[b]
Bloodwork	• HCT if significant blood loss • Repeat abnormal preoperative bloodwork • ± Blood gases, PT, PTT[b]
Estimated pain score	• Depends on type of surgery performed

[a]Patients with compromised cardiac output have longer drug onset times. Be patient and wait for adequate circulation time before repeating a dose.
[b]Should be considered in invasive abdominal/thoracic surgeries and other long procedures.
[c]Monitor for hyperthermia in cats.
[d]Buprenorphine is a better analgesic than morphine in cats.
BUN, Blood urea nitrogen; *CHF,* congestive heart failure; *Cr,* creatinine; *CRI,* constant-rate infusion; *CVP,* central venous pressure; *EBL,* estimated blood loss; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *MAC,* minimum alveolar concentration; *MAP,* mean arterial pressure; *NSAID,* nonsteroidal antiinflammatory drug; *OR,* operating room; *OTM,* oral transmucosal; *PRN,* as needed; *PT,* prothrombin time; *PTT,* partial thromboplastin time; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein; *U/O,* urine output.

be a clinically acceptable induction agent.[2] Ketamine combined with diazepam may be appropriate for induction of compromised patients. It should be avoided in animals with mitral insufficiency as it increases the regurgitant fraction by increasing peripheral vascular resistance. However, it is the induction agent of choice in animals with pericardial constriction. Opioids can be used for induction of very sick and compromised dogs; however, opioids do not induce hypnosis, so intubation may be difficult. Etomidate is not arrhythmogenic, maintains cardiac output, and offers rapid induction, although it is associated with longer and poorer recoveries. Mask induction is discouraged in all patients with cardiopulmonary disorders because the patient's reduced cardiac output will cause an increased time to achieve adequate induction. Additionally, inhalants cause marked hypotension, which is often undesirable. A balanced anesthetic approach using benzodiazepine, opioids, and modest amounts of inhalant is generally much safer. Atracurium (Box 27.2) is a short-acting muscle relaxant that is not dependent on metabolism or excretion to terminate its action; it may be used if further muscle relaxation is needed but requires the use of intermittent positive-pressure ventilation (IPPV).

BOX 27.2 Drugs for Inflow Occlusion

Lidocaine (Xylocaine)
Dogs: 50–100 µg/kg/min IV CRI (see also Box 27.1)
Cats: 10–40 µg/kg/min IV CRI

Dobutamine (Dobutrex)
Dogs: 5–15 µg/kg/min CRI
Cats: 2.5–5 µg/kg/min CRI

Epinephrine (Adrenaline, Generic)
Dogs: 0.01–0.4 µg/kg/min IV CRI

Fentanyl Citrate (Sublimaze)
2–30 µg/kg/h IV

Atracurium Besylate (Tracrium)
0.1–0.2 mg/kg IV

Dexamethasone (Azium)
1 mg/kg IV

IV, Intravenous; *CRI,* constant-rate infusion.

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Thoracic surgery always requires controlled ventilation. Controlled ventilation can be achieved by manually squeezing the reservoir bag or by attaching a mechanical ventilator to the anesthetic machine. Ideally, mechanical ventilation should achieve a tidal volume of 6 to 10 mL/kg of body weight at an inspiratory pressure of less than 20 cm H_2O. Ensuring adequate ventilation is accomplished by optimizing tidal volume, inspiratory pressure, and respiratory rate to achieve ventilation with the least risk of causing pulmonary injury or cardiovascular compromise. Ultimately, the goal of mechanical ventilation is to maintain normocapnia. Ventilation can be monitored by measurement of end-tidal CO_2 by capnography, or of arterial CO_2 by blood gas analysis.

Successful inflow occlusion requires meticulous anesthesia. Intraoperative and postoperative care may be complicated and may require multiple vasoactive medications. For further discussion of anesthesia techniques for these patients, refer to a cardiac anesthesia text. Balanced anesthetic techniques that minimize inhalation anesthetic agents are indicated (e.g., fentanyl citrate plus atracurium besylate combined with isoflurane [see Box 27.2]). Administration of a single dose of dexamethasone (see Box 27.2) after induction may be beneficial in reducing cardiac damage and improving postoperative outcomes. Mild hypothermia (32°C–34°C) reduces basal metabolic rate, allowing lengthening of occlusion time; however moderate hypothermia (<32°C) is associated with spontaneous ventricular fibrillation. Animals should be hyperventilated for 5 minutes before inflow occlusion. Ventilation is discontinued during inflow occlusion and is resumed immediately upon reestablishment of blood flow. Drugs and equipment for full cardiac resuscitation must be available immediately after inflow occlusion. Gentle cardiac massage may be necessary after inflow occlusion to reestablish cardiac function. Digital occlusion of the descending aorta during this period helps direct available cardiac output to the heart and brain. If ventricular fibrillation occurs, immediate internal defibrillation is necessary as soon as inflow occlusion is discontinued. Constant IV infusion of lidocaine (see Box 27.2) should be initiated before inflow occlusion and continued as necessary. Epinephrine, administered as a constant-rate infusion, should be given as the animal is being weaned off inflow occlusion or a pump (see Box 27.2). If long-term inotropic support is necessary, dobutamine or amrinone should be given (see Boxes 27.1 and 27.2 and Tables 27.1 to 27.3).

Transesophageal echocardiography (TEE) can be an invaluable tool for assessing both cardiac function and volume status in the cardiac patient. The use of TEE intraoperatively and immediately postoperatively can guide the choice of pharmacologic intervention and can assist in assessing the effectiveness of therapy. Central venous pressure measurements have been shown to poorly correlate with volume status. When TEE is used, ventricular filling can be visualized and volume replacement modified accordingly.

ANTIBIOTICS

Perioperative antibiotics are indicated for cardiac procedures lasting longer than 90 minutes. First-generation cephalosporins (e.g., cefazolin) can be administered intravenously at induction and repeated once or twice (Box 27.3). For cardiac procedures involving circulatory arrest or cardiopulmonary bypass, IV cefoxitin is a good choice to be administered before surgery and continued for 24 hours after surgery owing to impairment of humoral host defenses associated with these procedures (see Box 27.3).

BOX 27.3 Prophylactic Antibiotics for Cardiac Surgery

Cefazolin (Ancef, Kefzol)
22 mg/kg IV at induction

Cefoxitin (Mefoxin)
30–40 mg/kg IV at induction

IV, Intravenous.

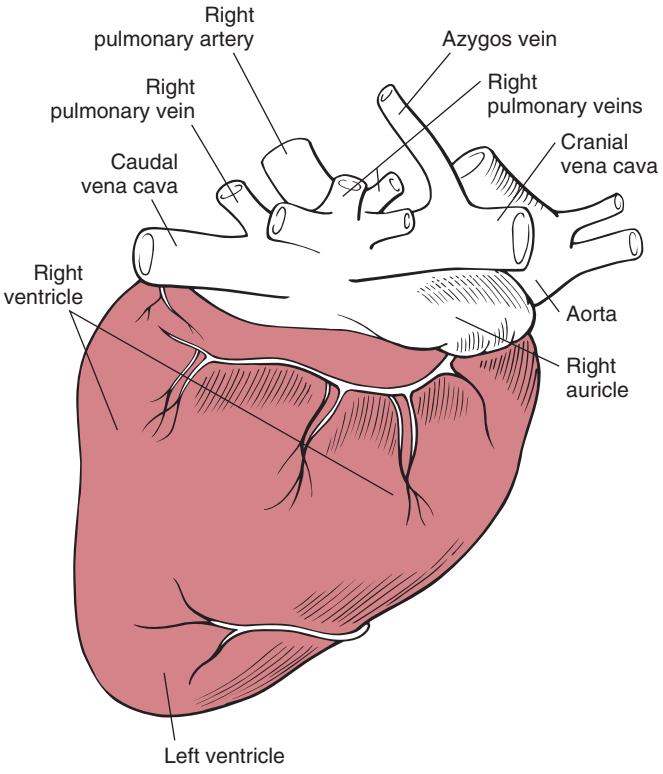


FIG. 27.1 Cardiac anatomy as viewed from the right side.

SURGICAL ANATOMY

The heart is the largest mediastinal organ. It generally extends from the third rib to the caudal border of the sixth rib; however, variations have been noted among breeds and between individuals. The heart base (i.e., the craniodorsal aspect [from where the great vessels originate]) faces dorsocranially, whereas the apex (i.e., formed by muscles of the left ventricle) points caudoventrally. Except for a portion of the right side of the heart (cardiac notch), most of its surface is covered by lung. The right ventricular wall accounts for approximately 22% of the total heart weight; the left ventricular wall accounts for nearly 40%.

The right atrium receives blood from the systemic circulation. The coronary sinus enters the left caudal aspect of the atrium, ventral to the caudal vena cava. The caudal vena cava returns blood from the abdominal viscera, the pelvic limbs, and a portion of the abdominal wall (Fig. 27.1). The cranial vena cava returns blood to the heart from the head, neck, thoracic limbs, and ventral thoracic wall, and from a portion of the abdominal wall.

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The azygos vein usually enters into the cranial vena cava; it carries blood from the lumbar regions and the caudal thoracic wall. The brachycephalic trunk is the first large artery from the aortic arch. The common carotid arteries usually arise from it as separate vessels. The left subclavian artery arises from the aortic arch distal to the brachycephalic trunk (the right subclavian is a branch of the brachycephalic trunk). The vertebral arteries, costocervical trunk, internal thoracic arteries, and axillary arteries branch from the subclavian vessels.

The pericardium is a thick, two-layer sac composed of outer fibrous and inner serous layers. The pericardial cavity is located between two layers (visceral and parietal) of serous pericardium and normally contains a small amount of fluid. The fibrous pericardium blends with the adventitia of the large vessels, and its apex forms the sternopericardiac ligament. Phrenic nerves lie in a narrow plica of pleura adjacent to the pericardium at the heart base. Complete pericardiectomy requires that these nerves be elevated to avoid incising them. The vagus nerves lie dorsal to the phrenic nerve. They divide to form dorsal and ventral branches that lie on the esophagus in the caudal thorax. The left recurrent laryngeal nerve leaves the vagus and loops around the aortic arch distal to the ligamentum arteriosum to run cranially along the ventrolateral tracheal surface.

SURGICAL TECHNIQUE

Cardiac surgery is not fundamentally different from other types of general surgery, and similar principles of good surgical technique (i.e., atraumatic tissue handling, good hemostasis, and secure knot tying) apply. Consequences of poor surgical technique are often devastating. Cardiac surgery differs from other surgeries in that motion from ventilation and cardiac contractions adds to the technical difficulty of performing these procedures. Approaches that provide limited access to dorsal structures (e.g., median sternotomy; see p. 891) require that surgeons incise, suture, and/or ligate structures located deep within the thorax. Ligature placement using hand ties (see p. 73) is useful in such situations, and the ability to place hand-tied knots (vs. instrument tying) should be considered a fundamental skill for cardiac surgeons. Secure knot tying is critically important for successful cardiac surgery. Hand tying of knots is fast and produces tighter and more secure knots than instrument tying. The one-handed knot tie technique (see p. 74) is best suited to the fine sutures used in cardiac surgery. Tight knots are facilitated by throwing the first two or three throws in the same direction before finishing with square knots for security.

Closure of cardiovascular structures requires precise suturing techniques and good instrument handling skills to minimize hemorrhage. Using fine suture with swaged-on atraumatic needles (see discussion on suture materials on p. 796) and carefully following the needle contour when suturing (to minimize the size of needle tracts) are important. "Palming" of needle holders is a good skill for fast suturing but should be avoided when suturing inside the thoracic cavity. Finer control is gained by grasping instruments with fingers placed in the instrument rings.

Inflow Occlusion

Inflow occlusion is a technique used for open heart surgery in which all venous flow to the heart is temporarily interrupted. Because inflow occlusion results in complete circulatory arrest, it allows limited time to perform cardiac procedures. Ideally, circulatory arrest in a normothermic patient should be less than

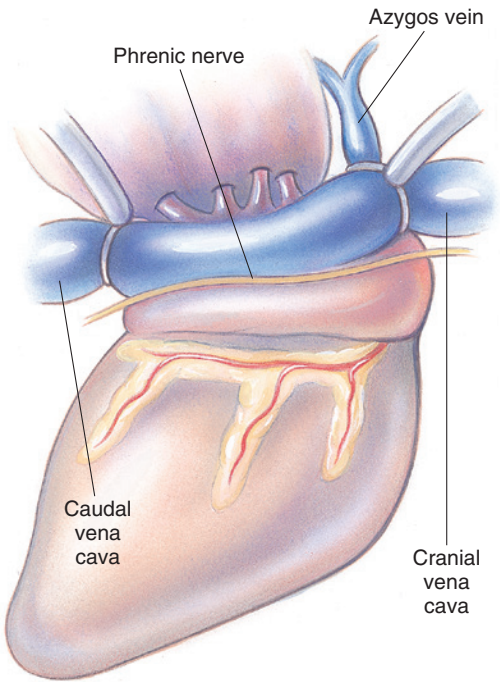


FIG. 27.2 To occlude cardiac inflow from the right side of the thorax, pass tapes around the caudal vena cava and the common drainage of the azygos veins and the cranial vena cava. Fashion tourniquets as described in Fig. 27.3.

2 minutes, but it can be extended to 4 minutes if necessary. Circulatory arrest time can be extended up to 8 minutes with mild, whole-body hypothermia (32°C–34°C). Temperatures below 32°C may predispose to spontaneous ventricular fibrillation and should be avoided. The advantage of inflow occlusion is that it does not require specialized equipment; however, the limited time available to perform the surgery requires that the procedure is well planned and executed with speed and expertise.

Depending on the cardiac procedure that is being done, perform a left or a right thoracotomy (see p. 890) or a median sternotomy (see p. 891). With a right thoracotomy or median sternotomy, occlude the cranial and caudal vena cava and the azygos vein with vascular clamps or Rumel tourniquets (Fig. 27.2). Make a Rumel tourniquet by passing umbilical tape around the vessel, then thread the umbilical tape through a piece of rubber tubing that is 1 to 3 inches (2.5–7.5 cm) long. When the umbilical tape has been adequately tightened to occlude the vessel, place a clamp above the rubber tubing to hold it securely in place. Take care to prevent injuring the right phrenic nerve during placement of the clamps or tourniquets. For left thoracotomies, pass separate tourniquets around the cranial and caudal venae cavae. Then, while dissecting dorsal to the esophagus and aorta, occlude the azygos vein by placing a tourniquet around it (Fig. 27.3).

Cardiopulmonary Bypass

Cardiopulmonary bypass is a procedure whereby an extracorporeal system provides flow of oxygenated blood to the patient while blood is diverted away from the heart and lungs. This greatly extends the time available for open cardiac surgery. Several advances (i.e., development of membrane oxygenators, improved methods of myocardial protection, increased availability of monitoring technologies, and improved veterinary critical care) have made cardiopulmonary bypass increasingly feasible in dogs.

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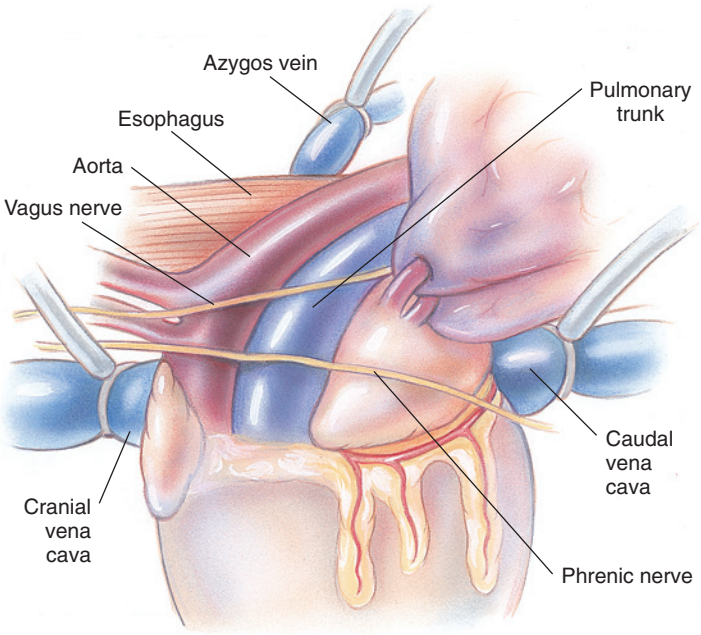


FIG. 27.3 During inflow occlusion from the left side of the thorax, pass tapes around the cranial and caudal vena cava and the azygos vein. Fashion tourniquets for inflow occlusion by passing the tapes through rubber tubing.

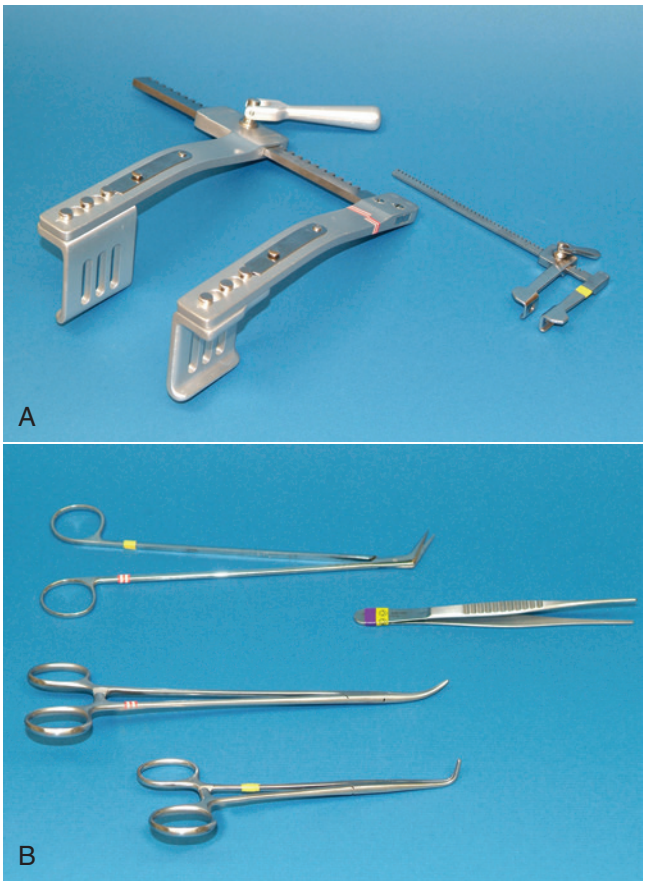


FIG. 27.4 Instruments for cardiovascular surgery. (A) Large and small Finochietto retractors. (B) *Top to bottom,* Potts scissors, DeBakey tissue forceps, angled thoracic forceps (45 and 90 degree). The 90-degree forceps is also called a *right-angle* or *Mixter forceps*; the 45-degree forceps is also called a *Julian thoracic artery forceps* or *Mixter forceps*.

Cardiopulmonary bypass can be used to treat dogs with congenital or acquired cardiac defects. The challenges facing cardiopulmonary bypass in dogs and cats include, but are not limited to, variations in heart position within the thoracic cavity between different breeds and at different ages; the variability in size and body conformation between individual animals; the limited blood volume in smaller patients; problems with coagulation, anticoagulation, and hemostasis; and the limited availability and cost of blood products.[3] Refer to a cardiovascular surgery text for details of performing cardiopulmonary bypass.

HEALING OF CARDIOVASCULAR STRUCTURES

Vascular structures heal quickly, forming a fibrin seal within minutes. Epithelialization and early endothelial regeneration occur in veins used for grafts. Thrombosis commonly occurs in small veins that have been traumatically occluded for short periods of time; however, thrombosis of large veins occluded during inflow occlusion or cardiac bypass procedures has not been a clinically recognized problem. To prevent thrombosis of vascular structures, they should be handled gently, because trauma may lead to the deposition of platelets, fibrin, and red cells on the intimal surface. If the torn intima is lifted upward, a flap may develop that partially or completely occludes the distal lumen. This in turn can lead to accumulation of blood within the vessel wall, vascular sludging, and thrombosis.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Polypropylene (e.g., Prolene, Surgipro) and braided polyester (e.g., Ticron, Mersilene) suture are the standard sutures used for cardiovascular procedures. The sizes most commonly used are 3-0 to 5-0, although smaller sizes may be needed for vascular anastomoses. Sutures should be available with swaged-on taper point cardiovascular needles in a variety of sizes. Some procedures require that the suture be double-armed (i.e., with needles at both ends). Teflon pledgets are useful for buttressing mattress sutures in ventricular myocardium or great vessels.

Successful cardiac surgery requires proper surgical instrumentation. Most of the basic instruments required for general surgery can be used for cardiac surgery; however, a few specialized instruments are desirable for thoracic surgery. The standard thoracic retractor is a Finochietto retractor (Fig. 27.4A). It is helpful to have retractors of at least two sizes to accommodate animals of different sizes. Self-retaining orthopedic retractors can substitute as thoracic retractors in small dogs and cats. The standard tissue forceps for thoracic surgery is a DeBakey tissue forceps (see Fig. 27.4B). At least two DeBakey forceps should be available, and it is helpful if one has a carbide inlay for grasping suture needles. Metzenbaum scissors are the standard operating scissors for cardiac surgery. Curved Metzenbaum scissors are more versatile than the straight design. Potts scissors (45-degree angle) are desirable for some cardiac surgery procedures (see Fig. 27.4B). Needle holders should be long and available in different sizes to accommodate a variety of suture needle sizes. Mayo-Hegar, Crile-Wood, and Castroviejo needle holders represent a good selection of sizes for thoracic surgery in animals.

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FIG. 27.5 Tangential (Satinsky) vascular clamps.

Angled thoracic forceps are an important instrument for cardiac surgery and should be available in a variety of sizes (see Fig. 27.4B). Vascular clamps are noncrushing clamps used for temporary occlusion of cardiovascular and pulmonary structures. They come in a variety of sizes and shapes, including straight, angled, curved, and tangential (Fig. 27.5). The most versatile shape for most cardiac surgery procedures is a medium-width tangential clamp.

POSTOPERATIVE CARE AND ASSESSMENT

Patient monitoring and postoperative care are the cornerstones of successful cardiac surgery. The level of supportive care required for cardiac surgeries depends on the patient and surgical procedure performed. A working knowledge of cardiopulmonary function and good patient observation skills are as important to successful patient management as advanced monitoring devices.

Evaluation of ventilation is important after any thoracic surgery. Poor ventilatory efforts may first be noted in the period after surgery, when the influence of anesthetic drugs is still present but ventilatory support has been discontinued. Hypoventilation may also result from uncontrolled pain. Total ventilation can be assessed directly by measuring the volume of expired gas with a respirometer. Tidal volume should be at least 10 mL/kg of body weight. Ultimately, the best measure of alveolar ventilation is arterial CO_2 partial pressure ($PaCO_2$). Alveolar hypoventilation is present when $PaCO_2$ is increased to above 40 mm Hg. Treatment of hypoventilation should be directed at correcting its underlying cause if possible. Drugs that are known to depress ventilation (i.e., opioids and muscle relaxants) should be used with caution in the perioperative period, and the risk of ventilatory depression weighed against the risk of hypoventilation due to pain (see p. 886 for analgesia after thoracotomy). Pleural air or fluid should be evacuated if present. Injury or dysfunction of the neuromuscular ventilatory apparatus should be corrected, if possible. If hypoventilation is severe and the cause is not immediately correctable, positive-pressure ventilation is indicated. If necessary, keep the animal intubated and ventilated postoperatively until partial pressure of arterial oxygen (PaO_2), $PaCO_2$, pH, blood pressure, and heart rate indicate that the patient is stable enough to extubate. Constant-rate infusions of fentanyl and propofol can assist in keeping the patient comfortable until complications such as significant ventilation/perfusion ($\dot{V}/\dot{Q}$) mismatch or acidosis have improved. If necessary, vasopressors may be administered to correct hypotension caused by propofol.

Under physiologic conditions, gas exchange between the alveolus and the pulmonary capillary blood is efficient, and alveolar oxygen tension (PAO_2) and arterial oxygen tension (PaO_2) are nearly equal. In patients with impaired gas exchange, hypoxemia occurs because PAO_2 and PaO_2 are not equal. The most common cause of impaired pulmonary gas exchange in the postoperative setting is alveolar ventilation/perfusion ($\dot{V}_A/\dot{Q}$) mismatch with formation of pulmonary shunts secondary to alveolar collapse. The importance of $\dot{V}/\dot{Q}$ ratios relates to how well the lungs resaturate venous blood with O_2 and eliminate CO_2. During procedures involving circulatory arrest or cardiopulmonary bypass, ventilation is temporarily ceased and the remaining oxygen is absorbed, resulting in collapsed alveoli (absorption atelectasis). With reperfusion of collapsed pulmonary tissues, blood passes through the lungs without becoming oxygenated. The result can be large shunts of deoxygenated blood returning to the left side of the heart. Patients with shunts are not responsive to higher concentrations of oxygen. Instead, they need assisted ventilation and positive end-expiratory pressure (PEEP). Therefore response to supplemental oxygen therapy must be evaluated for each individual patient, preferably by arterial blood gas analysis. The therapeutic goal of supplemental oxygen should be to keep PaO_2 above 80 mm Hg. Ventilation and PEEP are indicated for patients with severe gas exchange impairment that is not responsive to supplemental oxygen therapy alone.

Maintaining an adequate PaO_2 in a patient is important because it is the soluble oxygen that crosses membranes. Total oxygen content of the blood is the sum of the oxygen in solution (PaO_2) plus that carried by hemoglobin. The major determinant of hemoglobin oxygen saturation (SaO_2) is hemoglobin. SaO_2 can be measured by pulse oximetry. The therapeutic goal should be to maintain SaO_2 at or above 90%. Oxygen content of the blood is a function of SaO_2 and hemoglobin concentration. Thus maintenance of adequate oxygen content requires not only adequate pulmonary function but also an adequate hemoglobin concentration. Maintenance of the packed cell volume above 30% is an important therapeutic goal for animals undergoing cardiac surgery, especially if cardiopulmonary compromise is present.

Systemic blood pressure is directly proportional to cardiac output and systemic vascular resistance (SVR). Measurement of blood pressure provides a good assessment of cardiovascular function, especially during and immediately after surgery. Indirect techniques for measuring blood pressure include the oscillometric method, which serves as the basis of monitors such as the Dinamap, or Doppler, method. Doppler technique provides only systolic pressure but is useful for evaluating blood pressure trends during and after surgery. Indirect methods of blood pressure assessment are less invasive but are also less accurate than direct measurements. Direct measurement of blood pressure requires placement of an arterial catheter. Arterial catheters have the additional advantage of providing access for arterial blood gas analysis. An arterial catheter is typically placed percutaneously into a dorsal pedal artery. Direct blood pressure measurement also requires a pressure transducer and a monitor, or a manometer. The therapeutic goal is to maintain a mean blood pressure above 65 mm Hg and systolic blood pressure above 90 mm Hg. Blood pressure can be elevated by increasing cardiac output or SVR. In many instances (depending on the cause), a more appropriate therapeutic strategy to correct hypotension is to improve cardiac

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output. Maintenance of adequate vascular volume is the most important aspect of maintaining adequate cardiac output. Central venous pressure should be maintained at between 5 and 10 cm H_2O. If echocardiography is available, evaluation of ventricular filling is a more reliable way to detect hypovolemia. Indications for arterial pressor therapy are rare. Inotropic and pressor support can be obtained by constant IV infusion of epinephrine (see Box 27.2). Long-term inotropic support is maintained with dobutamine (see Box 27.2).

Monitoring the electrocardiogram (ECG) for disturbances in cardiac rhythm is important for animals undergoing cardiac surgery. Sinus tachycardia is the most common rhythm disturbance in surgery patients. Therapy for sinus tachycardia should be directed at correction of its underlying cause (e.g., hypovolemia, pain, anxiety, acidosis, hypotension, anemia, hypoxemia, drug induced) and improvement in cardiac output. Ventricular dysrhythmias, including premature ventricular complexes (PVCs) and nonsustained or sustained ventricular tachycardia, are frequently encountered during and after cardiac surgery. Frequent PVCs, particularly when they occur with a short coupling interval (i.e., R-on-T phenomena), and rapid ventricular tachycardia should be suppressed in the perioperative period. Continuous IV infusion of lidocaine is effective in most instances. Ventricular fibrillation is a form of cardiac arrest that requires immediate electrical defibrillation. If cardiac surgery is performed, equipment for defibrillation should be available. Recommendations for postoperative analgesics are provided in Chapter 13 (see Tables 13.2 and 13.3).

COMPLICATIONS

The major complication associated with cardiac surgery is hemorrhage. Severe hemorrhage may be encountered intraoperatively or postoperatively. Materials for blood transfusion should be available (see Table 4.5 and Box 4.1). Fresh whole blood should be collected as close as possible to the time that it is needed and should not be cooled because this may reduce platelet content. If possible, a compatible donor should be identified by cross matching the patient before surgery. Cell Saver 5+ autologous blood recovery systems (Haemonetics) are available for collection and processing of blood for procedures in which rapid bleeding or high-volume blood loss may occur. They can also be used to sequester platelets and plasma from a patient immediately before surgery, thereby reducing the need for donor blood.

SPECIAL AGE CONSIDERATIONS

Most animals undergoing surgery for congenital cardiac defects are young. Special care must be given to these animals during and after surgery. Young animals should not have food withheld for longer than 4 to 6 hours before surgery and should be fed as soon as they are fully recovered from anesthesia. If they cannot be fed, blood glucose concentration should be maintained by adding dextrose to IV fluids; blood glucose concentrations should be monitored intraoperatively. Hypothermia is common in young patients during thoracotomy and can be protective during cardiac procedures. However, the temperature should be monitored closely, and patients should be actively rewarmed postoperatively.

NOTE Remember, hypothermia decreases the minimum alveolar concentration of inhalants used for maintenance.

SPECIFIC DISEASES

MITRAL REGURGITATION

DEFINITIONS

MR occurs when blood leaks retrograde across the mitral valve into the left atrium during contraction of the left ventricle. Synonyms include *degenerative mitral valve disease* (DMVD) and *mitral insufficiency*. MR is due most often to endocardiosis of the mitral valve, which is also known as *myxomatous valvular degeneration* (MVD), *myxomatous mitral valve disease, DMVD,* and *chronic valvular heart disease*.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

MR is the most common form of acquired heart disease in dogs. Approximately 75% of dogs with chronic heart disease have MR attributable to MVD. It commonly occurs as the result of myxomatous degeneration of the valve and may be associated with one or more of the following: thickening and billowing of the leaflets, dilation of the mitral annulus, thickening and lengthening or rupture of the chordae tendineae, and flattening of the papillary muscles with left ventricular dilation. Morphologic changes associated with MVD in dogs are similar to those observed in humans with mitral valve prolapse. Tissue swelling occurs on the edge of valve leaflets, the chordae tendineae, and the chordal-papillary muscle junction. Damage to the valve complex endothelium is unevenly distributed. On rare occasions, annular dilation and MR occur without significant disease of the chordae or leaflets. Congenital valve dysplasia and dilated cardiomyopathy are other causes of MR.

The clinical presentation and progression of MVD are variable. Most dogs remain asymptomatic for their lifetime. For dogs that do have progression of disease, volume overload of the left ventricle occurs when blood regurgitates through the mitral valve, causing left atrial and left ventricular hypertrophy. As the mitral valve annulus dilates, MR typically becomes more severe, and left-sided CHF typically ensues. Atrial fibrillation may be associated with left atrial dilation, especially in large and giant breeds of dogs with sufficient atrial mass to sustain the arrhythmia. A classification system has been proposed (Box 27.4) to aid identification of dogs with cardiac disease and to link the extent of clinical signs with appropriate treatment and monitoring recommendations.

DIAGNOSIS

Clinical Presentation

Signalment

MVD typically occurs in older, small-breed dogs (<15 kg), whereas congenital mitral valve dysplasia occurs most commonly in cats and in large- and giant-breed dogs. When large-breed dogs are affected with MVD, progression of disease appears to be more rapid than in small-breed dogs. Cavalier King Charles spaniels are a well-recognized, predisposed breed and typically develop MVD at a relatively young age (5–7 years old). Male dogs are affected 1.5 times more often than females. Dilated cardiomyopathy may be associated with secondary MR as dilation of the valve annulus coupled with papillary muscle dysfunction causes valvular incompetence.

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BOX 27.4 Classification System for Dogs Affected by Myxomatous Valvular Degeneration

Stage A
Dogs at high risk (e.g., Cavalier King Charles spaniel) with no identifiable structural cardiac disorder

Stage B1
Dogs with MVD with no clinical signs and little or no radiographic or echocardiographic evidence of cardiac remodeling

Stage B2
Dogs with MVD with no clinical signs, but with radiographic or echocardiographic evidence of cardiac remodeling

Stage C
Dogs with MVD with current or past clinical signs of heart failure associated with structural cardiac remodeling

Stage D
Dogs with MVD and congestive heart failure refractory to standard medical therapy

MVD, Myxomatous valvular degeneration.
Data from Atkins C, Bonagura J, Ettinger S, et al. Guidelines for the diagnosis and treatment of canine chronic valvular heart disease. *J Vet Intern Med.* 2009;23:1142–1150.

History

Many animals are asymptomatic. Affected animals may have a history of exercise intolerance, coughing, and/or shortness of breath. A murmur associated with MR may be picked up on physical examination in an asymptomatic animal.

Physical Examination Findings

Affected animals typically have a holosystolic murmur heard best at the left cardiac apex. The intensity of the murmur may be correlated with the severity of disease in some dogs (usually small breeds). Pulmonary crackles may be heard if pulmonary edema is present. Electrocardiographic evidence of left atrial and/or left ventricular enlargement may be manifested by a P wave duration greater than 0.04 seconds (P mitrale) or tall R waves (>2–2.5 mV), respectively, in lead II.

Diagnostic Imaging

Consensus guidelines from the American College of Veterinary Internal Medicine regarding dogs with MVD recommend thoracic radiographs for all dogs with suspected MVD to assess the hemodynamic significance of the murmur, as well as to obtain a baseline image when the dog is asymptomatic. Echocardiography should be performed to confirm the suspected cause of an identified murmur and to document the presence or absence of cardiac chamber enlargement. There is increasing evidence of the value of echocardiography in dogs with preclinical disease.

Left atrial and left ventricular enlargement may be evident on thoracic radiographs. When CHF develops, additional radiographic features include pulmonary venous congestion and pulmonary parenchymal infiltrate, typically with a perihilar or caudal and dorsal distribution.

Echocardiography (Video 27.1)

In addition to characteristic changes in the mitral valve (mild to moderate thickening and irregularity), echocardiographic findings typically include left atrial dilation and left ventricular dilation. Initially, indices of systolic function (shortening fraction) are within normal limits and commonly supranormal in dogs with important volumetric regurgitation. Echocardiographic evidence of myocardial systolic failure is indicative of advanced disease. Occasionally, echocardiography documents chordal rupture or pericardial effusion secondary to an atrial tear or concurrent right heart failure.

Laboratory Findings

Laboratory abnormalities are typically unremarkable unless cardiac output is reduced sufficiently to reduce organ perfusion (e.g., prerenal azotemia). Evidence of chronic renal disease may be noted in many affected animals. Cavalier King Charles spaniels with mitral MVD appear to have an increased plasma fibrinogen concentration and low plasma von Willebrand factor. The latter is likely due to their destruction via shear stress to the blood. The risk these changes pose for thromboembolic risk is unknown.

DIFFERENTIAL DIAGNOSIS

Other causes of heart failure and cardiac murmur in mature dogs include dilated cardiomyopathy and previously undiagnosed or unrepaired congenital heart disease (patent ductus arteriosus [PDA], ventricular septal defect [VSD], mitral dysplasia, subvalvular aortic stenosis [SAS]). Primary differentials include chronic bronchitis, heartworm disease, tracheal collapse, pneumonia, and primary or metastatic pulmonary neoplasia.

MEDICAL MANAGEMENT

The benefits of medical intervention prior to the onset of clinical signs has been controversial; however, recent data suggest that the administration of pimobendan prior to the onset of CHF in dogs with MVD and cardiomegaly results in prolongation of the preclinical period by approximately 15 months.[4] Once CHF is documented, treatment with diuretics (e.g., furosemide; see Box 27.1), ACE inhibitors (e.g., enalapril, benazepril; see Box 27.1), and inodilators (e.g., pimobendan; see Box 27.1) is indicated. Additional diuretics (e.g., spironolactone, hydrochlorothiazide) and vasodilators (e.g., hydralazine, amlodipine) are frequently used in refractory cases. The use of β-blockers in dogs with MVD is controversial as they can exacerbate unstable CHF, although they may improve quality of life and functional class improvement in certain dogs without affecting echocardiographic variables. Inadequate control of atrial fibrillation in non-small-breed dogs has been shown to increase mortality rate.[5] In this study, the administration of diltiazem and digoxin increased median survival time when compared to diltiazem alone (130 vs. 35 days).

SURGICAL TREATMENT

Mitral valve replacement and repair have been reported in dogs. For mitral valve replacement, a mechanical or bioprosthetic heart valve is typically used. Disadvantages of these valves are that the animal must be placed on lifelong anticoagulation therapy, and early pannus formation and calcification occur with bioprosthetic valves. Valvular replacement requires cardiopulmonary bypass. Mitral valve repair may offer advantages over valve replacement in that long-term anticoagulation is not required and myocardial function is better preserved. However, results are variable and are highly dependent on the surgeon's experience.

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NOTE Techniques for mitral valve repair that use cardiopulmonary bypass are probably safer than those that do not, particularly in animals with severe heart failure. However, bypass procedures are expensive and are not readily available in veterinary medicine.

Preoperative Management

Preoperative arrhythmias should be controlled before surgery. Affected animals typically have signs of CHF, and treatment with positive inotropes (e.g., pimobendan), vasodilators (e.g., hydralazine, enalapril, or amlodipine), and diuretics (e.g., furosemide, spironolactone) is indicated (see discussion under Medical Management).

Anesthesia

The goal of managing the patient with MR while under anesthesia is to maximize forward flow of blood and minimize the regurgitant fraction (see Tables 27.1 and 27.2). To do this, heart rate needs to be kept at high normal values and blood pressure needs to be normal to 20% below normal. A slower heart rate allows for larger filling volumes, potentially leading to left ventricular distention and dilation of the mitral annulus. This can result in a larger regurgitant fraction. Treat bradycardia with anticholinergics (e.g., atropine, glycopyrrolate). Patients with mild or moderate disease usually have adequate left ventricular function and normal end-diastolic ventricular pressures. Therefore they are able to perfuse the myocardium with low-normal systemic blood pressures. With lowering of SVR, the pressure gradient across the aortic valve favors forward flow of blood into the aorta. This reduces the regurgitant fraction, increases cardiac output, and increases blood pressure. High systemic blood pressure should be avoided. If it does occur, hypertension should be treated immediately by deepening the anesthesia with an inhalational agent and giving a rapid-acting opioid (e.g., fentanyl). If the initial treatment is inadequate, nitroglycerin or nitroprusside may be titrated to effect as a constant-rate infusion.

Several drugs should be avoided in patients with MR. The alpha-2 agonists are contraindicated in affected animals. Hypertension can cause a marked increase in the regurgitant fraction, resulting in a decrease in cardiac output and potentially cardiovascular collapse. Although acepromazine causes a decrease in SVR, many of these patients are geriatric with hepatic congestion, and its metabolism may be prolonged. Therefore acepromazine should be used very cautiously and probably only in patients with mild valvular disease. Propofol causes a decrease in SVR that accounts for part of the hypotension seen with its use. However, it also causes a decrease in myocardial contractility. This is undesirable in patients with moderate to severe MR. In patients with mild valvular dysfunction, propofol or alfaxalone may be slowly titrated to effect as an induction drug. In moderate to severe cases of MR, etomidate is a better choice for induction. Because ketamine maintains sympathetic tone and thus SVR, it is a poor choice for induction in MR patients.

If hypotension needs to be treated while the patient is under anesthesia, ephedrine (0.1–0.25 mg/kg IV) is a good choice. Because ephedrine causes stimulation of beta receptors in the heart, it also causes a mild increase in contractility and heart rate. Phenylephrine in low doses causes venoconstriction with a subsequent increase in preload. However, at higher doses, the increase in SVR caused by phenylephrine is undesirable. For this reason, epinephrine is often the vasopressor of choice. It has less effect on SVR and a greater effect on both heart rate and contractility.

To avoid increases in pulmonary vascular resistance (PVR) and worsening pulmonary hypertension, avoid acidosis, nitrous oxide, hypoxemia, hypoventilation, and hypercarbia. Refer to Tables 27.1 and 27.2 for anesthetic recommendations for patients with MR undergoing cardiac or noncardiac procedures.

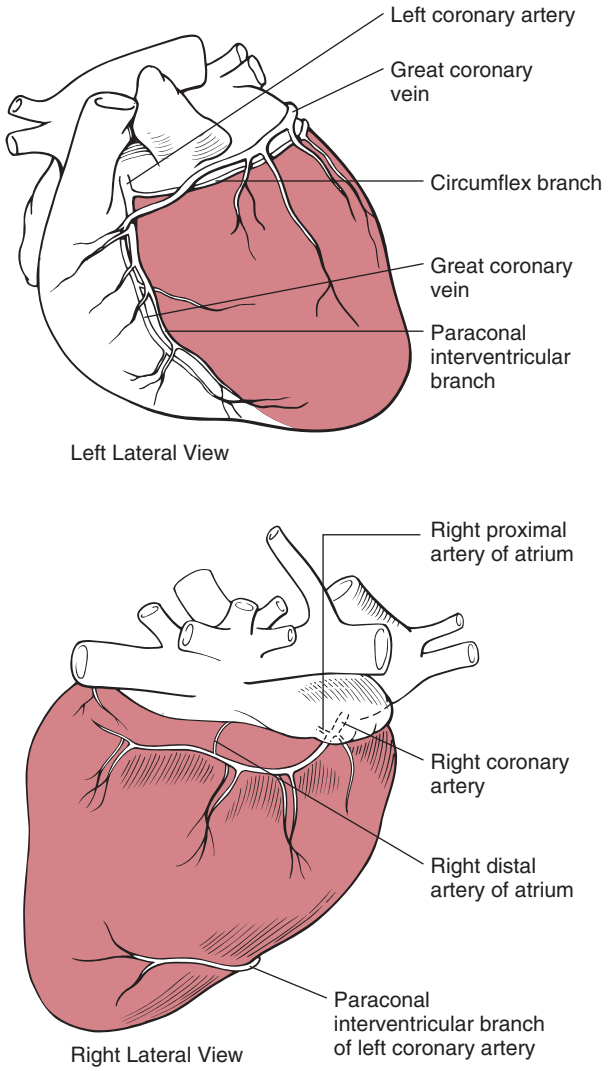


FIG. 27.6 Anatomy of the coronary vessels in a dog.

Surgical Anatomy

The right and left coronary vessels (Fig. 27.6) arise from the aortic bulb immediately distal to the aortic valve. The right coronary artery arises from the right sinus of the aorta and curves to the right and ventrocranially, lying in the fat of the coronary groove. Its initial part is bounded by the pulmonary trunk and the conus arteriosus craniolaterally; dorsally it is covered by the right auricle. The left coronary artery is a short trunk approximately 5 mm long and nearly as wide. It terminates in the circumflex and paraconal interventricular branches. The circumflex branch lies in the coronary groove as it extends to the left. On approaching the dorsal interventricular groove, it turns toward the apex of the heart and is known as the *subsinuosal interventricular branch*. The combined length of the circumflex

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and subsinuosal branches is approximately 8 cm in the dog. The paraconal interventricular branch is approximately 1.5 mm wide and 7 cm long. It winds obliquely and distally from left to right across the sternocostal surface of the heart in the paraconal interventricular groove.

Positioning

Mitral valve repair or replacement may be done via right- or left-sided thoracotomy. The entire thorax is prepped for aseptic surgery.

SURGICAL TECHNIQUE

AP For a description of mitral valve repair or replacement, readers are referred to a cardiac surgery textbook and recent literature.[6]

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative pain should be treated with systemic opioids (see Chapter 13) and local anesthetic techniques (see Box 30.2). Animals should be monitored for pulmonary edema after surgery. If pulmonary edema occurs, it should be treated with furosemide (see Box 27.1). Left ventricular failure should be treated as outlined in the Medical Management section.

PROGNOSIS

MVD is a common cardiac condition, but most dogs remain nonclinical during their lifetime. For dogs with progressive disease and heart failure, prognosis is related to cardiac size and severity of MR, type of pharmacologic and adjunctive therapy, cardiac cachexia, complications including arrhythmias and pulmonary hypertension, and other concomitant disease.

Mitral valve replacement with a mechanical valve prosthesis has been performed with a median survival after surgery of 4.5 months.[7] Although most dogs survive surgery, a high incidence of prosthetic valve thrombosis was reported, which limits application of this technique. In 48 small-breed dogs undergoing mitral valve repair (e.g., circumferential annuloplasty, placement of artificial chordae), 93% survived until hospital discharge with improved clinical signs and all survived for at least 5 months after surgery.[6] In this same study, 17 dogs survived 18 to 24 months and 12 dogs survived greater than 3 years.

PATENT DUCTUS ARTERIOSUS

DEFINITIONS

The **ductus arteriosus** is a fetal vessel that connects the main pulmonary artery and the descending aorta. During development, it shunts blood away from collapsed fetal lungs. Normally it closes shortly after birth during the transition from fetal to extrauterine life. Continued patency of the ductus arteriosus for longer than a few days after birth is called **patent ductus arteriosus (PDA).**

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

PDA is one of the most common congenital heart defects of dogs; it occurs infrequently in cats. PDA typically causes a left-to-right shunt that results in volume overload of the left ventricle and produces left ventricular dilation. Progressive left ventricular dilation distends the mitral valve annulus, causing secondary regurgitation and additional ventricular overload. This severe volume overload leads to left-sided CHF and pulmonary edema, usually within the first year of life. Atrial fibrillation may occur as a late sequela because of notable left atrial dilation.

Occasionally, animals with PDA develop suprasystemic pulmonary hypertension that reverses the direction of flow through the shunt, causing severe hypoxemia and cyanosis (Eisenmenger's physiology). Right-to-left PDA can occur as a late sequela (6 months) to untreated PDA. When right-to-left PDA is noted in very young animals, it may be due to persistent pulmonary hypertension after birth. Reversal of right-to-left PDA lessens the risk for developing progressive left-sided heart failure but causes severe debilitating systemic hypoxemia, exercise intolerance, and progressive polycythemia.

NOTE Animals with PDA should not be used for breeding, regardless of the breed.

DIAGNOSIS

Clinical Presentation

Signalment

Small-breed dogs (e.g., bichon frise, Chihuahua, poodle, Pomeranian, Yorkshire terrier) are most commonly affected; however, PDA also occurs commonly in German shepherd dogs and Shetland sheepdogs. Yorkshire terriers were the most common purebred dogs reported in a large retrospective study.[8] Females are more commonly affected than males. In the aforementioned study, median age at the time of diagnosis was 5.1 months.

Although PDA is a relatively rare condition in cats, it does occur. Males and females appear to be at similar risk and domestic shorthair cats are most commonly affected. In a 2017 retrospective study of 50 cats with PDA, the median age at presentation was 6 months.[9]

History

Most young dogs with PDA have no clinical signs; however, in clinically affected dogs, the most common complaint is cough, shortness of breath, or both as a result of pulmonary edema. Animals with right-to-left or reverse PDA may be nonclinical or may have exercise intolerance and pelvic limb collapse during exercise. Most cats with PDA have a cardiac murmur but are otherwise asymptomatic at presentation.

Physical Examination Findings

The most prominent physical finding associated with PDA is a characteristic continuous (machinery) murmur heard best at the high left heart base or left axillary region. The left apical cardiac impulse is prominent and is displaced caudally, and a palpable cardiac "thrill" is often present. Femoral pulses are strong or hyperkinetic (water hammer pulse) owing to a wide pulse pressure caused by diastolic runoff of blood through the ductus. Tall R waves (>2.5 mV in lead II) or wide P waves on a lead II ECG are supportive of the diagnosis, but they are not always present. Atrial fibrillation or ventricular ectopy may occur in advanced cases.

Physical examination findings in animals with right-to-left or reverse PDA differ from findings in those with left-to-right

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shunts. Differential cyanosis is typically present (i.e., most apparent in the caudal mucous membranes), but cyanosis may also be noted in the cranial half of the body in some animals. Cyanosis occurs as a result of mixture of nonoxygenated blood (from the pulmonary artery) with oxygenated aortic blood. Femoral pulses are normal. A systolic cardiac murmur, rather than a machinery murmur, may be present. However, a murmur may not be auscultated if polycythemia is present, if left- and right-sided pressures are nearly equal, and if shunting of blood through the ductus is minimal.

In one study in cats, continuous murmurs were found in 55% of cases, whereas 45% had systolic murmurs.[9] Pulmonary hypertension was reported in nearly half of affected cats; importantly, concurrent cardiac anomalies may be found in 25% to 50%.[9,10]

Diagnostic Imaging

Thoracic radiographs typically show left atrial and ventricular enlargement, pulmonary vasculature overcirculation, and characteristic dilation of the descending aorta and sometimes the main pulmonary artery on the dorsoventral view. With right-to-left PDA, thoracic radiographs show evidence of biventricular enlargement, notable dilation of the main pulmonary artery segment, and enlargement and tortuosity of lobar pulmonary arteries. Nuclear scintigraphy can be used to quantitate left-to-right shunts and to diagnose right-to-left shunts.

Echocardiography

Echocardiography provides information that further confirms PDA and helps exclude concurrent cardiac defects, but it is not invariably required to establish the diagnosis. Echocardiographic findings that support a diagnosis of PDA include left atrial enlargement, left ventricular dilation, pulmonary artery dilation, increased transaortic and transmitral flow velocities, and a characteristic reverse turbulent Doppler flow pattern in the pulmonary artery. Echocardiographic features of right-to-left PDA typically include right ventricular dilation and thickening, dilation of the main pulmonary artery, and flattening of the interventricular septum. A right-to-left PDA can be documented by performing a saline microbubble contrast echocardiogram. Observing microbubbles in the descending aorta, but not in any left-sided cardiac chamber, is diagnostic.

Angiography

Angiographic studies determine the ductal morphology and the minimal ductal diameter of the PDA (Box 27.5). This information is helpful for predicting procedural feasibility with intravascular coils or ductal occluders.

Laboratory Findings

Laboratory abnormalities are uncommon in animals with left-to-right shunting PDA; however, animals with right-to-left shunts are commonly polycythemic. Polycythemia occurs in response to increased erythropoietin production due to chronic hypoxemia.

DIFFERENTIAL DIAGNOSIS

The characteristic physical examination findings (i.e., continuous murmur and bounding arterial pulses) make diagnosis of PDA straightforward in most affected animals. Rarely, a combination of AS and/or aortic insufficiency (see p. 807), or VSD and/or aortic insufficiency, causes a to-and-fro murmur that may be difficult to differentiate from continuous PDA murmurs. In animals in which the diastolic component of the PDA murmur is difficult to detect, other differentials would include subaortic stenosis, pulmonic stenosis (PS), atrial septal defect (ASD), and VSD. Differentials for dogs with right-to-left PDA include tetralogy of Fallot (TOF), right-to-left shunting, ASD or VSD, and other rare complex forms of cyanotic heart disease.

BOX 27.5 Angiographic Classification of Patent Ductus Arteriosus Morphology

Type I
Diameter of the ductus gradually decreases in size from the aorta to the pulmonary artery

Type IIA (Most Common)
Walls of the ductus parallel to each other with abrupt decrease in diameter of the ductus at the pulmonary ostium

Type IIB
Diameter of the ductus markedly decreases in size from the aorta to the pulmonary artery

Type III
Ductus is tubular with little or no change in diameter throughout its length

PDA, Patent ductus arteriosus.
From Miller MW, Gordon SG, Saunders AG, et al. Angiographic classification of patent ductus arteriosus morphology in the dog. *J Vet Cardiol.* 2006;8:109–114.

MEDICAL MANAGEMENT

Animals with pulmonary edema should be given furosemide (see Box 27.1) for 24 to 48 hours before surgery. If atrial fibrillation is present, the ventricular response rate should be controlled with a β-adrenergic blocker or a calcium channel blocker (with or without digoxin) or amiodarone before surgery. If hemodynamically significant arrhythmias are present, they must also be controlled. Complete resolution of clinical signs of CHF may be difficult or impossible with medical management alone. Long-term medical management of dogs with right-to-left PDA has been described in only a small number of dogs using phlebotomy or hydroxyurea.

SURGICAL TREATMENT

Intravascular coils, vascular plugs, and ductal occluders are now used routinely for closure of PDA (Fig. 27.7). These techniques have the advantage of not requiring a thoracotomy and have less risk for major complications; however, mortality rates are comparable between transcatheter arterial occlusion and surgical ligation. Ductal occlusion is most commonly performed from access through the femoral artery, although coil embolization through the carotid artery has been described, as has a transvenous approach through the femoral vein. The coil(s), plugs, or occluders are placed in the ductus under fluoroscopic guidance, and extent of occlusion is verified by injection of contrast agent into the aorta (Fig. 27.8).

Amplatzer Vascular Plugs and the Amplatzer Canine Duct Occluder (ACDO; Fig. 27.9) are self-expanding multilayered nitinol devices that have been shown to effectively occlude a

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FIG. 27.7 Intravascular coil used to occlude a patent ductus arteriosus. Intravascular coils have largely been replaced by duct occluders (see Fig. 27.9). (Courtesy Dr. M. Miller, VetMed, Phoenix, AZ.)

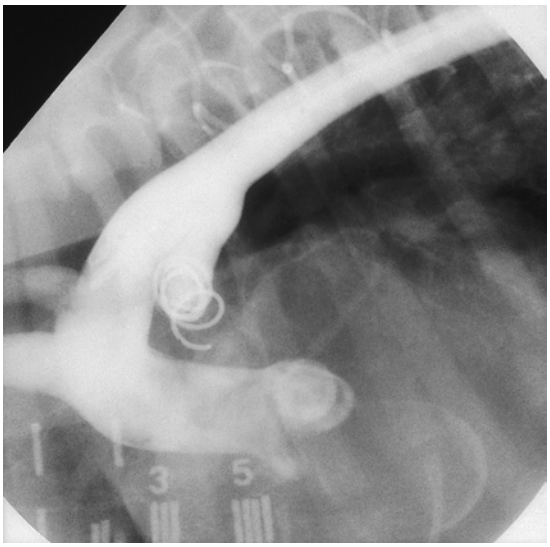


FIG. 27.8 Radiograph taken after deployment of a coil in a ductus. Contrast has been injected into the aorta at the site of coil deployment to verify complete occlusion of the ductus. (Courtesy Dr. M. Miller, VetMed, Phoenix, AZ.)

wide range of ductal shapes and sizes with better outcomes over surgical ligation and other methods of transcatheter occlusion. A study comparing occlusion devices and approaches found ACDO from a transarterial approach to be easier to perform, have significantly less complications, and have higher completeness of occlusion.[11] Patient size was found to be the only limiting factor for this device; however, successful PDA occlusion has been achieved in small dogs (1.5–4.2 kg) using a low-profile ACDO prototype.[12]

Surgical correction of PDA is most commonly accomplished by circumferential ligation of the ductus arteriosus. Ligation of PDA is considered curative and should be performed as soon as possible after diagnosis. Secondary MR usually regresses after surgery owing to reduction in left ventricular dilation. Ligation may be performed using a standard dissection technique, an intrapericardial approach, or the Jackson-Henderson method (see p. 804). The latter technique carries a higher risk of residual

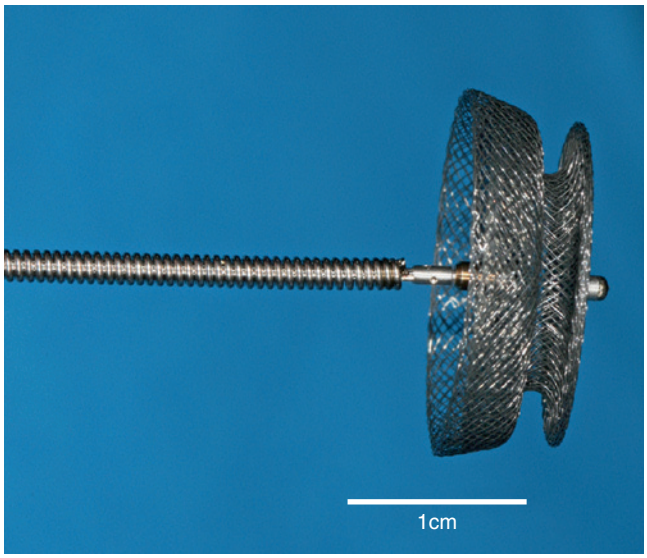


FIG. 27.9 Amplatzer Canine Duct Occluder. (Courtesy Dr. M. Miller, VetMed, Phoenix, AZ.)

flow and should be used only when bleeding or rupture associated with the standard dissection precludes its use.

Inadvertent ductal rupture during dissection is the most serious complication associated with PDA repair. The risk of this complication decreases with more experienced surgeons. Small ruptures, especially those on the medial aspect of the ductus, often respond to gentle tamponade, but tears may enlarge and worsen if dissection is continued. If bleeding occurs, sodium nitroprusside (5–25 µg/kg per minute IV to effect) may be administered to lower the systemic mean arterial pressure to 50 to 65 mm Hg within 5 to 10 minutes, so that ligation can be continued. If bleeding is severe, vascular clamps may be needed to occlude the aorta while the ductus is ligated. Once bleeding is controlled, a decision must be made whether to continue surgery if the ductus was not ligated or to stop in favor of repair at a later time. Second surgeries are more difficult owing to adhesions at the surgical site, so complete occlusion should be attempted during the initial procedure, if possible. Simple ductal ligation is often not possible after a rupture has occurred. In such instances, surgical alternatives include ductal closure with pledget-buttressed mattress sutures or ductal division and closure between vascular clamps. The divided ductal ends are closed with a continuous mattress suture oversewn with a simple continuous pattern. Ductal closure without division is safer than with surgical division, but recannulation of the ductus may occur. Because ductal division requires additional technical expertise, only experienced surgeons should perform this procedure.

Placement of hemoclips on the ductus to make medial dissection unnecessary has been described. Hemorrhage occurs at the same rate as for ligation methods and is associated with dissection of the cranial aspect of the ductus.

Preoperative Management

Preoperative arrhythmias should be controlled before surgery. If the animal has signs of CHF, treatment with inodilators (e.g., pimobendan), vasodilators (e.g., hydralazine or enalapril), and diuretics (e.g., furosemide; see Box 27.1) should be initiated preoperatively. Excessive diuretics and/or vasodilators may cause hypotension and should be avoided.

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Anesthesia

Bradycardia occasionally occurs during PDA ligation. An anticholinergic (e.g., atropine or glycopyrrolate) should be available and should be given if the heart rate drops to below 60 beats/min in the dog. Blood should be available for transfusion if excessive hemorrhage occurs during the surgical procedure. Techniques for anesthetic management of cardiovascular patients are discussed on p. 788. Bupivacaine may be injected intercostally during surgery to supplement analgesia (see pp. 152 and 918).

Surgical Anatomy

The ductus arteriosus in dogs and cats is usually wide (approximately 1 cm) but relatively short (<1 cm). It is located between the aorta and the main pulmonary artery, caudal to the origin of the brachycephalic and left subclavian arteries. The left vagus nerve always passes over the ductus arteriosus and must be identified and retracted during dissection. Often the left recurrent laryngeal nerve can be identified as it loops around the ductus.

Positioning

The animal is positioned in right lateral recumbency, and the left thorax is prepared for aseptic surgery.

SURGICAL TECHNIQUE

Standard Approach

Perform a left fourth space intercostal thoracotomy (see p. 889). Identify the left vagus nerve as it courses over the ductus arteriosus and isolate it using sharp dissection at the level of the ductus. Place a soft suture (e.g., silk or multifilament absorbable suture) or umbilical tape around the nerve and gently retract it (Fig. 27.10). Isolate the ductus arteriosus by bluntly dissecting around it without opening the pericardial sac. Pass a right-angle forceps behind the ductus, parallel to its transverse plane, to isolate the caudal aspect of the ductus. Then dissect the cranial aspect of the ductus by angling the forceps caudally approximately 45 degrees (Fig. 27.11). Complete dissection of the ductus by passing forceps from medial to the ductus in a caudal-to-cranial direction. Grasp a strand of suture (2-0, 0, or 1 silk) with right-angle forceps. Slowly pull the suture beneath the ductus. If the suture does not slide easily around the ductus, do not force it. Regrasp the suture and repeat the process while taking care not to include surrounding soft tissue in the forceps. Pass a second suture using the same maneuver. The suture may also be passed as a double loop and the suture cut so that the surgeon has two strands (Fig. 27.12) rather than passing behind the ductus twice. *Slowly tighten the suture closest to the aorta first. Then tighten the remaining suture.*

Alternatively, use an intrapericardial approach. Make an incision through the pericardium and mediastinal pleura perpendicular to the ductus at the level where the ductus enters the pulmonary artery. The proposed advantage of this approach is that it subjectively leads to increased exposure and access to the cranial and caudal aspects of the PDA for development of the dissection planes.[13]

Jackson-Henderson Approach

Approach the ductus as described previously. With scissors, incise the mediastinal pleura dorsal to the aorta from the origin of the left subclavian artery cranially to the origin of the first intercostal artery caudally. Bluntly dissect the loose areolar tissue on the medial aspect of the aorta. Insert a right-angled forceps immediately cranial to the ductus and pass it around the aorta from ventral to dorsal while gently elevating the aortic arch with a finger. Pass a loop of ligature from the dorsomedial aspect of the aorta to the cranial aspect of the ductus, ventral to the aorta. Then insert the right-angled forceps immediately caudal to the ductus, and pass it around the aorta from ventral to dorsal to pick up the two free strands of the

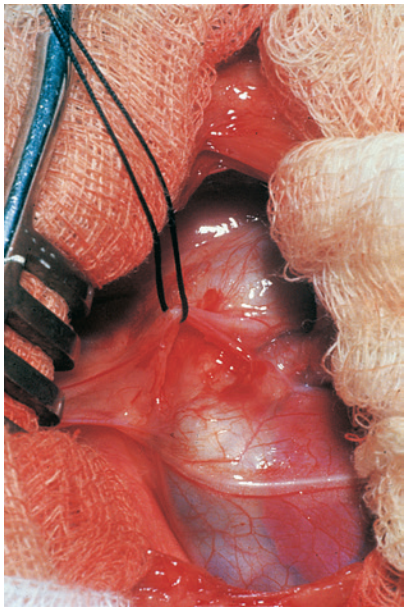


FIG. 27.10 During patent ductus arteriosus ligation, elevate and retract the left vagus nerve to expose the ductus arteriosus. The left recurrent laryngeal nerve may be seen as it separates from the vagus nerve and courses caudally around the ductus arteriosus.

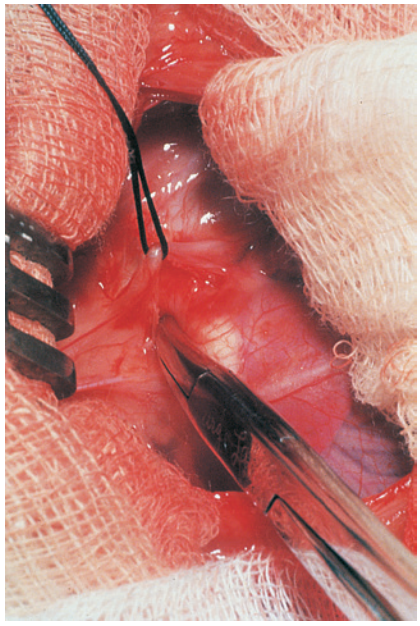


FIG. 27.11 Isolate the craniomedial aspect of the ductus arteriosus by bluntly dissecting with an angled forceps. The forceps should be directed at a 45-degree angle from the transverse plane.

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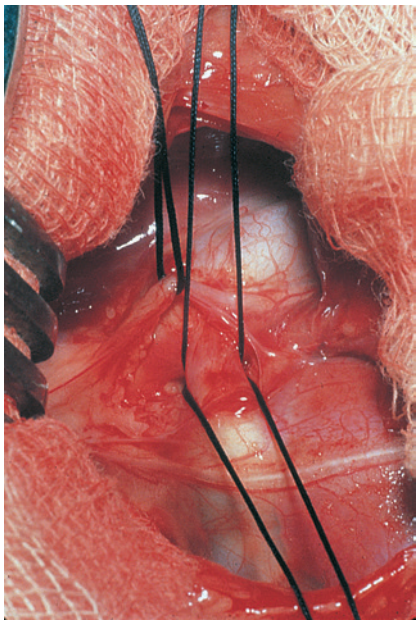


FIG. 27.12 Ligate the patent ductus arteriosus by passing two ligatures around the ductus arteriosus. Tie the ligatures separately.

ligature. Draw the strands ventral around the ductus, and divide the loop to form two individual strands. Tie the ligature as described earlier.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Heavy silk (No. 2-0, 0, or 1) and umbilical tape are suitable materials for ductal ligation. Large hemoclips have also been used. Right-angle forceps are best suited for blunt dissection of the PDA and for passing ligatures. Angled or tangential vascular clamps are required for surgical division of the PDA and for repair of inadvertent ruptures. Polypropylene mattress sutures (3-0 to 4-0) buttressed with Teflon pledgets are used for repair of ruptured PDA.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative pain should be treated with systemic opioids and local anesthetic techniques. Young animals should be fed as soon as they are fully recovered from surgery. Thoracostomy tubes are occasionally placed before thoracic closure (e.g., if intraoperative bleeding has occurred). They can generally be removed within 4 to 24 hours after surgery.

COMPLICATIONS

The most serious complication of PDA ligation is rupture. Ductal rupture and other major complications occur in 6% to 15% of cases, with mortality rates ranging from 0% to 5.6%. In a large retrospective study, the mortality rate within the first 3 days of 456 dogs was 2.6% ($n = 12$) and was not significantly different between catheter-based (5 of 276, 1.8%) and surgical (5 of 178, 2.8%; $P = .5$) closure.[8] Residual shunting may also be a complication but is more common with the Jackson-Henderson surgical approach and with transarterial coils. Pulmonary embolization of transcatheter coils or other devices used to embolize PDA occasionally occurs but does not appear to cause short- or long-term complications. Infection of cardiovascular implants is uncommon.

PROGNOSIS

Dogs with untreated PDA usually develop progressive left-sided CHF and pulmonary edema. Most dogs with untreated PDA die before 1 year of age, whereas the mortality rate within the first 3 days in 456 dogs was 2.6% ($n = 12$).[8] Excluding two dogs that died before starting a procedure, mortality rate was not significantly different between catheter-based (5 of 276, 1.8%) and surgical (5 of 178, 2.8%; $P = .5$) closure. Dogs with MR before surgery have similar survival times to dogs without MR; however, older animals and those with left atrial dilation on preoperative radiographs are less likely to survive. Dogs with PDA may also develop suprasystemic pulmonary hypertension that reverses the direction of the shunt, causing severe hypoxemia, cyanosis, and exercise intolerance. Ligation of a completely reversed PDA is contraindicated.

Although PDA is an uncommon congenital heart condition in cats when compared with dogs, there are several notable differences between the species. There is an increased prevalence of concurrent cardiac abnormalities (32%–55%) and concurrent pulmonary hypertension (37%–46%) in cats.[9,10,14] The presence of these concurrent conditions may affect long-term survival.

PULMONIC STENOSIS

DEFINITIONS

PS is a congenital narrowing of the pulmonic valve, pulmonary artery, or right ventricular outflow tract. Synonyms include *pulmonic valve dysplasia* and *right ventricular outflow tract obstruction*.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

PS was historically the third most common congenital heart defect in dogs after PDA and SAS; it is uncommon in cats. However, retrospective studies have found PS to be the most common congenital heart defect.[15,16] In dogs, the condition is usually valvular, although supravalvular and subvalvular defects have been reported. Subvalvular stenosis can occur as a primary isolated defect but more often occurs from infundibular hypertrophy secondary to primary valvular stenosis. Valvular stenosis may be simple, consisting of incomplete separation of valve leaflets, or it may be due to valve dysplasia characterized by a hypoplastic valve annulus and thickened immobile valve leaflets. More than 80% of dogs with valvular PS have some degree of valve dysplasia. Based on their echocardiographic valvular anatomy and aortic-to-pulmonary annular ratio, affected dogs may be classified as having **type A PS** (normal annulus diameter and aortic-to-pulmonary ratio ≤1.2) or **type B PS** (pulmonary annulus hypoplasia and aortic-to-pulmonary ratio >1.2). This classification is also associated with the outcome following valvuloplasty.

PS causes pressure overload and hypertrophy of the right ventricle. Right ventricular hypertrophy often compounds right ventricular outflow obstruction by narrowing the right ventricular outflow tract. Narrowing of the right ventricular outflow tract is greatest during systole, producing a dynamic obstruction that

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contributes to the fixed stenosis. Dynamic stenosis has important implications for surgical repair of PS. Dogs with mild to moderate obstruction may remain asymptomatic, whereas dogs with severe obstruction may show exercise intolerance, syncope, progressive right-sided CHF, or sudden death.

DIAGNOSIS

Clinical Presentation

Signalment

English bulldogs, Scottish terriers, wirehaired fox terriers, beagles, miniature schnauzers, cocker spaniels, Samoyeds, and mastiffs are at increased risk for developing PS. English bulldogs and boxers have a high concurrent incidence of aberrant left coronary artery (caused by a single right coronary artery), which has important surgical implications. Female English bulldogs and male bull mastiffs are more commonly affected; a sex predisposition has not been identified in other breeds. A hereditary form of pulmonary valve dysplasia has been found in beagles and Boykin spaniels.

History

Young animals with PS are often asymptomatic. Advanced cases may present with exercise intolerance, syncope, or abdominal distention from ascites.

Physical Examination Findings

The predominant physical finding is a systolic ejection murmur heard best at the left heart base. The ECG may show prominent S waves in leads I, II, III, and aVF, indicative of a right axis shift and right ventricular hypertrophy.

Diagnostic Imaging

Thoracic radiographs show varying degrees of right ventricular enlargement and main pulmonary artery segment enlargement. Diagnosis of PS can be confirmed by echocardiography. Cardiac catheterization is usually necessary only if abnormal coronary anatomy is suspected or if an interventional procedure (e.g., percutaneous balloon valvuloplasty) is performed.

Echocardiography

Echocardiographic findings include right ventricular hypertrophy, poststenotic dilation of the main pulmonary artery, malformation and reduced mobility of the pulmonic valve, and high pulmonary flow velocity. A systolic pressure gradient across the stenosis can be measured directly by right ventricular catheterization or can be calculated from the Doppler-derived peak systolic pulmonic flow velocity using the modified Bernoulli equation ($\Delta P = 4\ V^2$, where ΔP is instantaneous pressure gradient and V is velocity).

Laboratory Findings

Specific laboratory abnormalities are not found in animals with PS.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include SAS, VSD, ASD, and TOF.

MEDICAL MANAGEMENT

No specific medical therapy for PS is known other than symptomatic treatment for CHF, if it occurs.

INTERVENTIONAL TREATMENT

Percutaneous balloon valvuloplasty is a nonsurgical alternative for correction of moderate to severe PS, provided that facilities and skilled personnel for cardiac catheterization are available. Simple valvular PS is more amenable to balloon valvuloplasty than is severe pulmonic valve dysplasia or severe PS with dynamic obstruction. Balloon valvuloplasty has become the treatment of choice for dogs with amenable lesions (valvular dysplasia). Dogs with type A stenosis are most likely to benefit from balloon valvuloplasty. Balloon valvuloplasty is not recommended for patients with supravalvular PS, patients with coronary artery anomalies, or patients with substantial annular hypoplasia.

English bulldogs with PS present a therapeutic dilemma because of the possibility of concurrent aberrant left coronary artery. In dogs with this defect, the left coronary artery courses across the right ventricular outflow tract and is at risk for injury during valve dilation. Sudden death due to rupture of the coronary artery has occurred during balloon valvuloplasty. Conservative balloon valvuloplasty (using a balloon the size of the pulmonary annulus or smaller) was successfully performed in four English bulldogs; minimal change in the pressure gradient was achieved, but ratios of the pulmonary artery-to-aortic velocity time integral (indicator of PS progression) improved.[17]

SURGICAL TREATMENT

Therapy for PS is based on its degree of severity and the type of lesion present. Severity is judged by the presence of signs, the extent of right ventricular hypertrophy, and the magnitude of the systolic pressure gradient. Doppler-derived systolic pressure gradients measured in unsedated or unanesthetized animals are considered mild when they are less than 50 mm Hg, moderate when they are between 50 and 75 mm Hg, and severe when they are greater than 75 mm Hg. Animals with PS that have no signs, mild hypertrophy, and a pressure gradient less than 50 mm Hg generally do not require surgical intervention. If the pressure gradient is greater than 50 mm Hg, and if right ventricular hypertrophy is significant, correction should be considered.

Surgical options for correction of PS include valve dilation and patch-graft valvuloplasty. With the advent of balloon valvuloplasty, operative valve dilation is seldom indicated. Animals with severe annular hypoplasia, dysplastic valve lesions, or severe hypertrophy are less likely to respond to valve dilation. Aberrant left coronary artery precludes patch-graft valvuloplasty. In such cases, a valved or nonvalved conduit placed between the right ventricle and pulmonary artery is a possible surgical option for this condition. Open valvotomy, pulmonary valve commissurotomy, and biomembrane patch grafting under cardiopulmonary bypass has been reported in nine small dogs with severe PS.[18] Palliative stenting of the right ventricular outflow tract using balloon expandable stents has been reported in two dogs with dysplastic pulmonary valve stenosis and right-to-left intracardiac shunting defects.[19]

Preoperative Management

Right-sided CHF or cardiac arrhythmias should be managed medically before surgery is performed. See preoperative management of animals with cardiovascular disease on p. 788.

Anesthesia

Refer to p. 788 for anesthetic management of cardiac patients.

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Surgical Anatomy

The pulmonary valve is approached through a left fourth or fifth intercostal thoracotomy or median sternotomy. This valve consists of right, left, and intermediate semilunar cusps. Sounds associated with lesions of the pulmonary valve may be heard best at the fourth intercostal space, slightly below a line drawn through the point of the shoulder. See previous comments about concurrent aberrant left coronary arteries.

Positioning

Animals are positioned in right lateral recumbency, and the entire left hemithorax is prepared for aseptic surgery.

SURGICAL TECHNIQUE

Patch-graft valvuloplasty is indicated for severe PS, particularly if marked infundibular hypertrophy and dynamic stenosis are suspected. Patch-graft valvuloplasty can also be used effectively to relieve concurrent or isolated supravalvular PS. It may be performed with or without inflow occlusion (with mild hypothermia: 32°C–34°C) or using cardiopulmonary bypass. If inflow occlusion is used, circulatory arrest time should be less than 5 minutes.

Open Patch-Graft Correction

With Inflow Occlusion

AP *Perform a left fifth intercostal thoracotomy. Pass tape tourniquets around the venae cavae and azygos vein (see Inflow Occlusion on p. 795). Make a partial-thickness incision in the right ventricular outflow tract (Fig. 27.13A). Suture an autogenous pericardial or synthetic patch to the ventriculotomy incision and the cranial aspect of the pulmonary artery (Fig. 27.13B). Initiate venous inflow occlusion and make full-thickness incisions into the pulmonary artery and right ventricle (Fig. 27.13C). Incise or excise dysplastic pulmonic valve leaflets as necessary. Complete suturing of the pulmonary artery to the patch graft and discontinue inflow occlusion (Fig. 27.13D–E). Resuscitate the heart.*

It is important to remove air from the heart by discontinuing inflow occlusion just before tying the last suture.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Polypropylene (e.g., 3-0 Prolene or Surgipro) suture buttressed with Teflon pledgets is suitable for transventricular valve dilation. Valve dilation can be accomplished with a Cooley or Tubbs valve-dilating instrument or with hemostatic forceps of appropriate size. Synthetic materials such as polytetrafluoroethylene (PTFE) or autogenous pericardium can be used for the patch-graft procedure. Polypropylene (4-0) suture is appropriate for suturing the patch graft.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative pain should be treated with systemic opioids and local anesthetic techniques (see p. 886 for postthoracotomy analgesia). Animals should be monitored postoperatively for complications associated with low output failure due to reduced function of the right ventricle.

PROGNOSIS

The prognosis for dogs with PS depends on its severity. Animals with systolic pressure gradients greater than 60 mm Hg are likely to experience heart failure or sudden death early in life. In a study of 55 dogs with PS that did not undergo balloon valvuloplasty or surgery, 13% had a cardiac cause of death within the study time frame (median follow-up = 1096 days; interquartile range = 648–1926 days); dogs in this study with concurrent tricuspid regurgitation were at 16 times greater risk for cardiac death.[20] In a separate study dogs with severe PS not receiving balloon valvuloplasty or surgery had a one-year mortality rate of 53%.[21]

Balloon valvuloplasty is associated with minimal risk of complications and carries low operative mortality, and may be beneficial even in dogs with severe PS. The type of PS lesion may affect outcome with or without balloon dilation as dogs with type B lesions have a poorer survival. Balloon valvuloplasty results in clinical improvement in approximately 80% of previously clinical cases, but overall survival is worse compared with dogs without clinical signs.

Patch grafting is effective in relieving severe PS but is unforgiving of technical errors during surgery. Operative mortality for this procedure is approximately 15% to 20% in the hands of an experienced surgeon and increases to 30% in small dogs.[22] The most common problem is inability to resuscitate the heart after inflow occlusion. Successful patch-graft valvuloplasty results in substantial pulmonic valve insufficiency, but this has minimal consequence as long as the tricuspid valve is competent and pulmonary hypertension is not present. If facilities are available, cardiopulmonary bypass may lead to a better outcome, particularly if the dog has evidence of right ventricular failure.

AORTIC STENOSIS

DEFINITIONS

AS is a congenital narrowing of the aortic valve, aorta, or left ventricular outflow tract. The stenosis may be supravalvular, valvular, or subvalvular.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

AS is the most common congenital heart defect affecting large-breed dogs and occurs uncommonly in cats. SAS accounts for more than 90% of canine cases and occurs with widely disparate morphology and severity. The typical lesion is a discrete subvalvular fibrous ring that courses across the ventricular septum and reflects onto the anterior mitral valve leaflet. This lesion is often complicated by varying degrees of muscular septal hypertrophy and diffuse fibrosis of the outflow tract. The most severe lesions are associated with an immobile mitral valve leaflet that effectively results in a tunnel-like stenosis. In most cases, SAS is associated with some degree of aortic insufficiency; however, this is usually mild. Concurrent mitral valve insufficiency also occurs.

SAS causes pressure overload on the left ventricle. Varying degrees of left ventricular concentric hypertrophy may develop, depending on severity. Dogs with moderate to severe SAS are at substantial risk for sudden death, presumably as the result of myocardial ischemia and malignant ventricular arrhythmias. Dogs with SAS may also develop CHF, particularly if concurrent mitral insufficiency is present. Lastly, dogs with SAS are at increased risk for bacterial endocarditis of the aortic valve owing to turbulent blood flow and resultant valvular damage.

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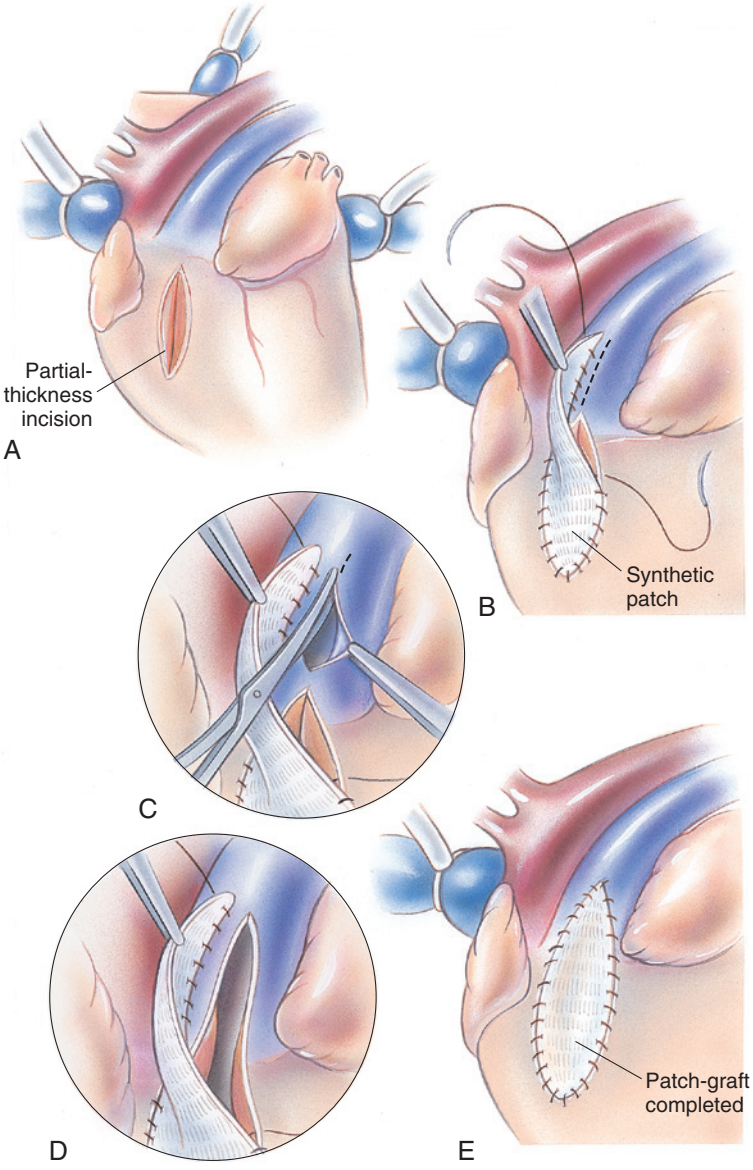


FIG. 27.13 To place a transvalvular patch for pulmonic stenosis, pass tapes for inflow occlusion as described in Fig. 27.3. (A) Partially incise the right ventricular outflow tract just ventral to the pulmonic valve. (B) Suture a synthetic or pericardial patch to the ventriculotomy and the cranial aspect of the pulmonary artery. (C) Initiate inflow occlusion and incise the pulmonary artery, extending the incision across the pulmonary valve. (D) Make the ventriculotomy incision full thickness. (E) Finish suturing the patch graft to the pulmonary artery.

DIAGNOSIS

Clinical Presentation

Signalment

Newfoundlands, boxers, golden retrievers, Rottweilers, German shepherds, Samoyeds, and Dogue de Bordeaux are at increased risk for developing SAS. A genetic basis for SAS has been established in Newfoundlands. Phenotypic expression of SAS may occur a short time after birth. The defect may not be clinically apparent until several weeks of age. SAS should be considered a progressive lesion until maturity.

History

Dogs with SAS may be asymptomatic or may exhibit exercise intolerance, collapse, or syncope. Lack of clinical signs is not an appropriate reason to delay diagnostic evaluation, because the first clinical evidence of SAS may be sudden death.

Physical Examination Findings

The predominant physical finding in animals with SAS is a systolic ejection murmur heard best at the left heart base. The murmur radiates well to the right base and thoracic inlet. In moderate to severe cases, femoral pulses are noticeably weak or hypokinetic, unless substantial concurrent aortic insufficiency is present. ECGs may show a left cranial axis shift or ventricular ectopy, but these findings are usually unremarkable.

Diagnostic Imaging

Thoracic radiographs may reveal a normal cardiac silhouette or mild left ventricular and left atrial enlargement. Enlargement of the ascending aorta is frequently evident.

Echocardiography

Definitive diagnosis of SAS is obtained by echocardiography. M-mode echocardiography demonstrates variable left ventricular free wall and septal thickening, depending on severity. With moderate to severe disease, left ventricular diameter is small unless substantial concurrent aortic or mitral insufficiency is present. Systolic anterior motion of the mitral valve is indicative of a dynamic component to the obstruction and may cause mitral insufficiency. Early closure of the aortic valve suggests that dynamic obstruction may be present. Two-dimensional

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echocardiography provides direct visualization of the various morphologic components of the lesion. Doppler-measured aortic velocities are increased. The peak systolic pressure gradient across the aortic valve can be calculated from the peak aortic velocity ($\Delta P = 4 V^2$). Systolic gradients obtained from the subxiphoid position of 25 to 50 mm Hg are mild, 50 to 75 mm Hg are moderate, and greater than 75 mm Hg are severe when measured in unsedated or unanesthetized animals.

Laboratory Findings

Dogs with SAS may have abnormalities in platelet function and von Willebrand factor multimer distribution. A form of platelet dysfunction detected at high shear rates was associated with MR and SAS in dogs; both are diseases in which turbulent, high-velocity blood flows are seen. The clinical significance of this is unknown.

DIFFERENTIAL DIAGNOSIS

AS must be differentiated from other conditions that cause systolic murmurs (e.g., PS, VSD, TOF). Physiologic (flow/innocent) systolic murmurs are commonly detected in large-breed dogs but are usually of low grade (i.e., I or II/VI).

MEDICAL MANAGEMENT

β-Adrenergic blockade therapy with propranolol or atenolol (see Box 27.1) may reduce the risk of sudden death by decreasing myocardial oxygen requirements and suppressing ventricular arrhythmias during exercise. However, a 2014 study found β-blocker treatment did not influence survival in dogs with severe SAS.[23] Symptomatic treatment (e.g., furosemide, enalapril; see Box 27.1) for CHF is indicated if it occurs. Other drugs for treatment of CHF are provided in Box 27.6. Balloon valvuloplasty may be beneficial in animals with moderate SAS if facilities for cardiac catheterization are available; however, although significant decreases in pressure gradients can be achieved, survival is not significantly different from that of dogs receiving medical management alone. More aggressive intervention using cutting and high-pressure balloons may improve catheter-based outcomes.

SURGICAL TREATMENT

Surgical intervention can be considered for dogs with substantial left ventricular hypertrophy and systolic gradients above 75 mm Hg. If surgery is undertaken, it should be done early to minimize degenerative myocardial changes. Surgical options for dogs with SAS include valve dilation and open resection. Open resection during cardiopulmonary bypass can be considered in dogs with severe SAS; however, long-term benefit of this procedure has not been established (see later under Prognosis). Direct visualization of the defect through an aortotomy, excision of the discrete fibrous ring, and septal myectomy can be performed (Fig. 27.14A). Surgical correction of SAS via right ventriculotomy and septal resection (see Fig. 27.14B) has been described in one dog with long-term benefits; however, too few dogs have undergone the procedure for its true benefit to be known.

Preoperative Management

Arrhythmias should be controlled before surgery with appropriate antiarrhythmic drugs (i.e., atenolol, procainamide, amiodarone, and sotalol) (see p. 789). β-Adrenergic blockade should be continued until the day of surgery. Administer the normal dose or a slightly reduced dose (no greater than 50% reduction) on the morning of the procedure.

BOX 27.6 Medical Management of Congestive Heart Failure

Benazepril (Lotensin)
0.25–0.5 mg/kg PO q12–24h

Enalapril (Vasotec)
0.25–0.5 mg/kg PO q12–24h

Furosemide (Lasix)
2–4 mg/kg PO, IV, SC q6–24h as needed

Pimobendan (Vetmedin)
0.25–0.3 mg/kg PO q12h

Spironolactone (Aldactone)
1–2 mg/kg PO q12h

IV, Intravenous; *PO,* orally; *SC,* subcutaneous.

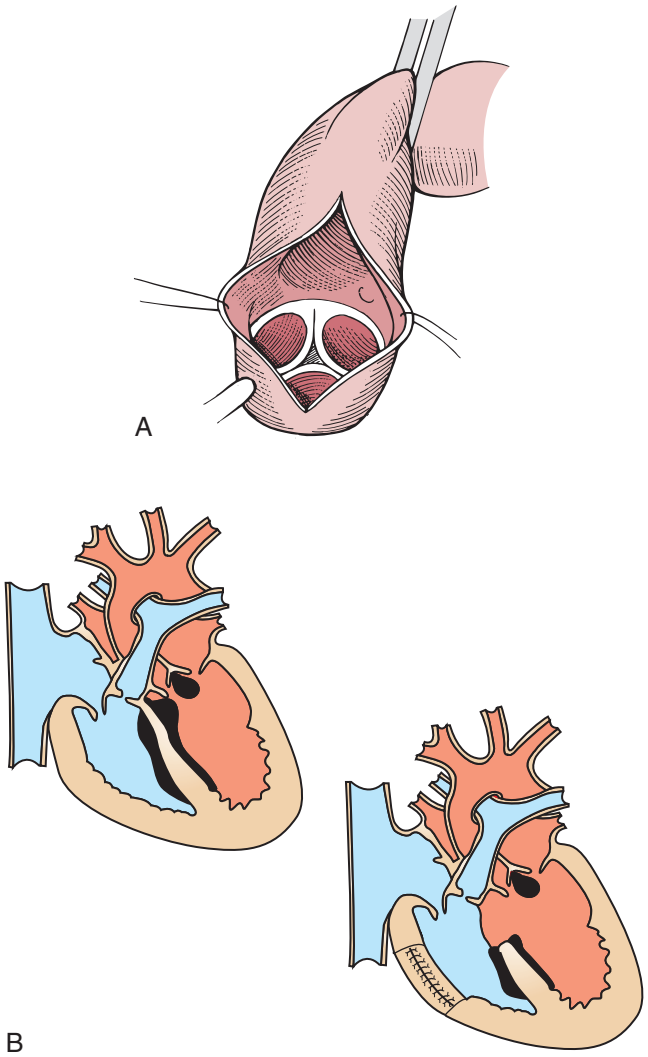


FIG. 27.14 Open resection for subvalvular aortic stenosis may be accomplished (A) through an aortotomy or (B) via a right ventriculotomy during cardiopulmonary bypass.

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Anesthesia

The goal of managing a patient with aortic or subaortic stenosis is to allow the heart sufficient time to eject as much blood as possible through the small valve opening (see Tables 27.1 and 27.3). This means that rate control is extremely important and elevated heart rates should be avoided. Unlike MR, in which elevations in systemic blood pressure can decrease forward blood flow, the ventricular outflow tract is preventing forward flow. Therefore lowering systemic blood pressure does not assist in moving blood out of the heart. In fact, low blood pressures can be catastrophic because the ventricular myocardium is often thickened and end-diastolic pressures are high, meaning that systemic pressures must be kept high enough to perfuse the myocardium during diastole. When the heart rate is elevated and blood pressure drops, the myocardium does not receive sufficient blood flow and oxygen. If this happens, a rapid drop in cardiac output and cardiovascular collapse can occur. Additionally, low systemic arterial pressure may predispose to development or worsening of dynamic left ventricular outflow tract obstruction.

Patients with SAS should be kept calm. To decrease anxiety and excitability, a benzodiazepine (e.g., midazolam, diazepam) may be given. Because these patients are very sensitive to hypotension, it is best not to give opioids as a premed but instead to administer them intraoperatively while blood pressure is being closely monitored. If the benzodiazepine causes some excitement, then small doses of opioid may be given. Avoid acepromazine because of the profound hypotension it can cause. Additionally, avoid alpha-2 agonists because of the potential for second-stage hypotension. Do not premedicate with anticholinergics (e.g., atropine, glycopyrrolate), and note that ketamine is probably best avoided.

The patient should be carefully induced with etomidate. Propofol can cause vasodilation and decreased contractility, a potentially devastating combination, and is not recommended in SAS patients. Monitors should be readily accessible and should be placed before or immediately after induction. Blood pressure and heart rate must be monitored carefully. Induction is a critical time, and these patients can decompensate quickly. Be prepared with phenylephrine, fentanyl, and esmolol for cardiovascular changes. If blood pressure drops, phenylephrine is usually the drug of choice. Other vasopressors may cause varying degrees of increased heart rate; they are usually not needed. If tachycardia occurs, treat with boluses of fentanyl or esmolol (see Box 27.1 and Table 27.1). If appropriate, deepen the anesthetic by increasing the inhalant concentration. Depending on the severity of the stenosis and the type of procedure to be performed, many patients benefit from use of a balanced anesthetic technique that relies on higher doses of opioids and less inhalant agent, usually one-half of the minimum alveolar concentration. Refer to Tables 27.1 and 27.3 for anesthetic recommendations for patients with aortic or subaortic stenosis undergoing cardiac or noncardiac procedures.

Surgical Anatomy

The aortic valve consists of right, left, and noncoronary semilunar cusps. The three aortic sinuses are dilations of the aorta on the vessel side of the valve; the right and left coronary arteries leave the right and left sinuses. The aortic bulb is a widening of the base of the ascending aorta formed by the aortic sinuses.

SAS usually consists of a discrete fibrous ring located 1 to 3 mm below the aortic valve leaflets. This ring generally extends across the septum and reflects onto the anterior mitral valve leaflet. The conduction system (His bundle) courses through the septum at the juncture of the right and noncoronary aortic leaflets.

Positioning

Open resection of AS is performed through a fourth right intercostal thoracotomy.

SURGICAL TECHNIQUE

AP The long-term benefits of surgical correction of SAS are unknown at this time. Cardiopulmonary bypass affords the best opportunity to resect the stenotic area. With additional experience, septal resection and patch grafting may prove beneficial in reducing transaortic pressures over the long term in affected dogs.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Cardiopulmonary bypass is required for open repair of SAS. A glutaraldehyde-fixed pericardial graft may be sutured into the defect caused by resecting the obstructing portion of the septum.

POSTOPERATIVE CARE AND ASSESSMENT

Ventilation should be monitored carefully in the early postoperative period. Poor ventilatory efforts may be associated with residual pneumothorax, hemorrhage, anesthetic agents, or pain. Heart rate and rhythm should be monitored postoperatively for 48 to 72 hours, and hemodynamically significant arrhythmias should be treated. Blood pressure should be measured by direct or indirect means until the animal is fully recovered from anesthesia. Analgesics (local anesthetic techniques and systemic opioids) should be given to decrease postoperative discomfort (see Box 30.2 and Tables 13.2 and 13.3). Urine output should be monitored closely, especially if hypotension occurred during or after surgery.

PROGNOSIS

Dogs with systolic gradients above 75 mm Hg are at substantial risk for sudden death in the first several years of life. Valve dilation can be performed at an early age with low operative mortality and without cardiopulmonary bypass. However, little evidence suggests that valve dilation results in sustained reduction in the systolic pressure gradient. The long-term benefit of this procedure is questionable (see previous discussion under Medical Management). Modest gradient reduction (30%–40%) has been achieved in approximately 33% of dogs that undergo valve dilation by balloon catheter. It is unclear whether valve dilation reduces the risk for sudden death.

Open resection of SAS under cardiopulmonary bypass may result in a substantial reduction of the systolic pressure gradient that is sustained for at least several years after surgery. However, in dogs undergoing open surgical correction of SAS, a positive benefit on survival was not found despite reduction of the systolic pressure gradient. These dogs appear to remain at high risk for sudden death associated with profound reflex vasodilation and bradycardia or fatal ventricular dysrhythmia. For balloon valvuloplasty, no clear benefit is seen in dogs with severe SAS that underwent this procedure, despite a significant decrease in the peak systolic pressure gradient.

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VENTRICULAR SEPTAL DEFECT

DEFINITIONS

VSD is a congenital defect that results from failure or incomplete development of the membranous or muscular interventricular septum.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

VSD is the second most common congenital heart defect in cats and accounts for 5% to 10% of congenital heart defects in dogs. The cause of VSD is not completely understood but is suspected to have a genetic component. VSD has been demonstrated to be a polygenic trait in keeshonden. Most VSDs in small animals occur in the membranous septum. Perimembranous defects are located in the membranous septum, medial to the septal tricuspid leaflet and inferior to the crista supraventricularis. Infundibular or supracristal defects are located in the right outflow tract superior to the crista supraventricularis.

The pathophysiology of VSD depends on the size of the defect and PVR. VSD typically causes a left-to-right shunt. A typical VSD overloads the left ventricle and, depending on its size and location, may overload the right ventricle as well. A large VSD can progress to left-sided CHF. Chronic overcirculation of the lungs can cause progressive pulmonary vascular remodeling, leading to severe pulmonary hypertension and right-to-left shunting of blood (Eisenmenger's physiology). Residence at high altitude likely accelerates the development of pulmonary hypertension.

Aortic insufficiency is a fairly common secondary abnormality associated with VSD, particularly infundibular VSD. Aortic insufficiency results from prolapse of the right coronary aortic leaflet into the defect. This prolapse is due to the Venturi effect associated with VSD flow and loss of support of the aortic annulus. Aortic insufficiency adds to the left ventricular volume overload; it is commonly progressive and may increase the patient's risk of developing endocarditis. Concurrent abnormalities with VSD are common; based on a 2015 retrospective study, solitary defects occur in 48.6% of dogs and cats.[24] The most common concomitant defect is PS, either as an isolated defect or part of TOF complex. Animals with solitary defects are often subclinical with low pulmonary-to-systemic flow ratios.

DIAGNOSIS

Clinical Presentation

Signalment

No breed predisposition has been clearly determined for VSD; however, French bulldogs, cocker spaniels, and terrier breeds may have a higher than expected incidence.

History

Young animals with VSD are often nonclinical at first presentation. Animals with large VSD may have signs of left-sided CHF (i.e., cough and shortness of breath).

Physical Examination Findings

The most prominent physical finding associated with VSD is a systolic murmur with the point of maximal intensity at the right sternum. The murmur is also heard well at the left heart base. When the defect is in the supracristal position, the murmur may actually be loudest at the left cardiac base. The murmur is ejection in quality if the defect is small and regurgitant if the defect is large. A diastolic blowing murmur at the left heart base can give the murmur a continuous or to-and-fro quality and suggests the presence of concurrent aortic insufficiency. Animals with right-to-left VSD may have no murmur owing to polycythemia.

Diagnostic Imaging

Thoracic radiographs reveal varying degrees of left or biventricular enlargement depending on the size and position of the defect. The degree of pulmonary vascular enlargement from overcirculation also depends on the size of the defect, volumetric shunting, and PVR.

Echocardiography

A VSD larger than 5 mm can usually be visualized directly on two-dimensional echocardiography. Color-flow Doppler is particularly useful for detecting small defects. The direction and velocity of shunt flow can be determined by spectral Doppler. A high-velocity left-to-right shunt suggests that the VSD is "restrictive" or hemodynamically insignificant and warrants a good prognosis. Large defects are usually associated with lower shunt velocities and suggest that the animal is at risk for development of progressive heart failure or pulmonary hypertension. The pulmonary-to-systemic flow ratio can be calculated from Doppler analysis of aortic and pulmonary flows. Pulmonary-to-systemic flow ratios (Qp:Qs) greater than 2:1 are indicative of a hemodynamically significant VSD.

Laboratory Findings

Polycythemia may be present in dogs with right-to-left shunts.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include SAS, PS, TOF, ASD, and atrioventricular (AV) septal defects.

MEDICAL MANAGEMENT

Medical management for VSD consists of symptomatic treatment for CHF. Useful drugs for CHF include ACE inhibitors (e.g., enalapril, benazepril, and lisinopril), diuretics (e.g., furosemide or spironolactone; see Box 27.1), and inodilators (e.g., pimobendan). No effective medical management for Eisenmenger's physiology is known. Vasodilator therapy generally causes increased right-to-left shunting owing to preferential dilation of systemic vessels over remodeled pulmonary vessels. Periodic phlebotomy and replacement with crystalloid fluids may be necessary to keep the hematocrit below 60%. Low-dose aspirin (1–2 mg/kg once daily) or clopidogrel (2–3 mg/kg orally once daily) is recommended to prevent thromboembolic complications.

SURGICAL TREATMENT

Surgical intervention can be considered for hemodynamically significant VSD. Concurrent aortic insufficiency is usually progressive and is an indication for surgical intervention. Pulmonary

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artery banding has been used successfully to palliate dogs and cats with VSD. The goal of pulmonary artery banding is to increase right ventricular systolic pressure, thereby decreasing shunt flow.

Definitive patch closure of VSD can be accomplished with the aid of cardiopulmonary bypass in dogs weighing more than 4 kg. A perimembranous VSD is corrected from the right side via a right atriotomy approach. An infundibular VSD is corrected via a right ventriculotomy from a left thoracotomy or median sternotomy approach. Percutaneous occlusion of a VSD using an Amplatzer muscular VSD occluder has been reported in isolated cases.[25]

Preoperative Management

If heart failure is present, attempts should be made to control it medically. See also p. 788 for preoperative management of animals with cardiovascular disease.

Anesthesia

Anesthetic management of animals undergoing cardiac surgery is described on p. 788.

Surgical Anatomy

The interventricular septum is composed of a dorsal, thin, membranous part and a large, ventral, muscular part. The membranous part is formed by fusion of the AV cushions. When the cushions fail to fuse, a VSD arises. The AV node and its bundle are usually closely associated with the caudal margin of a perimembranous VSD.

Positioning

The animal is positioned in right lateral recumbency for pulmonary artery banding. The entire left thorax is prepared for aseptic surgery.

SURGICAL TECHNIQUE

Pulmonary Artery Banding

Perform a left fourth intercostal thoracotomy. Open the pericardium and suture it to the thoracotomy incision. Separate the pulmonary artery from the aorta using a combination of sharp and blunt dissection. Pass a large cotton or Teflon tape around the pulmonary artery just distal to the pulmonic valve (Fig. 27.15). Tighten the tape to reduce the circumference of the pulmonary artery. Place a purse-string suture in the pulmonary artery wall distal to the ligature, and insert a catheter into the pulmonary artery to measure pressures. Constrict the pulmonary artery until the pulmonary artery pressure distal to the band is less than 30 mm Hg. In addition, monitor systemic artery pressures, which should increase during the banding. Optimal banding occurs when the increase in systemic arterial pressures just reaches a plateau.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Wide cotton or Teflon tape is used for pulmonary artery banding. A PTFE vascular graft is used for definitive closure of a VSD.

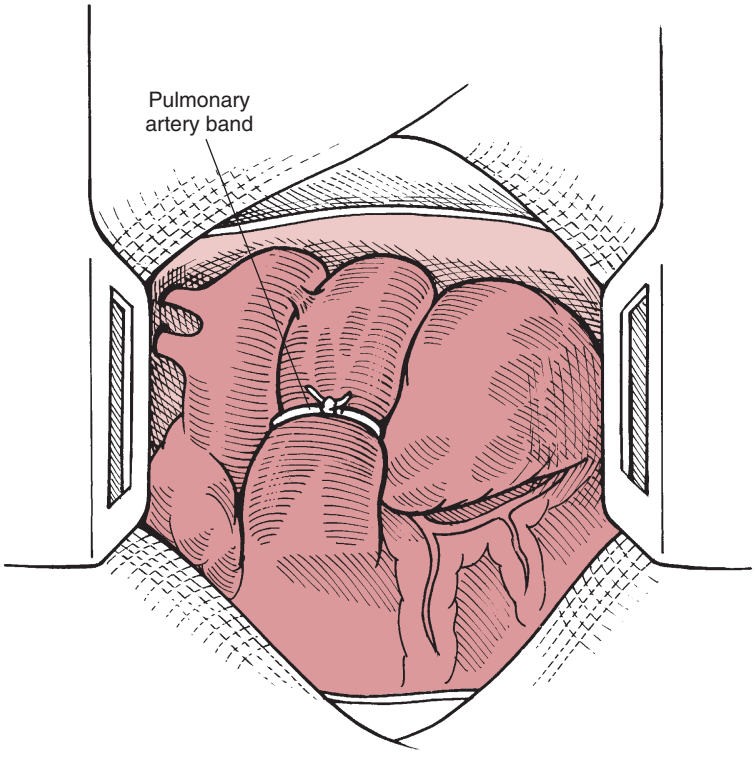


FIG. 27.15 For pulmonary artery banding, pass a tape around the main pulmonary artery. Tighten the tape until pulmonary artery pressure distal to the band is less than 30 mm Hg and systemic artery pressure increases and plateaus.

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POSTOPERATIVE CARE AND ASSESSMENT

Animals should be closely observed for worsening of heart failure secondary to anesthesia, surgery, or arrhythmias. Hypoxemia or cyanosis suggests that the band may have been placed too tightly, potentially reversing the shunt. Postoperative pain should be treated with systemic opioids (see Tables 13.2 and 13.3) and local anesthetic techniques (see Chapter 13 and Box 30.2).

PROGNOSIS

The prognosis for animals with VSD depends on the size of the defect. Animals with small, restrictive, isolated defects may tolerate the defect without ill effects, with a long survival time. Of 37 subclinically affected animals, none developed clinical signs and the median age of death from any cause was 12 years.[24]

Large defects (i.e., Qp:Qs greater than 2:1) will likely result in the development of progressive heart failure or pulmonary hypertension. Pulmonary artery banding is a reasonably effective procedure for palliation of the consequences of a hemodynamically significant VSD in both dogs and cats. Definitive closure of a VSD under cardiopulmonary bypass is considered curative. Aortic insufficiency places an added volume load on the left ventricle and generally indicates a poor prognosis.

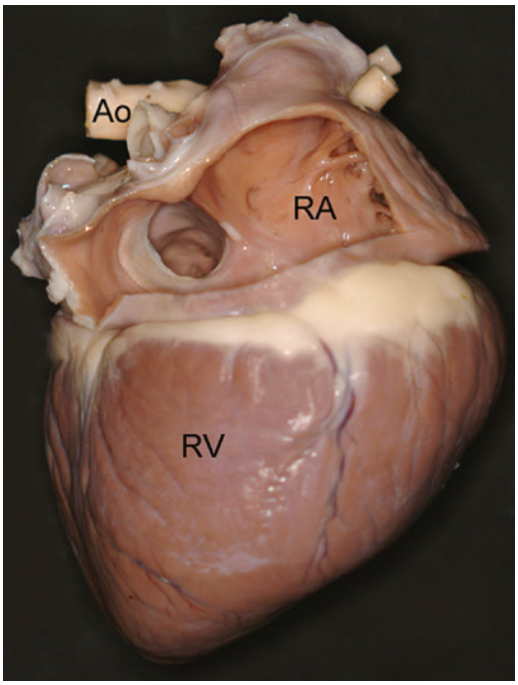


FIG. 27.16 The heart of a dog with a large ostium secundum atrial septal defect. *Ao,* Aorta; *RA,* right atrium; *RV,* right ventricle. (Courtesy Dr. M. Miller, VetMed, Phoenix, AZ.)

ATRIAL SEPTAL DEFECT

DEFINITIONS

ASD is a congenital defect that results from failure or incomplete development of the atrial septum.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

ASD is considered to be a rare congenital defect of dogs; however, it may be more common than previously considered, particularly in some breeds (e.g., standard poodles). The prevalence of feline ASD is unknown, but it may be more common than in dogs, most notably as a portion of an AV canal or endocardial cushion defect. The cause of ASD is not completely understood but is suspected to have a genetic component; therefore affected individuals and their parents should not be used for breeding. Siblings should be carefully screened via echocardiography to determine whether they are affected.

Four types of ASD are described according to the location of the septal defect. The most common type of ASD is an *ostium secundum,* which is dorsally located in the septum (region of the fossa ovalis) (Fig. 27.16). An *ostium primum* ASD is located in the lower part of the interatrial septum and originates from an altered fusion between the septum primum and the endocardial cushion. These defects are often large and may be associated with concurrent abnormalities resulting in a common AV channel between the left and right ventricular chambers. Rare defects include a sinus venosus and a coronary sinus defect. A *sinus venosus* ASD involves the dorsal part of the interatrial septum, close to the junction with the cranial vena cava and pulmonary veins; a coronary sinus defect originates from a disruption in the formation of the wall between the coronary sinus and the left atrium.

The pathophysiology of ASD depends on the size of the defect and cardiac compliance. Because the right atrium typically has a higher compliance than the left atrium, ASDs normally result in a left-to-right shunt. Depending on the size of the defect, the shunt may overload the right side of the heart and eventually, if the defect is large, may cause right-sided heart failure.

DIAGNOSIS

Clinical Presentation

Signalment

No breed predisposition has been clearly determined for ASD; however, standard poodles, boxers, Doberman pinschers, Samoyeds, and Old English sheepdogs may be at increased risk.

History

Young animals with ASD are often asymptomatic at first presentation. Animals with a large ASD may present with exercise intolerance or syncope, or they may have signs of CHF, depending on the size of their defect.

Physical Examination Findings

ASDs may be associated with a soft, midsystolic ejection murmur, which is loudest in the pulmonic area. Splitting of the second heart sound may be audible. Jugular distention and ascites may be noted in dogs with large defects and resultant right ventricular failure.

Diagnostic Imaging

Thoracic radiographs may be normal with small shunts. Right-sided cardiac enlargement and pulmonary overcirculation are common with larger shunts. The degree of pulmonary vascular enlargement from overcirculation depends on the size of the defect and PVR. An ECG may be normal, or it may show right ventricular and possibly right atrial enlargement. Deep S waves in leads I, II, III, and aVF; right axis deviation of the QRS; and

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delayed ventricular conduction are the main findings on ECG in dogs with ASD. Partial or complete right bundle branch is common. Atrial fibrillation may be noted occasionally.

Echocardiography

An ASD larger than 5 mm can usually be visualized directly on two-dimensional echocardiography. Right atrial and ventricular dilation, enlargement of the main pulmonary artery, left atrial enlargement, and reduced or normal left ventricular and aortic size are suggestive of ASD on two-dimensional and M-mode echocardiography. Color-flow Doppler is particularly useful for detecting small defects. The direction and velocity of shunt flow can be determined by spectral Doppler. The pulmonary-to-systemic flow ratio can be calculated from Doppler analysis of aortic and pulmonary flows. Pulmonary-to-systemic flow ratios (Qp:Qs) greater than 2:1 are indicative of a hemodynamically significant ASD; ratios greater than 2.5:1 may indicate the need for surgery or catheter occlusion.

NOTE The complex color-flow Doppler filling patterns of the left and right atria, coupled with false echo dropout of the interatrial septum in the area of the fossa ovalis, commonly result in an erroneous diagnosis of atrial septal defect by inexperienced echocardiographers.

Laboratory Findings

Laboratory abnormalities are not typically present.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include SAS, PS, TOF, and AV septal defects.

MEDICAL MANAGEMENT

Medical management for ASD consists of symptomatic treatment for CHF. Useful drugs for CHF include ACE inhibitors (e.g., enalapril), diuretics (e.g., furosemide, spironolactone; see Box 27.1), and inodilators (e.g., pimobendan).

SURGICAL TREATMENT

Surgical intervention should be considered for hemodynamically significant ASDs. Depending on the conformation of the defect (it must have a rim of tissue around at least 75% of the defect edge for the occluder to attach), a septal occluder device (e.g., Amplatzer Septal Occluder) may be used to close the defect (Figs. 27.17 and 27.18). If the defect does not have an appropriate rim of tissue for a septal occluder, definitive patch closure can be accomplished with the aid of cardiopulmonary bypass (Fig. 27.19). Surgical correction of an ASD is typically done from a right-sided thoracotomy via a right atriotomy incision.

Preoperative Management

If significant heart failure is present, attempts should be made to control it medically. See also p. 788 for preoperative management of animals with cardiovascular disease.

Anesthesia

Anesthetic management of animals undergoing cardiac surgery is described on p. 788.

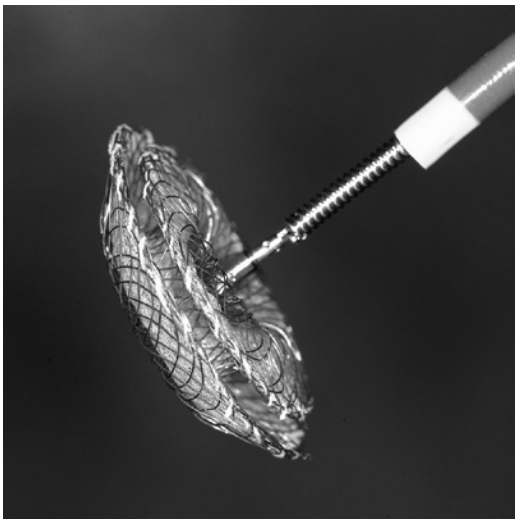


FIG. 27.17 Amplatzer device for transcatheter closure of an atrial septal defect. (Courtesy Dr. M. Miller, VetMed, Phoenix, AZ.)

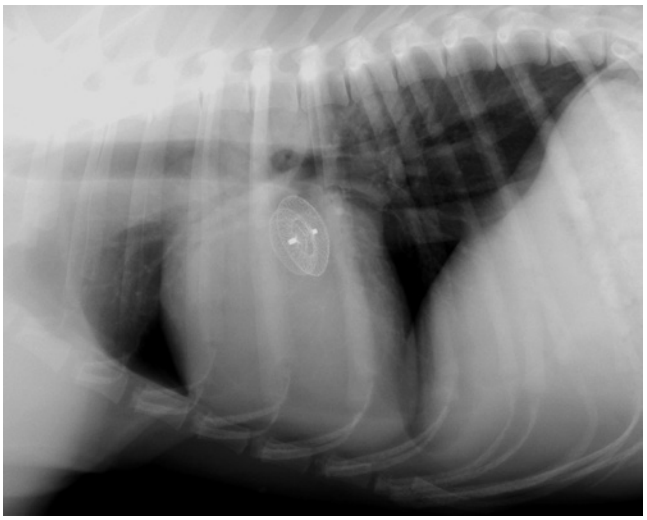


FIG. 27.18 Radiograph of a dog in which the device in Fig. 27.17 was placed to close a hemodynamically significant atrial septal defect. (Courtesy Dr. M. Miller, VetMed, Phoenix, AZ.)

Surgical Anatomy

During cardiac development, the atria and ventricles are joined as a common chamber. The foramen ovale is a slitlike passage in the atrial septum that persists between the atria to permit right-to-left atrial shunting in the fetus. The foramen ovale closes in the neonate once left atrial pressure increases. Eventually, the lower portion of the atrial septum connects to the upper ventricular septum by differentiation of the endocardial cushion. The AV node lies adjacent to the anterior edge of the defect and must be avoided when sutures are placed to close the defect.

Positioning

The animal is positioned in left lateral recumbency for definitive closure of the defect via a right atriotomy. The entire right-sided thorax is prepared for aseptic surgery.

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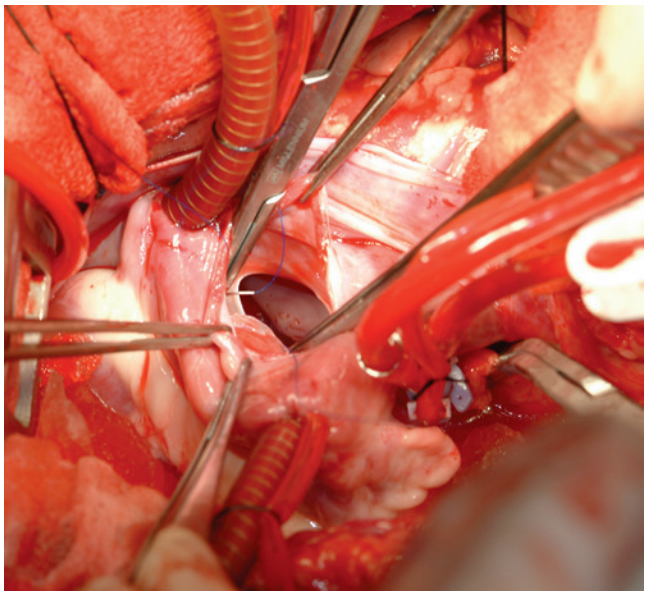


FIG. 27.19 Sutures being placed in a pericardial graft to close a large atrial septal defect.

SURGICAL TECHNIQUE

Definitive patch closure of ASD can be accomplished with the aid of cardiopulmonary bypass in dogs weighing more than 4 kg. A secundum ASD is corrected from the right side via a right atriotomy approach. Percutaneous transcatheter closure of ASD using an Amplatzer Atrial Septal Occluder was successful in 10 of 13 dogs in which it was attempted.[26]

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

A glutaraldehyde-fixed pericardial graft may be used to close the defect if it is too large for a direct suture technique. An Amplatzer Septal Occluder may be used for transcatheter closure of the defect.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative care of these patients differs depending on the technique used for closure. After transcatheter closure, animals are generally released from the hospital within 24 hours of the procedure. Open surgical repair of the defect requires a longer postoperative recovery period. Animals should be closely observed for worsening of heart failure secondary to anesthesia, surgery, or arrhythmias. Postoperative pain should be treated with systemic opioids (see Tables 13.2 and 13.3) and local anesthetic techniques (see Chapter 13 and Box 30.2).

PROGNOSIS

The prognosis for animals with ASD depends on the size of the defect. Animals with small restrictive defects typically tolerate the defect without ill effects. Large defects (i.e., Qp:Qs >1.5–2:1) will likely result in the development of progressive heart failure or pulmonary hypertension. Definitive closure of an ASD under cardiopulmonary bypass or via transcatheter occlusion is considered curative.

TETRALOGY OF FALLOT

DEFINITION

TOF is a complex congenital heart defect that consists of PS, VSD, dextropositioned overriding aorta, and right ventricular hypertrophy.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

TOF is the most common congenital heart defect that causes cyanosis in small animals. It occurs in cats and a variety of canine breeds. TOF can be simplified into two physiologically significant defects: PS and VSD. The pathophysiologic consequences of tetralogy depend on the relative magnitude of these two defects. If a large VSD and hemodynamically insignificant PS are present, the functional result is a left-to-right shunt and volume overload of the left ventricle similar to an isolated, large VSD. If severe PS, suprasystemic right ventricular pressures, and a right-to-left shunt are present, the result is moderate to severe cyanosis, exercise intolerance, and progressive polycythemia. A shortened life span is expected in these animals because of complications of hyperviscosity-induced thromboembolism or sudden death (caused by polycythemia). Animals that have PS and VSD that are somewhat balanced are functionally similar to those that have a VSD and a pulmonary artery banding performed (see p. 812). Animals with a predominantly left-to-right shunt are termed *acyanotic* or *pink tetralogy* and may function reasonably well as long as the shunt flow is insufficient to cause left ventricular failure. Progression of PS due to infundibular hypertrophy is possible and may cause acyanotic animals to become cyanotic as they age.

DIAGNOSIS

Clinical Presentation

Signalment

Breeds most commonly reported to have TOF include keeshonden, English bulldogs, poodles, schnauzers, terriers, collies, and Shetland sheepdogs. In keeshonden, TOF is genetically transmitted as part of the spectrum of conotruncal defects.

History

Clinical findings at presentation for a typical TOF include moderate to severe exercise intolerance, exertional tachypnea, collapse, and syncope.

Physical Examination Findings

Physical findings in animals with TOF include cyanosis unresponsive to supplemental oxygen and systolic murmurs heard well at the left ventricular base and right sternum. However, some animals may have no murmur.

Diagnostic Imaging

Thoracic radiographs typically show evidence of right ventricular enlargement, usually without main pulmonary artery enlargement. Pulmonary vessels are usually small, suggesting pulmonary undercirculation. ECGs usually show a right axis shift in the frontal plane suggestive of right ventricular hypertrophy.

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Echocardiography

Two-dimensional echocardiography demonstrates all the elements of TOF, including right ventricular hypertrophy, PS, VSD, and overriding aorta. Doppler interrogation of the pulmonic outflow tract and septal defect is useful in determining the direction and magnitude of the shunt.

Laboratory Findings

Polycythemia (i.e., packed cell volume >55%) is often present because of chronic hypoxemia.

DIFFERENTIAL DIAGNOSIS

Differentials include right-to-left shunting VSD, ASD, AV septal defect, complex cyanotic cardiac disease, and PDA.

MEDICAL MANAGEMENT

Periodic phlebotomy with crystalloid fluid replacement may be necessary to maintain the hematocrit below 60% in animals with severe cyanosis and progressive polycythemia. Caution must be taken during this procedure to avoid introducing IV air and causing air embolism. Low-dose aspirin (1–2 mg/kg once daily) or clopidogrel (2–3 mg/kg PO once daily) is also recommended to reduce the risk of thromboembolic complications.

β-Adrenergic blockade therapy with propranolol or atenolol has been advocated as a palliative treatment for TOF (see Box 27.1). Possible beneficial effects include reduced dynamic outflow obstruction, decreased heart rate, increased SVR, and decreased myocardial oxygen demand.

SURGICAL TREATMENT

Surgery should be considered for severely cyanotic animals to lessen clinical signs and prolong life. Animals with resting arterial oxygen saturation less than 70% should be considered candidates for surgery. Palliative surgeries for TOF include isolated correction of the PS or creation of a systemic-to-pulmonary shunt (e.g., Blalock-Taussig shunt; see later discussion). Correction of the PS risks overcorrection of the stenosis and an overwhelming left-to-right shunt. For this reason, valve dilation (see p. 806), achieved surgically or by balloon dilation, is preferred over a more definitive procedure, such as a patch graft (see p. 807). Definitive repair of TOF can be undertaken in medium- to large-breed dogs with cardiopulmonary bypass. Patch closure of the VSD and patch grafting of the pulmonary outflow tract are undertaken through a right ventriculotomy approach.

Preoperative Management

Although arrhythmias are uncommon, an ECG should be performed and hemodynamically significant arrhythmias controlled before surgery. Severe polycythemia should be corrected before surgery.

Anesthesia

Recommendations for anesthetic management of animals undergoing cardiac surgery are discussed on p. 788.

Surgical Anatomy

With TOF, the parietal portion of the infundibular septum attaches more cranial and leftward than normal, resulting in narrowing of the right ventricular outflow tract and dextropositioned overriding of the aorta. The magnitude of this shift determines which type of physiology is associated with the defect. The VSD is usually located high in the infundibular septum, just at the level of the crista supraventricularis, although supracristal septal defects do occur.

Positioning

The animal should be positioned in right lateral recumbency for Blalock-Taussig shunt surgery. The entire left hemithorax is prepared for aseptic surgery.

SURGICAL TECHNIQUE

AP Several types of systemic-to-pulmonary shunts have been used to palliate TOF. A modified Blalock-Taussig shunt is accomplished by harvesting the left subclavian artery as a free autogenous graft and placing it between the aorta and the main pulmonary artery (Fig. 27.20). Alternatively, a segment of carotid artery or a tube of expanded PTFE is sutured end-to-side between the left subclavian artery and the main pulmonary artery.

Perform a left fourth intercostal thoracotomy. Harvest an autogenous arterial graft by ligating and dividing the proximal left subclavian artery. Open the pericardium and suture it to the thoracotomy incision. Place tangential vascular clamps on the pulmonary artery and ascending aorta. Make incisions into both vessels by making a longitudinal incision in the vessel wall held within the clamp. Interpose the graft between the aorta and the pulmonary artery by end-to-side anastomoses, using simple continuous suture patterns. Be sure that the graft is not kinked. Release the clamps and verify hemostasis at the suture sites. The pulmonary artery clamp should be released first.

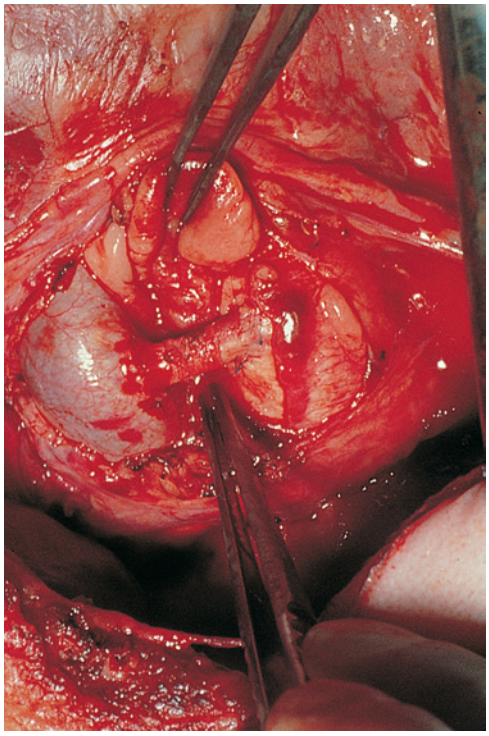


FIG. 27.20 Modified Blalock-Taussig shunt for tetralogy of Fallot.

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SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Polypropylene (e.g., 5-0 Prolene or Surgipro) suture is used for vascular anastomoses of the Blalock-Taussig shunt. Two tangential vascular clamps are required to control hemorrhage during the surgery.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative pain should be treated with systemic opioids (see Table 13.2) and local anesthetic techniques (see Chapter 13 and Box 30.2). Postoperative care of patients undergoing cardiac surgery is described on p. 797.

PROGNOSIS

Animals with reasonably balanced acyanotic TOF should be monitored for progression but otherwise generally do not require surgery. These animals have a reasonably good prognosis. In a 2016 retrospective study of dogs and cats with TOF, median age at cardiac-related death was 23.4 months, with no significant difference between dogs and cats.[27] Median survival time from time of diagnosis to cardiac-related death was briefer for animals with no or low-grade heart murmur (3.4 months) than for those with higher-grade heart murmur (16.4 months).

The prognosis for animals with cyanotic TOF depends on the shunt fraction, magnitude of hypoxemia, and degree of polycythemia. Some animals may live several years without surgical therapy despite moderate to severe exercise intolerance. Animals with severe hypoxemia and progressive polycythemia will likely succumb to the effects of the disease or will experience sudden death early in life. Modified Blalock-Taussig shunts are reasonably effective at reducing the magnitude of hypoxemia and palliating the consequences of TOF.

PERICARDIAL EFFUSION AND PERICARDIAL CONSTRICTION

DEFINITIONS

The **pericardium** is a fibroserous envelope that encompasses the heart and great vessels. **Pericardial effusion** is an abnormal accumulation of fluid within the pericardial sac. **Cardiac tamponade** refers to the decompensated phase of cardiac compression that results from an unchecked rise in intrapericardiac fluid pressure. **Pericardial constriction** results from restrictive fibrosis of the parietal and/or visceral pericardium that interferes with diastolic function of the heart. **Electrical alternans** is a beat-to-beat voltage variation of the QRS or ST-T complex. *Pericardial tamponade* is a synonym for cardiac tamponade; pericardial constriction is a synonym for *constrictive pericarditis*.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Diseases that affect primarily the pericardium account for approximately 1% of cardiovascular disease. Although primary pericardial disease represents a small percentage of the total number of cardiac diseases in small animals, it is an important cause of canine right-sided CHF. Pericardial diseases are uncommon in cats and are most often related to CHF. Several types of primary and secondary pericardial diseases may occur, the most common of which are those resulting in the accumulation of pericardial effusion. Pericardial effusion can be transudative, exudative (inflammatory), or sanguineous. Causes of pericardial transudation include right-sided CHF, severe hypoproteinemia, and incarceration of a liver lobe within the pericardial cavity (peritoneopericardial diaphragmatic hernia; see p. 931). Transudative pericardial effusions are usually subclinical.

Infectious pericarditis is an uncommon cause of pericardial effusion in dogs and cats, usually producing a purulent or fibrinous exudate. A variety of aerobic and anaerobic bacterial and fungal organisms have been associated with infectious pericarditis. Bacterial pericarditis can arise from bite wounds to the thorax, migrating foreign bodies, or hematogenous seeding. Coccidioidomycosis is an important cause of pericardial effusion in endemic regions. Feline infectious peritonitis (FIP) and toxoplasmosis are potential causes of feline inflammatory effusions.

The most common causes of pericardial effusion in dogs are neoplastic and benign idiopathic effusions. Neoplasms that produce pericardial effusion in dogs include hemangiosarcoma, chemodectoma, mesothelioma, ectopic (heart base) thyroid carcinoma, lymphoma, and metastatic carcinoma to the heart. Hemangiosarcomas may be multicentric, with simultaneous splenic or hepatic involvement (see also p. 645). Aortic body tumors (chemodectoma, nonchromaffin paraganglioma) sometimes invade the heart base and cause pericardial effusion. Lymphosarcoma, mesothelioma, and metastatic carcinoma have been implicated in cats with pericardial effusion. Neoplastic pericardial effusions are usually sanguineous.

Idiopathic (benign) pericardial effusions are common in dogs but have not been reported in cats. The effusion usually appears sanguineous and must be differentiated from neoplastic effusions. Coagulopathies and left atrial rupture secondary to chronic mitral insufficiency rarely cause acute pericardial hemorrhage.

Pathophysiologic alterations associated with pericardial effusion depend on the rate and volume of fluid accumulation and the distensibility or compliance of the pericardium. If effusion accumulates slowly, the pericardium will expand to accommodate the fluid, intrapericardial pressure will not increase initially, and cardiac filling will not be compromised. Slow progressive accumulation of pericardial fluid allows compensation. First, the fibrous pericardium can stretch and remodel over time to accommodate an increased volume of fluid with only a modest increase in intrapericardial pressure. Second, neurohumoral mechanisms are evoked, leading to retention of vascular volume and increased intracardiac diastolic filling pressures. Although the latter compensatory mechanism delays the onset of cardiac tamponade, it leads to progressive right-sided CHF, including jugular venous distention, ascites, peripheral edema, and/or pleural effusion. Cardiac tamponade eventually occurs despite compensatory mechanisms, leading to circulatory collapse.

Conversely, rapid or sudden accumulation of fluid (e.g., pericardial hemorrhage) causes acute cardiac tamponade. Rapid fluid accumulation in the pericardial space compresses the ventricles, restricts ventricular filling, and reduces cardiac output. Although significant volumes of fluid are usually required to raise pericardial pressure initially, additional small volumes may substantially increase intrapericardial pressure and greatly impede ventricular filling. The thin walls and low pressures of the right atrium and ventricle make these chambers particularly vulnerable to cardiac compression. Diastolic pressures (i.e., pulmonary wedge pressure, left and right ventricular end-diastolic pressures, and mean right atrial pressure) on both sides of the heart equilibrate.

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Pericardial constriction occurs when visceral and/or parietal pericardial layers become fused, thickened, densely fibrotic, or inelastic and form a rigid case around the heart. The pericardial space may become totally obliterated or may contain small amounts of fluid (constrictive-effusive disease). Constrictive pericardial disease may develop asymmetrically so that one ventricle is more affected than the other; however, the two ventricles are usually affected nearly equally. An audible "pericardial knock" may be heard during early diastolic filling and is attributed to vibrations produced by the sudden deceleration of blood as it strikes the encased nondistensible ventricular wall. As constriction worsens, cardiac output declines even though myocardial systolic function may be maintained. Fluid retention initiated by chronically reduced cardiac output further contributes to venous congestion, which is most commonly manifested as hepatomegaly and ascites accumulation.

DIAGNOSIS

Clinical Presentation

Signalment

Idiopathic benign and neoplastic pericardial effusions are more commonly observed in large- and giant-breed dogs. Right atrial hemangiosarcoma is especially common in German shepherd dogs and golden retrievers (see also p. 823). Idiopathic pericardial effusion has been reported most commonly in golden retrievers, German shepherd dogs, and other large-breed dogs. Aortic body tumors are most common in aged brachycephalic dogs. Medium- to large-breed, middle-aged dogs are most commonly affected with constrictive pericardial disease; however, the condition is uncommon.

History

Presenting complaints associated with pericardial effusion include weakness, lethargy, exercise intolerance, and/or collapse. Patients often have right-sided congestion, ascites, and/or pleural effusion. The most common owner complaint with constrictive pericarditis is abdominal enlargement. Dyspnea, tachypnea, weakness, syncope, and/or weight loss are noted less frequently. Occasionally, a previous history of idiopathic pericardial effusion is reported.

Physical Examination Findings

Clinical findings are related to the consequences of cardiac tamponade and right-sided CHF. The classic triad of signs of cardiac tamponade (e.g., rapid and weak arterial pulse, distended jugular veins, and diminished heart sounds) is usually present. Jugular venous distention or a positive hepatojugular reflux is commonly overlooked. Measurement of central venous pressure will document systemic venous hypertension and frequently exceeds 10 cm H_2O (normal, <5–6 cm H_2O). Lung sounds (especially ventrally) may be diminished if pleural effusion is present. Other auscultatory abnormalities (e.g., gallop rhythms, cardiac murmurs, arrhythmias) are uncommon. Ascites, hepatomegaly, and/or peripheral edema may also be noted.

Although no pathognomonic electrocardiographic findings are observed for pericardial disease, several abnormalities may occur. Electrical alternans may be recorded in as many as 50% of patients with pericardial effusion (Fig. 27.21). If present, electrical alternans is strongly suggestive of pericardial effusion. It is caused by swinging of the heart within large pericardial effusions rather than alterations in conduction within the heart and is most likely to occur at heart rates between 90 and 144 beats/min. Other findings supportive of pericardial effusion on ECGs are diminished QRS voltages and ST segment elevation. Sinus tachycardia is the predominant rhythm, although nonsustained ventricular tachycardia may occur.

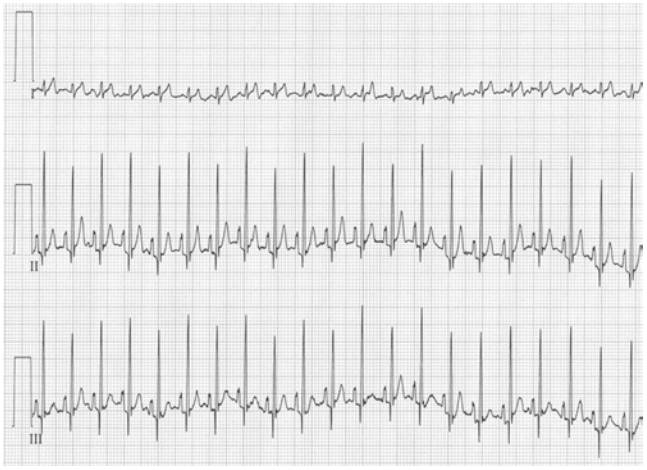


FIG. 27.21 Electrocardiogram of a dog with electrical alternans with pericardial effusion. Notice the overall small complex size and the variable R-wave amplitude. (Courtesy Dr. M. Miller, VetMed, Phoenix, AZ.)

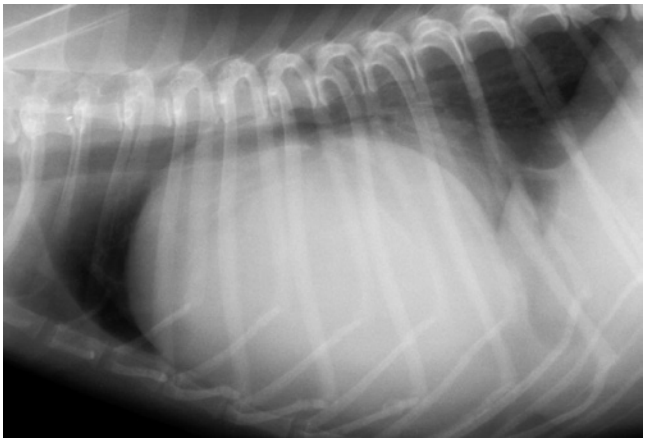


FIG. 27.22 Thoracic radiograph of a dog with pericardial effusion. Note the large, globoid heart.

Diagnostic Imaging

Thoracic radiographic findings in dogs with pericardial effusion are not sensitive or specific for diagnosis of cardiac tamponade.[28] Thoracic radiography usually demonstrates varying degrees of globoid enlargement (i.e., the cardiac silhouette loses its angles and waists and becomes globe shaped) of the cardiac silhouette (Fig. 27.22). Radiographic evidence of pulmonary congestion or edema, which helps distinguish pericardial effusion from dilated cardiomyopathy, is not expected. Pulmonary undercirculation is often noted. If right-sided congestion has developed, distention of the caudal vena cava, hepatomegaly, ascites, and pleural effusion are usually evident. Heart base tumors may deviate the trachea, typically cranial to the carina, producing a mass effect. Abnormal radiographic findings in animals with constrictive pericarditis are subtle; the cardiac silhouette may be rounded. Caudal vena cava dilation may be evident.

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Fluoroscopy may demonstrate reduced cardiac motion in animals with pericardial effusion. Pneumopericardiography may help identify intrapericardial mass lesions, although this technique is no longer used with the advent of echocardiography. Angiography will usually show filling defects or tumor vascularity if neoplasia is causing the effusion; furthermore, angiography will show increased endocardial-pericardial distance typical of pericardial effusion. Although these modalities are reliable when properly used, echocardiography has supplanted most indications for their use.

The diaphragmatico-hepatic view of the abdominal- and thoracic-focused assessment with sonography for triage is clinically helpful in the diagnosis of pericardial effusion.[29] Ultrasound-guided transthoracic fine needle aspiration of cardiac masses has been described in six dogs with definitive diagnosis and only minor complications.[30]

Echocardiography

Definitive diagnosis of pericardial effusion is obtained readily by echocardiography. The fibrous pericardium is easily identified as a thin, echo-dense structure, and any degree of separation or echo-free space between the pericardium and underlying cardiac structures on two-dimensional or M-mode echocardiography is diagnostic of pericardial effusion. Echocardiography is the most reliable procedure for identifying primary cardiac neoplasia; sensitivity and specificity for detecting a right atrial or heart base mass were found to be 82% and 100%, respectively.[31] Specific diagnosis of cardiac tumor type based on echocardiographic anatomic location is only moderately accurate.[32] Flattening of the left ventricular endocardium during diastole and abnormal diastolic (early notch) and systolic septal motion are often noted in patients with constrictive pericarditis. Differentiation of constrictive pericardial disease and restrictive myopathy may be difficult with echocardiography.

Laboratory Findings

The complete blood count may reveal inflammation, and increased numbers of circulating nucleated red blood cells or large numbers of acanthocytes are suggestive of cardiac or splenic hemangiosarcoma. Anemia and thrombocytopenia are also common findings and appear to be more common in dogs with pericardial effusion due to hemangiosarcoma. Mild increases in the activities of liver enzymes are the most common biochemical abnormality.

Cardiac enzymes may be elevated because of ischemia or myocardial invasion. Plasma cardiac troponin I was found to be higher in dogs with cardiac hemangiosarcoma relative to dogs with hemangiosarcoma at other sites, dogs with other tumors, and dogs with pericardial effusion from other causes. Other abnormalities may be associated with the primary disease or with CHF. Serum fungal titers (coccidioidomycosis) or enzyme-linked immunosorbent assay tests for feline leukemia virus (cats) may be positive when pericarditis is related to these infections. Chronic systemic venous congestion associated with pericardial effusion or constriction can cause splenic dysfunction (functional hyposplenism) and protein-losing enteropathy (intestinal lymphangiectasia). Hyposplenism can cause increased numbers of circulating activated platelets, whereas protein-losing enteropathy may exacerbate the splenic dysfunction and reduce circulating antithrombin III levels. Both conditions promote a hypercoagulable state and may make affected animals prone to pulmonary thromboembolism (PTE). Aspirin (1–2 mg/kg once daily), clopidogrel (2–3 mg/kg orally once daily), and low-molecular-weight heparin (dalteparin; 150 U/kg subcutaneously every 8–12 hours) have been used to prevent PTE in dogs at risk, but their value is uncertain (see also Box 29.1).

NOTE Pulmonary thromboembolism should be considered likely in dogs with pericardial disease with effusion, particularly those with constriction, that exhibit severe, sudden respiratory distress.

Cytologic and microbiologic analysis should be performed on pericardial fluid. An inflammatory exudate on cytologic examination suggests infectious pericarditis. The causative organism may be visible on cytologic examination or may be identified by bacterial or fungal culture. FIP can be strongly suspected based on serology or reverse transcription-polymerase chain reaction performed on the pericardial fluid (not the blood).

NOTE Warn the laboratory if you suspect coccidioidomycosis because it is hazardous to attempt culture of this organism.

Neoplastic effusions are usually sanguineous (i.e., characterized by large numbers of red blood cells and variable numbers of neutrophils and mononuclear cells). Cytology is unreliable in identifying neoplastic effusions because both false-positives and false-negatives occur; mesothelial cell reactivity is seen in dogs with idiopathic pericarditis and in those with mesothelioma. Idiopathic pericardial effusion produces a sanguineous effusion that is difficult to distinguish from neoplastic effusions on fluid analysis alone. Fluid analysis identified a specific cause in only 20 of 259 (7.7%) dogs with pericardial effusion; however, the diagnostic utility of cytology increased to 20.3% if the fluid hematocrit was <10%.[33] The use of fluid pH to differentiate between neoplastic and idiopathic pericardial effusion has been suggested; however, it has been found to be an unreliable indicator in most studies. Exploratory surgery or thoracoscopy and histopathology may be necessary to definitively diagnose neoplastic or idiopathic pericardial effusion.

DIFFERENTIAL DIAGNOSIS

In addition to pericardial effusion, differentials for a globoid-appearing heart on thoracic radiographs include dilated cardiomyopathy and peritoneopericardial diaphragmatic hernia (see p. 931). The latter may be associated with pericardial effusion, particularly when the liver is herniated. Ultrasonography is used to detect incongruities in the diaphragmatic silhouette and to identify abdominal contents within the pericardial sac. Echocardiography differentiates diffuse cardiac enlargement from pericardial effusion. If echocardiography is unavailable, nonselective angiography can be used.

MEDICAL MANAGEMENT

Pericardiocentesis is the treatment of choice for initial stabilization of dogs and cats with pericardial effusion and cardiac tamponade. When performed properly, pericardiocentesis is associated with minimal complications. It should be attempted in symptomatic animals with suspected pericardial effusion, even if echocardiography is not available to confirm the diagnosis.

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NOTE Monitor an electrocardiogram during pericardiocentesis because inadvertent cardiac contact usually produces premature ventricular complexes.

BOX 27.7 Sedation During Pericardiocentesis in Dogs

Hydromorphone
0.05–0.2 mg/kg IV PRN

Oxymorphone
0.1–0.2 mg/kg IV PRN

Fentanyl (Sublimaze)
0.005–0.01 mg/kg IV PRN

Midazolam[a]
0.2 mg/kg IV
Avoid acepromazine, propofol, and alpha-2 agonists.

[a]Consider adding to opioid if patient is still anxious.
IV, Intravenous; *PRN,* as needed.

Shave and surgically prepare a large area of the right hemithorax (sternum to midthorax, third to eighth rib). Perform a local block with lidocaine and, if necessary, sedate the animal (e.g., hydromorphone, fentanyl; Box 27.7). Infiltrate the pleura with lidocaine as pleural penetration causes significant discomfort. Place the animal in sternal (preferably) or lateral recumbency, depending on its demeanor. Pericardiocentesis can be accomplished in the standing animal, but adequate restraint is essential to prevent cardiac puncture or pulmonary laceration. *Determine the puncture site based on heart location on thoracic radiographs. This is most commonly between the fourth and fifth intercostal spaces at the costochondral junction. Attach a 14- to 18-gauge needle or over-the-needle catheter to a three-way stopcock, extension tubing, and syringe to allow constant negative pressure to be applied during insertion and drainage (Video 27.2). Once the catheter has been inserted through the skin, apply negative pressure.* If pleural effusion is present, it will be noted immediately on entering the thoracic cavity. Pleural effusion associated with heart disease is usually a clear to pale yellow color. *Advance the catheter until it contacts the pericardium and a scratching sensation is noticed. Then advance the catheter slightly to penetrate the pericardium. Stop advancing the catheter as soon as fluid is obtained. Withdraw the needle immediately if the epicardium is contacted and if cardiac motion is felt through the needle.*

Ultrasound guidance is seldom necessary when pericardiocentesis is performed unless the volume of fluid is small or it is compartmentalized; however, given the widespread availability of this modality, ultrasound-guidance of pericardial sac aspiration is advocated. Pericardiocentesis causes immediate clinical improvement in animals with cardiac tamponade. The pulse slows and strengthens as soon as an adequate volume of fluid has been removed. Pericardial effusion can usually can be differentiated from peripheral blood in that it rarely clots and the packed cell volume is significantly lower than that of peripheral blood. Approximately 50% of dogs with idiopathic effusion are managed successfully by one or more pericardiocenteses; other cases may require pericardiectomy. Fluid may reaccumulate rapidly (within several days) or may not recur for months or even several years. In patients requiring more than two centeses within a few months, subphrenic pericardiectomy is usually indicated. Although antiinflammatory doses of prednisolone and diuretics are commonly administered to dogs with idiopathic pericardial effusion, no controlled studies have confirmed the efficacy of these therapies. Diuretic therapy may put the animal at additional risk by decreasing preload, if cardiac tamponade should recur. Subtotal pericardiectomy is usually curative in dogs with idiopathic pericardial effusion. Recurrent effusion and pericardial constriction are possible late sequelae of idiopathic effusions if pericardiectomy is not performed.

Recent investigation into the use of Yunnan Baiyao (Chinese herbal remedy used to reduce bleeding) and epsilon aminocaproic acid (antifibrinolytic) in dogs with right atrial masses and pericardial effusion found no significant delay in recurrence of clinical signs or improvement in survival.[34]

SURGICAL TREATMENT

Although temporary relief of cardiac tamponade is provided by pericardiocentesis, long-term palliation of pericardial effusion often requires pericardiectomy. Pericardiectomy can be performed through an intercostal thoracotomy (see p. 890) or a median sternotomy (see p. 891), or it can be done with thoracoscopy. Concerns that removing only a small portion of the pericardium might allow the remaining pericardium to adhere to the heart and cause recurrence of pericardial effusion have not been borne out with thoracoscopic pericardiectomy. In a 2013 study, dogs with idiopathic pericardial effusion treated with a subtotal pericardectomy via thoracotomy had a significantly longer disease-free interval and median survival time compared with dogs treated by the thoracoscopic pericardial window procedure.[35] However, a more recent smaller study found that thoracoscopic creation of a pericardial window in dogs with idiopathic pericardial effusion provided good long-term control with a median survival time of 635 days.[36]

For open thoracotomy, it is technically easier to perform a pericardiectomy through a median sternotomy because access to both sides of the heart and both phrenic nerves is provided by this approach. If right atrial hemangiosarcoma is suspected, a right fifth intercostal thoracotomy or a median sternotomy should be used. Removal of right atrial tumors can be accomplished equally well with either approach (see p. 825) and has also been accomplished by thoracoscopy (Video 27.3).[37] Chemodectomas can arise on the left or the right heart base. Pericardiectomy in these cases should be performed through a thoracotomy on the side where the bulk of the tumor is suspected to be. If cardiac neoplasia is not identified before surgery, and if idiopathic pericardial effusion is suspected, then pericardiectomy should be performed through a right thoracotomy or a medial sternotomy so that the right atrium can be examined and resected, if necessary. Although total pericardiectomy can be performed, subphrenic pericardiectomy is usually adequate for animals with pericardial effusion. Total pericardiectomy may be indicated in some animals with neoplasia or infectious processes of the pericardium. Total pericardiectomy is best performed from a median sternotomy approach.

Pericardiectomy is the therapy of choice for constrictive pericarditis. Complications associated with surgery include development of arrhythmias (most notably atrial fibrillation or ventricular tachycardia). The outcome with surgery depends on the severity of the underlying disease. If the visceral pericardium

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(epicardium) is significantly involved, the surgical outcome is less favorable. Epicardial decortication may be necessary.

Preoperative Management

If hemodynamically significant quantities of pericardial effusion are present (i.e., cardiac tamponade as evidenced by jugular vein distention, ascites, and/or pleural effusion), the animal should have preoperative pericardiocentesis. Metabolic causes of pericardial effusion (e.g., hypoproteinemia) should be ruled out. Electrolyte and acid-base abnormalities (e.g., associated with high doses of diuretics) should be corrected before anesthetic induction.

Anesthesia

Refer to p. 788 for anesthetic management of cardiac patients. Before anesthesia, pericardiocentesis will typically have improved clinical signs substantially. Because pericardial fluid cannot be removed completely and may return quickly, it must be remembered that these patients are still compromised (see Table 27.1). Their cardiac output is very rate dependent, and care should be taken to avoid drops in heart rate and blood pressure. Bradycardia should be treated with an anticholinergic (i.e., atropine, glycopyrrolate), and blood pressure should be treated with ephedrine boluses. If necessary, epinephrine, dobutamine, or isoproterenol may be used with severely compromised and unstable patients with pericardial constriction. Ketamine is an excellent choice for induction because of its sympathetic stimulation. Propofol should be avoided owing to the hypotension and decrease in myocardial contractility that it causes. Fentanyl and midazolam are excellent premedicants. Fentanyl as a constant-rate infusion can be continued intraoperatively and postoperatively. Care should be exercised when the heart is manipulated because arrhythmias or hypotension may occur. In the unstable patient, avoid mechanical ventilation if possible. Positive-pressure ventilation of these patients can add just enough pressure to the thoracic cavity to decrease preload. During pericardiocentesis or before the chest has been opened, if the patient is able to breathe spontaneously, let him do so. In addition, avoid PEEP. This is one of the few times it is contraindicated; it should not be used until the pressure around the heart has been resolved. Unilateral bronchial intubation or alternating one-lung ventilation can be done for thoracoscopic pericardiectomy performed from a lateral approach. Avoid administering alpha agonists and acepromazine.

Surgical Anatomy

The pericardium envelops the heart in a strong flask-shaped sac with extensions that enclose the origins of the ascending aorta, pulmonary artery, distal pulmonary veins, and venae cavae. The adventitia of the great arteries blends with fibrous tissue of the pericardium, forming strong attachments. The pericardium is firmly attached to the diaphragm via the pericardiophrenic ligament. The pericardium is composed of two layers: a fibrous outer layer and an inner serous membrane composed of a single layer of mesothelial cells. The inner serous layer forms the epicardium or visceral pericardium. It reflects back on itself to line the outer fibrous layer, and together they form the parietal pericardium.

The pericardial cavity is filled with a variable amount of pericardial fluid. This fluid is an ultrafiltrate of serum containing between 1.7 and 3.5 g/dL of protein and having a colloidal osmotic pressure approximately 25% that of serum. The volume of pericardial fluid present in normal dogs ranges from 1 to 15 mL. Lymphatic drainage of the pericardium is similar to that of the myocardium, with most drainage via epicardial lymphatics rather than from parietal pericardium. Functions ascribed to the pericardium include the ability to fix the heart anatomically and prevent excessive motion associated with changes in body position, reduction in friction between the heart and surrounding structures, and prevention of extension of infection or malignancy from the pleural space.

Positioning

The animal should be placed in lateral recumbency for an intercostal thoracotomy or in dorsal recumbency for a median sternotomy. A sufficiently large area should be prepared to allow intraoperative placement of a thoracostomy tube.

SURGICAL TECHNIQUE

Partial, subtotal, or total pericardiectomy can be performed depending on the underlying cause and the surgical approach (thoracotomy, sternotomy, or thoracoscopy) chosen.

Subphrenic (Subtotal) Pericardiectomy via Right Thoracotomy

After opening the chest, open the pericardium and submit fluid samples for microbiologic examination, fungal culture, and/or cytology, if indicated. Make a T-shaped incision in the pericardium from cardiac base to apex and across the cardiac base ventral to the phrenic nerve (Fig. 27.23A). Extend the circumferential incision at the cardiac base around the venae cavae, taking care not to violate the vessel walls (Fig. 27.23B). Have an assistant elevate the heart and retract it as the circumferential incision is extended to the opposite side (see Fig. 27.23C). Take care not to injure the contralateral phrenic nerve. Divide the pericardiophrenic ligament with cautery or between ligatures (Fig. 27.23D). Check the remnants of the pericardium to ensure that no hemorrhage is occurring. Submit the pericardium for histologic analyses. Place a thoracostomy tube before thoracic closure. It may be necessary to only remove the pericardium on one side well below the phrenic nerve. Removal of large amounts of pericardium seems to only be necessary if the pericardium itself is diseased.

Total Pericardiectomy

Using blunt dissection, carefully elevate the phrenic nerves from the pericardial sac. Make a longitudinal incision in the pericardial sac and resect the pericardium as close to the base of the heart as possible. Place a thoracostomy tube before thoracic closure.

Thoracoscopic Pericardiectomy

Thoracoscopic pericardiectomy requires video assistance and can be performed from a lateral or subxiphoid approach; the latter is described below. *Position the patient in dorsal recumbency, sometimes tilting 10 to 15 degrees to the left, or in left lateral recumbency, depending on your preference. Typically, place the observation port substernally (paraxiphoid), entering the chest on the patient's right side.* If the patient is large enough, placing an operating scope (i.e., a 10-mm-diameter scope with a 5-mm biopsy channel through it) in this position allows the port to be used to simultaneously observe and manipulate the pericardium. *Usually place two ancillary ports on the right side, but one can be placed on the right and one on the left (the latter requires making a large window in the mediastinum).* It is helpful to place the

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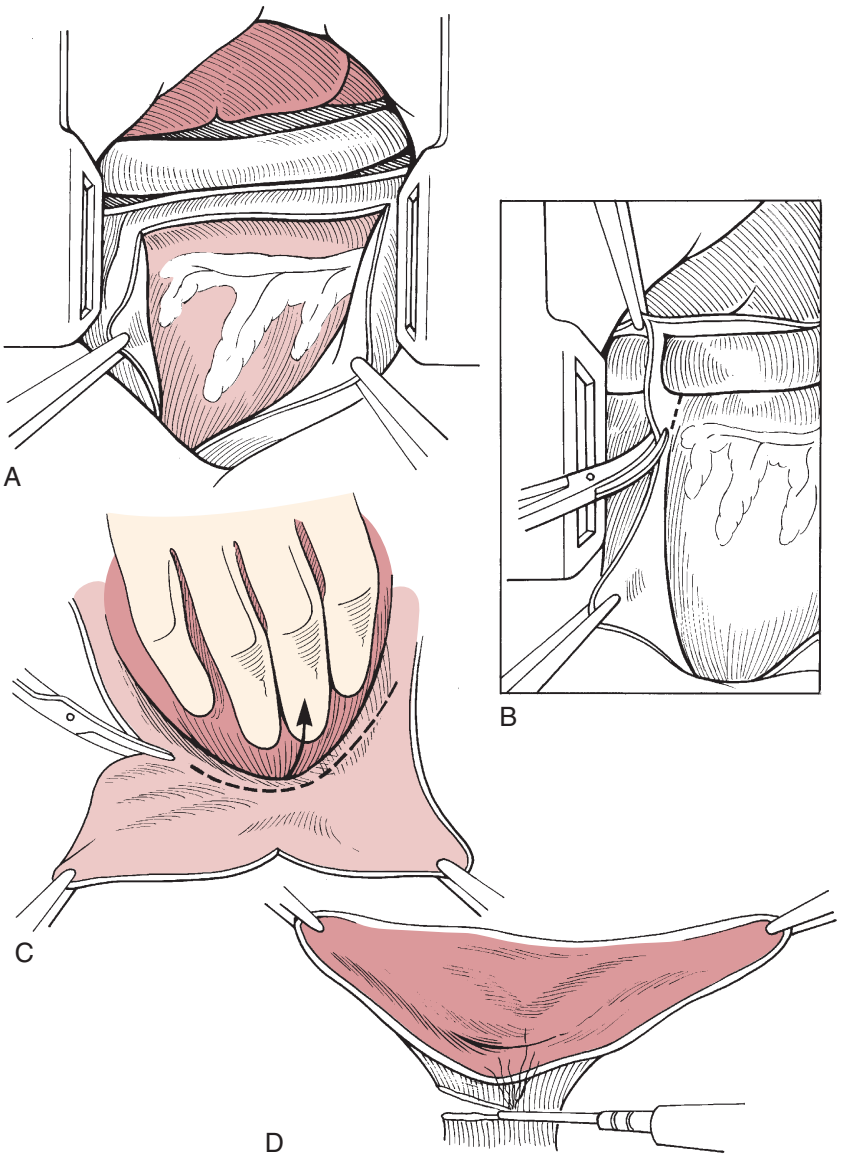


FIG. 27.23 (A) For subtotal pericardiectomy via a right fifth intercostal thoracotomy, incise the epicardium vertically and horizontally ventral to the right phrenic nerve. (B) Carefully extend the incision around the vena cava, taking care to identify the vessel wall while making the incision. (C) Gently retract the heart and extend the incision across the left side, ventral to the left phrenic nerve. (D) Divide the pericardiophrenic ligament with cautery or between ligatures. Removal of smaller portions of the pericardium may be equally as effective.

ports at or above (from the perspective of the endoscopist) the level of the costochondral junction (i.e., ventral to the costochondral junction from the perspective of the dog) to minimize the chance that the inflated lungs may obscure vision. *Place the ports around the sixth intercostal and ninth intercostal spaces (this can be varied based on the needs in a particular animal). Use Babcock forceps, hemostats, scissors, electrocautery, and/or a bipolar vessel-sealing device to make a large opening in the mediastinum if necessary to enhance visualization, to grasp the pericardium, and to incise and resect a portion (3 × 3 cm) of pericardium (Fig. 27.24). If possible, open the pericardium sufficiently that you can visualize the right atrium and see previously undetected right atrial masses or diffuse intrapericardial proliferations. Take care to avoid cutting the phrenic nerve. Place a thoracostomy tube after the procedure.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Electrocautery or a vessel-sealing device (e.g., LigaSure, Medtronic) are useful for pericardiectomy to decrease intraoperative and postoperative hemorrhage. Inflamed pericardium often has an increased number of blood vessels, and significant hemorrhage can occur after pericardiectomy if these vessels are not cauterized or ligated.

POSTOPERATIVE CARE AND ASSESSMENT

The thoracostomy tube should be aspirated every hour initially and the volume of pleural effusion quantitated. After 4 to 6 hours, frequency of drainage may be decreased to every 2 to 4 hours. Once the pleural effusion has decreased to levels consistent

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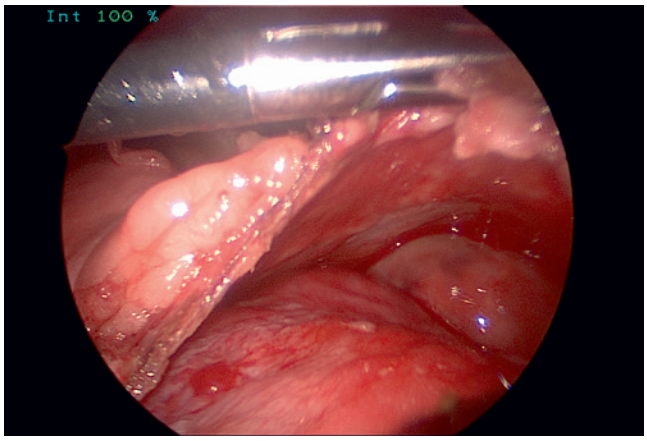


FIG. 27.24 Subxiphoid thoracoscopic view of creation of a pericardial window in a dog with pericardial effusion due to a heart base mass; the pericardial sac is grasped with Babcock forceps.

with those caused by the thoracostomy tube, the tube may be removed. If the patient develops acute respiratory distress without evidence of pleural effusion or significant pulmonary infiltrates suggestive of pulmonary edema, PTE should be suspected. Oxygen therapy may be beneficial in such cases. If PTE is diagnosed, thrombolytic agents may be used, but their value is uncertain (see Box 29.1). Postoperative pain should be treated with systemic opioids (see Tables 13.2 and 13.3) and local anesthetic techniques (see Chapter 13 and Box 30.2).

PROGNOSIS

Pericardiectomy is palliative for neoplastic pericardial effusion and curative for idiopathic pericardial effusion. The metastatic rate is 50% to 66% regardless of the type of tumor, but long-term palliation is possible for tumors other than hemangiosarcoma. Dogs with mesothelioma that have undergone subphrenic pericardiectomy survive to 1 year 80% of the time. Intracavitary chemotherapy may be considered for adjunctive therapy for mesothelioma for possible longer remission times.

Chemodectomas are slow-growing tumors, and long-term palliation with pericardiectomy and primary mass excision is possible. Regardless of the presence or absence of pericardial effusion at the time of surgery, dogs with aortic body tumors survive significantly longer if a pericardiectomy is performed (median survival, 730 days) than if one is not done (median survival, 42 days).

For dogs with cardiac hemangiosarcoma, median survival is approximately 4 months with pericardiectomy with or without resection of the right auricular tumor. Dogs presenting with collapse have significantly shorter survival times than those that do not.

CARDIAC NEOPLASIA

DEFINITIONS

Cardiac neoplasia includes any neoplastic condition involving the heart, great vessels, or pericardium. Synonyms for hemangiosarcomas include *angiosarcomas* and *malignant hemangioendotheliomas*. Tumors arising from the chemoreceptor aortic bodies have also been called *chemodectomas, heart base tumors, aortic body adenomas* or *carcinomas,* or *nonchromaffin paragangliomas*.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Cardiac neoplasia is relatively uncommon in small animals. The most important cardiac neoplasms in dogs are right atrial hemangiosarcoma and heart base chemodectoma. Hemangiosarcoma is the most common cardiac tumor, occurring nearly 10 times as frequently as the second most common tumor, aortic body tumor. A variety of primary intramural and intracavitary neoplasms have been reported in dogs, including hemangiosarcoma, fibrosarcoma, chondrosarcoma, rhabdomyosarcoma, ectopic thyroid carcinoma, fibroma, and myxoma. Lymphosarcoma and metastatic neoplasia are the most frequent causes of cardiac neoplasia in cats.

The right atrium is a common primary site for hemangiosarcoma and accounts for 40% to 50% of canine cases of hemangiosarcoma. Other reported primary cardiac sites for hemangiosarcoma include the right ventricular free wall, the interventricular septum, and the main pulmonary artery. Primary cardiac hemangiosarcoma has not been described in cats, but metastasis of hemangiosarcoma to the heart has been reported.

Chemodectomas can arise from the aortic body at the base of the heart (e.g., between aorta and pulmonary artery, between aorta and right atrium, between pulmonary artery and left atrium) or from the carotid body in the neck. Aortic body chemodectomas account for approximately 80% of chemodectomas and occur in older dogs. Chemodectomas occur rarely in cats. Residence at altitude and chronic hypoxia probably increase the risk for developing these tumors. Chemodectomas can cause pericardial effusion, which probably accounts for the most common clinical presentation of this disease. However, chemodectomas just as often are an incidental finding in older dogs undergoing thoracic radiography or echocardiography for other reasons. Ectopic thyroid adenomas and carcinomas account for approximately 5% to 10% of all heart base tumors in dogs.

DIAGNOSIS

Clinical Presentation

Signalment

German shepherd dogs and golden retrievers have been identified as having increased risk for developing hemangiosarcoma. Boxers, English bulldogs, and Boston terriers are the breeds most likely to develop chemodectomas.

History

Animals with cardiac neoplasia may be brought in for evaluation of dyspnea, cough, syncope, or CHF, or they may be asymptomatic.

Physical Examination Findings

The most common clinical presentation for right atrial hemangiosarcoma is acute or chronic cardiac tamponade resulting from intrapericardial hemorrhage (see the section on pericardial effusion, p. 817). Animals with chemodectomas may present for evaluation of CHF, signs of cardiac tamponade, or pleural effusion, or they may be asymptomatic.

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Diagnostic Imaging

Thoracic radiographs of animals with chemodectomas may show dorsal elevation of the terminal trachea, pleural or pericardial effusion, pulmonary edema, or increased perihilar density. Selective angiography has identified canine chemodectomas. Echocardiography has a high sensitivity and specificity for identifying right atrial and heart base masses (Fig. 27.25). However, presumptive specific diagnosis of cardiac tumor type based on echocardiographic anatomic location is only moderately accurate.[31] Computed tomography with or without three-dimensional reconstruction can be helpful to identify the extent of disease, as well as facilitate radiation planning (Fig. 27.26).

Laboratory Findings

Specific laboratory abnormalities are not found with cardiac neoplasia. Cytologic analysis of the pericardial fluid does not reliably differentiate neoplastic from idiopathic pericardial effusion (see p. 817).

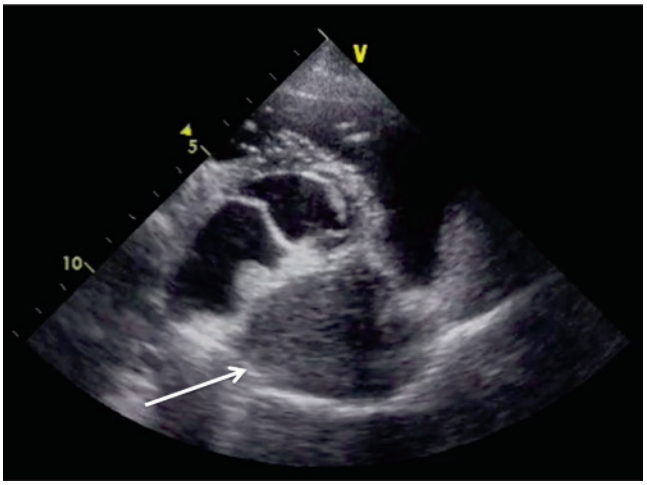


FIG. 27.25 Echocardiographic view of a large heart base mass *(arrow)* in a 9-year-old Labrador retriever.

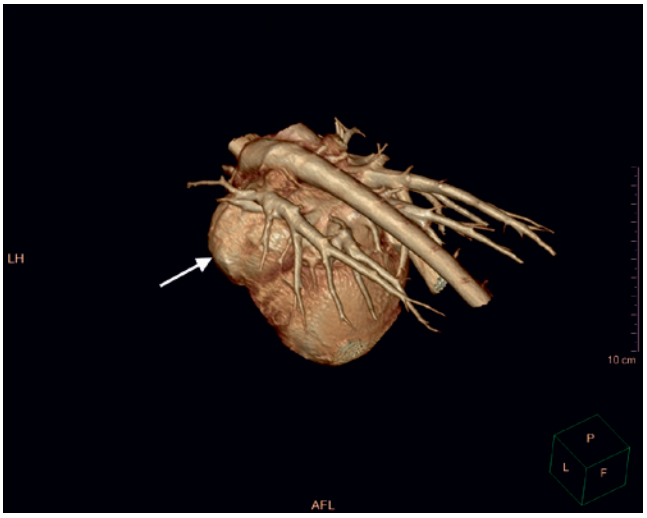


FIG. 27.26 Computed tomography three-dimensional reconstruction of the heart from the same dog in Fig. 27.25; heart base mass is designated by the arrow.

DIFFERENTIAL DIAGNOSIS

Cardiac neoplasia must be differentiated from other causes of pericardial effusion (see p. 819), CHF, and cardiac arrhythmias. Endomyocardial biopsy may be used to make a definitive diagnosis of intracardiac neoplasia. Differentials for radiographic masses near the heart base include hilar lymphadenopathy, left atrial enlargement, aberrant parathyroid or thyroid tissue, and fibrosing pleuritis or pericarditis.

MEDICAL MANAGEMENT

Various chemotherapeutic strategies can be used for cardiac neoplasia (as primary therapy or as an adjunct to surgery). Doxorubicin plus cyclophosphamide and vincristine have been used to attempt to palliate cardiac hemangiosarcoma but have had minimal effect on survival.

SURGICAL TREATMENT

Pericardiectomy and excision of the right atrial tumor are palliative for atrial hemangiosarcoma (see later discussion in the Prognosis section). Thoracoscopic subtotal pericardiectomy and right atrial mass resection has been reported in dogs.[36] Chemodectomas are highly vascular, slow-growing, and moderately locally invasive. Surgical excision of aortic body chemodectomas is possible depending on the size, location, and degree of invasiveness of the tumor. However, many animals with chemodectomas and clinical signs associated with pericardial effusion benefit from pericardiectomy without tumor excision.

Surgical excision of intramural or intracavitary primary cardiac tumors has been attempted rarely in small animals. Surgical excision of well-defined primary cardiac tumors using inflow occlusion or cardiopulmonary bypass is possible in selected cases. However, because of the high incidence of malignancy of most primary cardiac tumors, echocardiographic (Video 27.4), angiographic, and endomyocardial biopsy findings should be considered carefully when appropriate cases are selected for surgery.

Preoperative Management

Abdominal radiography or ultrasonography should be performed before surgery to detect concurrent intraabdominal neoplasia (especially splenic hemangiosarcoma). If hemodynamically significant quantities of pericardial effusion are present (e.g., cardiac tamponade as evidenced by jugular vein distention, ascites, and/or pleural effusion), pericardiocentesis should be performed before surgery.

Anesthesia

Refer to p. 788 for anesthetic management of cardiac patients.

Surgical Anatomy

Refer to p. 794 for surgical anatomy of the heart.

Positioning

The animal is positioned in dorsal recumbency for median sternotomy (see p. 891) or in lateral recumbency for intercostal thoracotomy (see p. 890). A sufficiently generous area should be prepped to allow intraoperative placement of a thoracostomy tube.

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SURGICAL TECHNIQUE

Right Atrial Hemangiosarcoma

Perform a median sternotomy or a right fourth space intercostal thoracotomy. Clamp the atrial appendage with a tangential vascular clamp and excise the appendage (Fig. 27.27). Close the atriotomy incision with a continuous mattress suture pattern. Remove the vascular clamp and oversew the incision with a simple continuous suture pattern. Perform a pericardiectomy if pericardial effusion is present (see p. 821). Alternatively, the right atrial appendage may be excised with a thoracoabdominal stapling instrument or an Endo GIA stapler via thoracoscopy.

Chemodectoma

The surgical approach for removing chemodectoma depends on the suspected location of the tumor.

Sharply dissect the tumor from the walls of the great vessels and atria. Use care to prevent rupturing these structures during dissection. Use electrocautery to decrease hemorrhage during excision of these highly vascular tumors. Perform a subtotal pericardiectomy (see p. 821) whether pericardial effusion is present or not.

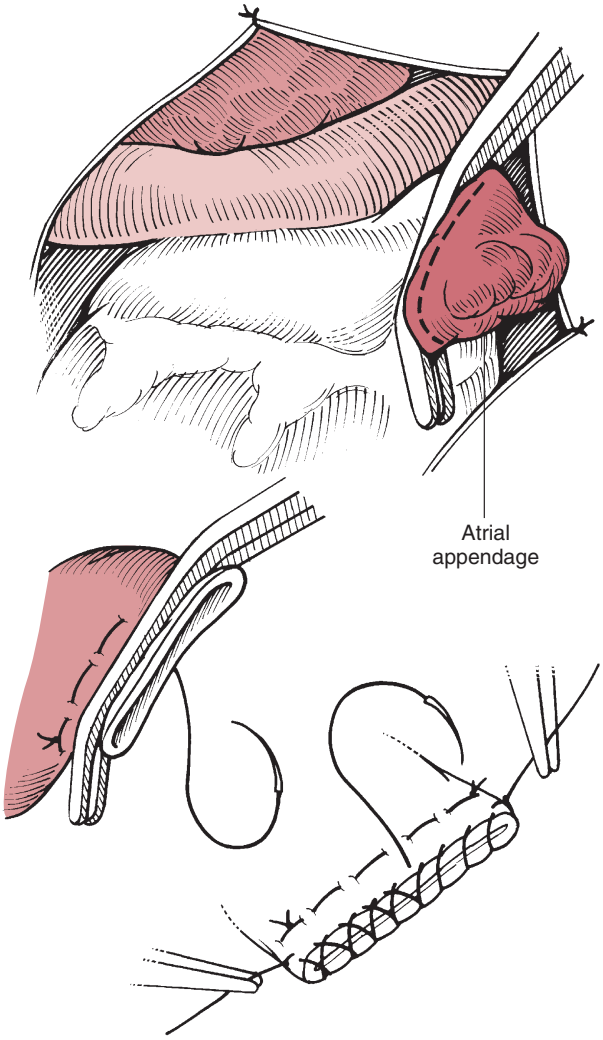


FIG. 27.27 To resect a right atrial hemangiosarcoma, place a tangential vascular clamp across the base of the right auricle and excise the tumor and auricle. Place a continuous horizontal mattress suture behind the vascular clamp. Remove the clamp and oversew the incision with a simple continuous suture.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

A tangential vascular clamp is useful for excision of right atrial hemangiosarcoma. Closure of the right atrium can be accomplished with polypropylene (e.g., 4-0 Prolene or Surgipro) suture.

POSTOPERATIVE CARE AND ASSESSMENT

The animal should be monitored carefully for postoperative hemorrhage (pleural effusion). Arrhythmias are common, and electrocardiography should be monitored for 36 to 72 hours postoperatively. Postoperative pain should be treated with systemic opioids (see Tables 13.2 and 13.3) and local anesthetic techniques (see Chapter 13 and Box 30.2).

PROGNOSIS

The prognosis for right atrial hemangiosarcoma is generally poor; however, surgery plus adjuvant chemotherapy has improved survival. Micrometastasis is considered present in virtually all cases at the time of diagnosis. Pericardiectomy and excision of the right atrium are palliative. Median survival after surgery alone is approximately 4 months.

Long-term survival of up to several years is possible after surgical removal of an aortic body chemodectoma. In older animals with incidental asymptomatic chemodectoma, the risks of surgical excision should be weighed against the likelihood that the tumor will be slow-growing and can remain asymptomatic for a long time.

BRADYCARDIA

DEFINITIONS

Bradycardia is a heart rate that is slower than normal. Bradycardia can be physiologic (e.g., sinus bradycardia) or can result from a variety of pathologic disorders, including sick sinus syndrome, atrial standstill, or AV block with ventricular escape.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

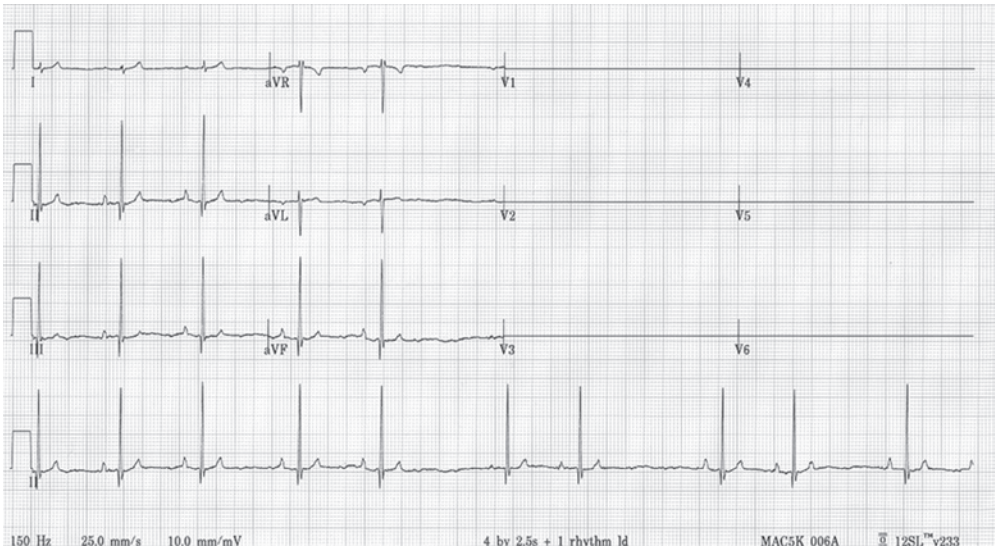
Bradycardia can result from extrinsic causes, such as exaggerated vagal tone or electrolyte imbalance, or from intrinsic degenerative disorders of the heart. Sinus bradycardia results from a predominance of parasympathetic influence (sometimes associated with cranial or abdominal disease) and is often is accompanied by other parasympathetically mediated rhythms (e.g., sinus arrhythmia, wandering pacemaker, low-grade, second-degree AV block). It is generally considered a physiologic rather than a pathologic rhythm.

Atrial standstill (Fig. 27.28A) occurs when the atria fail to conduct an electrical impulse. The cardiac impulse may arise in the sinus node and be conducted to the AV node via internodal pathways in the atria (i.e., sinoventricular rhythm), or an escape rhythm may develop. Transient atrial standstill is caused by hyperkalemia. Persistent atrial standstill occurs as the result of a heritable muscular dystrophy syndrome involving the cardiac atria, ventricles, and scapulohumeral skeletal muscles.

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A



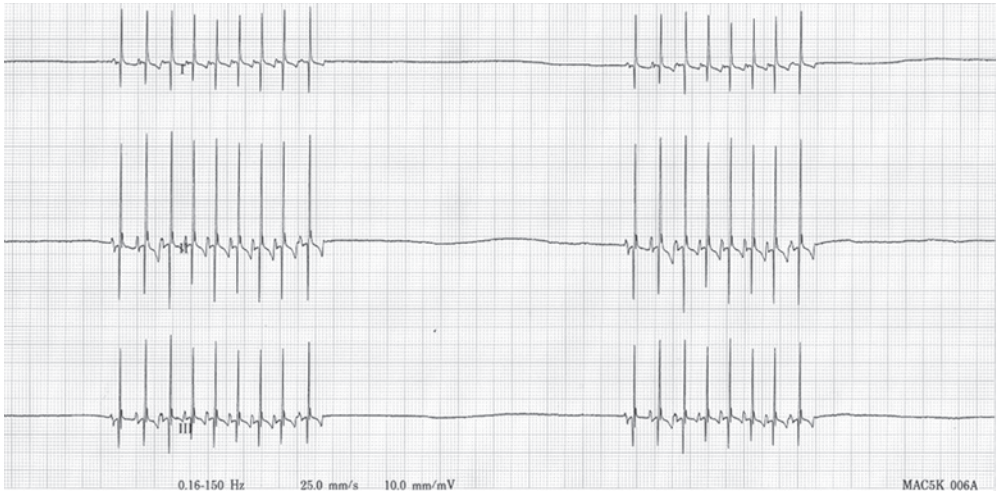
B



C

FIG. 27.28 Electrocardiograms from dogs with (A) atrial standstill, (B) first-degree atrioventricular (AV) block, (C) third-degree AV block, and

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D

FIG. 27.28, cont'd (D) bradycardia-tachycardia associated with sick sinus syndrome. (Courtesy Dr. M. Miller, VetMed, Phoenix, AZ.)

AV block results when a delay or block of cardiac impulse conduction occurs through the AV node. First-degree AV block, which occurs as a prolongation of conduction through the AV node, usually results from exaggerated parasympathetic influence on the AV node (see Fig. 27.28B). Second-degree (incomplete) AV block is characterized by intermittent failure of impulse conduction through the AV node. Low-grade (infrequent) second-degree AV block usually results from exaggerated parasympathetic influence on the AV node. High-grade (frequent) second-degree AV block is more likely the result of intrinsic disease of the AV node. Third-degree (complete) AV block (see Fig. 27.28C) is seen as complete failure of conduction through the AV node and strongly implies intrinsic degenerative or infiltrative disease of the AV node. Third-degree AV block causes complete AV dissociation and development of a slow ventricular escape rhythm. The result is low and unresponsive cardiac output.

Frequent and long sinus pauses may result from degeneration and malfunction of the sinus node (see Fig. 27.28D). *Sick sinus syndrome,* which is the clinical result of sinus node malfunction, is characterized by frequent syncopal and near-syncopal episodes. Sick sinus syndrome may also be accompanied by frequent supraventricular tachycardia.

DIAGNOSIS

Clinical Presentation

Signalment

English springer spaniels and Siamese cats are predisposed to persistent atrial standstill. Small-breed dogs, particularly miniature schnauzers, are predisposed to sick sinus syndrome. Third-degree AV block occurs in all breeds. The Labrador retriever is the most common breed to receive artificial pacing.

History

Signs due to bradycardia include weakness, exercise intolerance, collapse, and syncope. The relatively short duration of syncopal episodes (usually only a few seconds) and the lack of tonic-clonic motor activity or postictal signs can help distinguish syncope from neurologic seizures. However, syncopal and neurologic episodes can sometimes be difficult to distinguish, sometimes requiring ambulatory ECG monitoring for definitive diagnosis.

BOX 27.8 Atropine or Glycopyrrolate Response Test

Give 0.02–0.04 mg/kg atropine SC or IM; wait 15–20 min, then recheck cardiac rhythm, *or*
Give 0.01 mg/kg glycopyrrolate SC or IM; wait 15–20 min, then recheck cardiac rhythm

IM, Intramuscular; *SC,* subcutaneous.

Physical Examination Findings

Sinus bradycardia is recognized on ECG as a normal but slow rhythm with normal P-QRS-T complexes. It is often accompanied by other vagally mediated changes (i.e., sinus arrhythmia, wandering pacemaker, and low-grade, second-degree AV block). Sinus bradycardia is abolished by exercise or by administration of atropine or glycopyrrolate (Box 27.8).

Electrocardiography

Electrocardiographic abnormalities associated with transient atrial standstill from hyperkalemia are bradycardia, small or absent P waves, and shortening and widening of QRS complexes (see Fig. 27.28A). The major rule-outs for hyperkalemia are obstructive uropathy, acute renal failure, uroabdomen, adrenocortical insufficiency, and iatrogenic potassium intoxication. Electrocardiographic abnormalities associated with persistent atrial standstill are similar (e.g., an absence of P waves, a slow supraventricular or ventricular escape rhythm).

Electrocardiographic findings associated with sick sinus syndrome include intermittent severe bradycardia, sinus pauses that last several seconds, supraventricular escape complexes, and occasionally paroxysmal supraventricular tachycardia (see Fig. 27.28D). Sick sinus syndrome causes frequent syncopal attacks but rarely causes sudden death. Sick sinus syndrome usually is not responsive to acute administration of atropine.

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BOX 27.9 First-Degree Atrioventricular Block

Dogs: PR interval >0.14 seconds
Cats: PR interval >0.09 seconds

First-degree AV block (Box 27.9) is recognized by prolongation of the P-R interval on an ECG (see Fig. 27.28B). Second-degree AV block is intermittent failure of impulse conduction through the AV node. It is recognized on an ECG as a P wave that is not followed by a QRS-T complex. Low-grade, second-degree AV block is characterized by occasional "dropped complexes" after several normal complexes and is usually abolished by atropine. High-grade, second-degree AV block is characterized by more dropped complexes than are seen with conducted complexes and usually does not respond to atropine. Third-degree AV block is recognized on ECG by complete dissociation of the P waves and QRS-T complexes and by a slow ventricular escape rhythm (see Fig. 27.28C). Third-degree AV block is not atropine responsive.

Diagnostic Imaging

Thoracic radiographs are usually normal or show mild to moderate generalized cardiomegaly.

Echocardiography

With transient atrial standstill, echocardiography shows lack of atrial motion. Echocardiography is also used to rule out concurrent valvular or congenital abnormalities.

Laboratory Findings

Hyperkalemia may cause transient atrial standstill; however, persistent atrial standstill is usually associated with normal serum potassium concentrations. Other specific laboratory abnormalities are not found.

DIFFERENTIAL DIAGNOSIS

Non-cardiogenic causes of bradycardia (e.g., hyperkalemia, increased vagal tone from central nervous system disease [intracranial pressure] or abdominal disease) should be differentiated from intrinsic conduction system dysfunction.

MEDICAL MANAGEMENT

Therapy for atrial standstill secondary to hyperkalemia should be directed at immediately lowering serum potassium levels and correcting the underlying cause of hyperkalemia. Aggressive IV fluid therapy with physiologic saline solution will dilute the hyperkalemia, which is typically sufficient. If the animal has concurrent hyponatremia, 5% dextrose solutions (i.e., D5W) and half-strength saline should be avoided. Very severe hyperkalemia rarely needs additional therapy (e.g., sodium bicarbonate therapy (Box 27.10) which drives potassium into cells in exchange for hydrogen ions; or, insulin (see Box 27.10) and dextrose (2 g per unit of insulin) simultaneously administered to facilitate cellular uptake of potassium). If hyperkalemia appears immediately life-threatening, 10% calcium gluconate given slowly intravenously may protect the heart until other therapy lowers the plasma potassium concentration.

BOX 27.10 Therapy for Hyperkalemia

Physiologic Saline Solution (0.9%) Aggressive IV Administration
To dilute potassium and enhance renal excretion

Sodium Bicarbonate[a]
1–2 mEq/kg IV (give over 10–20 min)

Insulin[a]
0.2–0.4 U/kg regular insulin IV plus dextrose (2 g/U of insulin)

10% Calcium Gluconate[b]
0.5–1 mL/kg over 5–15 min, IV to effect with ECG monitoring

[a]Used for severe hyperkalemia.
[b]Temporary measure to sustain animal while other methods lower the blood potassium concentration (i.e., this drug does not affect blood potassium concentration itself); monitor the ECG while administering this drug. If bradycardia occurs, stop infusion. Give only to patients that are critical and may die shortly from hyperkalemia.
ECG, Electrocardiogram; *IV,* intravenous.

BOX 27.11 Drugs Used to Increase Heart Rate in Animals With Unresponsive Bradycardia

Isoproterenol (Isuprel)
0.01–0.08 µg/kg/min IV

Propantheline Bromide (Pro-Banthine)
0.25–0.5 mg/kg PO q6–8h

IV, Intravenous; *PO,* orally.

NOTE Never give calcium chloride intravenously.

Animals that have severe life-threatening bradycardia may require emergency therapy to increase their heart rate. Short-term anticholinergic therapy with atropine or glycopyrrolate may be attempted, but many clinically relevant bradycardias are not due to parasympathetic mechanisms and are not responsive to these drugs. IV adrenergic therapy with isoproterenol (Box 27.11) is sometimes effective as a short-term measure for increasing the heart rate associated with persistent atrial standstill or third-degree AV block.

The most reliable method for increasing heart rate in animals with unresponsive bradycardia is temporary transvenous pacing. This is accomplished by percutaneous jugular or lateral saphenous venous placement of a pacing electrode into the right side of the heart under sedation and local anesthetic (see later discussion under Anesthesia). The electrode is then connected to an external pulse generator. Long-term oral anticholinergic therapy with propantheline bromide (see Box 27.11) is sometimes advocated for various bradycardias. However, this drug is seldom effective for clinically relevant bradycardias and causes unpleasant side effects. Animals with sick sinus syndrome may require management of supraventricular tachycardia with digoxin, β-adrenergic blockade, or calcium channel blockade therapy after pacemaker implantation.

Permanent transvenous endocardial pacemakers offer a reasonable treatment option for dogs with symptomatic bradycardia; this is the most common route for pacemaker implantation.

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Transdiaphragmatic epicardial pacemakers are used less often but are elected when experience and/or equipment for transvenous placement is lacking, when complications or failure of the transvenous pacemaker occurs, or when patients have underlying metabolic predispositions to venous thrombosis (e.g., protein-losing nephropathy and hospital-acquired conditions).

SURGICAL TREATMENT

Cardiac pacemaker therapy is indicated for bradycardias caused by intrinsic cardiac disease that is not responsive to atropine and that is causing clinical signs.

Preoperative Management

Most bradycardias are exacerbated by anesthetic drugs. Therefore some accommodation for maintaining an acceptable cardiac rhythm during permanent pacemaker implantation is usually necessary. Preanesthetic medication with an anticholinergic drug (e.g., atropine, glycopyrrolate) is indicated but is rarely sufficient to prevent worsening of bradycardia during anesthesia. Temporary transvenous pacing is the most reliable method of maintaining an adequate heart rate during pacemaker implantation. Constant IV infusion of isoproterenol (see Box 27.11) is a less reliable means of maintaining heart rate during permanent pacemaker implantation. Perioperative antibiotic therapy (e.g., cefazolin) during pacemaker implantation is indicated to reduce the risk of implant-associated infection.

Anesthesia

Temporary pacemakers can be implanted in dogs under ketamine plus diazepam (Box 27.12) administered intravenously to effect for sedation; a local anesthetic is used to place the temporary pacemaker. Once the animal is paced, ketamine plus diazepam may be used for induction. Anesthesia should be maintained with isoflurane or sevoflurane and oxygen.

Surgical Anatomy

Refer to p. 794 for surgical anatomy of the heart.

Positioning

The animal is placed in dorsal recumbency for transdiaphragmatic pacemaker implantation. The entire abdomen and caudal thorax are prepared for aseptic surgery.

SURGICAL TECHNIQUE

Epicardial pacemaker implantation in small animals is accomplished through a midline celiotomy diaphragmatic incision. The transdiaphragmatic approach has several advantages, including avoidance of a thoracotomy and abdominal placement of the generator.

BOX 27.12 Anesthesia for Placement of a Temporary Pacemaker

Ketamine (5.5 mg/kg) Plus Diazepam (0.27 mg/kg)
Administer IV to effect for sedation
Use a local anesthetic at site of pacemaker insertion
Maintain anesthesia with isoflurane or sevoflurane and oxygen[a]

[a]Not a particularly painful procedure; low doses of inhalant are usually adequate.
IV, Intravenous.

Perform a celiotomy that extends cranially to the level of the xiphoid (Fig. 27.29A). Make a vertical midline incision in the diaphragm and expose the cardiac apex. Open the pericardium and retract it gently with tissue forceps to expose the apex of the left ventricle (see Fig. 27.29B). Implant a screw-in electrode into the left ventricular apex by turning the electrode tip a specified number of rotations (see instruction sheet accompanying pacemaker), usually 2.5 turns (Fig. 27.30A). Alternatively, directly suture the lead(s) to the epicardium using 3-0 or 4-0 nonabsorbable monofilament suture (e.g., polypropylene [Prolene], polybutester [Novafil]) (Fig. 27.30B). Bring the lead wire into the abdominal cavity through the diaphragmatic incision and connect it to the pulse generator using the small screwdriver or locking mechanism provided by the manufacturer. Place the pulse generator in a pocket created between the transverse abdominis and internal abdominal oblique muscles (Fig. 27.29C). Do not suture the pericardium. Place a thoracostomy tube. Close the diaphragm and abdomen in routine fashion (Fig. 27.29D).

NOTE If a unipolar system is used, the pulse generator does not begin to function until the generator casing is brought into contact with the patient to complete the electrical circuit.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Modern cardiac pulse generators are compact, have a long battery life, are programmable after implantation, and are generally capable of a variety of sophisticated pacing modes. A three-letter code identifies the intended site of cardiac sensing, the intended site of cardiac pacing, and the pacing mode. The most commonly used pacing mode in small animals is VVI, which stands for the ventricular-sensing, ventricular-pacing, inhibited mode. This means that the pacemaker is intended to pace the cardiac ventricles, but it will sense naturally occurring ventricular impulses and will inhibit its own output when they occur. This demand function prevents competitive rhythms between the heart and the pacemaker should spontaneous intrinsic ventricular activity occur. Most recent model pulse generators are powered by lithium cells that have a life of 8 to 12 years. Pacemakers that have exceeded their shelf life for implantation in humans but still have several years of useful battery life left can often be obtained for a fraction of the cost of new pacemakers. Modern pacemakers are programmable by radiofrequency after implantation for several indices (e.g., pacing rate, stimulus voltage, sensing voltage). Cardiologists and pacemaker technical representatives can usually provide appropriate programmers for setting pulse generator parameters before and after surgery. Dogs are paced at a rate of 70 to 110 beats per minute, depending on the size and nature of the animal. Ideally, the stimulus voltage should be approximately two times the measured stimulus capture threshold. A voltage of 4 to 5 V is usually adequate.

Pacemaker electrodes may be endocardial (transvenous) or epicardial. Endocardial electrodes may be unipolar or bipolar and are intended for placement in the right ventricle via a jugular vein. Endocardial electrodes may be used for temporary or permanent cardiac pacing. Endocardial electrodes offer the advantage of less invasive placement but require facilities for cardiac catheterization and have a higher incidence of catheter dislodgment. Permanent endocardial electrode placement requires pulse generator implantation in the neck. Epicardial leads are

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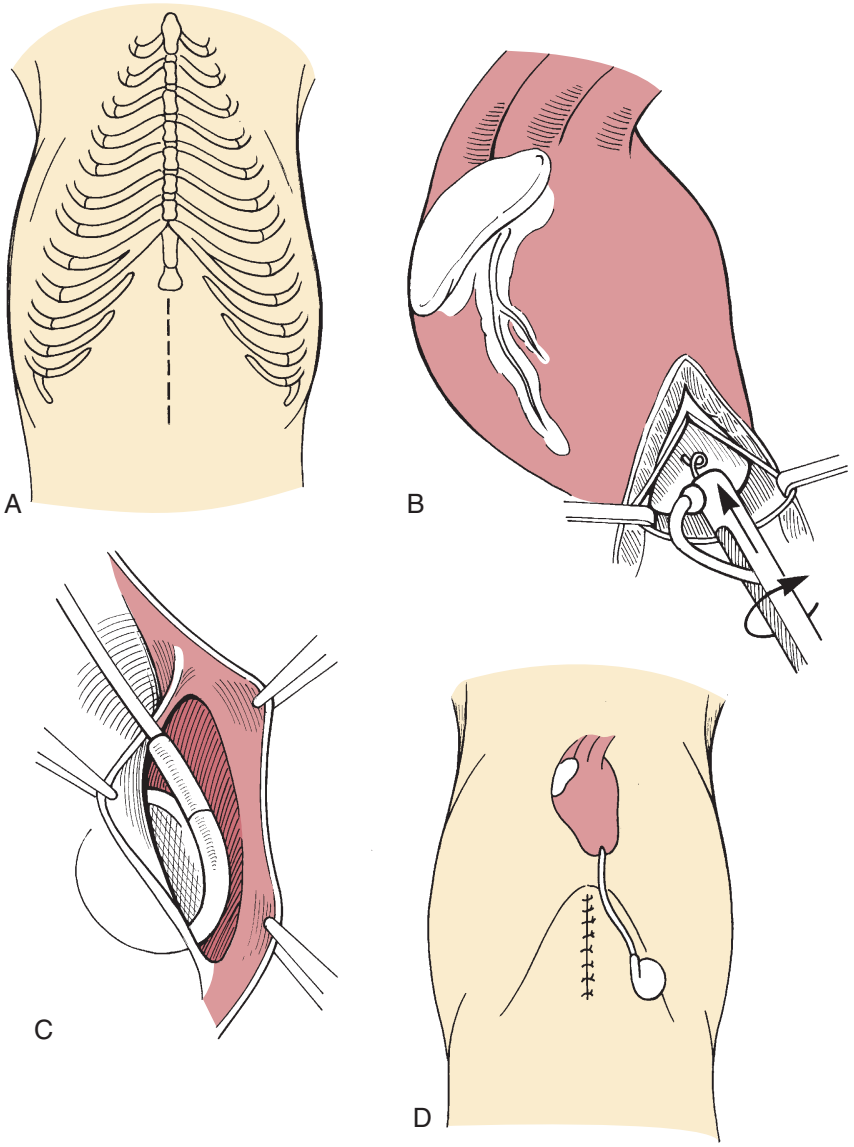


FIG. 27.29 (A) Transdiaphragmatic pacemaker implantation may be performed via midline celiotomy. (B) Incise the diaphragm on its midline and open and retract the pericardium to expose the cardiac apex. Implant a screw-in electrode into the left ventricular apex by turning the electrode the specified number of rotations. (C) Bring the lead wire through the diaphragmatic incision and connect it to the pulse generator. Place the generator in a pocket created between the transverse abdominis and internal abdominal oblique muscles. (D) Close the diaphragm and abdomen routinely.

unipolar and require open thoracic surgery for implantation on the epicardial surface. The screw-in epicardial electrode has the advantage of not requiring epicardial sutures and allows a minimal thoracic approach for implantation (Fig. 27.31).

POSTOPERATIVE CARE AND ASSESSMENT

Pacemaker function should be monitored closely for the first 48 hours postoperatively, and thereafter every 3 to 6 months. Recognition of normal pacemaker function is an important aspect of pacemaker management after surgery. Demand (VVI) pacemakers should be monitored both for their ability to pace or capture the heart and for their ability to sense intrinsic cardiac impulses and to inhibit their output when intrinsic rhythms occur. Failure of either of these functions can cause serious problems for the patient. Paced beats are recognized on ECG by the presence of a stimulus artifact just before the QRS-T. A stimulus artifact will be present on the ECG, regardless of whether the stimulus captures the heart.

Failure to pace is recognized on ECG by the presence of a stimulus artifact that is not followed by a QRS-T. Evaluation of paced beats for the presence of a T wave is important because artifacts that mimic the QRS complex may be present and can be misleading. Failure to pace is also recognized by its failure to generate an arterial pulse. Early failure to pace can be caused by an inadequate stimulus voltage or by a faulty connection between

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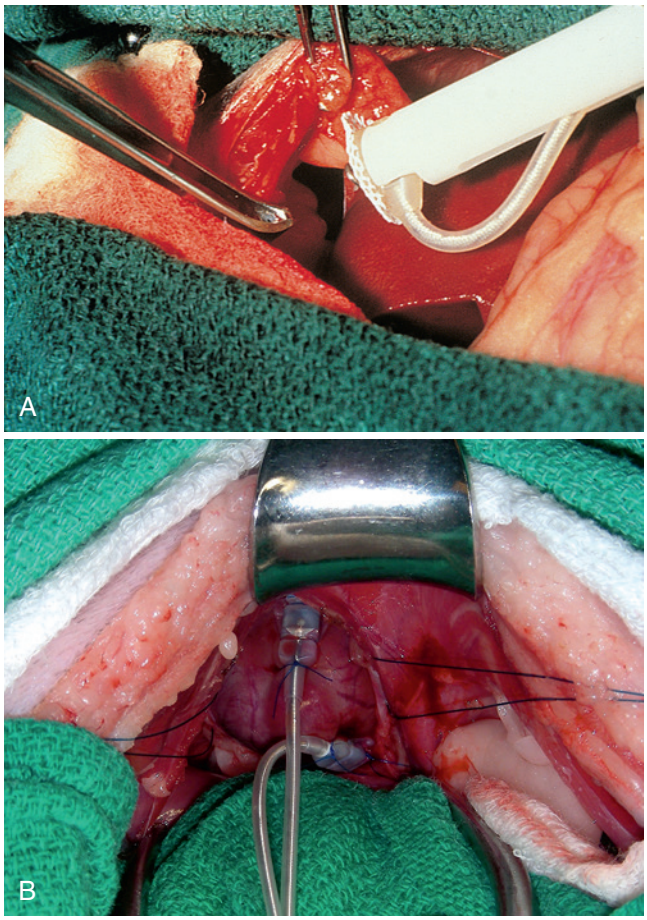


FIG. 27.30 Transdiaphragmatic pericardial electrode placement. (A) Screw-in epicardial electrode. (B) Sutured epicardial electrodes.

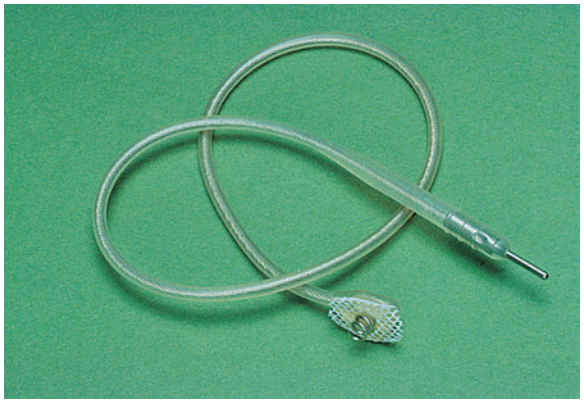


FIG. 27.31 Screw-in epicardial electrode.

the electrode and the generator. Late failure to pace can be caused by depletion of the generator battery, electrode breakage or dislodgment, or fibrosis, leading to increased impedance at the electrode-myocardial interface. Failure to pace may be correctable by adjusting the pacing stimulus voltage of the generator. Radiographs are useful for evaluating for lead breakage, disconnection, or dislodgment.

Failure to sense intrinsic cardiac impulses can lead to competitive rhythms between the heart and the pacemaker. Competitive rhythms are harmful because they result in tachycardia and place the patient at risk for ventricular fibrillation. Failure to sense is recognized on ECG by the presence of an intrinsic cardiac impulse between two paced impulses with a normal pacing interval. Failure to sense may or may not be accompanied by failure to pace. Failure to sense can be caused by a failing generator battery or by increased impedance at the electrode-myocardial interface. Failure to sense may be correctable by adjusting the sensing voltage threshold of the generator.

PVCs are often observed in the immediate postoperative period after pacemaker implantation. The origin of the ventricular complexes is usually consistent with the site of electrode implantation. Ventricular ectopy is usually self-limiting and is not a major problem as long as the pacemaker is sensing the premature complexes. Ventricular tachycardias that exceed 150 beats per minute should be suppressed with lidocaine therapy.

PROGNOSIS

Animals showing clinical signs of severe exercise intolerance or syncope as a result of bradycardia are at risk for sudden death or development of CHF. Pacemaker therapy is extremely effective in preventing these consequences and restoring reasonably normal activity to animals with clinically relevant bradycardia. Major complication rate and survival time are similar between animals receiving transvenous and epicardial pacemaker implantation.

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28

Surgery of the Upper Respiratory System

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Rhinotomy is an incision into the nasal cavity. **Tracheotomy** is an incision through the tracheal wall. **Tracheostomy** is the creation of a temporary or permanent opening into the trachea to facilitate airflow. The permanent tracheostomy opening is called a **tracheostoma. Tracheal resection and anastomosis** consists of removal of a segment of trachea and reapposition of the divided tracheal ends. **Ventriculocordectomy** *(debarking* or *devocalization)* is resection of the vocal cords.

PREOPERATIVE MANAGEMENT

Upper airway procedures are performed to remove, repair, or bypass areas of obstruction, injury, or disease (Box 28.1). Affected animals may have mild to severe respiratory distress. Mild or moderately dyspneic patients should initially be examined from a distance to prevent exacerbating the condition. Open-mouth breathing, abducted forelimbs, labored breathing, and restlessness indicate moderate to severe respiratory distress that may require emergency therapy. Minimal restraint should be used with severely distressed patients, and they should be allowed to maintain the position in which they feel most comfortable. Supplemental oxygen may be given by means of nasal insufflation, tracheostomy tube or catheter, endotracheal intubation, flow-by, face mask (including an Elizabethan collar that has had plastic wrap put over it to create an oxygen-rich environment [see Fig. 4.4]), or oxygen cage (see p. 29). Glucocorticoids, sedation, and/or cooling may help relieve some of the distress. Mild sedation may be beneficial for anxious patients (especially those with upper airway obstruction) with moderate to severe respiratory distress (see Chapter 12). Combinations of intravenous (IV) drugs are commonly given; hydromorphone or butorphanol and either acepromazine or diazepam are frequently used in dogs (Box 28.2). In cats, buprenorphine or midazolam is recommended (Box 28.3). To cool distressed animals, a fan may be directed at the patient; ice packs may be applied to the head, axilla, inguinal area, and extremities; and/or cooled fluids may be administered intravenously. Monitor temperature closely to avoid cooling the animal too rapidly.

NOTE In dogs, you may see some panting following administration of mu agonist opioids (i.e., hydromorphone); thus butorphanol may be preferred.

Diagnosis of upper respiratory disease is initially based on the history and clinical signs and physical examination findings. Upper airway disease typically manifests as an obstructive breathing pattern, which is relatively slow and deep compared with a rapid and shallow restrictive breathing pattern (Fig. 28.1). Additional diagnostics can include hematologic and serum biochemical parameters, blood gas analysis, oximetry, cervical and thoracic radiographs, fluoroscopy, endoscopy, cytologic studies, culture, and/or biopsy. The history and clinical signs may include abnormal respiratory noises (e.g., cough, inspiratory stridor, wheezing), exercise intolerance, hyperthermia, tachypnea, respiratory distress, cyanosis, restlessness, and/or collapse. Gagging and regurgitation of secretions are common with nasopharyngeal, laryngeal, and some tracheal abnormalities. Mucopurulent or bloody nasal discharges are common with obstructive or infectious nasal disease. Voice change may occur with laryngeal paralysis, and dysphagia may be noted with supraglottic obstruction. Subcutaneous (SC) emphysema occurs with penetrating laryngotracheal or nasal injury. Clinical signs may intensify or may be precipitated by excitement, stress, eating, drinking, or high ambient temperatures. Laboratory data should be evaluated to detect underlying metabolic disease and determine the advisability of general anesthesia.

Animals with nasal neoplasia, fungal infection, or foreign bodies may become anemic from epistaxis. Affected animals should be carefully evaluated for clotting abnormalities by assessment of platelet numbers, bleeding from venipuncture sites, or the presence of ecchymosis, petechiation, melena, hematuria, or retinal hemorrhage. If available, coagulative ability may be assessed by determining the activated clotting time, prothrombin time, partial thromboplastin time, and/or mucosal bleeding time. Blood transfusions should be considered before surgery if the packed cell volume is 20% or lower (see Box 4.1). Bleeding during rhinotomy may be severe, requiring intraoperative blood transfusion, carotid artery ligation, or both.

A single, preoperative antiinflammatory dose of a glucocorticoid (e.g., dexamethasone sodium phosphate 0.1–0.5 mg/kg IV) may reduce nasopharyngeal and upper airway edema secondary to surgical or diagnostic manipulations. Start with the lower dose and use the higher dose only if the low dose has failed to reduce swelling and if swelling is sufficiently severe that the animal cannot breathe. Large doses or repeated administration of glucocorticoids sometimes cause gastrointestinal erosion/ulceration, with or without hemorrhage. Glucocorticoids are routinely given for nasopharyngeal and intraluminal laryngeal procedures, provided no contraindications for administration are known.

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ABSTRACT

The upper respiratory system is composed of the nasal cavity, nasopharynx, larynx, and cervical trachea. Surgery in these areas is primarily used to relieve obstruction. The most common causes of upper airway obstruction are brachycephalic obstructive airway syndrome, laryngeal paralysis, and tracheal collapse; however, trauma and neoplasia should also be considered. Characteristic clinical signs of upper airway disease include nasal stertor and inspiratory stridor. Diagnosis is dependent on structural and functional pharyngeal and laryngeal examination, with treatment directed at removal or bypass of the obstruction.

KEYWORDS

nasal cavity
nasopharynx
larynx
cervical trachea
brachycephalic syndrome
laryngeal paralysis
tracheal collapse
rhinotomy
tracheostomy
stenotic nares
elongated soft palate
laryngeal saccule eversion
laryngeal collapse
epiglottic retroversion
nasal aspergillosis

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BOX 28.1 Indications for Upper Respiratory Tract Surgery

Brachycephalic syndrome
Devocalization
Laryngeal collapse
Laryngotracheal trauma
Laryngeal paralysis
Tracheal collapse
Laryngeal masses
Tracheal masses
Nasal masses or infection
Nasal trauma
Foreign bodies
Congenital abnormalities

ANESTHESIA

Patients with upper respiratory obstruction or disruption are extreme anesthetic risks. The periods of greatest danger are during induction of anesthesia and during recovery (see discussion under Postoperative Care and Assessment). For laryngeal examination, avoid drugs that inhibit laryngeal function. If the animal has already been sedated (see previous discussion), an anticholinergic drug (atropine or glycopyrrolate; Table 28.1) should be given if the patient has increased oral secretions or bradycardia. Preoxygenating the patient for 3 to 5 minutes before induction is recommended. If the procedure will be more extensive than a laryngeal examination, an opioid (e.g., hydromorphone, butorphanol, buprenorphine) may also be administered at induction (butorphanol may be preferred as it is associated with less panting and is antiemetic). Propofol is recommended for induction because

BOX 28.2 Sedation of Severely Dyspneic Dogs

1. Reconsider necessity for sedating a severely dyspneic patient.
2. Give oxygen by either flow-by or nasal insufflation.
3. Provide a calm, quiet, well-ventilated room that is neither hot nor humid. Depending on the breed, a cooler temperature may be required.
4. Allow the dog to assume a posture that it finds most conducive for breathing. If the dog struggles, back off and let the patient calm down.
5. Have intubating equipment available and checked prior to giving any sedative. Don't hesitate to intubate if respiratory distress doesn't improve or worsens.
6. Sedation:
   - Butorphanol, 0.2–0.4 mg/kg IV, IM, SC, *or*
   - Midazolam, 0.1–0.2 mg/kg IV, IM, *or*
   - Diazepam, 0.1–0.2 mg/kg IV

IM, Intramuscular; *IV,* intravenous; *SC,* subcutaneous.

BOX 28.3 Sedation of Severely Dyspneic Cats

1. Reconsider necessity for sedating a severely dyspneic patient.
2. Give flow-by oxygen.
3. Provide a calm, quiet, well-ventilated room that is not humid.
4. Allow the cat to assume a posture that it finds most comfortable. Be very careful to avoid overrestraining a cat. Do not hold the patient in any way that could compromise thoracic excursions. If the cat struggles, back off and let it calm down.
5. Have intubating equipment available and checked prior to giving any sedative. Don't hesitate to intubate if respiratory distress doesn't improve or worsens.
6. Benzodiazepines may cause an anxious, dyspneic cat to be more difficult to handle.
7. Sedation:
   - Butorphanol, 0.2–0.4 mg/kg IV, IM, SC, *or*
   - Buprenorphine, 0.005–0.02 mg/kg IV, IM, SC

IM, Intramuscular; *IV,* intravenous; *SC,* subcutaneous.

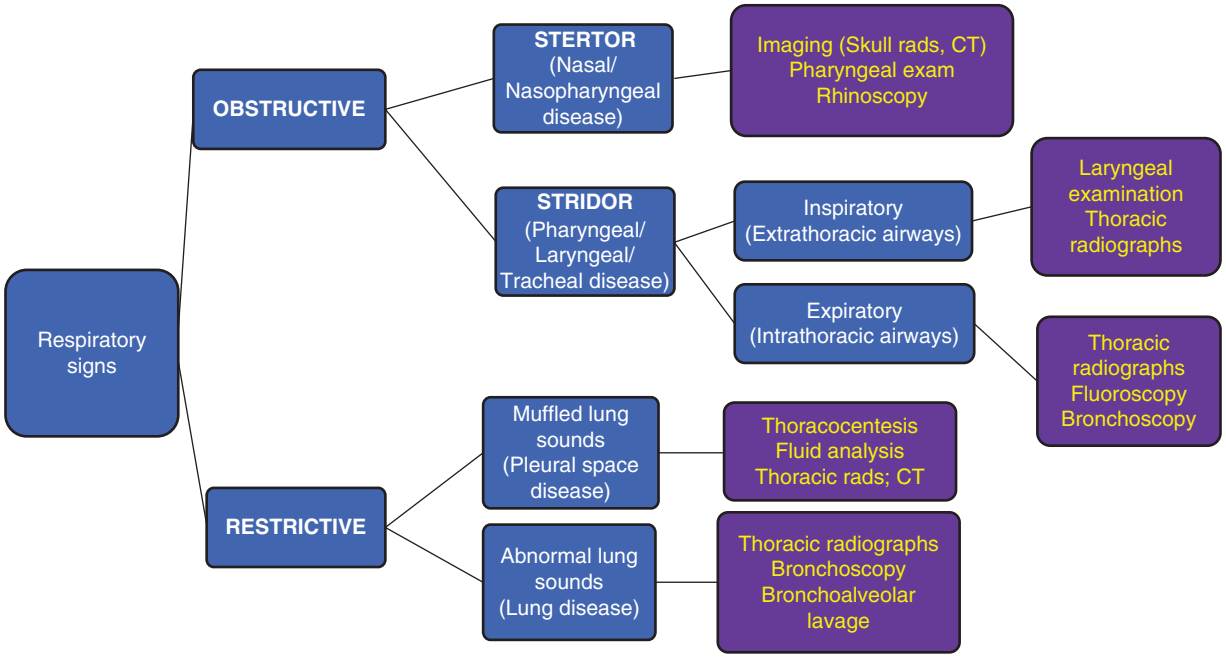


FIG. 28.1 Algorithm for diagnosis of respiratory distress. *CT,* Computed tomography.

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TABLE 28.1 Anesthetic Considerations for Upper Airway Examination

Preoperative Considerations	
Associated conditions	• Affected brachycephalic breeds are often young and otherwise healthy • Patients with nasal or sinus masses or collapsing tracheas are often older patients with comorbidities • May be obese
Bloodwork	• HCT • TP • CBC and chemistry profile in older patients
Physical examination	• Stridorous breathing • Open-mouth breathing or panting • Increased salivation • Restlessness and anxiousness • May have an orthopneic posture • May be easily stressed and become extremely dyspneic with minimal activity
Other diagnostics	• SpO_2 • Blood pressure • +/– ECG • +/– Radiographs, thoracic/cervical

Premedications	• Oxygen with flow-by or face mask
	• Avoid sedation in severely dyspneic patients
	• Glycopyrrolate (0.005–0.01 mg/kg IV, IM, SC), or atropine (0.04 mg/kg IM, SC or 0.02 mg/kg IV) *plus*
	• Dexamethasone (0.5–2 mg/kg IV, IM, SC); start with the lower dose and use the higher dose only if the low dose has failed to reduce swelling, and if swelling is sufficiently severe to impair ventilation, *plus*
	• In anxious patients, give:
	  • Midazolam (0.1–0.2 mg/kg IV, IM), *or*
	  • Diazepam[a] (0.1–0.2 mg/kg IV), *or*
	  • Butorphanol (0.2–0.4 mg/kg IV, IM, SC)
	• Before induction in extremely dyspneic patients, avoid respiratory depressants, including but not limited to xylazine, medetomidine, and dexmedetomidine. Avoid long-acting drugs such as acepromazine.

Intraoperative Considerations	May need oxygen support until fully recovered
Induction	• Preoxygenate for 3–5 min with face mask or flow-by oxygen • If normal cardiac function, give propofol (4–8 mg/kg IV) slowly to maintain spontaneous ventilation, *or* • If decreased cardiac function, give etomidate (0.5–1.5 mg/kg IV) Note: give a benzodiazepine (e.g., midazolam, diazepam) before giving etomidate

Maintenance	• Perform examination as soon as patient is sufficiently anesthetized to allow the mouth to be opened. Monitor SpO_2 and intubate when examination is over or when SpO_2 drops to <95%.
	• Use isoflurane or sevoflurane if needed
	• Ventilation parameters:
	  • SpO_2 >95%
	  • Respiratory rate 10–20/min
	  • Peak airway pressures <20–25 mm Hg (when intubated assess $EtCO_2$)

Fluid needs	• Often not necessary in these patients
Monitoring	• BP • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature if anesthesia longer than 30 minutes
Postoperative Considerations	May need oxygen support until fully recovered
Analgesia	• If procedure is only an examination under anesthesia, postoperative analgesia is not required
Monitoring	• SpO_2 • HR • Respiratory rate
Estimated pain score	None

[a]For a short and minimally painful procedure, midazolam is a better choice than diazepam because of the much longer half-lives of diazepam and its active metabolite.
BP, Blood pressure; *CBC,* complete blood cell count; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *SC,* subcutaneous; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein.

it has a rapid onset of action and is short acting. It should be administered in small incremental doses to attempt to maintain laryngeal function (Table 28.2). Even if laryngeal function is temporarily depressed with a rapid bolus of propofol, return to function usually occurs within several minutes. Meanwhile the patient can be intubated and given oxygen, and another look in the oropharynx can take place in a few minutes. In patients that have less respiratory compromise, combinations such as diazepam and ketamine or fentanyl and diazepam may be used for induction as these drugs maintain laryngeal function. Doxapram (Dopram, 1–2.2 mg/kg IV) administration during laryngeal examination increases intrinsic laryngeal motion and helps to differentiate

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normal dogs from dogs with functional laryngeal disease. Oxygen should be supplemented during the examination, and oxygen saturation should be monitored with pulse oximetry and by observation of mucous membrane color. As soon as the laryngeal examination is complete, the patient should be intubated and anesthesia maintained with an inhalant anesthetic and oxygen for additional diagnostics or surgery, or to facilitate a smooth recovery.

NOTE To provide supplemental oxygen during the laryngeal exam, attach a red rubber catheter to the fresh gas outlet of the anesthesia machine with an oxygen flow of 2 to 3 L/min and tape it to the blade of the laryngoscope.

General anesthesia is preferred for most upper respiratory procedures because it ensures a patent airway, allows controlled ventilation, facilitates asepsis, and is less stressful for patients. Local anesthesia may allow placement of a tracheostomy tube when the patient is comatose, cannot tolerate general anesthesia, or cannot be intubated. Respiratory patients should be managed with extreme care until intubation has been accomplished and ventilation can be assisted. Premedication with any drug that causes hypoventilation is contraindicated in moderately to markedly distressed patients. In addition, every attempt should be made to minimize stress to the patient before induction. Pulse oximetry with a sensor that can be secured to the tail can be especially helpful with perioperative monitoring of the patient in respiratory distress. Before the time of induction, the anesthesia provider should be prepared with airway devices, an

TABLE 28.2 Anesthetic Considerations for Patients With Brachycephalic Syndrome

Preoperative Considerations

Associated conditions	• Often young and otherwise healthy • May be obese
Bloodwork	• HCT • TP
Physical examination	• Stridorous breathing • Open-mouth breathing or panting • Increased salivation • Restlessness and anxiety • May have an orthopneic posture • May be easily stressed and become extremely dyspneic with minimal activity
Other diagnostics	• SpO_2 • Blood pressure • +/– Radiographs, thoracic

Premedications	• Oxygen with flow-by or face mask
	• Avoid sedation in severely dyspneic patients
	• Glycopyrrolate (0.005–0.01 mg/kg IV, IM, SC) or atropine (0.04 mg/kg IM, SC or 0.02 mg/kg IV), *plus*
	• Dexamethasone (0.5–2 mg/kg IV, IM, SC); start with the lower dose and use the higher dose only if the low dose has failed to reduce swelling, and if swelling is sufficiently severe to impair ventilation, *plus*
	• In anxious patients, give:
	  • Midazolam (0.1–0.2 mg/kg IV, IM), *or*
	  • Diazepam[a] (0.1–0.2 mg/kg IV), *or*
	  • Butorphanol (0.2–0.4 mg/kg IV, IM, SC) *or*
	  • Buprenorphine[b] (0.005–0.015 mg/kg IV, IM, SC)
	Before induction in extremely dyspneic patients, avoid respiratory depressants, including but not limited to xylazine, medetomidine, and dexmedetomidine.

Intraoperative Considerations

Induction	• Preoxygenate for 3–5 min with face mask or flow-by oxygen • Propofol (4–8 mg/kg IV) given rapidly to expedite intubation and manual/mechanical ventilation

Maintenance	• Isoflurane or sevoflurane, *plus*
	  • Fentanyl (2–10 µg/kg IV PRN in dogs and 1–4 µg/kg IV PRN in cats) for short-term pain relief, *plus*
	  • Hydromorphone[c] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Oxymorphone (0.1–0.2 mg/kg IV PRN), *or*
	  • Buprenorphine[b] (0.005–0.015 mg/kg IV PRN), *plus*
	  • Ketamine (low dose) (0.5–1 mg/kg IV)
	• Ventilation parameters:
	  • SpO_2 >95%
	  • Respiratory rate 10–20/min
	  • Peak airway pressures <20–25 mm Hg (when intubated assess $EtCO_2$)

Fluid needs	• 5–10 mL/kg/h
Monitoring	• BP • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature

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TABLE 28.2 Anesthetic Considerations for Patients With Brachycephalic Syndrome—cont'd

Postoperative Considerations	May need oxygen support until fully recovered

Analgesia	• Oxygen with flow-by or face mask if patient showing signs of sedation
	• Consider using NSAIDs as the main analgesic therapy to minimize potential for airway obstruction due to sedation
	• In dogs:
	  • Carprofen (2.2 mg/kg q12h PO), *or*
	  • Deracoxib (3–4 mg/kg q24h for <7 days PO), *or*
	  • Meloxicam (0.1–0.2 mg/kg once SC or PO, then 0.1 mg/kg PO q24h), *or*
	  • Tramadol (2–5 mg/kg PO q6–8h)
	• In cats:
	  • Meloxicam[d] (0.05–0.1 mg/kg SC or PO once), *or*
	  • Buprenorphine[b] (0.005–0.02 mg/kg IV, IM q4–8h or 0.01–0.02 mg/kg OTM q6–12h)
	• Butorphanol (0.2–0.4 mg/kg IV, IM q1–3h) if mild pain, *or*
	• Hydromorphone[c] (0.05–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats) only if moderate to severe pain is present, *or*
	• Oxymorphone (0.1–0.2 mg/kg IV, IM q2–4h), *plus*
	• Ketamine CRI (2 µg/kg/min IV. If no previous loading dose, give 0.5 mg/kg IV before CRI) if moderate to severe pain is present

Monitoring	• SpO_2 • HR • Respiratory rate • Temperature
Estimated pain score	Mild to moderate

[a]For a short and minimally painful procedure, midazolam is a better choice than diazepam because of the much longer half-lives of diazepam and its active metabolite.
[b]Buprenorphine is a better analgesic than morphine in cats.
[c]Monitor for hyperthermia in cats.
[d]Black box warning added by the Food and Drug Administration in October 2010 identified cases of renal failure and death in cats with repeated uses of meloxicam. Meloxicam is approved for single use only in cats in the United States.
BP, Blood pressure; *CRI,* constant-rate infusion; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *NSAID,* nonsteroidal antiinflammatory drug; *OTM,* oral transmucosal; *PO,* orally; *PRN,* as needed; *SC,* subcutaneous; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein.

anesthesia machine, monitors, and induction and emergency drugs. Preoxygenating the patient for 3 to 5 minutes usually provides adequate oxygen reserve for a safer intubation period. The animal should be allowed to remain in sternal recumbency, and oxygen provided by face mask, flow-by, or nasal insufflation if the animal will tolerate it (see Chapter 4). Induction should be rapid (e.g., propofol, alfaxalone, etomidate), and intubation should be performed immediately. Mask induction is not recommended. A benzodiazepine must be given prior to administering etomidate to avoid muscle rigidity (see Chapter 12). Anesthesia should be maintained with an inhalant anesthetic and oxygen. Laryngeal or tracheal procedures may require temporary retraction of the endotracheal tube from the surgical site, placement of an endotracheal tube distal to the surgical site through a tracheotomy, or use of injectable anesthetic drugs. When breathing spontaneously during surgery, the animal may be "sighed" (i.e., given an extra-large breath) every 5 minutes to reexpand distal airways. Oxygen saturation or blood gases (or both) should be monitored from induction until recovery and until abnormalities have been corrected. Selected anesthetic protocols are provided in Table 28.2.

ANTIBIOTICS

Because the respiratory tract has a normal bacterial flora, prophylactic antibiotics (e.g., cefazolin; Box 28.4) are frequently given before surgery. However, animals with normal immune function undergoing short procedures (e.g., nares resection, laryngeal saccule resection, vocal cordectomy) may not need them. Streptococci, *Escherichia coli, Pseudomonas* spp., *Klebsiella* spp., and *Bordetella bronchiseptica* are the bacteria most commonly isolated from normal dogs. A majority of tracheal cultures are sterile, whereas most pharyngeal cultures are not.

Gram-negative organisms that cause most canine respiratory tract infections are often resistant to commonly used antibiotics. Antimicrobial drug selection is best based on cytologic and culture results of tracheobronchial, pulmonary parenchymal, and/or pleural secretions. Bland aerosol therapy (e.g., sterile 0.9% saline) helps loosen secretions and facilitates their clearance in dogs with tracheostomies; addition of antibiotics to the aerosol is generally unnecessary. However, intratracheal or aerosolized antibiotics may be effective in some dogs with chronic respiratory infection. Lipid-soluble antibiotics that contain a benzene ring reach the highest levels in the normal trachea and bronchus; however, increased permeability associated with inflammation allows numerous antibiotics to achieve high levels during infection. Antibiotics commonly recommended for treatment of upper respiratory disease include ampicillin, fluoroquinolones, cephalosporins, doxycycline, azithromycin, and potentiated sulfonamides (see Box 28.4).

SURGICAL ANATOMY

The nasal cavity extends from the nostrils to the nasopharyngeal meatus and is separated into two halves by the *nasal septum* (Fig. 28.2). The septum is mostly cartilaginous but also has bony and membranous portions. The *nasal conchae* develop from the lateral and dorsal walls of the nasal cavity. Air passages between the conchae are known as the *meatus.* The *paranasal sinuses* include a maxillary recess, a frontal sinus, and a sphenoidal sinus. The *frontal sinus* occupies the supraorbital process of the frontal bone (see Fig. 28.2). The two sides are separated by a median

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BOX 28.4 Antibiotic Choices for Upper Respiratory Infection

Ampicillin
22 mg/kg IV, IM, SC q8h

Amoxicillin
22 mg/kg PO, IM q12h

Cefazolin (Ancef, Kefzol)
22 mg/kg IV, IM, SC q8h

Enrofloxacin (Baytril)[a]
Dogs: 7–20 mg/kg PO, IV (administer dilute solution slowly, over 30 min) q24h
Cats: 5 mg/kg PO q24h

Doxycycline
5 mg/kg PO, IV q12h[b]

Azithromycin (Zithromax)
Dogs: 5–10 mg/kg PO q24h or q12h for 5–20 d
Cats: 5–15 mg/kg PO q24–48h to q12h for 3–5 d

[a]Doses >5 mg/kg may be associated with blindness in cats.
[b]Do not administer with milk products. Be sure to have the patient drink or eat after receiving the medication; if the pill sticks in the esophagus, it can cause severe esophagitis.
IM, Intramuscular; *IV,* intravenous; *PO,* orally; *SC,* subcutaneous.

septum, and in dogs each side is divided into rostral, medial, and lateral compartments.

The *thyroid cartilage* forms the ventral and lateral walls of the larynx (Fig. 28.3). It surrounds the lateral aspect of the cricoid cartilage and articulates with it at its dorsolateral margin. Cranially, the thyrohyoid bones articulate with the thyroid cartilage, whereas ventrally, the cricothyroid ligament joins the caudal border of the thyroid cartilage to the cricoid cartilage. The *cricoid cartilage* is a complete ring that is five times wider dorsally than it is ventrally (see Fig. 28.3). It forms the dorsal wall of the larynx and cranially lies within the wings of the thyroid cartilage. The cricoid cartilage articulates at its cranial dorsolateral margin with the paired *arytenoid cartilages* (see Fig. 28.3). At the entrance to the glottis (laryngeal inlet), the arytenoid cartilages have two *cuneiform processes* ventrally and two corniculate processes dorsally. The *vocal folds* attach to the vocal process of the arytenoid cartilages at its ventral aspect. The muscular processes of the arytenoid cartilages are dorsolateral at the caudal aspect of the arytenoid.

The *glottis* (laryngeal inlet) consists of the vocal folds, the vocal processes of the arytenoid cartilages, and the rima glottidis (see Fig. 28.3). The vocal folds extend dorsally from the vocal processes of the arytenoids to the thyroid cartilage ventrally. Rostral and lateral to the vocal folds are the laryngeal saccules (ventricles). The laryngeal saccules are mucosal diverticula

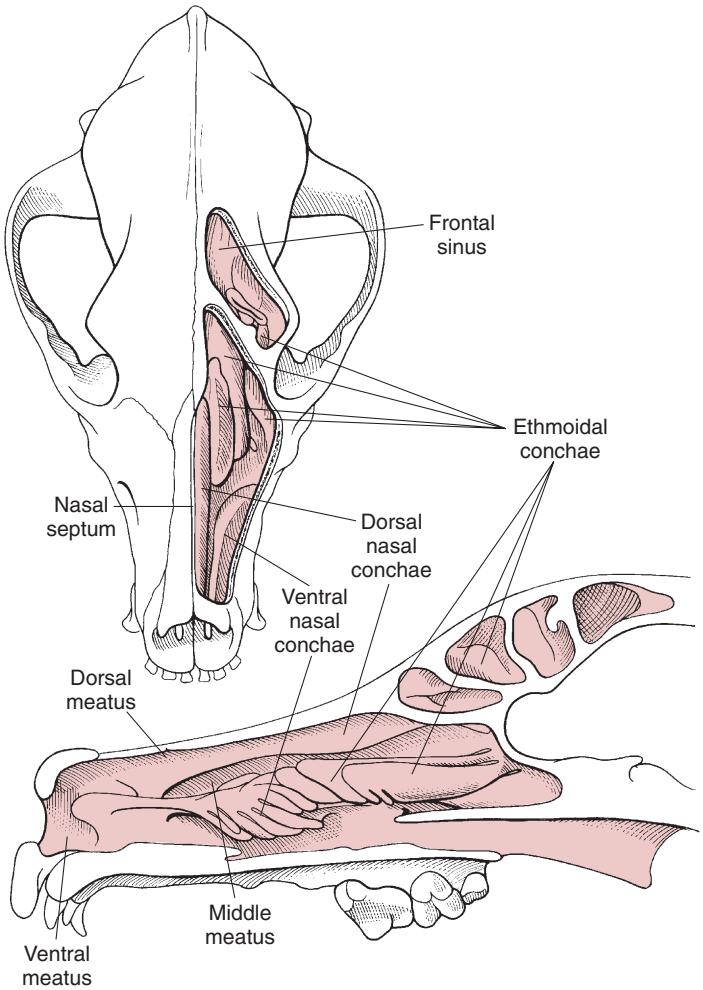


FIG. 28.2 Anatomy of the canine nasal cavity.

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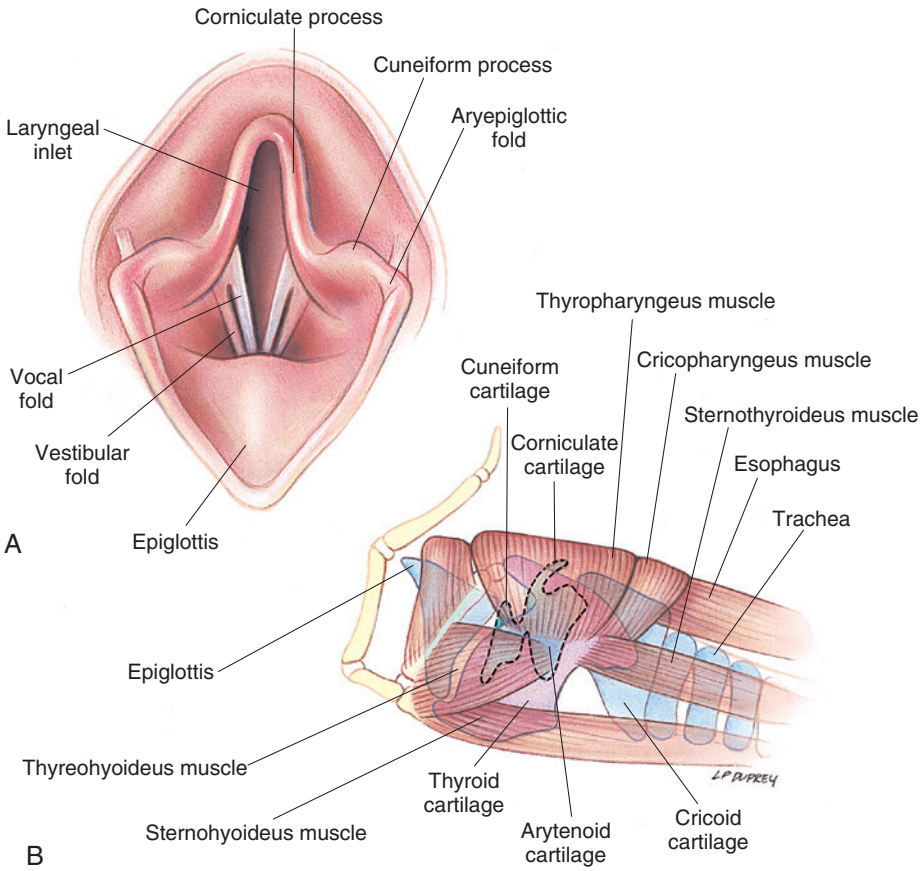


FIG. 28.3 Laryngeal anatomy. (A) Oral view. (B) Lateral view.

bounded laterally by the thyroid cartilage and medially by the arytenoid cartilage. The vestibular fold (false vocal cord) forms the rostral border of the laryngeal saccule and attaches to the cuneiform process.

The intrinsic muscles of the dog's larynx are innervated by somatic efferent axons from the vagus nerve. Some axons leave the vagus in the *cranial laryngeal nerve* to innervate the cricothyroid muscle; others provide sensory innervation to the mucosa. The *recurrent laryngeal nerve,* a branch of the vagus, terminates as the caudal laryngeal nerve, which innervates the remaining intrinsic muscles of the larynx. The *caudal laryngeal nerve* travels along the dorsolateral surface of the trachea and continues over the lateral surface of the cricoarytenoideus dorsalis before deviating to the medial surface of the thyroid cartilage lamina. The *cranial laryngeal artery,* a branch of the external carotid artery, travels with the cranial laryngeal nerve. It is the main blood supply to the larynx. The cranial laryngeal vein empties into the hyoid venous arch and then the external jugular vein. Lymphatics drain into the retropharyngeal lymph node.

The *trachea* is a semirigid, flexible tube that extends from the cricoid cartilage to the mainstem bronchi at approximately the fourth or fifth thoracic vertebra. Thirty-five to 45 incomplete C-shaped hyaline cartilages, joined by annular ligaments ventrally and laterally and by the trachealis muscle *(dorsal tracheal membrane)* dorsally, form the trachea. The tracheal vessels and nerves found in the lateral pedicles supply the trachea segmentally. Loose areolar connective tissue surrounds the trachea and forms the lateral pedicles. The cranial and caudal thyroid arteries and veins, the bronchoesophageal arteries and veins, and the internal jugular veins supply vascular branches to the trachea. Innervation is provided by the autonomic nervous system. Sympathetic fibers from the middle cervical ganglion and the sympathetic trunk inhibit tracheal muscle contraction and glandular secretions, whereas parasympathetic fibers from the vagus and recurrent laryngeal nerves cause tracheal muscle contraction and glandular secretions.

SURGICAL TECHNIQUE

Surgical techniques for the management of animals with upper respiratory disease include rhinotomy (see later), tracheotomy (see p. 842), tracheostomy (see p. 842), tracheal resection and anastomosis (see p. 844), ventriculocordectomy (see p. 845), tracheoplasty (see p. 871), arytenoid cartilage lateralization (see p. 861), partial laryngectomy (see p. 867), and surgery for stenotic nares (see p. 854), elongated soft palate (see p. 854), and everted laryngeal saccules (see p. 855).

Rhinotomy

The nasal cavity may be approached through dorsal, ventral, or lateral approaches. The dorsal approach is most commonly used for exploration and biopsy; however, the ventral approach can be used to explore the region caudal to the ethmoid turbinates and the ventral aspect of the turbinates. Lateral approaches are limited to lesions in the rostral aspect of the nasal cavity. Recently a combined rostrolateral approach (vestibulotomy) was described to address squamous cell carcinoma of the rostral nasal septum.[1]

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Dorsal Approach to the Nasal Cavity and Paranasal Sinuses

With the animal in ventral recumbency, make a dorsal midline skin incision from the caudal aspect of the nasal planum to the medial canthus of the orbit. Either or both sides of the nasal cavity can be entered through a single midline skin incision. *To explore the frontal sinus, extend the incision caudal to a line that connects the zygomatic processes of the frontal bone. Incise the SC tissue and periosteum on the midline. Elevate the periosteum and reflect it laterally on either or both sides of the nasal cavity. Use a bone saw to create and then elevate a flap of bone over the proposed site of entry into the nasal cavity (Fig. 28.4A). Save the bone flap (if healthy) and replace it after the nasal cavity has been explored. As an alternative, drill a hole to one side of the nasal septum with a Steinmann pin. Use rongeurs to enlarge the hole, and discard the bone fragments. If necessary, extend the bone removal bilaterally. Gently lavage the nasal passages and remove abnormal tissue. Submit tissues for histologic examination and culture. Use cautery, iced saline, and/or digital pressure to control hemorrhage. If continued hemorrhage is a problem, pack the nasal cavity with cotton gauze (see p. 848). If a bone flap was made, suture it in place with 3-0 or 4-0 nonabsorbable suture or 20- to 22-gauge wire placed through predrilled holes in the bone flap and adjacent bone (Fig. 28.4B). Do not use wire to replace the bone flap if radiation therapy is planned. Close the periosteum and SC tissues with absorbable suture material in a simple continuous pattern. Close the skin routinely. If rongeurs were used, close the periosteum and SC tissues, leaving a stoma at the caudal aspect of the incision. Close the skin similarly, leaving a stoma (se Fig. 28.4C).*

Ventral Approach to the Nasal Cavity

With the animal in dorsal recumbency (Fig. 28.5), make a midline incision in the hard palate. Elevate the mucoperiosteum of the hard palate laterally to the alveolar ridge. Be careful to spare the palatine nerves and vessels as they emerge from the major palatine foramen. Incise the mucoperiosteum and soft palate attachments to the caudal edge of the palatine bone, and extend the incision as far caudally as necessary into the soft palate (full thickness). Retract the edges of the incision with stay sutures. Remove the palatine bone with a power-driven burr or rongeurs and discard it (Fig. 28.6A). Explore the nasal cavity and remove abnormal tissues. Submit tissues for histologic examination and culture. Close the nasal mucosa of the soft palate with absorbable material in a simple continuous or simple interrupted pattern. Then close the submucosa-periosteum of the hard palate with absorbable suture in an interrupted pattern (Fig. 28.6B). Finally, close the oral mucosa of the hard and soft palates with monofilament nonabsorbable sutures in a simple continuous pattern.

Lateral Approach to the Rostral Nasal Cavity

This approach gives access to the nasal vestibule. Position the animal in lateral or dorsal recumbency.

Make an incision with a scalpel or Mayo scissors. When using scissors, insert one blade into the nostril and position it so the incision

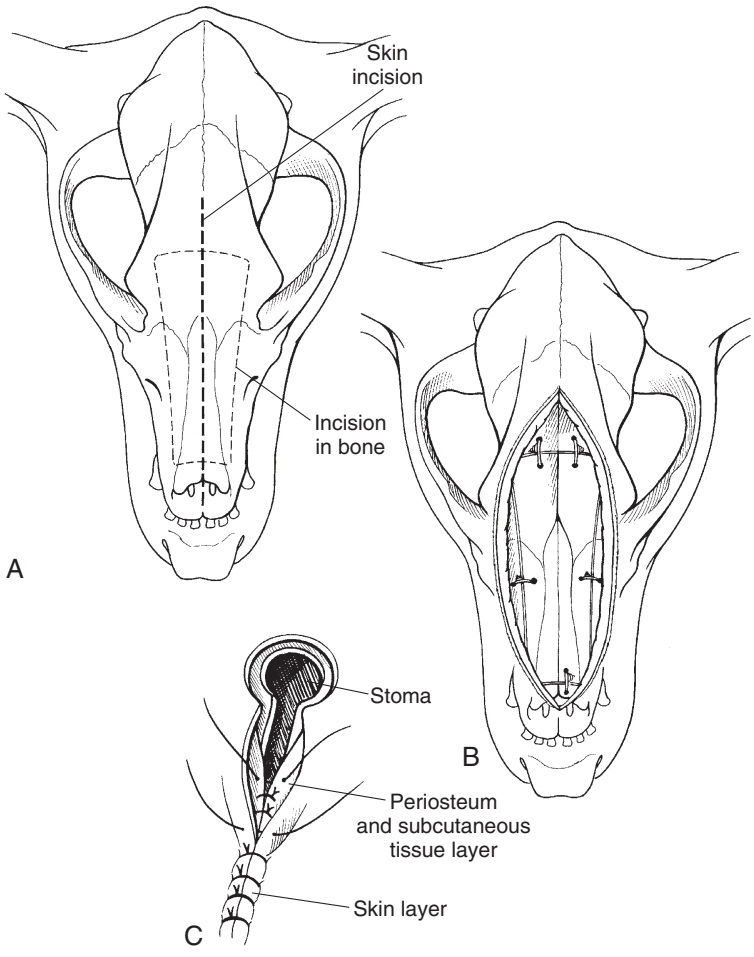


FIG. 28.4 Dorsal approach to the nasal cavity. (A) Make a skin incision from the caudal aspect of the nasal planum to the medial canthus of the orbit *(bold dashed line)*. Elevate a bone flap *(dashed rectangle)* or use rongeurs to remove bone. (B) If a bone flap was made, suture it in place with wire or sutures placed through predrilled holes in the bone flap and adjacent bone. (C) If rongeurs were used to remove bone, close the periosteum and subcutaneous tissue, leaving a stoma at the caudal aspect of the incision. Close the skin similarly, leaving a stoma.

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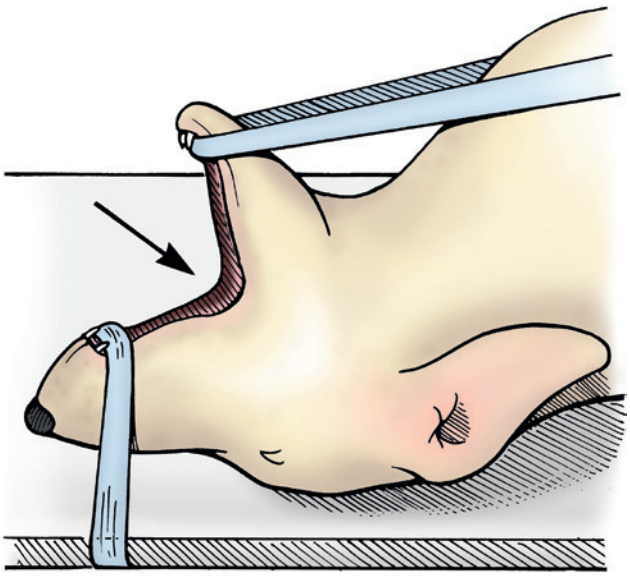


FIG. 28.5 Positioning for a ventral approach to the nasal cavity.

will be made ventral to the nasal planum and the dorsal lateral nasal cartilage (Fig. 28.7). Angle the incision in a dorsocaudal direction toward the nasomaxillary notch. Incise through all layers and retract the tissue dorsally to expose the vestibule. Explore and resect or biopsy abnormal tissue. Appose the nasal mucosa with 3-0 or 4-0 monofilament absorbable suture. Place two to four sutures in the musculocartilage layer (3-0 or 4-0 monofilament absorbable), and then reappose the skin (3-0 or 4-0 monofilament nonabsorbable).

Intraoral Approach to the Rostral Nasal Cavity

This approach has been used to remove foreign bodies and may be useful for removal or biopsy of rostral nasal masses. *Palpate the ridge along the rostrolateral aspect of the right (or left) nasal and incisive bone (Fig. 28.8). Incise the alveolar mucosa along this ridge from the nasal bone to the rostral end of the interincisive suture. Using a periosteal elevator, reflect the mucosa from these bones. Then retract the dorsal lateral nasal cartilage and the ventral lateral nasal cartilage medially. Penetrate the nasal mucosa and explore the rostral nasal cavity. Biopsy abnormal tissue and lavage the area. Close the gingival and buccal mucosa with 3-0 or 4-0 monofilament absorbable suture.*

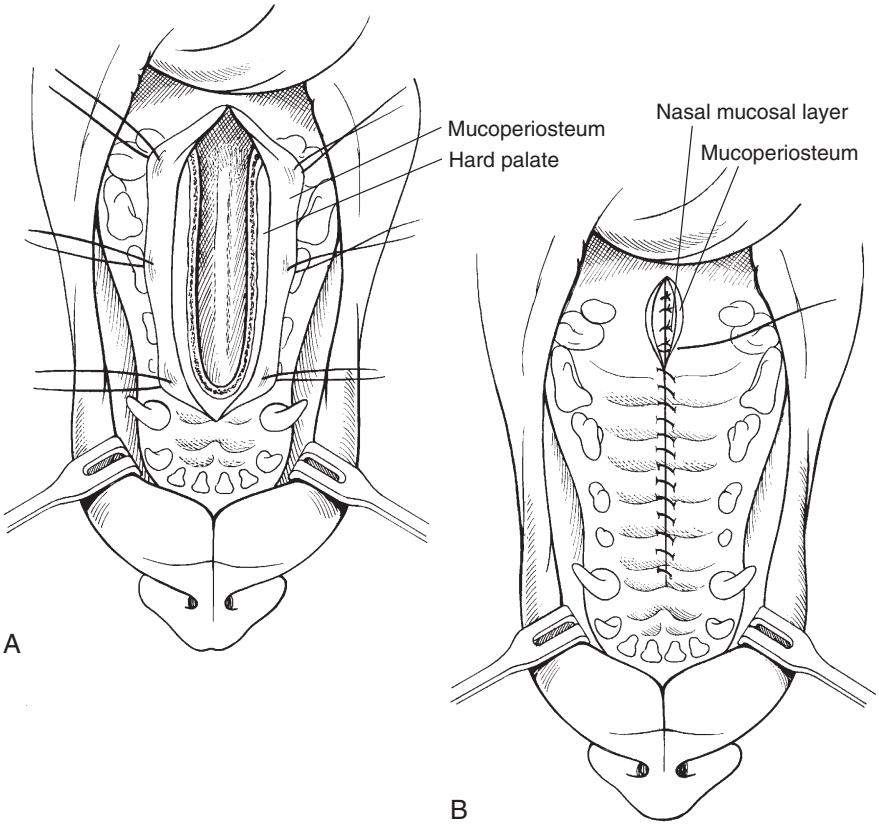


FIG. 28.6 Ventral approach to the nasal cavity. (A) Incise the mucoperiosteum of the hard palate. Remove the palatine bone with a power-driven burr or rongeurs, and discard it. (B) Close the nasal mucosa of the soft palate and the submucosa-periosteum of the hard palate with absorbable suture in an interrupted or continuous pattern. Close the oral mucosa of the hard and soft palates with monofilament, nonabsorbable suture in a simple continuous pattern.

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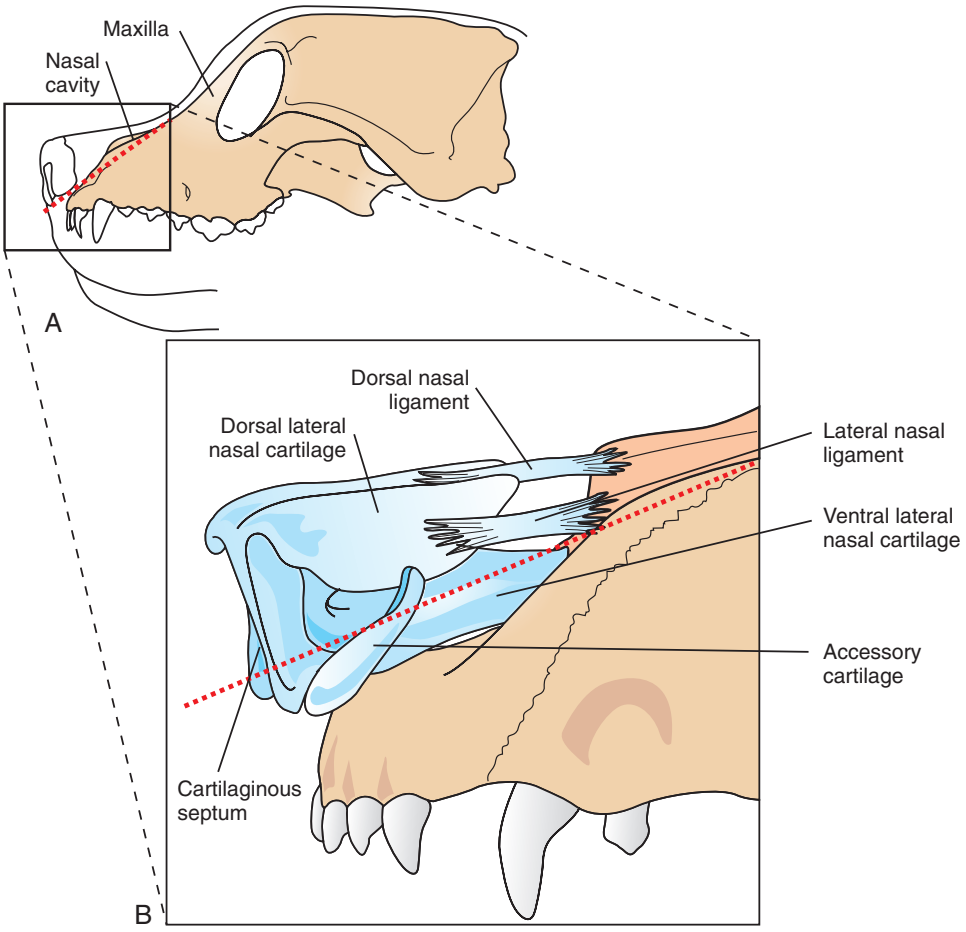


FIG. 28.7 Lateral approach to the rostral nasal cavity. (A) Make the incision for lateral rhinotomy in a dorsocaudal direction from the nasal planum toward the nasomaxillary notch. (B) Direct the incision between the dorsal lateral nasal cartilage and the ventral lateral nasal cartilage. Transection of the accessory cartilage cannot be avoided.

Tracheotomy

Tracheotomy is performed to gain access to the tracheal lumen to remove obstructions, collect specimens, or facilitate airflow. The tracheal incision may be closed or allowed to heal by secondary intention. Position the patient as described on p. 871.

Approach the cervical trachea through a ventral cervical midline incision. Extend the incision from the larynx to the sternum as needed to allow adequate exposure. Separate the sternohyoid muscles along their midline, and retract them laterally (Fig. 28.9A). Dissect the peritracheal connective tissue from the ventral surface of the trachea at the proposed tracheotomy site. Take care to prevent traumatizing the recurrent laryngeal nerves, carotid artery, vagosympathetic trunk, jugular vein, thyroid vessels, or esophagus. *Immobilize the trachea between the thumb and the forefinger. Make a horizontal or vertical incision through the wall of the trachea (see Fig. 28.9A). With a horizontal incision, place cartilage-encircling sutures (2-0 polypropylene [Prolene] or polybutester [Novafil]) around adjacent cartilages to separate the edges and allow lumen inspection or tube insertion. Suction blood, secretions, and debris from the tracheal lumen. After completion of the procedure, appose the tracheal edges with simple interrupted 3-0 or 4-0 polypropylene sutures or leave the trachea to heal by second intention. To close the tracheal incision, place sutures through the annular ligaments encircling adjacent cartilages or through the annular ligaments only. Lavage the surgical site with saline. Appose the sternohyoid muscles in a simple continuous pattern with 3-0 or 4-0 absorbable suture (e.g., polydioxanone [PDS], polyglyconate [Maxon], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], polyglactin 910 [Vicryl]). Appose SC tissues and skin routinely.*

Tracheostomy

Tracheostomy allows air to enter the trachea distal to the nose, mouth, nasopharynx, and larynx. A tracheotomy is performed to insert a tube (temporary tracheostomy) or create a stoma (permanent tracheostomy) to facilitate airflow. A nonreactive tube that is no larger than half the size of the trachea should be selected. Cuffed or cannulated autoclavable silicone, silver, or nylon tubes are recommended. In an emergency situation, a standard endotracheal tube can be used, but make sure the tube is not inserted too far into the respiratory tree and that the cuff is not inflated. Polyvinyl chloride and red rubber tubes are irritating and should be avoided. If the animal is to be placed on a ventilator, a cuffed tube is necessary.

Temporary Tracheostomy

A temporary tracheostomy is most commonly performed to provide an alternate airflow route during surgery or as an

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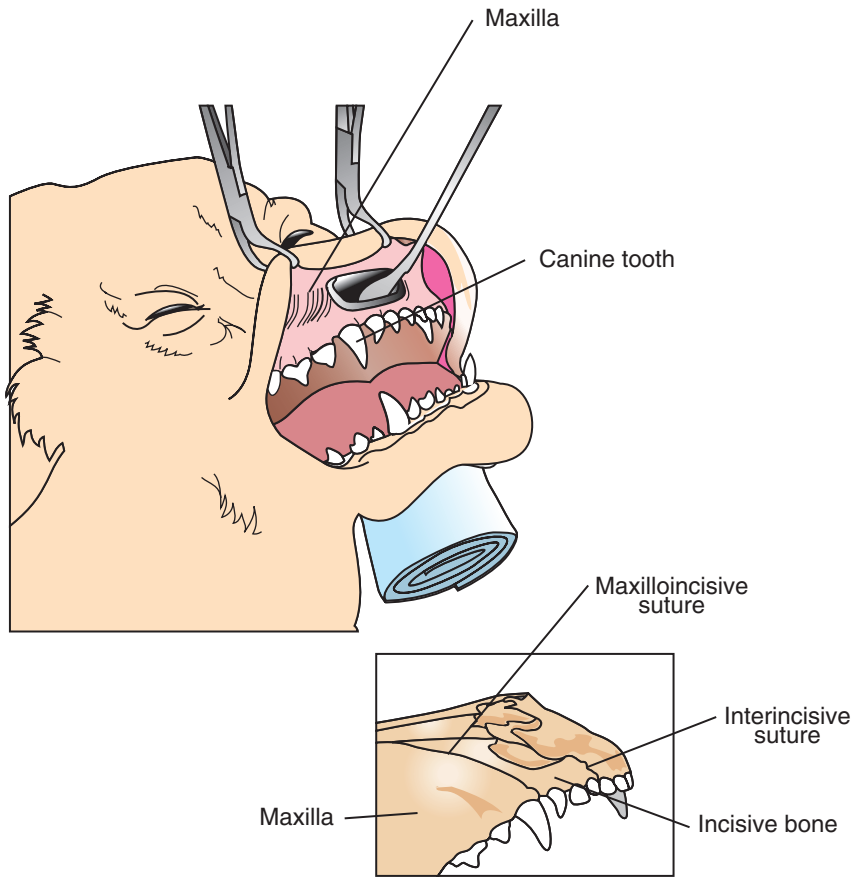


FIG. 28.8 Oral approach to the rostral nasal cavity. Elevate the lip and incise along the ridge at the rostrolateral aspect of the right (or left) nasal bone to the rostral end of the interincisive suture. Using a periosteal elevator, reflect the mucosa from these bones. Retract the dorsal lateral nasal cartilage and the ventral lateral nasal cartilage medially. Close the gingival and buccal mucosae with 3-0 or 4-0 monofilament absorbable suture.

emergency procedure in severely dyspneic patients. Tube tracheostomies are usually maintained for only a short time (12–72 hours).

Make a ventral midline incision from the cricoid cartilage extending 2 to 3 cm caudally. Separate the sternohyoid muscles and make a horizontal (transverse) tracheotomy through the annular ligament between the third and fourth or fourth and fifth tracheal cartilages (see Fig. 28.9A). Do not extend the incision around more than half the circumference of the trachea. As an alternative, make a vertical tracheotomy across the ventral midline of the third through fifth cartilages. *Place cartilage-encircling sutures (2-0 polypropylene [Prolene] or polybutester [Novafil]) around adjacent cartilages to separate the edges and allow for tube insertion. Suction blood and mucus from the lumen, widen the incision if necessary, and insert the tracheostomy tube. Facilitate tube placement by opening a hemostat in the incision, or depress the cartilages cranial to the horizontal incision (Fig. 28.9B). Alternatively, place tension on the caudal suture to open the incision (Fig. 28.9C). Appose the sternohyoid muscles, SC tissue, and skin cranial and caudal to the tube. Secure the tube by suturing it to the skin or tying it to gauze that is tied around the neck.*

Permanent Tracheostomy

Permanent tracheostomy is the creation of a stoma in the ventral tracheal wall by suturing tracheal mucosa to skin. Tracheostomas are maintained for life, or until the stoma is surgically closed. Tracheostomy tubes are not needed to maintain lumen patency after this procedure. Permanent tracheostomies are recommended for animals with upper respiratory obstruction causing moderate to severe respiratory distress (e.g., laryngeal paralysis, laryngeal collapse, upper airway neoplasia) that cannot be successfully treated by other methods. Owners should be warned that these animals must be restricted from swimming, that vocalization is diminished or absent after this procedure, and the patient is at increased risk for respiratory infections. Furthermore, ongoing care of the site will be necessary to keep it clean.

Expose the proximal cervical trachea with a ventral cervical midline incision. Create a tunnel dorsal to the trachea in the area of the third to sixth tracheal cartilages. Take care to prevent traumatizing the recurrent laryngeal nerves, carotid artery, vagosympathetic trunk, jugular vein, thyroid vessels, or esophagus. Using this tunnel, appose the sternohyoid muscles dorsal to the trachea with horizontal mattress sutures (2-0 or 3-0 nonabsorbable monofilament suture) to create a muscle sling to reduce tension on the mucosa-to-skin sutures (Fig. 28.10A). Beginning with the second or third tracheal cartilages, outline a rectangular segment of tracheal wall three to four cartilage widths long and one-third the circumference of the trachea in width. Incise the cartilage and annular ligaments to the depth of the tracheal mucosa (Fig. 28.10A). Elevate a cartilage edge with thumb forceps and dissect the cartilage segment

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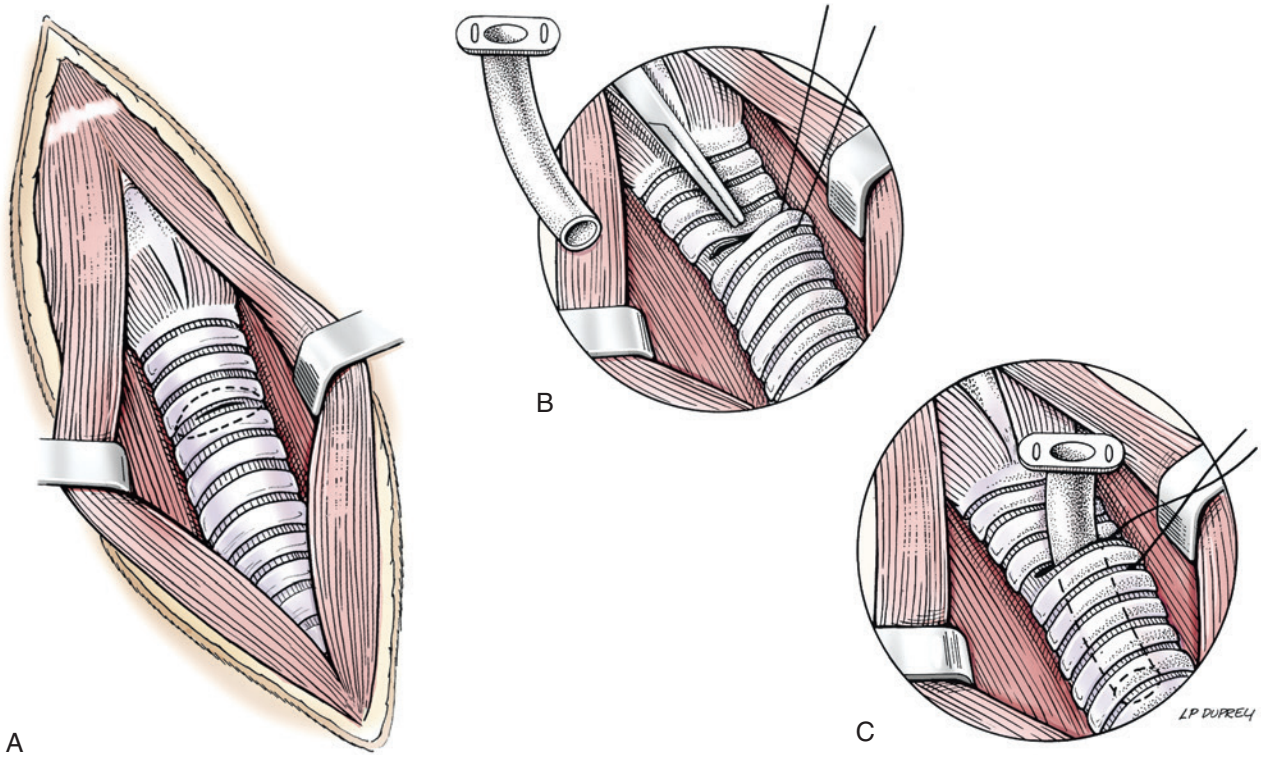


FIG. 28.9 Tube tracheostomy. (A) Make a transverse incision through the annular ligament. Excise a small ellipse of cartilage from each tracheal cartilage adjacent to the tracheotomy incision to minimize tube irritation *(dotted line)*. Facilitate tube placement by (B) depressing the proximal cartilages with a hemostat and (C) elevating the distal cartilages with an encircling suture. Insert a tracheostomy tube that does not completely fill the lumen.

from the mucosa. If there is concern that the tracheal cartilages are weak and may have a tendency to collapse, place one or two prosthetic tracheal rings cranial and caudal to the stoma (see p. 871). Excise a similar segment of skin adjacent to the stoma (excise larger segments of skin if the animal has loose skin folds or abundant SC fat). Make an I- or H-shaped incision in the mucosa. Fold the mucosa over the cartilage edges and suture it to the edges of the skin with approximating sutures to complete the tracheostoma (Fig. 28.10B). Use simple interrupted sutures at the corners and a simple continuous pattern to further appose skin and mucosa (4-0 monofilament suture) (Fig. 28.10B). Alternatively, make a full-thickness H-shaped incision in the ventral trachea to create two tracheal flaps that are reflected cranially and caudally and sutured directly to the skin.

Tracheal Resection and Anastomosis

Removal of a tracheal segment may be necessary to treat tracheal tumor, stenosis, avulsion, or trauma. Depending on the extent of injury, tears in the tracheal wall that occur as a consequence of bite wounds or endotracheal intubation may be allowed to close spontaneously, may be closed primarily, or may be resected and the tracheal ends anastomosed. Depending on the degree of tracheal elasticity and tension, approximately 20% to 50% of the trachea in an adult dog (approximately 8–10 rings) may be resected and direct anastomosis achieved. The split-cartilage technique is preferred because it is easier to perform and results in more precise anatomic alignment with less luminal stenosis than many other techniques. Imprecise anastomosis and tension across the suture site are considerable risk factors in the development of tracheal stenosis. Accurate and meticulous surgical technique is crucial for reconstruction of the trachea. Diseased trachea that exceeds the limits of resection and anastomosis may be managed with permanent tracheostomy, intraluminal silicone tubes, grafts, or prostheses with variable success.

Expose the involved trachea through a ventral cervical midline incision, lateral thoracotomy (see p. 890), or median sternotomy (see p. 891). Mobilize only enough trachea to allow anastomosis without tension. Preserve as much of the segmental blood and nerve supply to the trachea as possible. Place stay sutures around cartilages cranial and caudal to the resection sites before transecting the trachea. Resect the diseased trachea by splitting a healthy cartilage circumferentially at each end, or by incising annular ligaments adjacent to the intact cartilages (Fig. 28.11A). Use a No. 11 blade to split the tracheal cartilages at their midpoint. Transect the dorsal tracheal membrane with Metzenbaum scissors. Preplace and then tie three or four simple interrupted sutures (3-0 or 4-0 polypropylene [Prolene], polybutester [Novafil], polydioxanone [PDS], or polyglyconate [Maxon]) in the dorsal tracheal membrane (Fig. 28.11B). Retract the endotracheal tube into the proximal trachea during resection and placement of sutures in the dorsal tracheal membrane. Remove blood clots and secretions from the lumen, and advance the tube distal to the anastomosis after placing dorsal tracheal membrane sutures. Complete the anastomosis by apposing the split cartilage halves or adjacent intact cartilages with simple interrupted sutures beginning at the ventral midpoint of the trachea. Space additional sutures 2 to 3 mm apart. Place three or four retention sutures to help relieve tension on the anastomosis. Place and tie these sutures so that they encircle an intact cartilage cranial and

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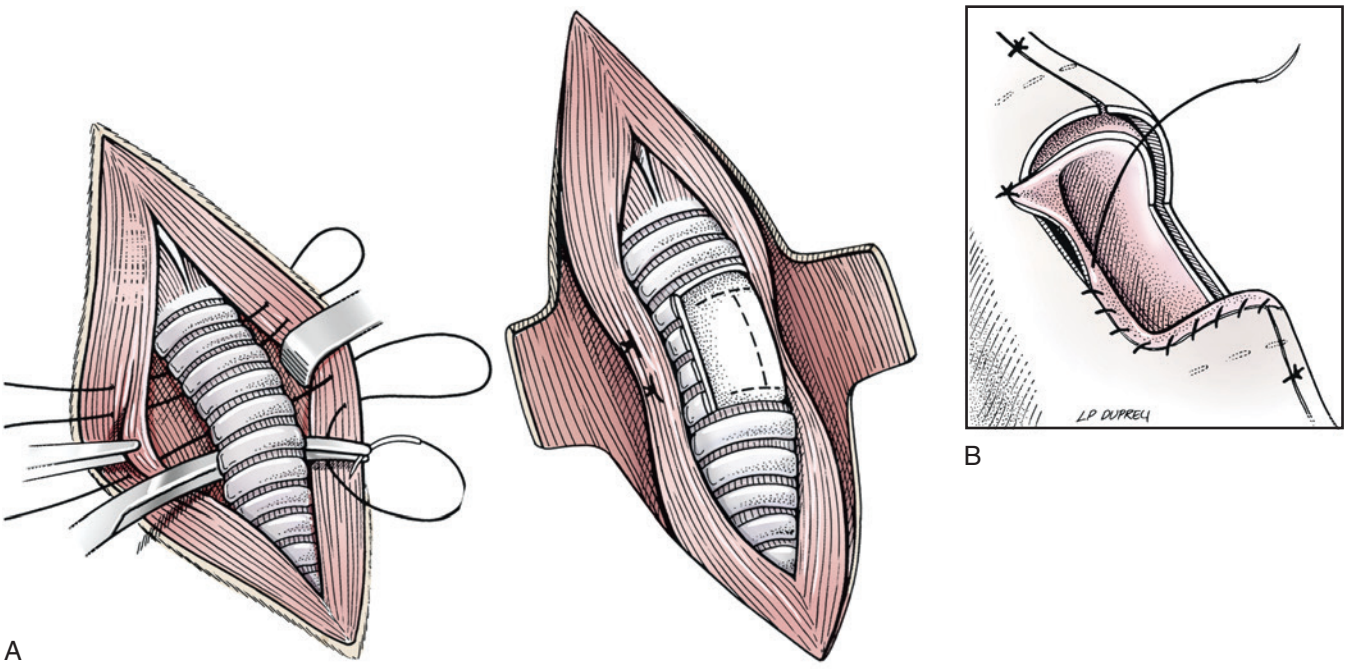


FIG. 28.10 Permanent tracheostomy. (A) Deviate the trachea ventrally by apposing the sternohyoid muscles with mattress sutures dorsal to the trachea. Excise a rectangular segment of ventral tracheal wall without penetrating the mucosa. Note the dotted line where the I-shaped incision is made after the cartilage segment is removed. Excise loose skin adjacent to the stoma. (B) Use intradermal sutures to appose the skin to the annular ligaments and peritracheal tissues *(dashed lines)*. Appose the tracheal mucosa to the skin with three or four interrupted sutures; complete the closure in a simple continuous pattern.

caudal to the anastomosis, crossing external to the anastomotic site (Fig. 28.11C). Lavage the area and appose the sternohyoid muscles in a simple continuous pattern. Close the SC tissues and skin routinely. Alternatively, consider a simple continuous pattern for anastomosis as it has been shown to have similar biomechanical strength to a simple interrupted pattern augmented with encircling sutures in the neighboring cartilage rings.

If tension-relieving sutures do not adequately relieve tension at the anastomosis, further mobilize the trachea by making partial-thickness incisions through annular ligaments proximal and distal to the anastomosis, or restrict head and neck movement after surgery. Prevent full extension of the neck by placing a suture from the chin to the manubrium or by fixing a muzzle to a harness to maintain mild to moderate cervical flexion. Maintain the muzzle for 2 to 3 weeks.

Ventriculocordectomy

Ventriculocordectomy is removal of the vocal cords to alter vocalization, remove masses, or enlarge the ventral glottis for dogs with laryngeal paralysis. The policy of the American Veterinary Medical Association regarding canine devocalization is that it should only be performed by qualified, licensed veterinarians as a final alternative to euthanasia after behavioral modification to correct excessive vocalization has failed and after discussion of potential complications from the procedure with the owner. Ventriculocordectomy may be performed through an oral or ventral (laryngotomy) approach. The ventral approach is recommended. Anesthesia is maintained by using a tube tracheostomy, manipulating the endotracheal tube to the contralateral side of the larynx, or using injectable anesthetic agents. Ventriculocordectomy performed to widen the ventral glottis requires that more vocal fold be resected than is required for debarking.

Oral Approach

Position the patient in ventral recumbency with the neck extended. Suspend the maxilla and pull the mandible ventrally to maximally open the mouth. Extend the tongue from the mouth to get maximum exposure of the glottis. Retract the cheeks laterally to improve visualization. Avoid placing padding or hands in the region of the larynx because this may distort the nasopharynx. Remove the central margin of the vocal cord for debarking with a laryngeal or uterine cup biopsy forceps (Fig. 28.12A). To widen the glottis, use long-handled Metzenbaum scissors and remove as much of the vocal fold extending into the laryngeal lumen as possible (Fig. 28.12B). With either technique, maintain 1 to 2 mm of mucosa at the dorsal and ventral aspects of the vocal cord. *Control hemorrhage with pressure. Remove blood clots and secretions with suction or sponges. Allow the incision to heal by secondary intention.*

Ventral Laryngotomy Approach

Position the patient in dorsal recumbency with the neck extended over a rolled towel (Fig. 28.13). Expose the larynx using a ventral midline cervical approach, beginning rostral to the basihyoid bone and extending caudally to the proximal trachea. Separate and retract the paired sternohyoid muscles. Identify the midline of the thyroid cartilage. Ligate and divide the laryngeal impar vein if necessary. Incise the cricothyroid ligament with a No. 15 or No. 11 blade. Extend the incision along the midline of the thyroid cartilage as

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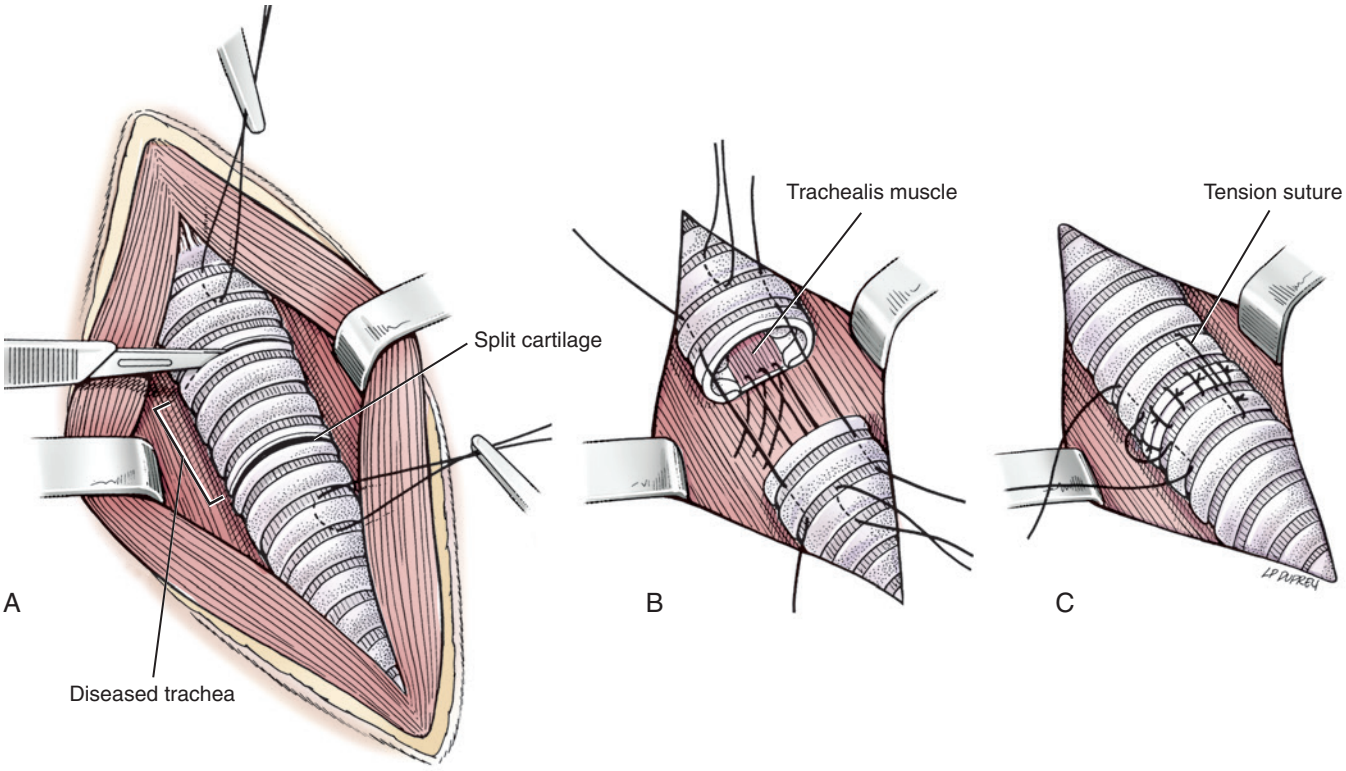


FIG. 28.11 Tracheal resection and anastomosis. (A) Place stay sutures cranial and caudal to the resection sites. Split the cartilages with a No. 11 blade, and transect the trachealis muscle with Metzenbaum scissors. (B) Appose the trachealis muscle with three or four interrupted sutures, then approximate the split cartilages. (C) Place three or four tension-relieving sutures around cartilages adjacent to the anastomosis.

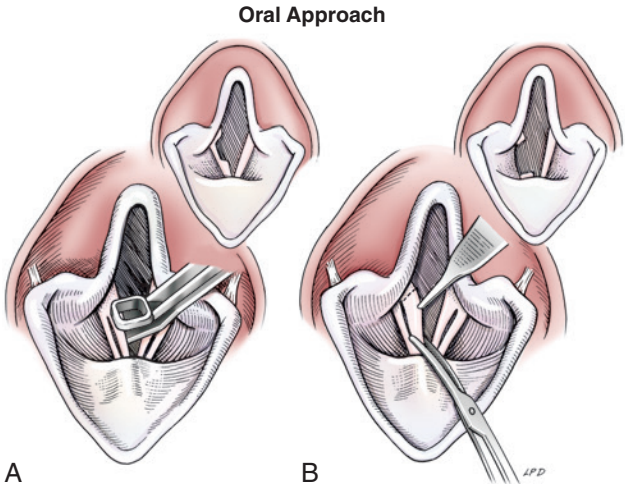


FIG. 28.12 For ventriculocordectomy an oral approach may be used. (A) Remove the central portion of the vocal fold with laryngeal cup forceps or uterine biopsy forceps. (B) For laryngeal paralysis or devocalization, remove most of the vocal fold with Metzenbaum scissors. Dorsal and ventral commissures remain intact.

needed to expose the vocal folds. Excise the entire vocal fold from the arytenoid cartilage dorsally and the thyroid cartilage ventrally using Metzenbaum scissors (see Fig. 28.13). Close the defect by apposing the mucosa in a simple continuous appositional pattern using 4-0 to 5-0 rapidly absorbable monofilament suture (poliglecaprone 25 or glycomer 631). Appose the cricothyroid ligament and thyroid cartilage with simple interrupted sutures. Appose the sternohyoid muscles in a simple continuous pattern with 3-0 or 4-0 monofilament absorbable suture. Close the SC tissues and skin routinely.

HEALING OF THE RESPIRATORY TRACT

Laryngeal wounds heal by reepithelialization if mucosal edges are in apposition. Epithelial cells at the wound margins extend and spread over the wound until it is covered. Constant motion associated with breathing and head movement inhibits primary healing. Laryngeal wounds with gaps heal by secondary intention, first filling with granulation tissue and then reepithelializing. Secondary intention healing may cause scarring across the glottis. Restricting surgery to one side of the larynx and leaving epithelium at the dorsal and ventral commissures intact may prevent scarring.

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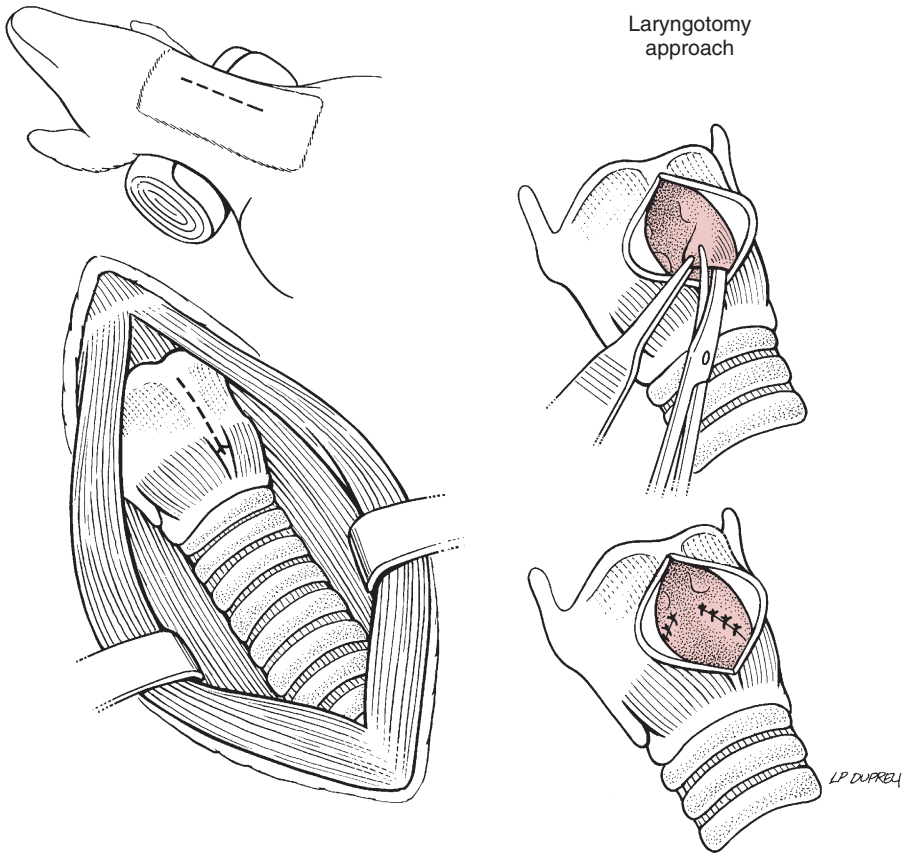


FIG. 28.13 For ventriculocordectomy by a laryngotomy approach, position the patient in dorsal recumbency with the neck extended over a rolled towel. Expose the larynx, identify the midline of the thyroid cartilage and the cricothyroid ligament, and then incise with a scalpel blade *(dashed lines)*. Expose the vocal folds and excise them. Close the defect by apposing the mucosa in a simple continuous appositional suture pattern.

Tracheal epithelium responds immediately to irritation or disease by increased production of mucus. If the insult continues, cells desquamate and goblet cell hyperplasia occurs to increase the protective mucous layer. Superficial wounds heal by reepithelialization. Healing begins within 2 hours after sloughing of superficial cells. Intact ciliated columnar cells surrounding the defect flatten, lose their cilia, and migrate over the wound. Mitosis begins approximately 48 hours after injury in the ciliated columnar and basal epithelial cells. Organization and differentiation begin after 4 days. Squamous cells replace ciliated and goblet cells if injury recurs without healing. Full-thickness tracheal mucosal wounds with a gap between mucosal edges fill with granulation tissue before reepithelialization. Full-thickness wounds may heal with scar tissue protruding into the lumen. Scar tissue narrows the lumen and may interfere with transport of mucus. A 20% reduction in lumen diameter may reduce mucociliary clearance by more than 50%.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

An assortment of long-handled instruments is beneficial. Skin hooks, laryngeal or uterine cup biopsy forceps, and tracheal prostheses (see p. 871) are needed for some procedures. Rhinotomy requires an oscillating saw, chisel and mallet, or rongeurs, as well as periosteal elevators and curettes. Nonreactive monofilament suture (e.g., polypropylene [PDS], polybutester [Novafil], polydioxanone [PDS], polyglyconate [Maxon], poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn]) is recommended for surgery of the upper respiratory tract.

POSTOPERATIVE CARE AND ASSESSMENT

Patients must be closely monitored during anesthetic recovery for hemorrhage, coughing, gagging, or aspiration. They should be kept intubated as long as possible and reintubated or have a tracheostomy tube placed if respiratory distress occurs after extubation. When reintubating these patients, consider using an endotracheal tube a half size smaller than was used initially to make intubation easier if airway swelling is present. Supplemental oxygen should be provided if necessary during recovery, and excitement and pain should be minimized by postoperative analgesics (see Table 13.2). Inserting a nasal oxygen catheter at the conclusion of surgery facilitates oxygen delivery (see Box 4.4). Alternatively, the animal may be placed in an oxygen cage. Positioning the patient in sternal recumbency may facilitate respiration. Postoperative glucocorticoids (e.g., dexamethasone sodium phosphate 0.1–0.5 mg/kg IV) may reduce nasopharyngeal and upper airway edema secondary to surgical or diagnostic

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manipulations. Start with the lower dose and use the higher dose only if the low dose has failed to reduce swelling and if swelling is sufficiently severe that the animal cannot breathe. Single large doses or repeated administration of dexamethasone typically causes gastrointestinal erosions, ulcers, and/or hemorrhage. Prophylactic antibiotics can be discontinued immediately after surgery. Water may be offered 6 to 12 hours after surgery; soft food made into meatballs may be offered 12 to 24 hours postoperatively if gagging, regurgitation, or vomiting does not occur. Meatballs should be fed one at a time for 7 to 14 days after nasopharyngeal or laryngeal procedures to slow ingestion. Exercise should be restricted for 2 to 4 weeks. A harness rather than a collar should be used lifelong to prevent incisional, tracheal, or laryngeal trauma.

After rhinotomy, animals should be recovered in a slightly head-down position, they should be kept quiet, and some sneezing should be expected. Breathing sounds are typically harsh and resonant after this procedure. Initially, nasal discharge is expected to be bloody, but it should gradually become serous and diminish in volume after the nasal cavity has reepithelialized (generally within a week). If bleeding continues after surgery, the nasal cavity can be packed with sterile gauze. If gauze strips or umbilical tape are used (see p. 840), the end of the gauze or tape can be exited from the nostril or a dorsal stoma and sutured to the side of the face. The packing is removed 1 or 2 days after surgery. A blood transfusion may be needed if hemorrhage is significant (see Box 4.1). Movement of the skin flap covering the bony defect is expected following dorsal rhinotomy, and SC emphysema may occur but should be self-limiting. SC air accumulates if the bone flap is not replaced adequately, or if an adequate stoma is not left in the SC tissues and skin for air to exit. SC air accumulation is generally not a problem if an adequate stoma is left. The stoma will contract and heal within 5 to 10 days. Do not allow the animal to chew on hard objects for at least 3 to 4 weeks following ventral rhinotomy until the palate incision is healed. Feed soft food for a minimum of 7 to 14 days following surgery. Animals may occasionally be depressed and unwilling to eat for several days; in such cases, consider placement of an enteral feeding tube (e.g., esophagostomy; see p. 95).

Intensive postoperative care is required after tube tracheostomy. The animal must be observed closely to prevent asphyxiation secondary to tube obstruction or dislodgment. Clearance of mucus is inhibited in these animals, and mucosal irritation leads to increased production of mucus. Tube cleaning may be required up to every 15 minutes, particularly in smaller animals. Sterile technique (i.e., gloves and instruments) should be used to clean tracheostomy tubes. Secretions may be removed by inserting a sterile suctioning cannula into the tube lumen and distal trachea. When cannulated (double-lumen) tubes are used, the inner cannula may be removed and cleaned while the outer tube is suctioned. Nebulization and injection of sterile saline (1 mL) into the tube a few minutes before suctioning help in loosening secretions. A new tube should be used if these techniques do not adequately remove secretions. Tracheostomy tubes may be removed when an adequate airway and spontaneous ventilation have been established. Occasionally the tube can be occluded and the patient observed while breathing around the tube to determine whether the tube can be removed. This should not be done in animals with cuffed tubes or in those that have large tubes that fill the tracheal lumen. After removal of the tube, the tracheostomy site is allowed to heal by secondary intention.

Management of permanent tracheostomy in the short term is usually less demanding than management of tube tracheostomy, yet still requires diligent in-hospital observation for the first 3 to 5 days. Initially the tracheostoma should be inspected every 1 to 3 hours for accumulation of mucus. When mucus begins to occlude the tracheostoma, or when respiratory effort increases, the site should be suctioned as described previously for tube tracheostomy. Mucus at the stoma may be removed by aspiration or by gentle wiping with a sponge or applicator stick. Only a moderate amount of mucus is expected to accumulate during the first 7 to 14 days after surgery unless the animal has severe tracheitis. By 7 days, the cleaning interval usually increases to every 4 to 6 hours, and after 30 days, twice-daily stomal cleaning is usually sufficient. However, smoke and other noxious stimuli increase mucus production and necessitate more frequent cleaning. Hair should be clipped as needed from around the stoma to prevent matting of the hair with mucus. Exercise and housing should be restricted to clean areas.

After tracheal resection and anastomosis, exercise and neck extension should be restricted for 2 to 4 weeks. Animals should be kept quiet and observed for signs of respiratory distress after ventriculocordectomy. Some animals gag and cough. Vocalization should be discouraged for 6 to 8 weeks.

COMPLICATIONS

Acute respiratory obstruction caused by mucosal swelling, edema, irritation, and increased mucus production and/or laryngeal or tracheal collapse may occur after upper respiratory surgery and must be relieved promptly (Box 28.5). Infection can be a problem because the nose, nasopharynx, larynx, and trachea have a resident bacterial flora. Using strict aseptic technique and lavaging contaminated tissues usually prevent infection. Injury to the recurrent laryngeal nerve may cause laryngeal spasms, paresis, or paralysis, leading to aspiration pneumonia. Mucostasis may occur after nerve damage. Gentle tissue handling, appropriate dissection, and careful tissue retraction prevent nerve damage.

Complications associated with rhinotomy include excessive blood loss, SC emphysema, gagging, coughing, and/or vomiting associated with aspiration of blood and exudates. Bone flaps that have been replaced following rhinotomy may sequester or harbor infectious organisms or tumor cells, leading to recurrence of disease. Caudal choanal stenosis may occur following severe rhinitis associated with infection or after extensive debridement of nasal epithelium. Signs include those of nasal obstruction with minimal nasal discharge and stridor. These stenotic lesions can be difficult to resolve; however, scar tissue may be perforated and then dilated with a balloon catheter or excised and covered with a mucosal flap. Most patients will ultimately require stenting.

BOX 28.5 Common Errors in Managing Animals With Upper Respiratory Disease

- Failing to diagnose and treat upper respiratory disease before secondary problems develop (i.e., aspiration pneumonia)
- Failing to recognize laryngeal collapse
- Causing trauma to the recurrent laryngeal nerves
- Rough handling or overhandling of tissue, causing excessive swelling
- Failing to monitor the patient intensively after surgery

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Intensive monitoring of a patient with a temporary tracheostomy tube is required to avoid life-threatening complications, particularly in smaller animals. Complications associated with tube tracheostomy include gagging, vomiting, coughing, tube obstruction, tube dislodgment, emphysema, tracheal stenosis, tracheal malacia, and tracheocutaneous or tracheoesophageal fistula. Some animals occlude the tracheostomy tube when the neck is flexed and when they sleep with bedding. Major complications (e.g., occlusion, dislodgment) were reported in 44% of cats with temporary tracheostomies.[2] Cuffed tracheostomy tubes and endotracheal tubes may cause pressure necrosis of the tracheal mucosa or cartilages, which may result in tracheal strictures.

Animals with permanent tracheostomy have high complication and mortality rates primarily caused by occlusion of the stoma from mucus, blood, or stricture. Both dogs and cats are at risk for acute occlusion and sudden death, but it is more notable for cats. Median survival times following permanent tracheostomy in dogs and cats are 328 and 20.5 days, respectively.[3,4] The long-term main complication of permanent tracheostomy is stomal occlusion from accumulated mucus, skin folds, or stenosis, which may require revision of the tracheostoma. Animals are also at lifelong risk for aspiration pneumonia.

Complications after tracheal resection and anastomosis may include hemorrhage, voice change, fistula formation, and cartilage malacia. Malacia is uncommon. The other complications are manageable. Dehiscence occurs after tracheal anastomosis if excessive postoperative tension or neck movement is present. SC emphysema, acute respiratory distress, hemoptysis, and SC swelling suggest dehiscence. Excessive anastomotic tension and secondary intention healing may cause tracheal stenosis. Excessive dissection may cause ischemic necrosis of the remaining trachea. Traumatizing the recurrent laryngeal nerves may cause laryngospasm, laryngeal paresis, or laryngeal paralysis.

After ventriculocordectomy, scar tissue may form within the larynx and trachea, causing obstruction weeks postoperatively. Clinical signs of obstruction are not usually apparent until luminal compromise approaches 50%. Scar tissue forms across the larynx as the result of mucosal damage or with second intention healing near the dorsal and ventral commissures. Other complications include edema, hemorrhage, cough, gag, stenosis, and altered vocalization. Mucosal edema may partially obstruct the glottis and can be reduced by pretreatment with glucocorticoids. Stenosis may occur at the dorsal or ventral commissures of the glottis after ventriculocordectomy if intact mucosa is not preserved in these areas, and healing occurs by secondary intention. Approximating mucosa over the ventriculocordectomy sites also minimizes stenosis. Ventriculocordectomy is expected to alter the normal bark, making it lower pitched and harsher. Resumption of a near-normal bark may occur within months after removal of only the vocal fold margin and secondary intention healing.

SPECIAL AGE CONSIDERATIONS

The tracheal and laryngeal cartilages of very young animals have a high water content, and these cartilages may not hold sutures well. Therefore a lesser length of trachea can be resected in young animals (20%–25%) than in older animals. Congenital abnormalities involving the respiratory tract should be treated early in the animal's life (within the first year) to prevent progressive respiratory distress and to improve the animal's quality of life. Older animals may have ossified, inelastic, brittle cartilages that are difficult to manipulate during surgery.

SPECIFIC DISEASES

BRACHYCEPHALIC SYNDROME

DEFINITIONS

Brachycephalic syndrome refers to the combination of the surgically addressable conditions of stenotic nares, elongated soft palate, everted laryngeal saccules, and laryngeal collapse causing upper airway obstruction in brachycephalic breeds; it is also referred to as **brachycephalic obstructive airway syndrome** or **brachycephalic airway syndrome.** ***Stenotic nares*** are nostrils with abnormally narrow openings that make the nostrils appear pinched together. An **elongated soft palate** is one that extends more than 1 to 3 mm caudal to the tip of the epiglottis. **Everted laryngeal saccules** are protrusions of the mucosal diverticula rostral to the vocal folds (Video 28.1); they are also referred to as *laryngeal saccule eversion* or *stage 1 laryngeal collapse.*

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Brachycephalic syndrome refers to upper airway obstruction attributable to a combination of anatomic abnormalities seen in brachycephalic dogs (Boxes 28.6 and 28.7). Concurrent tracheal hypoplasia, everted tonsils, nasopharyngeal turbinates, and other abnormalities often contribute to respiratory distress. Brachycephalic animals typically have a compressed face with poorly developed nares and a distorted nasopharynx. The head shape is the result of an inherited developmental defect in the bones of the base of the skull. These bones grow to a normal width but have a reduced length. The soft tissues of the head are not proportionately reduced and often appear redundant.

Stenotic nares are congenital malformations of the nasal cartilages that result in medial collapse and partial occlusion of the external nares (Figs. 28.14 and 28.15). Airflow into the nasal cavity is restricted and greater inspiratory effort is necessary, causing mild to severe dyspnea. Resistance to airflow through the nasal cavity in normal dogs is 76% to 80% of total resistance, depending on the volume of airflow. As more and more negative pressure is exerted to breathe, intratracheal and intrapharyngeal pressures can become high enough to cause surrounding tissues to collapse.

Elongated soft palate is the most common component of brachycephalic syndrome and is frequently diagnosed in brachycephalic dogs (Fig. 28.16). The elongated soft palate is pulled caudally during inspiration, obstructing the dorsal aspect of the glottis. It is sometimes drawn between the corniculate processes

BOX 28.6 Canine Breeds Commonly Affected With Brachycephalic Syndrome

English bulldog
French bulldog
Pug
Boston terrier
Shar Pei
Cavalier King Charles spaniel
Lhasa apso
Shih tzu
Boxer
Pekingese

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BOX 28.7 Upper Airway Abnormalities Associated With Brachycephalic Breeds

Classic Components of Brachycephalic Syndrome

- Elongated soft palate
- Stenotic nares
- Everted laryngeal saccules

Common Concurrent Findings

- Hypoplastic trachea
- Aryepiglottic collapse

Other Findings

- Corniculate collapse
- Tracheal collapse
- Tonsil eversion
- Nasopharyngeal turbinates
- Macroglossia
- Pharyngeal collapse
- Epiglottic collapse

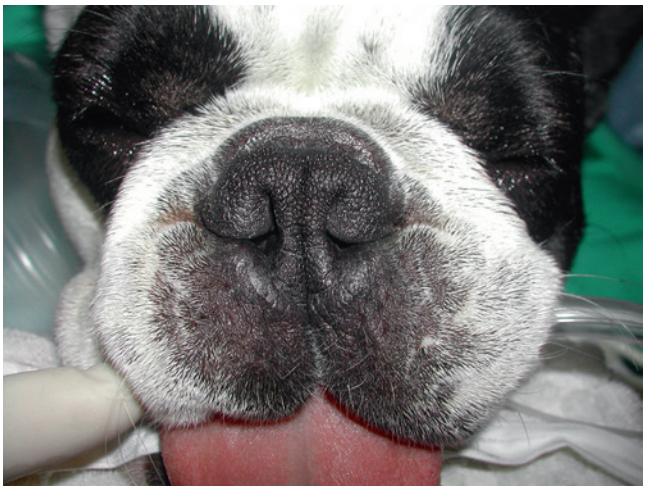


FIG. 28.14 Four-year-old Boston terrier with stenotic nares.

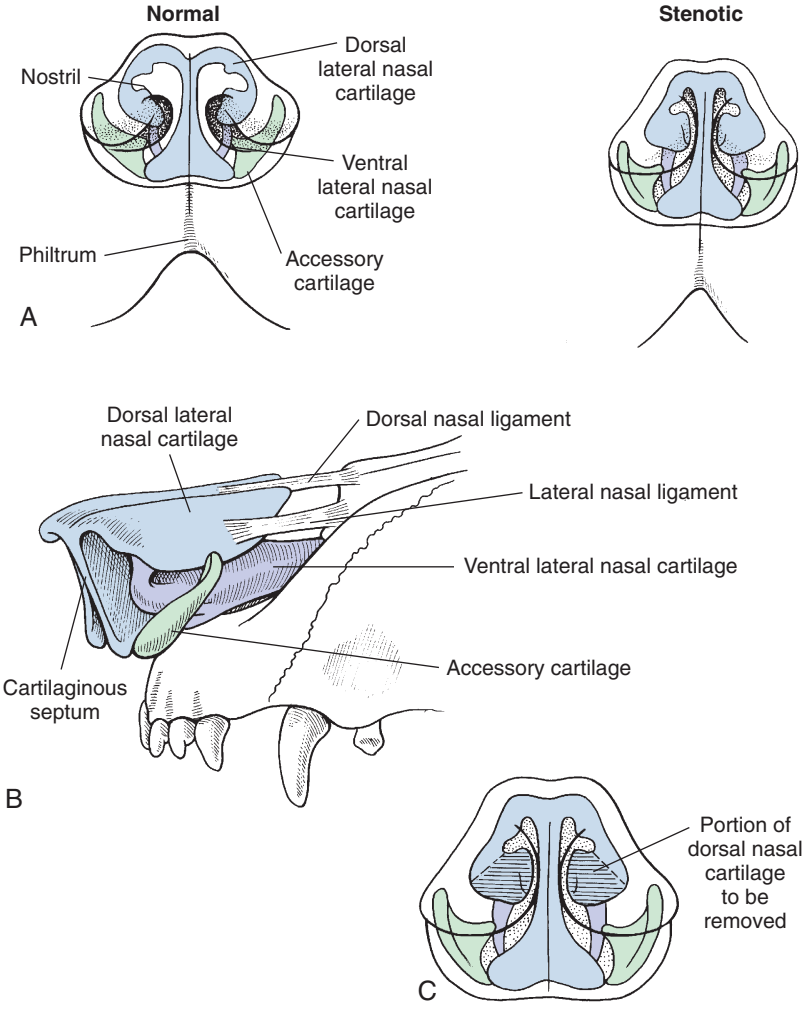


FIG. 28.15 (A) Normal and stenotic appearance of the nares. (B) The dorsolateral, ventrolateral, and accessory nasal cartilages form the nostrils. (C) To widen the nares, resect a portion of the dorsolateral nasal cartilages.

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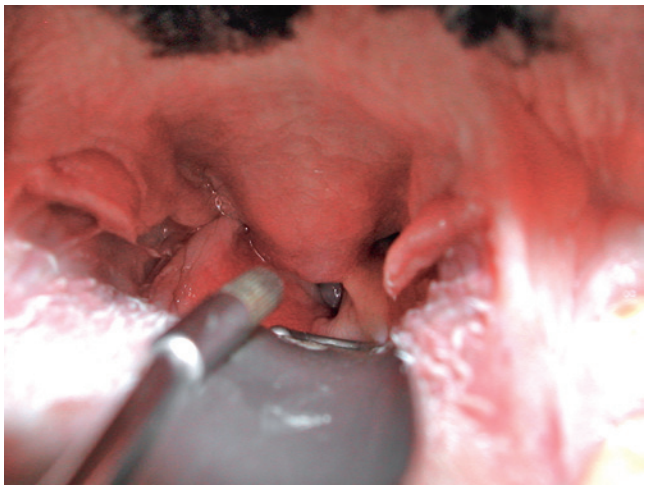


FIG. 28.16 Laryngoscopic view of an elongated soft palate.

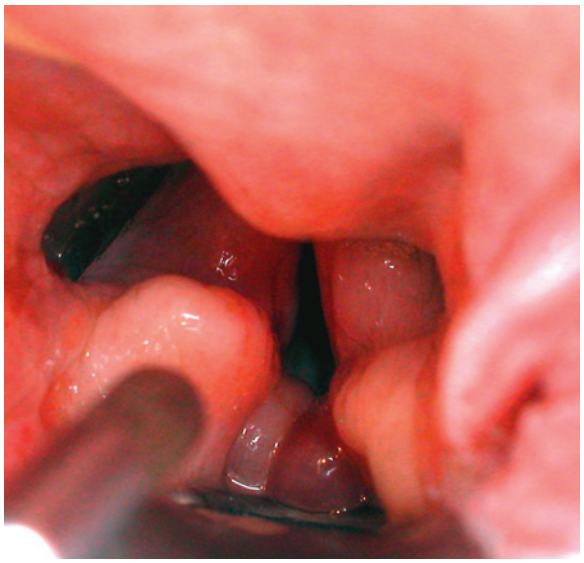


FIG. 28.17 Laryngoscopic view of everted laryngeal saccules in a 6-year-old bulldog.

of the arytenoid cartilages, which increases inspiratory effort and causes more turbulent airflow. The laryngeal mucosa becomes inflamed and edematous, further narrowing the airway. The tip of the soft palate is blown into the nasopharynx during expiration. Affected dogs may have trouble swallowing because normal occlusion of the airway during deglutition compromises ventilation. Dysfunctional swallowing may produce aspiration pneumonia. Some affected animals with concurrent gastrointestinal problems (e.g., esophagitis, hiatal hernia) have improved following surgical treatment of the brachycephalic syndrome.

Laryngeal saccule eversion (Fig. 28.17) is diagnosed less often than elongated soft palate or stenotic nares, but has been in reported in over half of dogs with brachycephalic syndrome. It is uncommon to have everted laryngeal saccules as the only abnormality in brachycephalic dogs. Eversion of the laryngeal saccules is considered the first stage of laryngeal collapse. Increased airflow resistance and increased negative pressure generated to move air past obstructed areas (stenotic nares, dorsal glottis) pull the saccules from their crypts, causing them to swell. Once

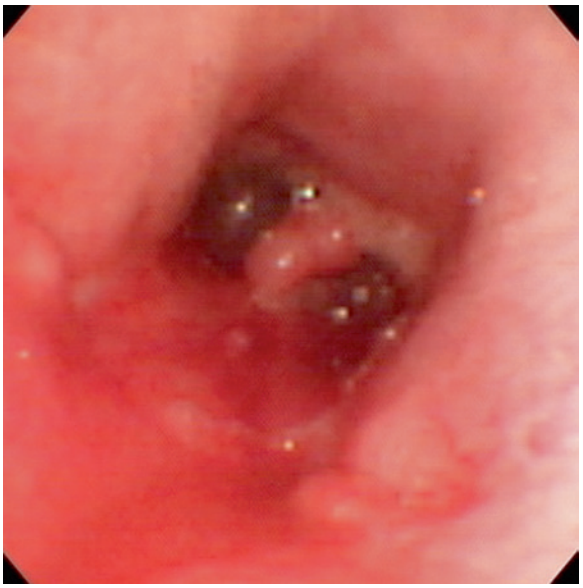


FIG. 28.18 Retroflexed endoscopic view of nasopharyngeal turbinates in a 2-year-old bulldog.

everted, the saccules are continuously irritated by turbulent airflow and become increasingly edematous. The saccules obstruct the ventral aspect of the glottis and further inhibit airflow. It may be difficult for an inexperienced examiner to differentiate everted laryngeal saccules from the vocal folds because of their close proximity. Everted saccules partially or completely obscure the vocal folds and may be unilateral or bilateral.

In addition to components already described, everted tonsils, aryepiglottic collapse, corniculate collapse, tonsil eversion, redundant pharyngeal folds, nasopharyngeal collapse, macroglossia, nasopharyngeal turbinates (Fig. 28.18), and laryngeal collapse may contribute to the severity of respiratory distress. Laryngeal collapse as a primary disease has been reported in English bull terriers, Norwich terriers, and other small-breed dogs.[5–7]

DIAGNOSIS

Clinical Presentation

Signalment

Brachycephalic breeds (particularly English bulldogs, Boston terriers, pugs, Pekingese, French bulldogs, and Cavalier King Charles spaniels) are predominantly affected. Dogs are more commonly affected than cats (e.g., Himalayans and Persians). The condition can affect either gender. Although affected animals may have some abnormalities since birth, many animals present for evaluation between 2 and 4 years of age.

History

Patients with upper airway obstruction usually have noisy, stridorous breathing, particularly on inspiration, with varying levels of respiratory distress. Some animals are brought in for evaluation because of frequent retching or gagging. Dogs may have trouble swallowing caused by production of thick salivary secretions and by the fact that normal occlusion of the airway during deglutition compromises ventilation. Some animals have concurrent gastrointestinal signs such as dysphagia, ptyalism, regurgitation, and vomiting. Exercise intolerance, cyanosis, restless sleeping (sleep-disordered breathing), and collapse are often reported.

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Excitement, stress, and increased heat and humidity frequently make clinical signs worse.

Physical Examination Findings

Pharyngeal and laryngeal auscultation reveals prominent snoring (stertor) and inspiratory stridor that obscures other respiratory sounds. Signs of increased inspiratory effort include retraction of lip commissures, open-mouth breathing or constant panting, forelimb abduction, and exaggerated use of abdominal muscles. Paradoxical movement of the thorax and abdomen, recruitment of accessory respiratory muscles, inward collapse of the intercostal spaces and thoracic inlet, and orthopneic posture (extended head and neck and reluctance to lie down) may be apparent. The mucous membranes are normal in color with mild or moderate dyspnea but are pale or cyanotic with severe dyspnea. Affected animals are often restless and anxious, especially when restrained. Animals may be hyperthermic as a result of ineffective cooling. Careful thoracic auscultation is difficult because of referred upper airway noise. Gastrointestinal tract distention may occur secondary to aerophagia associated with open-mouth breathing.

Stenotic nares are identified on physical examination. The nares may be mildly, moderately, or severely deviated medially. During inspiration, the nares may be pulled medially or may remain relatively stationary rather than abducting. Airflow through the nares can be objectively assessed by holding a glass slide or piece of cotton near the nares. It is difficult to visualize the oropharynx and larynx in brachycephalic animals because their tongues are thick, and restraint may accentuate respiratory distress.

Diagnostic Imaging

Thoracic radiographs should be evaluated to detect underlying cardiopulmonary abnormalities (e.g., cardiomegaly, pulmonary edema, pneumonia) or other concurrent conditions (e.g., hypoplastic trachea, hiatal hernia, heart base tumor). Lateral cervical radiographs allow evaluation of the nasopharynx, the soft palate, the larynx, and the entire length of the trachea. An elongated soft palate may appear thickened and elongated (Fig. 28.19). Nasopharyngeal, laryngeal, and tracheal masses may be identified (Fig. 28.20).

Hypoplastic trachea is congenital tracheal stenosis that affects the entire length of the trachea (Fig. 28.21). In addition, rigid tracheal cartilages are apposed or overlap, and the dorsal tracheal membrane is narrow or obscured. Tracheal hypoplasia can be associated with continuous respiratory distress, coughing, and recurrent tracheitis but may be tolerated in the absence of concurrent respiratory or cardiovascular disease. Tracheal hypoplasia can be diagnosed radiographically by determining the tracheal-to-thoracic inlet diameter ratios (Box 28.8). No definitive treatment for hypoplastic trachea is known, only symptomatic therapy and correction of other anatomic defects to alleviate clinical signs. The extent to which hypoplastic trachea

BOX 28.8 Radiographic Diagnosis of Hypoplastic Trachea

- Ratio of tracheal lumen diameter at the thoracic inlet to the thoracic inlet diameter (TD/TI) <0.2
- Ratio of tracheal lumen diameter at the midpoint between the thoracic inlet and carina to the width of the third rib (TT/3R) <3



FIG. 28.20 Lateral cervical radiograph demonstrating a soft tissue mass impinging on the proximal trachea in a 6-year-old Portuguese water dog.

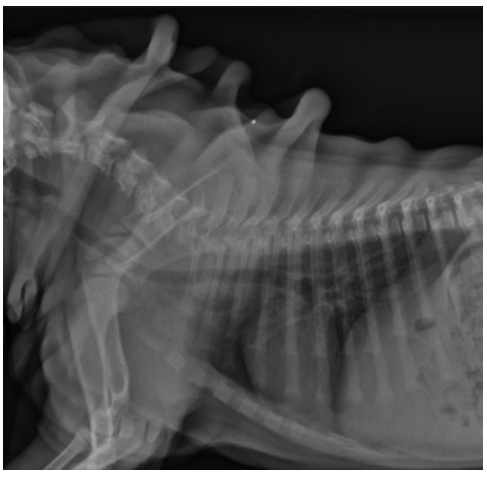


FIG. 28.19 Lateral cervical radiograph demonstrating an elongated soft palate in a 14-week-old shar pei.

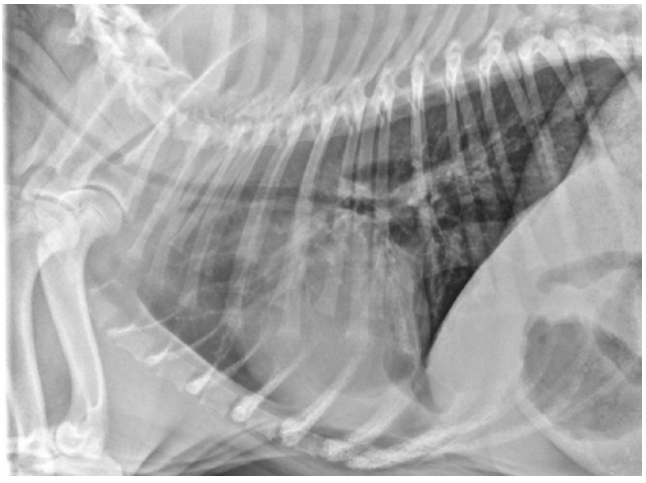


FIG. 28.21 Lateral thoracic radiograph showing hypoplastic trachea in a 2-year-old English bulldog.

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contributes to the severity of clinical signs in dogs with brachycephalic airway syndrome is unknown.

Computed tomography (CT) has been used to assess for tracheal hypoplasia, laryngeal collapse, and other forms of upper airway obstruction. A 2015 study compared CT tracheal dimensions with radiographic measurements and tracheoscopy scores in a group of clinically normal English bulldogs.[8] CT measurements averaged 19% greater than radiographic measurements, but neither were comparable with tracheoscopy scores because hypoplastic changes were identified in all dogs. CT, in conjunction with anterior and posterior rhinoscopy, is also useful in assessing for nasopharyngeal turbinates or excessive caudal aberrant turbinate protrusion. In the same group of clinically healthy English bulldogs described earlier, the prevalence of caudal aberrant nasal turbinates was 100%.[9]

Pharyngoscopy/Laryngoscopy

General anesthesia is necessary to evaluate the pharynx and larynx (see p. 834). This is initially provided with a rigid laryngoscope. An elongated soft palate overlies the epiglottis by at least a few millimeters but often by more than 1 cm (see Fig. 28.16). The soft palate is often thickened and has a roughened, inflamed tip. Dorsally displacing the soft palate with a tongue depressor improves visualization of the arytenoid cartilages. The arytenoids are frequently inflamed and edematous. Tonsils may be inflamed and everted from their crypts. Everted laryngeal saccules appear as spherical protrusions of tissue that obscure the vocal folds (see Fig. 28.17). Functional assessment of the larynx rules out concurrent laryngeal paralysis (p. 860), and laryngeal anatomy should be evaluated for other signs of collapse (p. 857).

Flexible Endoscopy

A flexible endoscope allows complete evaluation of the nasopharynx (to assess for nasopharyngeal turbinates, caudal aberrant nasal turbinates, choanal occlusion, masses), trachea (to assess for hypoplastic trachea or tracheal collapse [p. 869]), and bronchial tree. Bronchial abnormalities are often present in brachycephalic dogs, yet bronchial collapse or stenosis does not seem to affect long-term outcome. Similarly, upper gastrointestinal tract clinical signs and lesions are often identified in dogs with brachycephalic syndrome. Endoscopy of the upper gastrointestinal tract should be considered in brachycephalic dogs with signs of gagging, retching, regurgitation, or vomiting.

Laboratory Findings

Hematologic and serum biochemistry findings are usually normal. Blood gas evaluation may reveal hypoxemia and respiratory alkalosis. Oxygen saturation that acutely falls to below 80% may cause syncope and collapse. Polycythemia may occur with chronic moderate to severe hypoxia. In a small study comparing brachycephalic dogs with mesocephalic or dolichocephalic dogs, brachycephalic dogs are more likely to have lower partial pressure of arterial oxygen, higher carbon dioxide partial pressure, and higher arterial blood pressure.[10]

DIFFERENTIAL DIAGNOSIS

Other abnormalities that may cause or contribute to upper respiratory obstruction in brachycephalic dogs include tracheal collapse, hypoplastic trachea, obesity, laryngeal paralysis, masses obstructing the glottis, larynx, or trachea, and traumatic disruption of the airway.

MEDICAL MANAGEMENT

A weight reduction program should be instituted for overweight and obese animals. Exercise restriction and elimination of precipitating causes may be beneficial when clinical signs are mild. Sedation (see Box 28.2), glucocorticoids, supplemental oxygen (see Table 4.4), and cooling may be necessary for moderate to severe respiratory distress. An antiinflammatory dose of a glucocorticoid (e.g., dexamethasone sodium phosphate 0.1–0.5 mg/kg IV) may reduce nasopharyngeal and upper airway edema secondary to surgical or diagnostic manipulations. Start with the lower dose and use the higher dose only if the low dose has failed to reduce swelling and if swelling is sufficiently severe that the animal cannot breathe. Large doses or repeated administration of dexamethasone typically causes gastrointestinal erosion/ulceration.

SURGICAL TREATMENT

Multiple procedures (e.g., stenotic nares resection, resection of elongated soft palate, resection of everted laryngeal saccules) are usually required to alleviate signs of brachycephalic syndrome. Animals with upper respiratory obstruction are anesthetic and postoperative risks (see pp. 834 and 847). Stenotic nares resection should be performed as soon as the animal is old enough to be safely anesthetized, and when the animal's nasal tissues have matured enough to hold sutures (as early as 3–4 months of age). Resection of an elongated soft palate is best performed when the animal is young (i.e., 4–24 months old), before laryngeal cartilages deform and collapse. If present, everted laryngeal saccules are often excised at the same time as soft palate correction is performed, although some surgeons will choose to leave them in young dogs with mild to moderate clinical signs.

Preoperative Management

These animals should be monitored carefully for decompensation and progressive respiratory distress. Emergency therapy (e.g., temporary tracheostomy; see p. 842) may be necessary if dyspnea worsens acutely. Animals undergoing concurrent laryngeal or nasopharyngeal procedures can be treated with antiinflammatory doses of glucocorticoids (e.g., dexamethasone sodium phosphate 0.1–0.5 mg/kg IV), which may reduce nasopharyngeal and upper airway edema secondary to surgical or diagnostic manipulations. A tracheostomy tube may be placed before surgery; however, this is usually unnecessary unless the animal has severe pharyngeal and laryngeal edema or collapse. In addition, tracheostomy tubes are associated with significant complications (e.g., tube obstruction, tube dislodgement, aspiration pneumonia, and stoma swelling), and English bulldogs are more likely to have a negative outcome compared with other breeds.[11]

Anesthesia

Anesthesia or sedation must be done carefully in these animals (see p. 834). Virtually all sedatives and anesthetic agents relax the upper airway dilating muscles while allowing the diaphragm to continue contracting. This allows the upper airway to collapse. Additionally, most sedatives act centrally to decrease respiratory drive. Airway collapse is worsened by negative inspiratory pressure that draws the pharyngeal walls medially. Anesthetics also relax the muscles used by brachycephalic animals to facilitate breathing (e.g., geniohyoid, genioglossus, sternohyoid). Oxygen saturation

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can drop rapidly during anesthesia or sedation; therefore pulse oximetry should be monitored after premedication and during induction, oral examination, anesthesia, and anesthetic recovery. Preoxygenation of these animals before induction for 3 to 5 minutes by face mask, flow-by, or nasal insufflation is usually necessary. Anesthesia should be rapidly induced and the animal intubated as quickly as possible, if a laryngeal examination is not necessary. Being prepared with a variety of sizes of endotracheal tubes is helpful, especially if tracheal hypoplasia is present. Because these patients can rapidly desaturate or become bradycardic, monitoring equipment, anesthetic machines, and intubation and emergency drugs should be prepared in advance. General anesthetic recommendations and selected anesthetic protocols for animals with upper respiratory disease are given on p. 834.

Surgical Anatomy

The dorsal and ventral lateral nasal cartilages unite laterally to create a cartilage tube, forming the nostril (see Fig. 28.15A, B). The nostrils are supported medially and ventrally by the nasal septum and dorsally by the dorsal lateral nasal cartilages. The dorsal lateral cartilage also forms the lateral wall of the nostril. The lateral accessory cartilage contributes ventral support to the nostrils.

The soft palate extends from the hard palate to the tip of the epiglottis, effectively separating the oropharynx from the nasopharynx. The palatine muscle, which is covered by mucosa and innervated by the pharyngeal plexus (cranial nerves IX and X), shortens the soft palate during contraction. Palatine glands keep the mucosa moist. Blood supply is provided via the palatine vessel. The epiglottis is a curved, triangular cartilage at the entrance to the larynx. The apex of the epiglottis points to the oropharynx and lies just dorsal to the soft palate (see Fig. 28.3). The lingual aspect of the base of the epiglottis is attached to the basihyoid bone. Mucosa attaches the lateral aspects of the epiglottis to the cuneiform process of the arytenoid cartilage, forming the aryepiglottic fold (see Fig. 28.3). The epiglottis attaches to the body of the thyroid cartilage.

The end of the soft palate just covers the tip of the epiglottis in a normal dog. It generally extends no farther than the mid to caudal aspect of the tonsillar crypt. The distal end in a normal dog is concave; however, the distal end of an elongated soft palate is frequently sucked into the larynx, giving it a more pointed or pinched appearance.

The laryngeal saccule is a slight dorsoventral depression between the vestibular and vocal folds (see Fig. 28.3). Everted saccules lie just rostral to the vocal folds and should not be mistaken for the vocal cords. The surgical anatomy of the larynx is provided on p. 838.

Positioning

The patient is positioned in sternal recumbency with the mouth fully open. The maxilla should be suspended from a bar positioned several feet above the operating table and the mandible secured ventrally with tape. The chin should not be allowed to rest on the table or pads. For maximum visualization, the cheeks should be retracted laterally and the tongue pulled rostrally (Fig. 28.22). The oral cavity should be gently lavaged with dilute antiseptic solutions, and sponges should be placed around the endotracheal tube at the glottis to prevent fluids from entering the airway. To prevent irritation and edema, the mucosal surfaces should not be scrubbed.

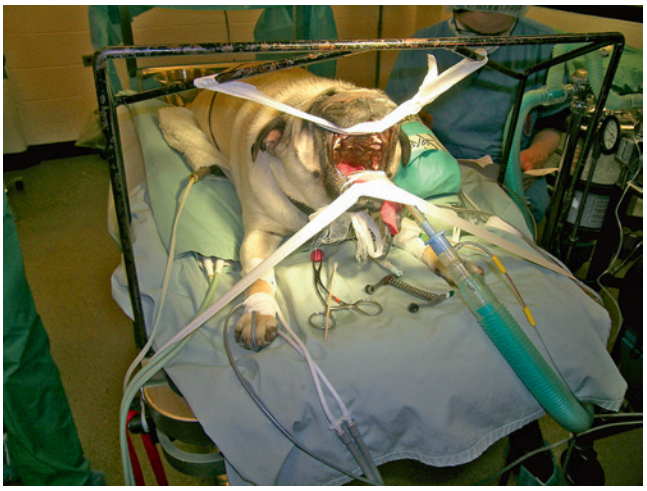


FIG. 28.22 For resection of an elongated soft palate, position the patient in sternal recumbency with the maxilla suspended and the mouth wide open.

Alternatively, correction of elongated soft palate and everted laryngeal saccules can be performed with the animal in dorsal recumbency.

For correction of stenotic nares alone, position the patient in sternal recumbency with the chin resting on a pad. Tape the head to the table to prevent rotation. The planum nasale should be gently scrubbed with antiseptic soap and solutions.

SURGICAL TECHNIQUE

Stenotic Nares Resection (Rhinoplasty)

Stenotic nares are easily correctable. Various surgical techniques are used, and all have the same end result: permanent enlargement of the external nares. A No. 11 scalpel blade is preferred for making deep and even cuts in the wing of the nostril; however, correction can also be performed with a skin biopsy punch tool or with electrosurgical or radiofrequency units with a fine-tipped blade or laser. Surgical procedures described to treat stenotic nares include nares amputation (see Fig. 28.15C), wedge resection, and alapexy.

Grasp the margin of the nares with a Brown-Adson thumb forceps. While maintaining this grip, make a V-shaped incision around the forceps with a No. 11 scalpel blade, approximately 1 to 3 mm deep into the alar fold. Make the first incision medially and the second incision laterally. Remove the vertical wedge of tissue (Fig. 28.23 and Video 28.2). Control hemorrhage with digital pressure and by reapposing the cut edges. Align the ventral margin of the nares and the mucocutaneous junction, and place three or four simple interrupted sutures using synthetic rapidly absorbable material (e.g., 3-0 or 4-0 poliglecaprone 25 [Monocryl], glycomer 631 [Biosyn], polyglactin 910 [Vicryl]) to reappose the tissues (Video 28.3). Repeat the procedure on the opposite side, taking care to excise a tissue wedge of the same size.

Elongated Soft Palate Resection (Staphylectomy)

Soft palate resection may be done with scissors, carbon dioxide laser, electrosurgery, or a bipolar sealing device (e.g., LigaSure), although electrosurgery may increase postoperative swelling. Electrosurgery and lasers may ignite oxygen if proper safety

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FIG. 28.23 Intraoperative image of a 2-year-old English bulldog after vertical wedge resection of the right nasal cartilage.

precautions are not taken. Surgical time for laser resection is significantly shorter than time for traditional resection with scissors; however, no difference in outcome has been noted between the two methods. Hemorrhage is generally mild to moderate after resection and can be controlled with gentle pressure. The caudal margin of the soft palate should be shortened so that it contacts the tip of the epiglottis and when pushed dorsally contacts the roof of the nasopharynx. Resection of too little soft palate does not optimally relieve respiratory distress, whereas resection of too much soft palate may result in nasal regurgitation, rhinitis, and sinusitis. More recently it has been recommended to resect the soft palate to the level from the caudal aspect of the tonsil or as far as the level of the cranial commissure of the tonsillar crypt, such that there is no interaction between the soft palate and the tip of the epiglottis.

Visually mark the site of proposed resection using the tip of the epiglottis and the caudal margin or the midpoint of the tonsils as landmarks. Handle the soft palate gently and as little as possible to prevent excessive mucosal swelling. Grasp the midline tip of the soft palate with thumb forceps or Allis tissue forceps, or place a stay suture. Place additional stay sutures at the proposed site of resection on the right and left borders of the palate. Place hemostats on these sutures and have an assistant apply lateral traction. Transect across one-third to half of the width of the soft palate with curved Metzenbaum scissors. Begin a simple continuous suture pattern (4-0 absorbable monofilament suture) at the border of the palate, apposing the oropharyngeal and nasopharyngeal mucosa (Fig. 28.24). Continue transecting and suturing until the excess palate has been resected (Video 28.4). Alternatively, the palate can be transected fully before suturing is begun; however, visualization of the nasopharyngeal mucosa will be more challenging.

Alternatively, a folded-flap palatoplasty has been described for dogs that have excessive length and excessive thickness of the soft palate. In this technique the soft palate is thinned by excision of a portion of the oropharyngeal mucosa, underlying soft tissues, and part of the levator veli palatini muscle. The palate is made shorter by bringing the caudal edge of the palate rostrally, folding it onto itself until the caudal nasopharyngeal opening is readily visible transorally (Fig. 28.25).

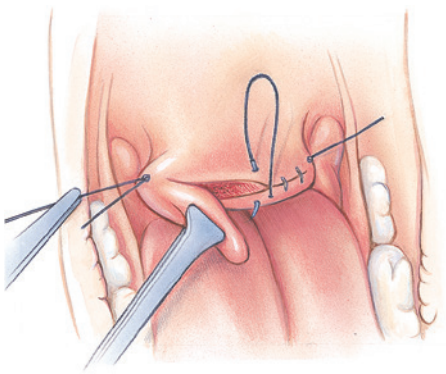


FIG. 28.24 To shorten the soft palate, place stay sutures at the proposed site of resection. Transect one-third of the palate, then appose the mucosa with sutures. Continue alternating excision and suturing until the resection is complete.

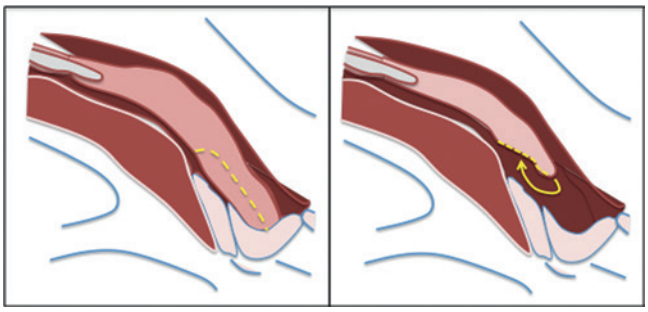


FIG. 28.25 Folded-flap palatoplasty. The dotted line shows the part of the thickened soft palate that is resected, allowing for the caudal edge of the palate to be folded rostrally and thus shortening and thinning the caudal soft palate.

Everted Laryngeal Saccules

Resection of the everted laryngeal saccules is relatively simple; however, whether resection of the saccules is needed in dogs with brachycephalic syndrome can be debated. Because complications with resection are possible (e.g., laryngeal swelling, laryngeal webbing, regrowth), it has been recommended that saccules be removed only when believed to contribute significantly to obstruction.[12] The challenge of the technique lies in obtaining good visualization of the larynx and glottis, and minimally handling the tissues. Excessive manipulation can cause local obstructive edema postoperatively. Use extreme care if laser or electrosurgical excision is used, to prevent trauma to surrounding tissues and igniting of flammable gases.

Retract the endotracheal tube dorsomedially so that the saccule on one side can be better visualized. Grasp the everted saccule with long-handled forceps or a tissue hook. Position the tip of a long-handled, curved Metzenbaum scissors at the base of the everted tissue and transect (Fig. 28.26). Biopsy forceps or laryngeal cup forceps may also be used. *Control hemorrhage with gentle pressure. Repeat the procedure on the opposite side. Handle the tissues gently.*

Laser-Assisted Turbinectomy

Removal of obstructive nasal turbinate tissue using diode laser as part of multilevel surgery for brachycephalic airway syndrome has recently been described.[13] Nasal concha rostrally, ventrally, and caudally are removed to shape a patent nasal airway.

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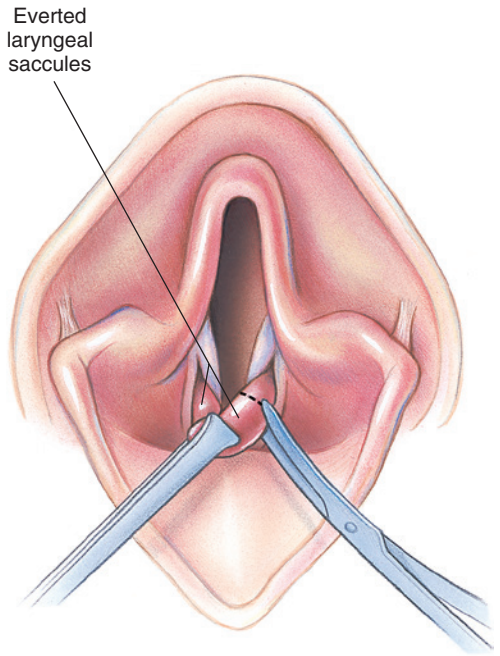


FIG. 28.26 To remove everted laryngeal saccules, grasp the protruding saccule and resect it with Metzenbaum scissors.

POSTOPERATIVE CARE AND ASSESSMENT

These patients require constant monitoring during recovery from anesthesia (see p. 847), and they should be closely observed for signs of respiratory distress until they are completely awake. Extubation should be delayed as long as possible, and the endotracheal tube should not be removed until the patient clearly objects to its presence. It is helpful to recover the animal in a quiet environment while oxygen saturation is being monitored with pulse oximetry. Optimally, the animal should be placed in sternal recumbency with the front limbs extended out and forward. In addition, the head and neck should be placed in extension with the tongue pulled forward to assist in opening up the airway. Before extubation, induction drugs, laryngoscope, endotracheal tube, and oxygen need to be immediately available. When the patient will no longer tolerate the endotracheal tube, it may be removed; however, reintubation needs to be considered if oxygen saturation does not stay in the nineties. Oxygen may be administered by nasal insufflation (see Box 4.4). Mild hemorrhage from the resection sites may lead to coughing, gagging, and hematemesis. Postoperative swelling and edema may cause severe laryngeal obstruction. Reintubation or placement of a tracheostomy tube may be necessary if respiratory obstruction or severe distress occurs. Glucocorticoids may be administered postoperatively if swelling is severe and if respiratory obstruction persists. IV fluids should be maintained until oral intake resumes. Hospital observation is recommended for 24 to 72 hours after surgery. Excessive mucosal swelling may cause asphyxiation. Postoperative coughing and gagging are common. Water, ice water, or ice chips may be offered when the animal is fully recovered from anesthesia; however, food should be withheld for 12 to 24 hours (see p. 848). Offering food soon after surgery may traumatize swollen tissues, causing swelling, airway obstruction, aspiration, or all of these.

COMPLICATIONS

If stenotic nares are the patient's only abnormality, complications are minimal. Dehiscence may occur if the patient frequently licks or rubs its nose; healing then occurs by secondary intention and may cause a pink scar. Respiratory distress may persist if other areas of the airway are obstructed (especially from postoperative edema) or if insufficient palate was resected. Transient intraoperative hemorrhage is a common complication of laser-assisted turbinectomy. Regrowth of obstructive turbinate tissue is also possible and typically occurs within 6 months of surgery. Acute respiratory distress and death following surgery are possible given the high anesthetic risk of these dogs.

PROGNOSIS

Surgical correction of brachycephalic syndrome will alleviate signs of respiratory distress and improve quality of life in most dogs. The outcome is dependent on the age of the animal at the time of surgery and how severely the dog is affected preoperatively. Recent studies describe a good to excellent long-term outcome in the majority of dogs; major complications, including death or euthanasia, occur in approximately 5%. The most beneficial result of multilevel surgery for airway obstruction in brachycephalic dogs is the marked reduction in life-threatening events of choking and collapse.[14] English bulldogs have been found to have a worse response to surgery when compared with all other breeds combined and are more likely to develop aspiration pneumonia postoperatively. Without surgery, the prognosis for dogs with brachycephalic airway syndrome is guarded because respiratory signs and laryngeal collapse progress over time. If advanced laryngeal collapse has developed, the prognosis is often poor unless additional surgery is considered.

LARYNGEAL COLLAPSE

DEFINITIONS

Laryngeal collapse is a form of upper airway obstruction caused by loss of cartilage rigidity that allows medial deviation of the laryngeal cartilages. Collapse of the cuneiform process of the arytenoid cartilage is referred to as *aryepiglottic collapse* or *stage 2 laryngeal collapse*. Collapse of the corniculate process of the arytenoid cartilage is referred to as corniculate collapse or *stage 3 laryngeal collapse*.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Laryngeal collapse most commonly occurs secondary to chronic upper airway obstruction or trauma. Trauma may fracture or disrupt the laryngeal cartilages and allow medial collapse. Laryngeal collapse is most often caused by chronic upper airway obstruction (e.g., brachycephalic syndrome, laryngeal paralysis) and cartilage fatigue or degeneration. The obstruction causes increased airway resistance, increased negative intraglottic luminal pressure, and increased air velocity. These forces displace laryngeal structures medially, with permanent cartilage deformation, and also fatigue the cartilages. Increased inspiratory effort irritates the mucosa, causing inflammation and edema. This further obstructs the airway, causing greater airflow resistance and increasing the effort of breathing.

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Laryngeal collapse is described in three stages: stage 1 is commonly referred to as *laryngeal saccule eversion* (see p. 851); stage 2 collapse is medial deviation of the cuneiform cartilage and aryepiglottic fold, or aryepiglottic collapse; stage 3 collapse is medial deviation of the corniculate process of the arytenoid cartilages, or corniculate collapse. Stages 2 and 3 are advanced stages of laryngeal collapse.

The diagnosis of laryngeal collapse that occurs concurrently with other upper respiratory abnormalities (i.e., elongated soft palate [see p. 849] and stenotic nares [see p. 849]) may be easily overlooked on oral and laryngeal examination. The incidence of laryngeal collapse in brachycephalic dogs varies widely and may be related to regional differences and the subjective assessment of laryngeal collapse. If the response to treatment is less than expected after appropriate surgery for these abnormalities, laryngeal collapse may be present.

DIAGNOSIS

Clinical Presentation

Signalment

Animals with brachycephalic syndrome (see p. 849) or laryngeal paralysis (see p. 858) are predisposed to laryngeal collapse. Advanced laryngeal collapse usually occurs in animals older than 2 years of age; however, it may be seen in younger animals (<6 months) with severe upper airway obstruction. Concurrent laryngeal paralysis and laryngeal collapse has been reported in a small set of nonbrachycephalic small-breed dogs.[7] Laryngeal collapse as a primary disease has been reported in English bull terriers, Norwich terriers, and other small-breed dogs.[5–7]

History

Inspiratory stridor, stertor, and other signs of upper airway obstruction have usually been present in affected animals for years but may have gradually or acutely worsened. Laryngeal collapse should be suspected in patients that had responded well to surgery for upper airway obstruction but later relapsed with moderate to severe respiratory distress.

Physical Examination Findings

Stridorous, labored breathing is the most consistent finding. Animals with advanced laryngeal collapse (stage 2 or stage 3) usually have moderate to severe respiratory distress (i.e., retraction of lip commissures, open-mouth breathing or constant panting, forelimb abduction, exaggerated use of abdominal muscles, paradoxical movement of the thorax and abdomen, recruitment of accessory respiratory muscles, inward collapse of the intercostal spaces and thoracic inlet, and orthopneic posture). See also p. 852.

Diagnostic Imaging

The thorax and the neck should be evaluated for evidence of concurrent abnormalities (see p. 853). A lateral cervical radiograph may show laryngeal cartilage ossification, cartilage fracture, or pharyngeal, laryngeal, or tracheal masses.

Laryngoscopy

Laryngeal evaluation requires general anesthesia (see p. 834 for general anesthesia during laryngeal examination). A rigid laryngoscope is best suited for this examination. Patients with laryngeal collapse have a reduced glottic lumen aperture. Stage 1 laryngeal collapse (lateral saccule eversion) is recognized as prolapsed, edematous mucosa just rostral to the vocal cords at the ventral aspect of the glottis (see p. 851). Stage 2 laryngeal collapse is present when one or both aryepiglottic folds are deviated medially and obstruct the ventral aspect of the glottis (Fig. 28.27). Stage 3 laryngeal collapse occurs when the corniculate processes of the arytenoid cartilages deviate medially from their normal paramedian position and are not adequately abducted during inspiration. The cartilages often have a flaccid appearance.

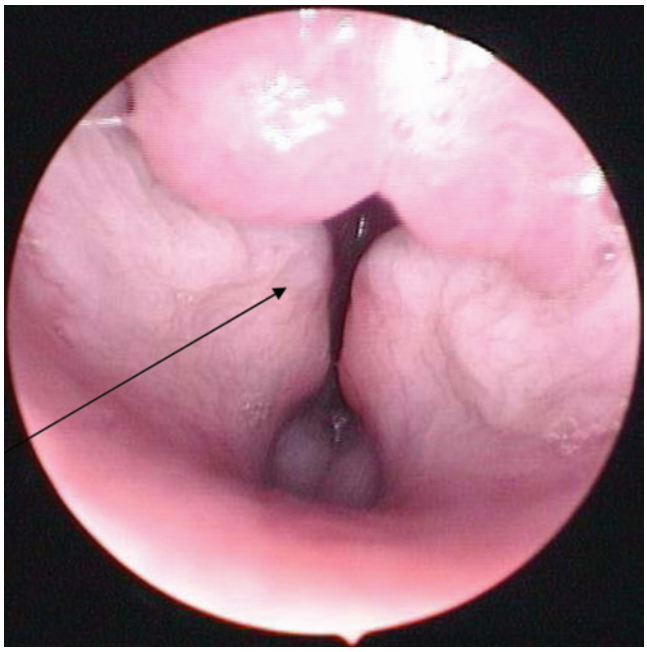


FIG. 28.27 Stage 2 laryngeal collapse is seen in this young English bulldog, along with elongation of the soft palate and everted laryngeal saccules. Cuneiform processes in the aryepiglottic folds collapse medially with stage 2 laryngeal collapse *(arrow)*.

Flexible endoscopy can be used to evaluated the nasopharynx, trachea, and bronchial tree for concurrent disease.

Laboratory Findings

Hematologic and serum biochemistry findings are usually normal (see also p. 853).

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include laryngeal or tracheal obstruction caused by masses, paralysis, everted laryngeal saccules, elongated soft palate, and stenotic nares.

MEDICAL MANAGEMENT

Medical therapy is recommended to alleviate acute respiratory distress. A weight reduction program should be instituted for obese animals. Exercise restriction and elimination of precipitating causes may be beneficial when clinical signs are mild.

Sedation (see Box 28.2), glucocorticoids, supplemental oxygen (see Table 4.4), and cooling may be necessary for moderate to severe respiratory distress. A single, preoperative antiinflammatory dose of a glucocorticoid (e.g., dexamethasone sodium phosphate 0.1–0.5 mg/kg IV) may reduce nasopharyngeal and upper airway edema secondary to surgical or diagnostic manipulations. Start

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with the lower dose and use the higher dose only if the low dose has failed to reduce swelling and if swelling is sufficiently severe that the animal cannot breathe. Note that large doses or repeated administration of dexamethasone typically causes gastrointestinal erosion/ulceration. Prolonged medical therapy for the respiratory signs may allow progression of degenerative laryngeal changes.

SURGICAL TREATMENT

In stable patients, the first step in treatment of laryngeal collapse is to treat concurrent abnormalities (e.g., resection of stenotic nares [see p. 854], elongated soft palate [see p. 854], and everted laryngeal saccules [see p. 855]). Resection of the aryepiglottic fold (see later discussion) may be performed in patients with mild to moderate stage 2 laryngeal collapse but is rarely needed. Permanent tracheostomy (see p. 843) is recommended for patients with advanced laryngeal collapse and moderate to severe respiratory distress that do not or are not expected to respond to resection. Partial laryngectomy (see p. 861) or lateralization (see p. 861) procedures are seldom beneficial because the weakened cartilages generally continue to collapse medially. Creation of a modified castellated laryngofissure that widens the ventral glottis is an alternative procedure proposed by some. Unilateral arytenoid laryngoplasty (i.e., cricoarytenoid lateralization combined with thyroarytenoid caudolateralization) has been reported to have reasonable long-term outcomes in a small number of brachycephalic dogs with laryngeal collapse, but this technique should be used with caution as the opposite cartilage may continue to collapse medially.[7]

Preoperative Management

These patients should be stabilized before surgery (see discussion under Medical Management) and observed closely for worsening dyspnea. Pretreatment with an antiinflammatory dose of a glucocorticoid (e.g., dexamethasone sodium phosphate 0.1–0.5 mg/kg IV) may reduce nasopharyngeal and upper airway edema secondary to surgical or diagnostic manipulations. Start with the lower dose and use the higher dose only if the low dose has failed to reduce swelling and if swelling is sufficiently severe that the animal cannot breathe.

Anesthesia

Anesthesia of animals with brachycephalic syndrome is described on p. 853. General anesthetic recommendations for animals with upper respiratory disease are given on p. 834. Recommended anesthetics for laryngeal examination are provided in Table 28.1.

Surgical Anatomy

The surgical anatomy of the larynx is discussed on p. 838.

Positioning

For resection of the soft palate, laryngeal saccules, and aryepiglottic fold, the animal should be positioned in sternal recumbency with the maxilla suspended and the mouth wide open. Permanent tracheostomy is performed with the animal in dorsal recumbency (see p. 843).

SURGICAL TECHNIQUE

Aryepiglottic fold resection is accomplished through an oral approach. It is performed unilaterally in conjunction with resection of the nares, soft palate, and everted laryngeal saccules. *Grasp and stabilize the fold with forceps. Transect the fold and cuneiform process with Mayo scissors or uterine biopsy forceps. Allow secondary intention healing.*

Unilateral arytenoid lateralization is described on p. 861.

POSTOPERATIVE CARE AND ASSESSMENT

These patients should be monitored continuously during anesthetic recovery for signs of airway obstruction. See postoperative recommendations for animals with brachycephalic syndrome on p. 856. Patients with advanced laryngeal collapse may develop acute respiratory obstruction after surgery. Tracheostomas should be managed as described on p. 848 to prevent occlusion of the stoma with mucus.

PROGNOSIS

Moderate to severe respiratory distress will persist without surgical intervention. Acute inflammation may cause cyanosis, collapse, and respiratory arrest. The prognosis for improvement with stage 3 laryngeal collapse after surgery is poor if resection of the nares, soft palate, and laryngeal saccules is not done concurrently, but may be fair to good in animals with stage 2 collapse. The prognosis in advanced laryngeal collapse (stage 3) is fair to good if concurrent permanent tracheostomy is performed.

LARYNGEAL PARALYSIS

DEFINITION

Laryngeal paralysis is complete or partial failure of the arytenoid cartilages and vocal folds to abduct during inspiration.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Laryngeal paralysis causes upper respiratory obstruction and mild to severe dyspnea. It occurs because of dysfunction of the laryngeal muscles, recurrent laryngeal or vagus nerves, or cricoarytenoid ankylosis; acquired or congenital neurologic causes are most common. The intrinsic laryngeal abductor and adductor muscles are innervated by the recurrent laryngeal nerves (see p. 839). Atrophy of the cricoarytenoideus dorsalis muscle causes the cartilages to remain in a paramedian position during inspiration, preventing maximal air intake and increasing airflow resistance. The narrowed rima glottidis increases resistance to airflow and creates turbulence, which gives rise to laryngeal stridor. To maintain the same flow rate through the paralyzed larynx as is seen in the remainder of the respiratory tract, the speed of airflow through the larynx must increase. Consequently, intraglottic pressure drops, and the arytenoid cartilages and vocal cords are sucked in medially, further increasing laryngeal obstruction. Ineffective laryngeal adduction and closure during swallowing predispose the patient to aspiration of food and secretions, resulting in subsequent aspiration pneumonia.

Congenital, inherited laryngeal paralysis occurs in Bouviers des Flandres, bull terriers, Siberian huskies, and white-coated German shepherd dogs. Laryngeal paralysis in Bouviers is due to degeneration of the nucleus ambiguus. A congenital laryngeal paralysis–polyneuropathy complex associated with dying back

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BOX 28.9 Proposed Etiologies of Laryngeal Paralysis

Congenital
Genetic trait
Laryngeal paralysis: polyneuropathy complex

Accidental Trauma
Cervical penetrating wounds
Strangulating trauma

Iatrogenic Surgical Trauma
Cranial thoracic surgery
Thyroidectomy/parathyroidectomy
Tracheal surgery
Ventral slot

Cervical/Intrathoracic Neoplasia
Lymphoma
Thymoma
Thyroid carcinoma/ectopic thyroid carcinoma

Neuromuscular Disease
Geriatric-onset laryngeal paralysis polyneuropathy syndrome
Endocrinopathy (hypothyroidism, hypoadrenocorticism)

Immune Mediated
Infectious
Myasthenia gravis
Polymyopathy

Systemic Lupus Erythematosus
Toxins (lead; organophosphates)

of peripheral nerves has been recognized in Dalmatians, Rottweilers, Leonberger dogs, and Pyrenean mountain dogs.

Acquired laryngeal paralysis is caused by damage to the recurrent laryngeal nerve or intrinsic laryngeal muscles most often attributed to polyneuropathy, polymyopathy, accidental or iatrogenic trauma, or intrathoracic or extrathoracic masses, although many other causes have been proposed (Box 28.9). It has been shown that many dogs thought to have idiopathic acquired laryngeal paralysis develop systemic neurologic signs within 1 year following diagnosis of laryngeal paralysis, which is consistent with progressive generalized neuropathy.[15] Abnormalities in the results of electrodiagnostic tests and histopathologic analysis of nerve and muscle biopsy specimens reflecting generalized polyneuropathy have been documented in a small number of dogs with acquired laryngeal paralysis.[16] Therefore it has been proposed that dogs previously believed to have idiopathic laryngeal paralysis may in fact have a progressive generalized polyneuropathy.[17,18] The abbreviation GOLPP (geriatric onset laryngeal paralysis polyneuropathy) has been proposed as a more accurate term for dogs with acquired laryngeal paralysis where other causes have been ruled out.[15]

DIAGNOSIS

Clinical Presentation

Signalment

Laryngeal paralysis is more common in large-breed than in small-breed dogs. Males are affected two to four times more often than females. Acquired idiopathic laryngeal paralysis is most common in middle-aged or older dogs. The Labrador retriever is by far the most common breed reported, but golden retrievers, Saint Bernards, Newfoundlands, Irish setters, and Brittany spaniels are overrepresented. Congenital laryngeal paralysis should be suspected in young (<1 year of age) large-breed dogs with upper airway obstruction (see previous discussion).

Laryngeal paralysis is an uncommon condition in the cat. Clinical presentation is similar to that of the dog; however, cats with unilateral laryngeal paralysis can present with significant clinical signs, unlike dogs, which are rarely symptomatic. A prevalence of left-sided unilateral laryngeal paralysis has been noted in cats, similar to what is reported in humans and horses. The specific cause of laryngeal paralysis in cats is unknown, but several cases have been associated with trauma, neoplastic invasion, and iatrogenic damage.

History

Laryngeal paralysis frequently causes progressive inspiratory stridor, voice change, and exercise intolerance. These animals may also have increased stridor, dyspnea, cyanosis, coughing, gagging, vomiting, restlessness, and anxiety. Some animals are asymptomatic at rest. Obesity, exercise, excitement, and high ambient temperatures may exacerbate the clinical signs. Laryngeal paralysis occurs in approximately one-third of dogs with tracheal collapse. All animals with laryngeal paralysis are at risk for aspiration of food and saliva.

Physical Examination Findings

Physical examination findings are nonspecific for laryngeal paralysis. The animal may have labored breathing (from upper airway obstruction and/or pulmonary edema), continuous panting, and/or hyperthermia (from increased respiratory effort). Inspiratory stridor is often obvious. Patients with acquired laryngeal paralysis may have other neurologic signs and evidence of muscle wasting and weakness. Neurologic examinations should be performed to detect concurrent abnormalities. Coexistent cardiac, neurologic, gastrointestinal, and metabolic abnormalities may be recognized, making management more difficult.

Diagnostic Imaging

Lateral cervical and thoracic radiographs should be evaluated for abnormalities to rule out other causes of abnormal respiratory noises and dyspnea. Postobstruction pulmonary edema may occur in dogs and can be recognized as an interstitial pattern (sometimes coalescing to an alveolar pattern) on thoracic films. Laryngeal paralysis cannot be diagnosed radiographically. Animals with laryngeal paralysis are at risk for aspiration pneumonia. Ultrasound can also be used to evaluate laryngeal function.

Corrective surgery in animals with proximal esophageal dysfunction can cause devastating aspiration; therefore it is critical to define esophageal function preoperatively. Survey films of the esophagus are rarely adequate. Dogs may have substantial esophageal dysfunction with no history of regurgitation or plain radiographic evidence of esophageal dilation. Contrast barium esophagrams using fluoroscopy can be performed in dogs with clinical signs (dysphagia, regurgitation) reflective of esophageal dysfunction. However, acquiring information from esophagography should be weighed against the potential for risk of aspiration.

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Laryngoscopy

Laryngoscopy requires induction of light general anesthesia (e.g., propofol [preferred], alfaxalone, diazepam plus ketamine; see p. 834). Transnasal evaluation is possible in large, cooperative dogs using only sedation and topical lidocaine, and echolaryngography can be performed in conscious dogs; however, neither technique is as effective as direct laryngeal observation. Apparent laryngeal motion must be compared with the phase of respiration for interpretation. Intubation should be delayed until the larynx has been examined because placement of the endotracheal tube obscures visualization.

Laryngeal examination is usually best performed with a rigid laryngoscope but can be done with flexible scopes as well. In affected dogs, the laryngeal cartilages are located in a paramedian position and do not abduct during inspiration. Fluttering of the vocal folds and arytenoid cartilages during turbulent airflow must not be mistaken for purposeful abduction. Paradoxical vocal fold movement may occur and may be confused with normal movement; however, a normal larynx maximally abducts during inspiration, not expiration. Diagnosis may be additionally challenging with the presence of paradoxical movement of the arytenoids, resulting in a false-negative result. In this scenario, the arytenoid cartilages move inward during inspiration due to negative intraglottic pressure that is created by breathing against the glottic obstruction. The cartilages then passively return to their original position during the expiratory phase, which gives the impression of abduction. In questionable cases, give doxapram (Dopram, 1–2.2 mg/kg IV) to stimulate laryngeal motion if topical stimulation is unsuccessful in eliciting reflex activity, and have an assistant state the phase of ventilation during laryngoscopy to help in distinguishing normal from abnormal motion.

Laboratory Findings

Hematologic and serum biochemistry findings are usually normal. Hypothyroid neuropathy (see p. 615) should be excluded by evaluation of serum total thyroxine (T_4) and endogenous canine thyroid-stimulating hormone concentrations; approximately 30% to 40% of dogs with acquired laryngeal paralysis have concurrent hypothyroidism. Acquired myasthenia gravis can be diagnosed by measuring circulating antibodies to acetylcholine receptors. Administration of edrophonium chloride may be diagnostic for generalized myasthenia, but the test is not as sensitive or as specific as measuring antibodies to acetylcholine receptors (Box 28.10). The clinician must be aware of possible complications associated with the use of edrophonium (e.g., paralysis).

Additional Diagnostics

Electromyography using bipolar concentric needle electrodes can detect denervation of the laryngeal muscles (dorsal cricoarytenoid, ventricular, and thyroarytenoid). Nerve conduction studies help identify generalized neuromuscular disease. Histopathology may show loss of large-caliber nerve fibers and axonal degeneration in nerve biopsies and neurogenic atrophy in muscle specimens, consistent with generalized polyneuropathy.[16]

DIFFERENTIAL DIAGNOSIS

Other upper respiratory obstructions must be ruled out (e.g., brachycephalic syndrome, laryngeal collapse, tracheal collapse, masses or trauma involving the upper airway).

MEDICAL MANAGEMENT

For dogs that are presented in acute respiratory distress, initial treatment is directed at improving ventilation, reducing laryngeal edema, and minimizing the animal's stress. A typical treatment regimen involves oxygen supplementation and administration of short-acting glucocorticoids (e.g., dexamethasone sodium succinate) and sedatives (e.g., acepromazine). Additional administration of buprenorphine or butorphanol also may be considered (Box 28.11). These dogs are often also hyperthermic, and appropriate cooling procedures should also be instituted. If respiratory distress cannot be abated, intubation or a temporary tracheostomy should be considered. However, the use of a temporary tracheostomy tube in dogs with laryngeal paralysis has been shown to be a negative prognostic indicator following surgery, as dogs that received a temporary tracheostomy preoperatively were more likely to experience major complications. The presence of a tube within the tracheal lumen causes epithelial erosion, submucosal inflammation, and inhibition of the mucociliary apparatus from the level of the tracheostomy to the bifurcation. Mucus production dramatically increases, and the tube must be suctioned or cleaned at very frequent intervals to prevent it from clogging. Therefore intensive monitoring of a patient with a temporary tracheostomy tube is required to avoid life-threatening complications. These patients can also have postobstructional pulmonary edema, in which case furosemide may be helpful. Some dogs may have aspiration pneumonia, which necessitates aggressive antibiotic therapy (see Box 18.3).

Dogs that are mildly affected by laryngeal paralysis often require no treatment if they maintain a sedentary lifestyle and avoid excessive weight gain and stress. Small dogs are more successfully managed with medical therapy than large dogs. Medical therapy is recommended to alleviate acute respiratory distress. A weight reduction program should be instituted for obese animals. Exercise restriction and elimination of precipitating causes may be beneficial when clinical signs are mild. Hypothyroidism should be appropriately treated; however, no

BOX 28.10 Tensilon Test for Generalized Myasthenia Gravis

Edrophonium Chloride (Tensilon)[a]
Dogs: 0.1–0.2 mg/kg IV
Cats: 2.5 mg/cat IV

[a]Use with caution. Have oxygen available in case of respiratory arrest. Some advocate pretreating with glycopyrrolate.
IV, Intravenous.

BOX 28.11 Drugs Used for Acute Respiratory Distress

To Decrease Laryngeal Swelling:
Dexamethasone sodium phosphate: 0.1–1.0 mg/kg IV

To Abate Anxiety:
Acepromazine 0.01–0.02 mg/kg IV
Buprenorphine 0.0005–0.001 mg/kg IV
Butorphanol 0.1–0.25 mg/kg IV
Dexmedetomidine 1–2 µg/kg IV

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improvement in clinical signs related to laryngeal paralysis should be expected.

SURGICAL TREATMENT

Surgical treatment is recommended for patients with laryngeal paralysis that have moderate to severe signs of respiratory distress. The goal of treatment is to enlarge the glottis without promoting aspiration of food or saliva. Surgical intervention is indicated in dogs severely affected by laryngeal paralysis. Numerous techniques have been described to treat laryngeal paralysis. Unilateral arytenoid lateralization is the current technique of choice for most surgeons, but various types of partial laryngectomy (bilateral vocal fold resection, partial arytenoidectomy) are also performed. Bilateral arytenoid lateralization is not recommended as it results in unacceptable morbidity. Other techniques include castellated laryngofissure, reinnervation of the laryngeal musculature, and permanent tracheostomy. Castellated laryngofissure is performed rarely because of the technical difficulty of the procedure and inconsistent outcomes. Reinnervation does not provide immediate clinical relief, so it is therefore not a practical treatment option in dogs. Permanent tracheostomy is considered a salvage procedure for dogs most at risk of aspiration pneumonia, but it is associated with a high rate of major and minor complications and requires diligent postoperative and long-term care.

Preoperative Management

These patients should be observed closely before surgery for progressive respiratory distress. They should be kept cool, calm, and quiet. Pretreatment with an antiinflammatory dose of a glucocorticoid (e.g., dexamethasone sodium phosphate 0.1–0.5 mg/kg IV) immediately before surgery may be considered. Perioperative use of gastrointestinal motility modifiers is frequently advocated to reduce the incidence of postoperative aspiration pneumonia. However, two 2016 studies found no benefit in the use of metoclopramide to decrease the incidence of postoperative complications.[19,20]

Anesthesia

General anesthetic recommendations for animals with upper respiratory disease are given on p. 834. See Table 28.1 for anesthetic recommendations for laryngeal examination.

Surgical Anatomy

The cricoarytenoideus dorsalis is the abductor muscle of the larynx. It extends from the muscular process of the arytenoid cartilage in a dorsomedial direction to the cricoid cartilage. The surgical anatomy of the larynx is described on p. 838.

Positioning

For arytenoid cartilage lateralization, the animal should be positioned in lateral or dorsal recumbency with the neck over a rolled towel and rotated to elevate the ipsilateral mandible. The head is stabilized by taping it to the table. For partial laryngectomy via an oral approach, the patient is positioned in sternal recumbency with the head suspended by the maxilla and the mandible held open by an assistant or taped to the table. For partial laryngectomy via a ventral approach, the animal should be positioned in dorsal recumbency with the head extended and secured to the operating table. For arytenoid cartilage lateralization and partial laryngectomy via a ventral laryngotomy, the entire cervical area should be clipped and prepared for aseptic surgery.

SURGICAL TECHNIQUE

Unilateral Arytenoid Lateralization

AP Several variations of unilateral arytenoid lateralization have been described, including differences in the number and positioning of sutures, variations in the degree of disarticulation, and alterations in the amount of abduction. The most common technique involves suturing the cricoid cartilage to the muscular process of the arytenoid cartilage. This mimics the directional pull of the cricoarytenoid dorsalis muscle and rotates the arytenoid cartilage laterally.

Make a skin incision just ventral to the jugular vein, beginning at the caudal angle of the mandible and extending over the dorsolateral aspect of the larynx to 1 to 2 cm caudal to the larynx (Fig. 28.28A). Incise and retract the SC tissues, platysma, and parotidoauricularis muscles. Retract the sternocephalicus muscle and the jugular vein dorsally and the sternohyoid muscle ventrally to expose the laryngeal area. Palpate the dorsal margin of the thyroid cartilage. Incise the thyropharyngeus muscle along the dorsolateral margin of the thyroid cartilage lamina. Place a stay suture through the thyroid cartilage lamina to retract and rotate the larynx laterally. Identify and transect the cricoarytenoideus dorsalis muscle. Disarticulate the cricothyroid articulation with a No. 11 blade or scissors (Fig. 28.28B); however, this step is often unnecessary. Palpate, identify, and disarticulate the cricoarytenoid articulation at the muscular process. Place a nonabsorbable monofilament suture (e.g., 2-0 to 0 polypropylene [Prolene], polybutester [Novafil]) from the caudal one-third of the cricoid cartilage near the dorsal midline to the muscular process of the arytenoid cartilage to mimic the direction of the cricoarytenoid dorsalis muscle (Fig. 28.28C). A 2014 in vitro study found that placement of the cricoarytenoid suture through the cricoid cartilage at the caudal border of the cricoarytenoid articulation was appropriate to sufficiently reduce laryngeal airway resistance without increasing the risk of aspiration pneumonia through overabduction of the arytenoid cartilage.[21] As an alternative or in addition, a suture can be placed through the most caudodorsal aspect of the thyroid cartilage and the muscular process. Muscular process–to–thyroid cartilage sutures tend to pull the arytenoid laterally, whereas muscular process–to–cricoid cartilage sutures tend to rotate the arytenoid laterally.

Tie the suture with enough tension to abduct the arytenoid cartilage moderately. Have an assistant verify abduction by intraoral visualization of the larynx. If abduction is insufficient, the suture can be repositioned to achieve better abduction. *Lavage the surgical site. Appose the thyropharyngeus muscle in a cruciate or simple continuous pattern with 3-0 absorbable suture. Appose the SC tissues and skin routinely.*

Partial Laryngectomy

AP Partial laryngectomy may be done by an oral approach or by a ventral laryngotomy approach; however, in dogs with laryngeal paralysis, the ventral approach is recommended. Bilateral vocal fold resection (ventriculocordectomy) alone or in conjunction with unilateral resection of the corniculate, cuneiform, and vocal processes of the arytenoid cartilage may be performed; however, bilateral vocal fold resection alone results in fewer complications and better postoperative outcome than other partial laryngectomy techniques.

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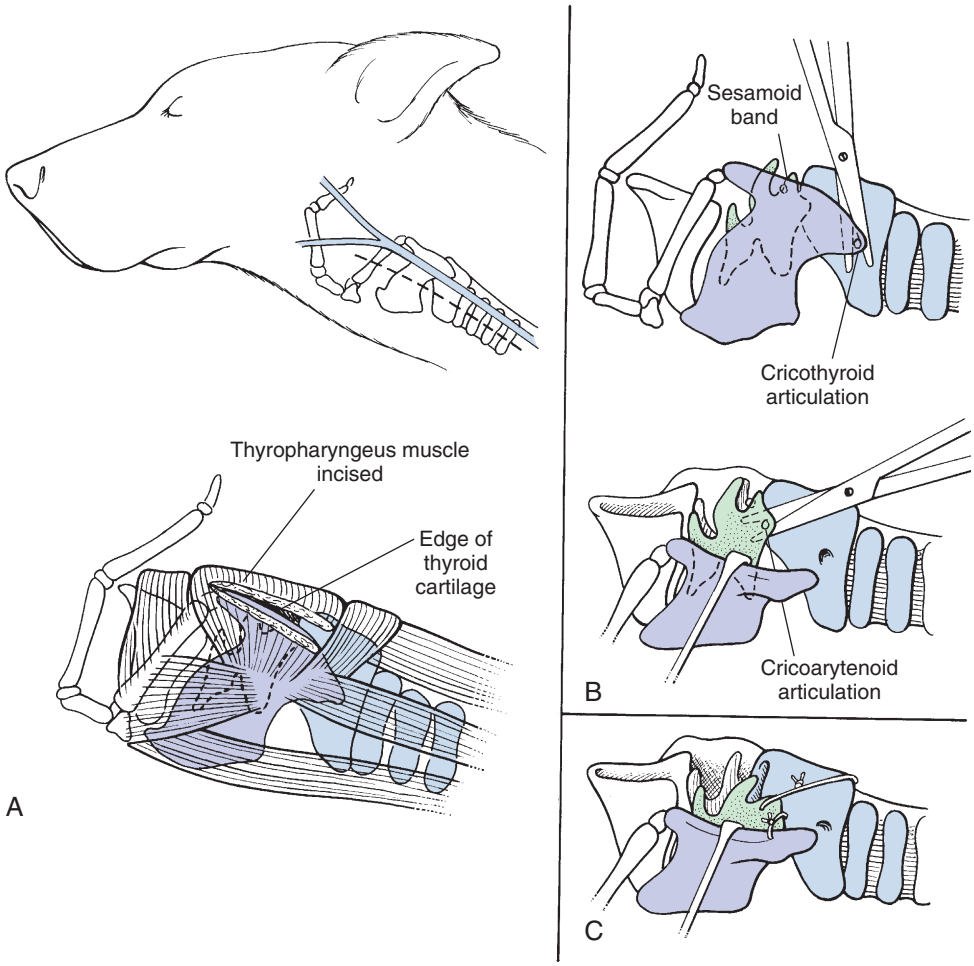


FIG. 28.28 (A) The larynx is exposed through a lateral cervical approach during arytenoid lateralization. Incise the skin and subcutaneous tissue ventral to the jugular vein. Then incise the thyropharyngeus muscle at the dorsal edge of the thyroid cartilage. (B) Separate the cricothyroid (if inclined) and cricoarytenoid articulations. (C) Place a suture from the muscular process of the arytenoid to the dorsocaudal aspect of the cricoid or thyroid cartilage.

Bilateral Ventriculocordectomy via Ventral Laryngotomy

Make a ventral midline incision over the larynx. Separate the sternohyoid muscles and incise the cricothyroid membrane and thyroid cartilage on the midline to expose the laryngeal lumen. Retract the edges of the thyroid cartilage with small Gelpi or Weitlaner retractors to visualize the vocal folds. Sharply excise the vocal fold and vocalis muscle on one side. Suture the mucosal defect with 4-0 to 5-0 monofilament rapidly absorbable suture in a simple continuous pattern. Repeat the procedure on the opposite side. Reappose the thyroid cartilage with partial-thickness interrupted monofilament sutures that do not penetrate the laryngeal lumen. Close the muscle, SC tissues, and skin routinely.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperatively, these animals should be monitored closely for respiratory distress caused by airway obstruction. Analgesics should be given as necessary. Gagging and coughing may occur in the early postoperative period. Swallowing discomfort may occur, and impaired glottic function may persist. IV fluids should be maintained until the animal is drinking. Soft food in meatball form may be offered shortly after the procedure, but the animal should be observed for evidence of aspiration (coughing, dyspnea). Exercise restriction should be enforced for 6 to 8 weeks and barking minimized. Occasional coughing occurs in most dogs. The bark is expected to be quiet and hoarse.

COMPLICATIONS

Early complications of suture lateralization include hematoma formation, swallowing discomfort, temporary glottic impairment, incisional issues, and coughing after eating and drinking. These complications usually resolve within a few days unless aspiration occurs. Coughing and gagging after surgery may indicate mucosal irritation or aspiration. Although bilateral arytenoid lateralization enlarges the glottis to a greater extent than unilateral lateralization, it is not routinely recommended because postoperative coughing, pneumonia, and death occur more often. Severe mucosal inflammation and swelling are rare after lateralization; therefore acute

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respiratory distress is unlikely. The cartilages of congenitally affected dogs may be insufficiently mineralized to retain sutures. Mineralized cartilages of older dogs may fracture or avulse the muscular process, causing failure of abduction and recurrence of clinical signs. If these events occur, the procedure may be repeated on the opposite side of the larynx.

Aspiration pneumonia occurs in approximately 10% to 20% of dogs after surgery for laryngeal paralysis. It may occur any time after surgery. Factors associated with a high risk of developing aspiration pneumonia include increasing age, temporary tracheostomy, progressive neurologic disease, postoperative megaesophagus, esophageal disease, concurrent neoplastic disease, and postoperative administration of opioids.

PROGNOSIS

Animals with mild or no clinical signs at rest do well without surgery; however, those with moderate to severe clinical signs may develop laryngeal collapse and acute respiratory obstruction. The prognosis after unilateral lateralization is good; more than 90% of patients have less respiratory distress and improved exercise tolerance. Reported 1-, 2-, 3-, and 4-year survival rates were 93.6%, 89.1%, 84.4%, and 75.2%, respectively, following unilateral arytenoid lateralization.[20]

EPIGLOTTIC RETROVERSION

DEFINITIONS

Epiglottic retroversion is the spontaneous retroflexion of the epiglottis during inspiration causing obstruction of the rima glottidis. *Epiglottopexy* is a method of adhering the epiglottis, either temporarily or permanently, to the base of the tongue to prevent laryngeal obstruction. *Subtotal epiglottectomy* is removal of portion of the epiglottis.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Epiglottic retroversion has been an infrequent cause of upper airway obstruction in dogs but is being recognized more commonly. Based on information from humans and horses, the theories about etiology in dogs include hypothyroid-associated peripheral neuropathy, epiglottic fracture or malacia, and denervation of the hypoglossal nerve or the glossopharyngeal nerve or both. Evaluation of this condition found that almost 80% of dogs diagnosed with epiglottic retroversion had concurrent or historical upper airway disorders.[18] Therefore epiglottic retroversion may most likely occur secondary to chronic increased inspiratory airway pressures that can occur with other causes of upper airway obstruction.

DIAGNOSIS

Clinical Presentation

Signalment

The most common breed reported to have epiglottic retroversion is the Yorkshire terrier. In general, mesocephalic and brachycephalic small- and medium-sized breeds have been most frequently reported to have this condition. Dogs tend to present at 8 years of age or older, although it has been reported in dogs less than 3 years old.

History

Dogs present with acute or chronic signs of upper airway obstruction: respiratory distress caused by inspiratory stridor. Signs may be intermittent, with dogs being clinically normal between episodes. Respiratory distress may be precipitated by stress or exercise. In some dogs, clinical signs may be worse when the animal is sleeping.

Physical Examination Findings

Physical examination findings are nonspecific for epiglottic retroversion. Inspiratory stridor may be obvious, but as it can be a sporadic condition, evidence of upper airway obstruction may not be apparent at initial examination.

Laboratory Findings

Hematologic and serum biochemistry findings are usually normal. Hypothyroid neuropathy (see p. 615) should be excluded, as a weak association has been reported in two dogs, although none of the dogs screened in the most recent study were hypothyroid.[22,23]

Diagnostic Imaging

Thoracic radiographs should be evaluated for abnormalities to rule out other causes of abnormal respiratory noises and dyspnea, as well as to rule out concurrent aspiration pneumonia or noncardiogenic pulmonary edema. Fluoroscopic examination of the larynx can be utilized to diagnose epiglottic retroversion, demonstrating entrapment of the epiglottis during inspiration.

Laryngoscopy

Laryngoscopy requires induction of light general anesthesia (e.g., propofol [preferred], alfaxalone, diazepam plus ketamine; see p. 834). Doxapram (Dopram, 1–2.2 mg/kg IV) can be used to stimulate respiration. Epiglottic retroversion is diagnosed when the epiglottis is noted to move caudally during inspiration resulting in obstruction of the rima glottidis. Flattening of the epiglottis and loss of epiglottic concavity can also be observed. In severely affected cases, the epiglottis may require manual manipulation to return it to its normal position. Dogs should also be evaluated for laryngeal paralysis, laryngeal collapse, elongated soft palate, tracheal collapse, and bronchial collapse.

DIFFERENTIAL DIAGNOSIS

Other upper respiratory obstructions must be ruled out (e.g., brachycephalic syndrome, laryngeal collapse, tracheal collapse, masses or trauma involving the upper airway).

MEDICAL MANAGEMENT

Symptomatic therapy may decrease the frequency or severity of clinical signs in dogs with mild clinical signs or when epiglottic retroversion is diagnosed as an incidental finding. Concurrent medical treatment may also improve response to surgical intervention. Such therapies include cough suppression, antimicrobial therapy, gastroprotection, antiinflammatory glucocorticoid therapy, and sedation.

SURGICAL TREATMENT

Surgical treatment is recommended for most patients diagnosed with epiglottic retroversion. Surgical interventions include

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temporary epiglottopexy, permanent epiglottopexy, and subtotal epiglottectomy. *Temporary epiglottopexy* is most often recommended for dogs with concurrent upper airway obstruction that can be successfully addressed. *Permanent epiglottopexy* can be used in animals with no concurrent airway condition, or when temporary fixation markedly improved clinical signs but then failed. *Subtotal epiglottectomy* is a salvage procedure that may be considered when epiglottopexy fails.

Preoperative Management

These patients should be managed as described earlier for animals with respiratory distress. Pretreatment with an antiinflammatory dose of a glucocorticoid (e.g., dexamethasone sodium phosphate 0.1–0.5 mg/kg IV) immediately before surgery may be considered. See p. 861.

Anesthesia

General anesthetic recommendations for animals with upper respiratory disease are given on p. 834. See Table 28.1 for anesthetic recommendations for laryngeal examination.

Surgical Anatomy

The surgical anatomy of the larynx is described on p. 838.

Positioning

The animal should be positioned in sternal recumbency with the head suspended by the maxilla and the mandible held open by an assistant or taped to the table.

SURGICAL TECHNIQUE

For temporary epiglottopexy, place two to six absorbable or nonabsorbable monofilament sutures in a horizontal mattress pattern between the lingual surface of the epiglottis and the base of the tongue. Ensure that each suture engages the epiglottic cartilage. For permanent epiglottopexy, perform the procedure as described for temporary epiglottopexy, except remove a crescent-shaped wedge of mucosa from the lingual surface of the epiglottis prior to suture fixation. You may also remove a corresponding section of tissue from the base of the tongue.

For subtotal epiglottectomy, use Metzenbaum scissors, a radiofrequency surgical unit, or a carbon dioxide laser to transect the epiglottis at the widest part, approximately 0.5 to 1 cm from the tip of the epiglottis. Close incised mucosal edges using 3-0 to 4-0 rapidly absorbable suture in a simple continuous pattern to cover the exposed cartilage.

COMPLICATIONS

Epiglottopexy, temporary or permanent, is associated with a high rate of failure due to suture breakage, pull-out, or loosening. Surgical failure results in return of clinical signs, or potentially a respiratory crisis. Based on a relatively small number of cases, postoperative aspiration pneumonia has been reported to occur within 2 to 270 days after surgery in approximately 33% of dogs.[22] In the few cases of subtotal epiglottectomy that have been reported, no episodes of postoperative aspiration pneumonia have occurred.

PROGNOSIS

Following surgery, marked improvement of clinical signs occurred in 52% of cases; overall median survival was 875 days.[22]

LARYNGEAL AND TRACHEAL TUMORS

DEFINITION

Oncocytomas arise from epithelial cells called *oncocytes,* small numbers of which are found in various organs, such as the larynx, thyroid, pituitary, and trachea.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Tumors of the larynx are uncommon in the dog and cat. Numerous types of tumor have been reported in the dog, including rhabdomyosarcoma, squamous cell carcinoma, adenocarcinoma, osteosarcoma, chondrosarcoma, chondroma, myxochondroma, lipoma, fibrosarcoma, undifferentiated carcinoma, extramedullary plasmacytoma, and mast cell tumor. Squamous cell carcinoma and lymphoma are the most common tumors of the larynx in the cat, but adenocarcinoma and other poorly differentiated round cell tumors have been reported (Box 28.12). Rhabdomyomas and oncocytomas are laryngeal tumors that appear histologically similar with light microscopy; electron microscopy and immunocytochemistry are necessary to distinguish them. Oncocytomas have been reported in young dogs and warrant special consideration because long-term survival of patients without metastasis has been reported after surgical resection (Fig. 28.29).

Inflammatory laryngeal disease is an uncommon nonneoplastic condition of the arytenoid cartilages of the larynx that has been reported in both dogs and cats. It can be granulomatous, lymphocytic-plasmacytic, or eosinophilic in nature, with multiple factors likely contributing to development of the disease. Severe cases can result in laryngeal stenosis and significant upper airway obstruction. Biopsy of the laryngeal mass is crucial to differentiate this disease from neoplasia, although it is still possible that inflammatory changes may represent a secondary response to underlying neoplasia. Treatment of inflammatory laryngeal disease is palliative and consists of debulking of the mass, steroid therapy, or permanent tracheostomy. Benign laryngeal cysts have been described in isolated feline cases. Cysts are typically epithelial in origin and stem from the ventral aspect of the larynx.

Tracheal tumors are even less common than laryngeal masses. Malignant and benign tracheal tumors have been reported

BOX 28.12 Laryngeal Tumors

Malignant
Squamous cell carcinoma
Lymphoma
Osteosarcoma
Fibrosarcoma
Rhabdomyosarcoma
Melanoma
Mast cell tumor
Other sarcomas
Granular cell myoblastoma
Adenocarcinoma
Undifferentiated carcinoma

Benign
Lipoma
Oncocytoma
Rhabdomyoma

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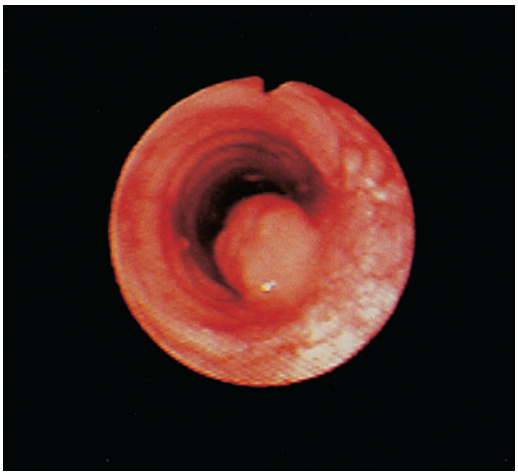


FIG. 28.29 Tracheal oncocytoma in a 7-year-old basset hound that was brought in for treatment of acute respiratory distress.

BOX 28.13 Tracheal Tumors

Malignant
Osteosarcoma
Chondrosarcoma
Lymphoma
Mast cell tumor
Adenocarcinoma
Squamous cell carcinoma
Rhabdomyosarcoma
Undifferentiated carcinoma
Melanoma

Benign
Osteochondroma
Oncocytoma
Leiomyoma
Chondroma
Extramedullary plasmacytoma
Polyp

(Box 28.13). Tracheal osteochondromas may occur in dogs younger than 1 year of age. These masses probably reflect a malfunction of osteogenesis and are benign. Their growth is expected to stop with skeletal maturity. In cats, tracheal squamous cell carcinomas, adenocarcinomas, and lymphosarcomas have been reported. Metastatic thyroid carcinomas, melanomas, lymphomas, and pharyngeal rhabdomyosarcomas may also involve the larynx and trachea. The incidence of metastasis of laryngeal and tracheal tumors in dogs and cats is unknown.

Oslerus osleri (Filaroides osleri) is a nematode that forms neoplastic-appearing nodules in the canine trachea and mainstem bronchi (Fig. 28.30). Nonendoscopic diagnosis of filaroidosis (i.e., finding larvae by Baermann fecal examination) is difficult because larvae are intermittently shed in the feces. Diagnosis is best made by finding larvae or adults by tracheal cytology. Anthelmintic therapy and surgical resection have met with varying success. Aberrant *Cuterebra* larvae and associated tissue trauma may obstruct the laryngeal or tracheal lumen. Other nonneoplastic differentials for tracheal masses include inflammatory nodules, including lymphoplasmacytic inflammation, lymphoid hyperplasia, and granulomatous tracheitis. All of these lesions must be differentiated from neoplastic lesions.

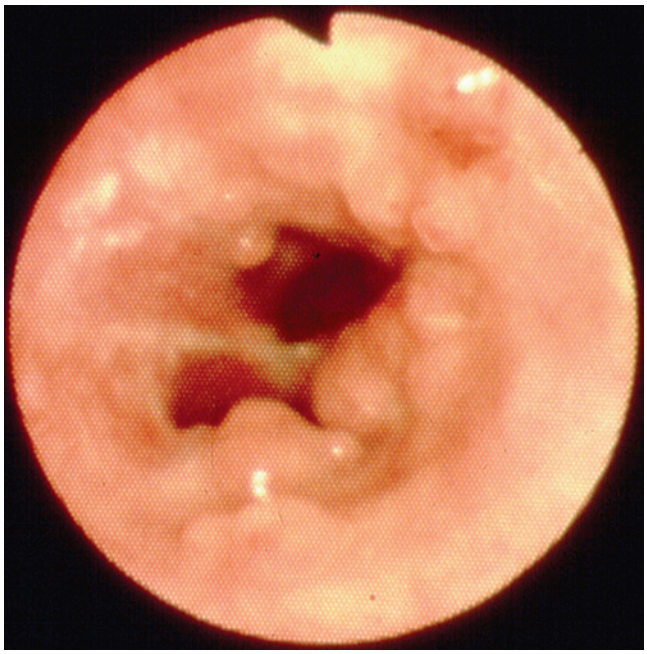


FIG. 28.30 *Oslerus osleri (Filaroides osleri)* nodules at the carina of the trachea.

Laryngeal and tracheal masses cause luminal obstruction by occupying space or compressing the lumen externally. As lumen size decreases, signs of respiratory distress become apparent (see p. 870). Acute respiratory distress, cyanosis, or collapse (or all of these) may occur if excitement, stress, high temperature, or infection causes mucosal swelling of an already compromised lumen.

DIAGNOSIS

Clinical Presentation

Signalment

Laryngeal and tracheal tumors occur most often in middle-aged to older animals (i.e., 5–15 years), with a median age of 12 years for cats and 10 years for dogs, with the exception of osteochondromas, which usually occur before 2 years. Tumors arising in older animals are more likely to be malignant; however, laryngeal oncocytomas and tracheal osteochondromas may occur in young animals. Benign tracheal osteocartilaginous tumors (osteochondromas) are most common in animals with active osteochondral ossification; they grow with the rest of the musculoskeletal system and may be recognized before 1 year of age. Siamese cats may be prone to lymphoma.

History

Animals with laryngeal or tracheal tumors may have an acute or progressive history of upper airway obstruction. Signs may include dysphonia, stridor, dyspnea, cough, decreased exercise tolerance, voice change, hyperthermia, ptyalism, gagging, dysphagia, cyanosis, or syncope, or all of these. Weight loss and lethargy may also be reported. Development of a mass in the ventral neck may be reported.

Physical Examination Findings

Besides respiratory difficulties, the physical examination is usually normal unless concurrent diseases or abnormalities are present.

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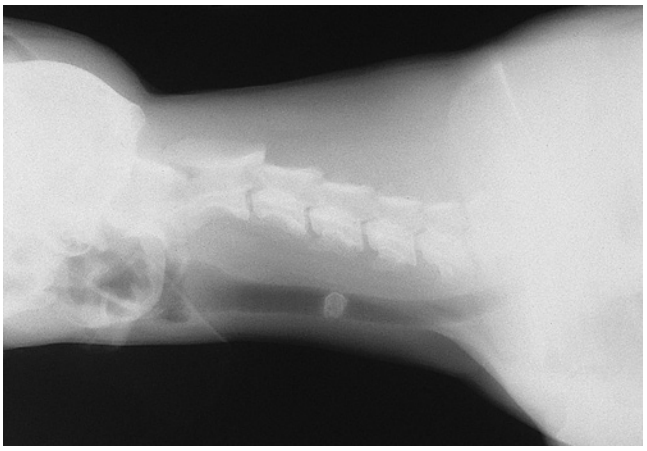


FIG. 28.31 Tracheal osteochondroma in a 6-month-old dog.

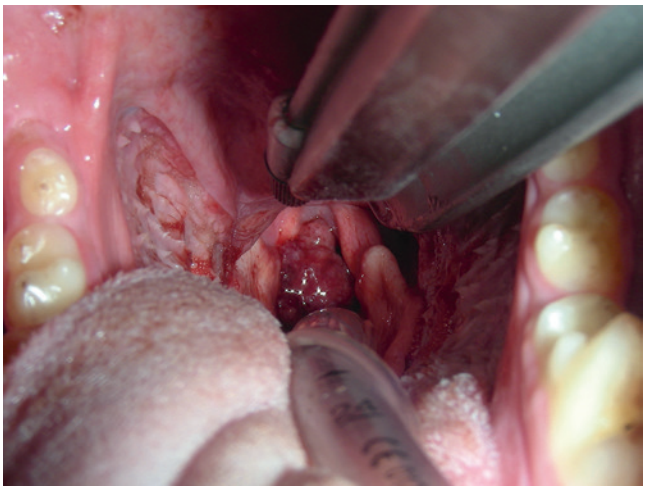


FIG. 28.32 Laryngoscopic view of a laryngeal squamous cell carcinoma in a dog.

Occasionally, extraluminal masses may be palpated along the ventral neck, and tracheal palpation may elicit coughing or increased dyspnea. A voice change may be noted in animals with laryngeal masses. Enlarged, accessible lymph nodes should be aspirated or a biopsy taken (see p. 633) to help stage the disease. The medial retropharyngeal lymph nodes drain the larynx and proximal trachea but are usually inaccessible.

Diagnostic Imaging

Pharyngeal and cervical radiographs should be evaluated to determine the location and extent of the tumor. Laryngeal and tracheal masses may appear as soft tissue densities in the airway (Fig. 28.31). Laryngeal distortion, decreased margination of laryngeal structures, and decreased laryngeal space (stenosis) may also be seen. Extraluminal masses may compress the tracheal lumen.

Thoracic radiographs should be evaluated for metastasis and bronchopneumonia. In rare cases, the lungs may appear to be hyperinflated if a tracheal or bronchial mass is large enough to act as a one-way valve. Occasionally, contrast esophagrams or esophagoscopy is needed to rule out esophageal involvement. Cervical ultrasound can be also used to assess masses and regional lymph nodes in this region and guide fine needle aspiration or biopsy. CT and magnetic resonance imaging (MRI) are expected to more precisely demonstrate the mass and its invasiveness.

Laryngoscopy

Laryngoscopy is preferred for examination and biopsy of pharyngeal and laryngeal masses. If a mass or an abnormal structure is seen (Fig. 28.32), it may be aspirated or brushed, or a biopsy taken with punch-type biopsy forceps. Hemorrhage may be controlled with direct pressure.

Tracheoscopy

For tracheal masses, one should use a flexible bronchoscope with a small enough outer diameter to allow the patient to breathe easily around it, thus minimizing the likelihood of obstructing or traumatizing the trachea. If the patient is at risk for hypoxia, or if the procedure will take longer than a few minutes, one may attach a tube from the anesthetic machine to the biopsy port on the bronchoscope so that oxygen can be insufflated during the procedure. It is critical to use a judicious flow rate so as not to cause barotrauma. Alternatively, one may repeatedly insert and withdraw the scope until an adequate evaluation has been performed.

> **NOTE** If electrocautery through the endoscope will be used, room air must be insufflated instead of oxygen to prevent the mucosa from being ignited.

Most laryngeal and tracheal tumors are inflamed or edematous pink masses protruding into the lumen (see Fig. 28.32); however, some laryngeal tumors appear as a diffuse thickening. The size and consistency of the tumor and the nature of its attachment to the tracheal wall should be noted. Biopsy may be performed using biopsy forceps or cytology brushes. It is usually advisable to first attempt brush cytology because this is faster and less risky. If cytology is not diagnostic, then one may take a biopsy of the mass; however, one should inspect the mass first and note the blood supply. If numerous vessels are seen on the mass, then hemorrhage is a greater risk, and suction (usually through the endoscope) must be readily available.

Laboratory Findings

Complete blood count, serum biochemistry profile, and urinalysis are indicated to evaluate the patient's overall status and to look for paraneoplastic syndromes. If lymphoma is suspected, a bone marrow aspiration and (in cats) feline leukemia virus test are warranted.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include obstruction of the larynx caused by elongated soft palate, laryngeal collapse, laryngeal paralysis, nasopharyngeal polyp, foreign bodies, inflammation, cysts, granulomatous masses, and parasites *(O. osleri)*. Inflammatory lesions include lymphoplasmacytic and lymphoid hyperplastic masses. Other causes of tracheal obstruction include tracheal collapse or hypoplasia, foreign bodies, congenital malformations,

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cysts, inflammation, granulomatous masses, nodular amyloidosis, and excess respiratory secretions.

MEDICAL MANAGEMENT

Radiation therapy may help treat squamous cell carcinomas, mast cell tumors, and lymphomas, but little information is available. Some tumors (e.g., lymphomas, mast cell tumors, adenocarcinomas) often respond to chemotherapy. Permanent tracheostomy (see p. 843) can palliate signs of respiratory distress during medical therapy.

SURGICAL TREATMENT

Surgical excision may be curative if the tumor is benign, localized, and small. Small lesions may be resected by mucosal resection or partial laryngectomy through a ventral laryngotomy. Excision of cartilaginous tumors (e.g., chondroma or chondrosarcoma) can be excised with reasonable success. Aggressive surgical intervention involves complete laryngectomy with permanent tracheostomy, but has been reported only in isolated cases. Permanent tracheostomy (see p. 843) is a palliative option for obstructive laryngeal masses.

For removal of tracheal tumors, tracheal resection and end-to-end anastomosis are required (see p. 844). Depending on its elasticity, 20% to 50% of the trachea (i.e., usually eight to ten rings) may be resected. Resection of large tumors with a minimum of 1 cm of normal trachea on both sides of the mass is not always possible. When resection would be too extensive to achieve, end-to-end anastomosis, tracheal replacement, or prostheses may be considered but are rarely successful. Resection of a segment of the tracheal wall without complete transection (i.e., wedge resection) with reapposition of the cut edges is not recommended because it narrows, or kinks, the trachea, which interferes with airflow and mucociliary transport.

Preoperative Management

The animal should be kept calm to prevent progressive dyspnea; sedation may be necessary in some animals (see p. 834). An antiinflammatory dose of glucocorticoids may be administered if dyspnea is severe (e.g., dexamethasone sodium phosphate 0.1–0.5 mg/kg IV). Distressed animals should be oxygenated before surgery. An emergency tracheostomy may be necessary in severely dyspneic animals (see p. 842).

Anesthesia

Animals with laryngeal masses may require intubation via a pharyngostomy or tracheotomy incision (see p. 842). Intubation and ventilation of patients with intraluminal tracheal masses may require insertion of a small-diameter tube or tracheostomy distal to the obstruction. Specific anesthetic recommendations for animals with respiratory disease are given on p. 834.

Surgical Anatomy

The surgical anatomy of the larynx and trachea is discussed on pp. 838 and 839.

Positioning

Patients requiring laryngotomy or cervical tracheal resection should be positioned in dorsal recumbency with the neck deviated ventrally with a dorsally placed pad or roll (see Fig. 28.13). The entire caudal mandibular area, ventral neck, and cranial thorax should be prepared for aseptic surgery. Partial laryngectomy is performed with the patient in dorsal recumbency. Total laryngectomy may be performed with the patient initially positioned in ventral recumbency for oropharyngeal mucosal incision and then repositioned in dorsal recumbency to allow removal of the larynx and permanent tracheostomy.

SURGICAL TECHNIQUE

Partial Laryngectomy

AP Partial laryngectomy is performed using an oral or preferably ventral laryngotomy approach (see p. 862). Mucosal closure after tumor resection helps prevent the formation of scar tissue and is more readily achieved when a laryngotomy approach is used.

Using sharp dissection, remove the mass with a margin of normal tissue. If possible, preserve the lateral margin of the corniculate process to allow appropriate epiglottic protection of the glottis. Avoid bilateral disruption of the dorsal and ventral laryngeal commissures to reduce the risk of postoperative glottic stenosis.

Complete or Total Laryngectomy

AP Total laryngectomy requires the creation of a permanent tracheostomy. It is a difficult procedure that is not often performed. *Expose the larynx by a ventral midline cervical incision. Transect the right and left sternohyoid muscles from their insertion on the basihyoid bone. Disarticulate the hyoid apparatus between the keratohyoid and basihyoid articulations with the thyrohyoid bones. Dissect dorsolaterally and excise the thyropharyngeus and cricopharyngeus muscles bilaterally from their insertion on the thyroid cartilage. Incise the pharyngeal mucosa at the base of the epiglottis, preserving as much mucosa as possible while keeping tumor-free margins. Free the larynx by transecting between the cricoid and first tracheal cartilage or between the first and second tracheal cartilages. Remove additional tissue as necessary to achieve an en bloc resection. Lavage the surgical field. Begin reconstruction by closing the pharyngeal submucosa in a continuous pattern with 3-0 absorbable (e.g., polydioxanone [PDS], polyglyconate [Maxon]) suture.* This suture line will be under tension. *Attach the sternohyoid muscles to the basihyoid bone dorsal to the trachea. Place a closed suction or Penrose drain if dead space is not completely eliminated. As an alternative, incise and appose the pharyngeal mucosa through an oral approach.*

The technique for permanent tracheostomy must be varied when a complete laryngectomy is performed. This procedure is rarely performed and may be challenging. *Close the end of the proximal trachea with a series of interrupted horizontal sutures and then perform a permanent tracheostomy as described on p. 843, or divert and incorporate the transected proximal trachea to create the tracheostoma.*

To close the end of the proximal trachea, place a series of interrupted horizontal mattress sutures from the annular ligament or tracheal cartilage through the dorsal tracheal membrane. As an alternative, preserve a flap of dorsal tracheal membrane during resection, fold it over the end of the trachea, and secure it with interrupted sutures. To incorporate the proximal trachea in the tracheostoma, create the tracheostoma by first apposing the sternohyoid muscles dorsal to the trachea. Remove the ventral third of four to six tracheal cartilages, taking care to preserve the underlying tracheal mucosa. Elevate the dorsal tracheal membrane, apposing and suturing it

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directly to the skin proximally using 4-0 nonabsorbable suture. Excise excess skin surrounding the stoma and place intradermal sutures from the skin to the peritracheal tissues to create adhesions and prevent skin flaps. Incise the mucosa and suture it laterally and distally to the skin in a simple continuous suture pattern.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

A laryngoscope, bronchoscope, alligator biopsy instrument, needle biopsy instrument, and endoscopic biopsy forceps are used for laryngeal and tracheal biopsy. Instruments and suture material for laryngeal and tracheal surgery are discussed on p. 847.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperatively, these patients should be monitored carefully for signs of airway obstruction. Supplemental oxygen and glucocorticoids may be given if needed. Water should be offered 6 to 12 hours postoperatively and food 18 to 24 hours after surgery if gagging, regurgitation, or vomiting does not occur. The animal should be kept quiet without exercise for 2 to 4 weeks. Endoscopic reevaluation is recommended at 4 to 8 weeks to identify tumor recurrence or stenosis. Stenosis of greater than 20% leads to mucostasis and infection, whereas a decrease in lumen size of approximately 50% causes respiratory distress. Periodic physical and radiographic evaluation is recommended to check for metastasis or recurrence.

COMPLICATIONS

Dysphagia, gagging, and pharyngeal dehiscence may occur after complete laryngectomy. Some patients benefit from an esophageal (preferred) or gastric feeding tube (see pp. 95 and 97, respectively). Vocalization is absent after laryngectomy. Tracheostomas must be monitored closely to maintain patency and prevent self-trauma (see p. 848). Other complications of laryngectomy include fistulae secondary to pharyngeal dehiscence, hypoparathyroidism secondary to ischemia, and tumor recurrence or metastasis.

Dehiscence may occur after tracheal anastomosis if tension is excessive and if head and neck motion are not restricted. To relieve tension, the neck should be kept mildly to moderately ventroflexed by attaching a muzzle to a harness with a lead or by placing a suture from the chin to the manubrium for 2 weeks. SC emphysema may be evident with dehiscence or anastomotic leakage. Infection and fistula formation are possible. Mild stenosis (<20%) is expected with the split-cartilage technique, in which anastomotic tension is minimal.

PROGNOSIS

Although undoubtedly related to the tumor's histologic type, the prognosis is excellent with some tracheal tumors (e.g., oncocytomas, osteochondromas). The prognosis for laryngeal tumors is guarded because most cases are advanced at the time of diagnosis; only isolated cases of successful management have been reported. Little information is available on the biological behavior of laryngeal tumors; however, the long-term prognosis is generally poor. Without surgery, complete obstruction of the tracheal or laryngeal lumen and subsequent asphyxiation may occur. Radiation therapy may be a valuable adjuvant after surgery for patients with malignant tumors.

TRACHEAL COLLAPSE

DEFINITION

Tracheal collapse is a form of tracheal obstruction caused by cartilage flaccidity and flattening. Tracheal collapse is sometimes erroneously referred to in older reports as *congenital tracheal stenosis.*

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The cause of tracheal collapse is unknown and probably multifactorial. Proposed causes include genetic factors, nutritional factors, allergens, neurologic deficiency, small airway disease, and degeneration of cartilage matrices. Affected tracheal cartilages become hypocellular, and their matrices degenerate. Normal hyaline cartilage is replaced by fibrocartilage and collagen fibers, and the quantities of glycoprotein and glycosaminoglycans are diminished. The cartilages lose their rigidity and their ability to maintain normal tracheal conformation during the respiratory cycle. Tracheal collapse may be confined to an isolated segment or may involve the entire trachea and bronchial tree. Collapse typically occurs in a dorsoventral direction as the cartilages weaken and the dorsal tracheal membrane thins and lengthens; however, lateral collapse of the tracheal walls has been reported. Collapse in the cervical tracheal and thoracic inlet classically occurs on inspiration as pressure within the lumen drops and the walls are susceptible to atmospheric pressure; the intrathoracic trachea collapses on expiration. Although pressure within the trachea decreases on inspiration, luminal pressure still exceeds intrapleural pressure, which keeps airways open. On expiration, intrapleural pressure becomes less negative and exceeds intraluminal pressure. Dogs with weakened cartilages lack sufficient strength to withstand the increased intrapleural pressure. The thoracic inlet is most susceptible to tracheal collapse because this is the site of the equal pressure point: where intrapleural pressure equals intraluminal airway pressure, and where the transition from intrapleural to atmospheric pressure occurs (Fig. 28.33).

Collapse reduces the lumen size and interferes with airflow to the lungs. Abnormal respiratory noises, exercise intolerance, gagging, and varying degrees of dyspnea occur with tracheal



FIG. 28.33 Lateral radiograph of a 2-year-old Yorkshire terrier with tracheal collapse *(arrow)* at the thoracic inlet.

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collapse. Chronic inflammation of the tracheal mucosa causes coughing, which exacerbates inflammation. Persistent inflammation leads to squamous metaplasia of the respiratory epithelium and interferes with mucociliary clearance; therefore coughing becomes an important tracheobronchial clearing mechanism.

DIAGNOSIS

Clinical Presentation

Signalment

Typically, tracheal collapse occurs in toy- and miniature-breed dogs, most commonly toy poodles, Yorkshire terriers, Pomeranians, Maltese, and Chihuahuas. Males and females are affected equally. Tracheal collapse in larger dogs is usually associated with trauma, deformity, or intraluminal or extraluminal masses and should not be equated with tracheal collapse in toy-breed dogs. Tracheal collapse is classically described as occurring in middle-aged or older toy breeds (average, 6–8 years). However, it is frequently diagnosed in dogs with respiratory problems between 6 months and 5 years of age.

History

The onset of clinical signs often occurs before 1 year of age. Clinical signs often progress with age and include abnormal respiratory noise, dyspnea, exercise intolerance, cyanosis, and syncope. Some dogs never suffer respiratory distress, and others die of asphyxiation. Clinical signs are more severe in obese animals. Respiratory noises include wheezing, hacking, coughing, and stridorous breathing. Some dogs do not make abnormal respiratory noises. The cough may be productive or nonproductive but is classically a "goose honk" cough (Video 28.5). Coughing often becomes cyclic and paroxysmal. Gagging after coughing may occur in as many as 50% of cases. Signs may be elicited or exacerbated by tracheal infection, tracheal compression, exercise, excitement, eating, drinking, or hot, humid weather. Noxious stimuli (i.e., smoke and other respiratory irritants) may also precipitate clinical signs.

Dogs with tracheal collapse may suffer from a variety of concurrent problems. Almost 50% of dogs suffer from a degree of obesity that will worsen clinical signs. Laryngeal paresis or paralysis has been reported in 20% to 30% of dogs, and one-third of dogs have concurrent systolic heart murmur consistent with mitral valve insufficiency. Upper respiratory signs may be aggravated by an enlarged left atrium putting pressure on the carina and mainstem bronchi. At least 40% of dogs are thought to have a degree of dental or periodontal disease. Aspiration of oral bacteria into diseased airways is hypothesized to contribute to exacerbation of clinical signs caused by increased airway inflammation or increased coughing.

Concurrent hepatomegaly and hepatopathy are common in dogs with tracheal collapse. The reason for this association is unclear, although speculative theories include passive hepatic congestion and centrilobular liver cell necrosis secondary to chronic hypoxia.

Physical Examination Findings

Flaccid tracheal cartilages with prominent lateral borders are occasionally evident on palpation of the cervical trachea. Palpation may elicit paroxysmal coughing. Auscultation may localize abnormal respiratory noises and may identify mitral valve disease. A soft end-expiratory snapping together of the tracheal wall may be auscultated in dogs with intrathoracic tracheal collapse. Abnormal heart sounds may be associated with concurrent cardiac disease. Hepatomegaly has been associated with this syndrome in some patients and may result from venous congestion caused by cor pulmonale or fatty change.

Diagnostic Imaging

Inspiratory and expiratory lateral radiographs of the neck and thorax are diagnostic in approximately 60% of patients with severe tracheal collapse (>50% of the lumen). The cervical trachea is expected to collapse on inspiration and the thoracic trachea on expiration. Thoracic radiographs often reveal cardiomegaly and pulmonary disease as well; bronchiectasis was noted on 30% of thoracic radiographs from dogs with tracheal collapse. Fluoroscopy facilitates evaluation of dynamic movement of the trachea and mainstem bronchi through all phases of respiration and finds many cases missed by survey radiographs. However, fluoroscopy will miss collapse of the trachea in the lateral dimension. Confirming the presence or absence of tracheal or bronchial collapse can require multiple imaging modalities as well as bronchoscopy.[24]

Laryngoscopy

Laryngoscopy (see Table 28.1 and p. 860) should usually be done at the same time as tracheoscopy because laryngeal paralysis or collapse is present in approximately 30% of dogs with tracheal collapse.

Tracheoscopy/Bronchoscopy

Tracheobronchoscopy is indicated in animals with suspected tracheal collapse to confirm and grade the severity of collapse, to evaluate the entire tracheobronchial tree, and to collect airway samples for cytology and culture. One should use a rigid or flexible bronchoscope with a small enough outer diameter to allow the patient to easily breathe around it, thus minimizing the likelihood of obstructing or traumatizing the trachea. If the patient is at risk for hypoxia, or if the procedure will take longer than a few minutes, one may attach a tube from the anesthetic machine to the biopsy port on the bronchoscope so that oxygen can be insufflated during the procedure. However, it is critical to use a judicious flow rate so as not to cause barotrauma. Alternatively, one may repeatedly insert and withdraw the scope until an adequate evaluation has been performed. Cytology and cultures performed during tracheobronchoscopy are useful to the clinician in selecting antibiotics and determining further medical therapy.

Tracheal conformation should be evaluated to determine the location and severity of the collapse. It is often easier to insert the scope to the carina and then evaluate the trachea as it is withdrawn, but this may not be possible in critically ill patients. The entire trachea may be collapsed; however, one area of the trachea is often more severely affected and is used for classification purposes (Box 28.14). Grade I tracheal collapse is a 25% reduction in lumen diameter, with the trachealis muscle being slightly pendulous and the cartilages maintaining a somewhat circular shape. Grade II collapse is a 50% reduction in lumen diameter, with the trachealis muscle stretched and pendulous and the cartilages beginning to flatten. Grade III collapse is a 75% reduction in lumen diameter, with the trachealis more stretched and pendulous and the cartilages nearly flattened (Fig. 28.34). Grade IV collapse means that the lumen is essentially obliterated, and the tracheal cartilages are completely flattened and may invert to contact the trachealis muscle. Collapse of mainstem bronchi

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BOX 28.14 Grading Scheme for Tracheal Collapse

Grade 1
Relatively normal tracheal cartilage anatomy; redundant dorsal tracheal membrane decreases luminal diameter up to 25%

Grade 2
Mild to moderate flattening of tracheal cartilages; 50% loss of luminal diameter

Grade 3
Severe flattening of tracheal cartilages; 75% loss of luminal diameter

Grade 4
Complete obstruction; tracheal lumen is obliterated

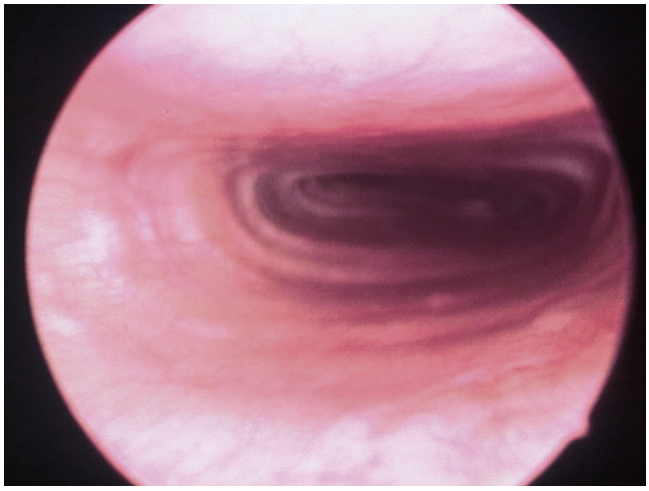


FIG. 28.34 Endoscopic view of a grade III dorsoventral tracheal collapse.

and bronchomalacia were identified by bronchoscopy in 50% of dogs with tracheal collapse.

Laboratory Findings

Hematologic and serum biochemistry findings are usually normal or insignificant unless concurrent systemic disease is present. Many dogs have mild to moderate increases in serum alkaline phosphatase. Positive tracheobronchial cultures are found in more than 50% of animals with tracheal collapse. Bacterial infection of the airways is generally accompanied by cytologic evidence of suppurative inflammation and detectable intracellular bacteria. Infection is also suggested by substantial growth of a single bacterial species in culture media. Electrocardiograms (ECGs) may reveal sinus arrhythmia, cor pulmonale, or left ventricular enlargement.

DIFFERENTIAL DIAGNOSIS

Other causes of chronic coughing or respiratory distress include brachycephalic syndrome, tonsillitis, laryngeal collapse, laryngeal paralysis, bronchitis, tracheobronchitis, allergies, heartworm disease, pulmonary disease, cardiac disease (especially mitral valve insufficiency), hypoplastic trachea, tracheal stenosis, and tracheal neoplasia or mass.

Although the clinical signs may be similar, tracheal collapse should not be confused with *tracheal stenosis*. Tracheal stenosis is an abnormal narrowing of the tracheal lumen caused by congenital malformation or cicatrix posttrauma. Trauma (e.g., penetrating or blunt wounds, foreign bodies, indwelling tubes) or surgery may cause segmental tracheal stenosis when the wound heals by secondary intention, when excess fibrosis and scarring cause luminal narrowing, or when tracheal cartilages heal with an abnormal shape. Traumatic stenosis is treated by balloon dilation or resection and anastomosis. Treatment of tracheal hypoplasia consists of symptomatic medical therapy (i.e., antibiotics and cough suppressants) and correction of other airway obstructions (e.g., resection of nares, palate, and saccules).

MEDICAL MANAGEMENT

Medical therapy is recommended for all animals with mild clinical signs and for those with less than 50% collapse because it results in improvement in clinical signs in most dogs. Weight loss is critical to the success of other medical therapies. Environmental modifications, such as use of a harness instead of a collar and the creation of a nonsmoking atmosphere, may help some dogs, as will management of concurrent underlying conditions. Medical therapy for dogs with tracheal collapse includes antitussives (e.g., butorphanol tartrate, hydrocodone bitartrate, and diphenoxylate; Box 28.15), antibiotics (e.g., ampicillin, cefazolin, clindamycin, and enrofloxacin), bronchodilators (e.g., extended-release theophylline, aminophylline, albuterol, and terbutaline), and/or antiinflammatory agents (i.e., prednisolone). Sedation with acepromazine (0.025–0.1 mg/kg [maximum, 1 mg] IV, intramuscular [IM], or SC q8h) and/or diazepam (0.2 mg/kg IV q12h) and supplemental oxygen (see Table 4.4) may be required in severely dyspneic patients. Pediatric metered dose inhalers used with spacers and face masks may be used in cooperative dogs to administer aerosolized bronchodilators (i.e., albuterol) and corticosteroids. Mucolytics and saline nebulization may benefit those with excess mucus production and infection. Response to medical therapy is usually transient, and the disease typically progresses.

SURGICAL TREATMENT

Surgery is recommended for all dogs with moderate to severe clinical signs, 50% or greater reduction of the tracheal lumen, or clinical signs refractory to medical therapy. Dogs brought in for treatment with laryngeal paralysis or collapse, generalized cardiomegaly, bronchial collapse, chronic pulmonary disease, or concurrent collapsed mainstem bronchi are poor surgical candidates. Coughing and dyspnea caused by laryngeal, pulmonary, or cardiac disease are not expected to improve without appropriate therapy. Respiratory distress and death may occur in animals with severe laryngeal dysfunction or bronchopulmonary disease.

The goal of surgery is to support the tracheal cartilages and trachealis muscle while preserving as much of the segmental blood and nerve supply to the trachea as possible. Although other techniques have been described, surgical goals for tracheal collapse are currently achieved with the use of extraluminal ring prostheses or endoluminal stenting. Extraluminal tracheal ring prostheses can be implanted in dogs with cervical tracheal collapse or proximal intrathoracic tracheal collapse. Intraluminal stents can address collapse at any level, and placement involves less risk of interrupting innervation and blood supply; however, implants are expensive, require fluoroscopic or endoscopic

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BOX 28.15 Drugs Used in Treatment of Tracheal Collapse

Butorphanol Tartrate (Torbutrol)
0.5–1.0 mg/kg PO q6–12h or 0.055 mg/kg SC q6–12h

Hydrocodone Bitartrate (Hycodan)
0.2–0.5 mg/kg PO q8h

Diphenoxylate With Atropine (Lomotil)
(2.5 mg diphenoxylate hydrochloride + 0.025 mg atropine per 5 mL or per oral tablet)
0.2–0.5 mg/kg diphenoxylate PO q8–12h

Ampicillin
22 mg/kg IV, IM, SC q8h

Amoxicillin
22 mg/kg PO q12h

Cefazolin (Ancef, Kefzol)
22 mg/kg IV, IM, SC q8h

Clindamycin (Antirobe, Cleocin)
Dogs: 11–33 mg/kg q12h PO, or 11 mg/kg IV (dilute and give slowly when administering IV)
Cats: 11–33 mg/kg PO, SC q12–24h

Doxycycline[a]
5 mg/kg PO q12h

Aminophylline[b]
Dogs: 10 mg/kg PO, IM, IV q8h (if given IV, give slowly over 5 min)
Cats: 5 mg/kg PO q12h

Theophylline (Extended Release)
Dogs: 10 mg/kg PO q12h (dose may vary with preparation)
Cats: 25 mg/kg PO q24h (dose may vary with preparation)

Dexamethasone Sodium Phosphate (Azium)
0.2 mg/kg IV, IM, SC q12h; can give up to 5 mg/kg for emergency treatment[c]

Prednisolone
1.1–2.2 mg/kg PO q12–24h

[a]Do not dry-pill cats with doxycycline; follow with at least 6 mL of water or use compounded slurry.
[b]Do not give as a rapid IV bolus.
[c]Large or repeated doses are typically associated with gastrointestinal erosion, ulceration, and/or hemorrhage.
IM, Intramuscular; *IV,* intravenous; *PO,* orally; *SC,* subcutaneous.

placement, and are associated with serious complications. Currently, intraluminal stents are best used as a salvage procedure for severe, refractory tracheal collapse involving the thoracic trachea. Patients with concurrent laryngeal paralysis or laryngeal collapse may also require arytenoid lateralization (see p. 861) or permanent tracheostomy (see p. 843), respectively.

Preoperative Management

These patients should be observed closely for signs of progressive dyspnea after hospitalization. Prophylactic antibiotics (e.g., cefazolin 22 mg/kg IV, repeated every 90 minutes to 2 hours until completion of surgery) should be given at the time of induction. Glucocorticoids may be given to dogs with very small tracheas (i.e., those weighing less than 2–4 kg) to minimize tracheal mucosal swelling.

Anesthesia

These patients should be preoxygenated, and they should be induced and intubated quickly. Manipulation of the endotracheal tube by an assistant is necessary during placement of ring prostheses to ensure that sutures have not been placed into the endotracheal tube or cuff. Extubation should be delayed as long as possible after surgery. Laryngeal paralysis may occur secondary to recurrent laryngeal nerve damage (see p. 858). Supplementation of oxygen via nasal insufflation (see Table 4.4) is beneficial, particularly if laryngeal paralysis or severe tracheal inflammation occurs. General anesthetic recommendations for animals with upper respiratory disease are given on p. 834.

Surgical Anatomy

Surgical anatomy of the trachea is discussed on p. 839. The segmental blood and nerve supply to the trachea travels in the lateral pedicles on each side of the trachea. Minimal mobilization of the trachea is necessary to maintain a good blood supply after surgery. The left recurrent laryngeal nerve is located in the lateral pedicle; the right is sometimes located within the carotid sheath.

Positioning

For extraluminal ring placement, the animal should be positioned in dorsal recumbency with the neck extended and elevated over a pad (to deviate the trachea ventrally). The caudal mandibular area, ventral neck, and cranial thorax should be clipped and prepared for aseptic surgery.

SURGICAL TECHNIQUE

Extraluminal Stents

AP Prosthetic tracheal rings or spirals are made by cutting 3 mL polypropylene syringe cases. To create individual rings, a pipe cutter can be used to divide a syringe case into cylinders 5 to 8 mm wide. Five or more staggered holes should be drilled through each ring for suture placement, and the ring should be split ventrally to allow placement. Rough edges of the rings can be smoothened by firing or trimming with a No. 11 blade or file. The rings should be autoclaved before implantation. Gas sterilization is possible, but the rings must be aerated for at least 72 hours to prevent toxic tissue reactions and tracheal necrosis. Narrower notched rings of variable diameter are preferred by some surgeons; these are commercially available.

Incise the skin and SC tissues along the ventral cervical midline from the larynx to the manubrium. Separate the sternohyoid and sternocephalicus muscles along their midline to expose the cervical trachea. Examine the trachea for evidence of collapse and deformity (Fig. 28.35). Identify and protect the recurrent laryngeal nerves. Place the first tracheal prosthesis one or two cartilages distal to the larynx. Dissect the peritracheal tissues and create a tunnel immediately around the trachea only in the areas of prosthetic ring placement. Guide and position a prosthetic ring through the tunnel and around the trachea with a long, curved hemostat (Fig. 28.36A–B). Position the prosthetic ring with the split on the ventral aspect of the trachea. Chondrotomy is occasionally necessary to allow deformed, rigid cartilages to conform to the prosthesis. *Secure the prosthesis with sutures ventrally, laterally, and dorsally (Fig. 28.36.C). Place three to six sutures (3-0 or 4-0 polypropylene [Prolene], polybutester [Novafil], or polydioxanone [PDS]) to*

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secure each prosthesis. Direct sutures around rather than through cartilages, and engage the trachealis muscle in at least one suture. Place four to six additional ring prostheses 5 to 8 mm apart along the trachea (Fig. 28.37). Cranial traction on the prostheses around the cervical trachea allows one or two rings to be placed at the thoracic inlet or beyond. *Preserve the blood vessels and nerves between the rings. Manipulate the endotracheal tube or trachea after the placement of each prosthesis to ensure that the tube cuff has not been engaged by a suture. Place a radiopaque marker on the last ring to radiographically identify its location postoperatively, if desired. Lavage the surgical site with sterile saline. Appose the sternohyoid and sternocephalicus muscles with simple continuous sutures (3-0 or 4-0 polydioxanone), and appose the SC tissues and skin routinely.*

Endoluminal Stent Placement

The minimally invasive technique used for intraluminal prosthesis placement varies with the type of self-expanding elastic implant selected. Stents are placed under general anesthesia using tracheoscopy or fluoroscopy. Selection of implant size must be accurate and may be determined from measurements taken during tracheoscopy or imaging. However, one study showed

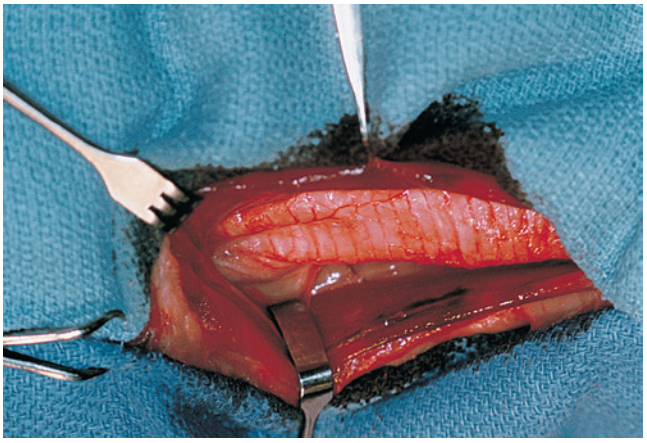


FIG. 28.35 Intraoperative appearance of a grade IV tracheal collapse.



FIG. 28.37 Appearance of the trachea after placement of six tracheal ring prostheses.

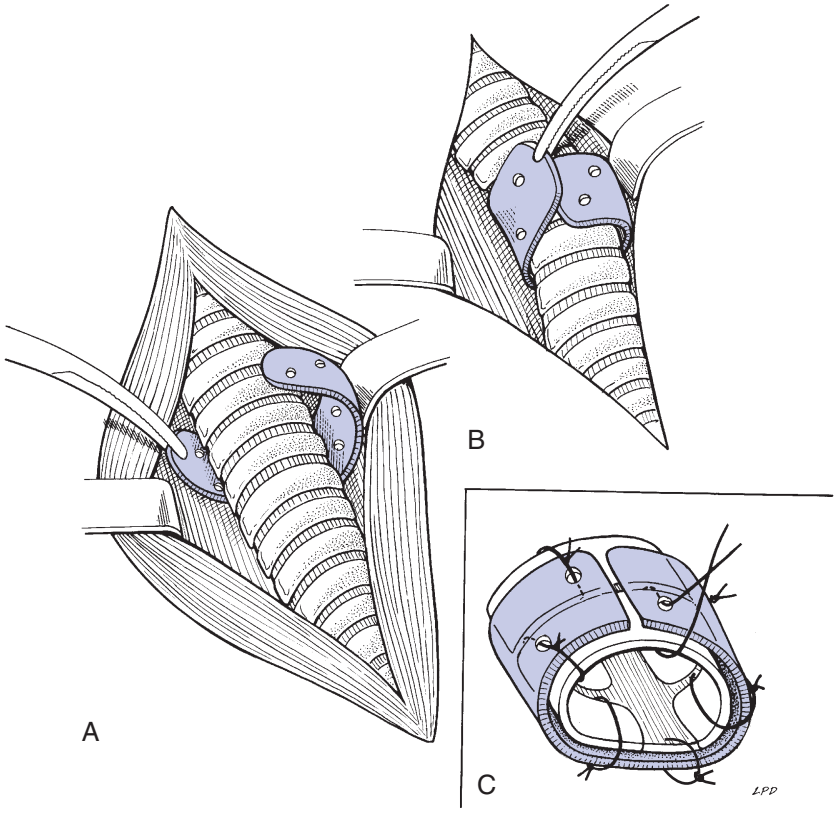


FIG. 28.36 (A) To place prosthetic rings on the trachea, dissect a tunnel around the trachea at each implantation site, and then guide the prosthesis through the tunnel. (B) Rotate the prosthesis around the trachea. (C) Secure the prosthesis with several sutures.

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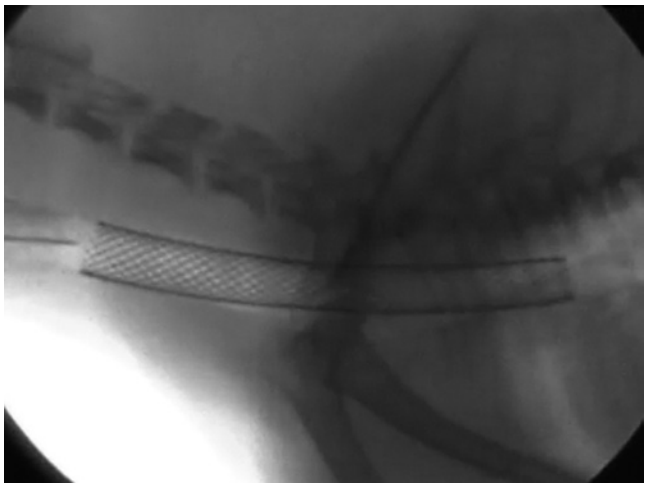


FIG. 28.38 Radiographic appearance of a 2-year-old Yorkshire terrier following endoluminal stent placement.

that measurements taken from CT images in healthy dogs had a consistently greater diameter when compared with measurements from fluoroscopy.[25] Additional work needs to be performed to determine whether CT is the imaging modality of choice for tracheal measurements for endoluminal stenting.

NOTE Place a metric radiopaque ruler beneath the patient to account for magnification error when measurements are taken from radiographs.

The implant should remain approximately 10 mm from the larynx and the carina in most cases to prevent excess irritation and granulation (Fig. 28.38). If the stent is near the thoracic inlet, constant movement at that point often leads to early fracture of the stent. Some have recommended measuring the diameter from films captured while positive-pressure airway insufflation to 10 mm Hg (20 cm H_2O) is applied with the end of the tube positioned at the end of the larynx. Open-looped or knitted implants are preferred because they promote coverage of the stent with tracheal epithelium.

After the implant is selected, anesthetize the dog, insert the implant, and deploy it according to the manufacturer's instructions with the aid of fluoroscopy or endoscopic positioning. Place multiple stents if necessary. Repositioning of stents is not usually possible after they have been deployed.

NOTE Because intraluminal stents cannot be easily retrieved and many potential complications are associated with their use, these stents are generally not recommended in the early management of tracheal collapse.

POSTOPERATIVE CARE AND ASSESSMENT

These animals should be continuously monitored during recovery. Acute respiratory distress may occur postoperatively secondary to inflammation, edema, and/or laryngeal paresis or paralysis. Animals with laryngeal paralysis may require surgery to widen the glottis (see p. 861), and those with collapse may require a permanent tracheostomy (see p. 843) within the first 24 hours to relieve respiratory distress. Nasal insufflation of oxygen and an antiinflammatory dose of glucocorticoids may be beneficial in animals with edema and inflammation. Mucolytics and saline nebulization may be appropriate for those with severe inflammation. Antibiotics should be continued for 7 to 10 days if bacterial tracheitis is present. Antitussives, bronchodilators, analgesics, and sedatives may be given as necessary to control coughing and excitement (see Box 28.15). These animals should have strict exercise restriction (cage rest) for 3 to 7 days. Thereafter exercise may be increased gradually. A harness rather than a collar should be used for leash walking. Weight reduction is important in obese patients. Tracheoscopy is recommended 1 to 2 months after surgery and later if respiratory signs deteriorate.

Immediate improvement in clinical signs may be seen with both extraluminal and endoluminal stents; however, coughing and lack of marked improvement in clinical signs should be expected for several weeks postoperatively because of tracheitis, peritracheal swelling, and suture irritation. However, significant clinical improvement (e.g., decreased respiratory noise, less respiratory effort, increased exercise tolerance, fewer tracheobronchial infections) should be noted within 2 to 3 weeks of surgery (Video 28.6). Some animals have nearly complete remission of clinical signs after surgery, whereas others continue to have episodes of coughing or other respiratory noises. The quality of life is improved for most patients, but neither surgery nor stents cure the condition.

COMPLICATIONS

Death may result if the trachea is obstructed by severe inflammation or is damaged by severe infection or necrosis with extraluminal or intraluminal prostheses. Coughing after surgery is expected until inflammation subsides. Infection is a possible problem because the trachea contains bacteria that may be harbored in implants.

Bruising and mild cervical swelling are expected postoperatively after placement of extraluminal prostheses. Recurrent laryngeal nerve damage may result in laryngospasm, laryngeal paresis, or paralysis. Tracheal necrosis may occur if too much dissection strips the blood supply away from the trachea, or if improperly aerated prostheses (gas sterilized) are implanted.

Incorrect placement or sizing of intraluminal implants may result in death. Failure to stent the entire involved trachea typically results in collapse proximal and/or distal to the stent. A stent that is too narrow may migrate, whereas one that is too wide may cause pressure necrosis. Stents placed too close to the larynx may cause intractable laryngospasm. Tracheal obstruction caused by granuloma formation, which may be steroid responsive, occurs in approximately 20% to 30% of cases. Other reported complications include cough, expectoration, tracheal hemorrhage, emphysema, pneumomediastinum, infection, mucous obstruction, tracheal rupture, squamous metaplasia and ulceration of the tracheal epithelium, implant shortening, implant fracture, and implant collapse or deformation. Regardless, the most recent report on the incidence of major complications between extraluminal and endoluminal stenting found them to be similar (42%–43%).[26]

PROGNOSIS

Clinical signs can sometimes be controlled medically if tracheal collapse is not severe, patients do not become obese, and a

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sedentary lifestyle is practiced. The prognosis is more dependent on concurrent respiratory problems, such as laryngeal paralysis or collapse and bronchial disease, than on the location or severity of tracheal collapse. Outcome studies are quite variable with regard to extraluminal versus endoluminal stenting. Recent median survival times reported for dogs receiving extraluminal stenting range from 1460 to 1680 days.[26,27] Survival times in dogs receiving endoluminal stenting appear to be markedly shorter; however, when accounting for effects of age, there was no significant difference. Regardless of treatment type, the presence of mainstem bronchi collapse is associated with shorter survival times.[26]

NASAL TUMORS

DEFINITIONS

Nasal tumors or sinonasal tumors are tumors that arise from the nasal cavity or paranasal sinuses. **Rhinotomy** is an incision into the nasal cavity.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Neoplasms of the nasal cavity and paranasal sinuses are rare in most domestic species; the reported incidence varies from 0.3% to 2.4% of canine tumors. They occur more commonly in dogs than in cats. Sinonasal tumors may be classified histologically as epithelial, nonepithelial, or miscellaneous (Box 28.16). Neoplasms of epithelial origin are most common, with adenocarcinoma being the single most frequent histologic diagnosis in dogs. Nonepithelial tumors of skeletal origin (e.g., chondrosarcoma and osteosarcoma) account for approximately one-fifth of canine nasal tumors. In cats, lymphoproliferative and epithelial tumors are most prevalent.

The metastatic rate of nasal tumors has traditionally been considered low, with metastasis occurring late in the natural course of these tumors; however, many dogs with sinonasal tumors have metastasis. The most common site of metastasis is the brain, followed in decreasing order of frequency by regional lymph nodes, lungs, and liver. Esthesioneuroblastomas and neuroendocrine tumors of the nasal cavity are most likely to metastasize to the brain, whereas epithelial tumors usually metastasize to the regional lymph nodes and lungs. Osseous tumors in this region have a low incidence of metastasis. Prolonged survival rates in cats with lymphoreticular nasal tumors have been reported after radiotherapy, suggesting that metastasis of these tumors is slow.

BOX 28.16 Histologic Classification of Sinonasal Neoplasms

Epithelial

Squamous cell carcinoma
- Nonkeratinizing
- Keratinizing

Nonepithelial

Skeletal
- Chondrosarcoma
- Osteosarcoma

Soft tissue
- Lymphosarcoma
- Fibrosarcoma
- Hemangiosarcoma
- Muscular origin
- Fibrous histiocytoma
- Malignant nerve sheath

Miscellaneous

Adenocarcinoid
Esthesioneuroblastoma
Carcinoid
Melanoma

DIAGNOSIS

Clinical Presentation

Signalment

Male dogs and cats appear to have a higher incidence of sinonasal neoplasms than females, irrespective of histologic diagnosis. Intranasal tumors generally occur in older animals, with a median age of approximately 10 years in dogs and cats. However, the mean age varies according to the histologic diagnosis; the mean age of dogs with chondrosarcoma (7 years) is younger than that of dogs with other tumor types (9 years). In dogs 1 to 4 years of age, chondrosarcomas are more commonly diagnosed than all other tumor types. Soft tissue tumors involving the nasal cavity have even been reported in dogs as young as 1 year of age.

History

Most affected dogs are brought in for evaluation of nasal discharge, often with epistaxis. Tumors may cause paroxysmal sneezing (which may be violent enough to produce epistaxis). Probably less than 10% of affected dogs do not have obvious nasal discharge or have only modest crusting at the nares. Clinical signs in cats are similar; most are evaluated for sneezing and nasal discharge and occasionally epistaxis. The duration of the clinical signs varies, but most animals have signs for longer than 1 month and many for longer than 6 months before definitive diagnosis. The clinical signs are often intermittent at first and gradually become persistent as the tumor progresses. Infections associated with nasal tumors often respond transiently to antibiotics and other drugs, delaying definitive diagnosis.

Physical Examination Findings

Clinical findings in dogs with nasal tumors include epistaxis, swelling of the facial region (including exophthalmos), nasal discharge, crust at the nares, sneezing or snuffling, dyspnea, ocular discharge, and/or bleeding from the oral cavity. Neurologic signs (e.g., seizures, behavior changes, obtundation, paresis, ataxia, circling, visual deficits, and/or proprioceptive deficits) may be preponderant. Clinical signs may vary according to the histologic type of the tumor. Seizures are more common in dogs with carcinoids and esthesioneuroblastomas than in those with tumors of epithelial origin, presumably because of differences in metastatic patterns. Dyspnea may be more typical with epithelial neoplasm, and sneezing has been reported most commonly with chondrosarcoma.

Diagnostic Imaging

Thoracic radiography is indicated to evaluate for metastasis. Skull imaging requires general anesthesia to obtain satisfactory positioning. Good quality nasal radiographs help define the extent and location of disease and should be performed before

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rhinoscopy (see later section on endoscopy of the choana and nares for the exception to this rule), nasal flushes, and surgical biopsies. Lateral, dorsoventral, open-mouth ventrodorsal, and frontal sinus views are suggested. Oblique views may be necessary occasionally to outline lesions that are masked by or are superimposed over bony structures. The open-mouth ventrodorsal view consistently provides the most information by allowing visualization of the entire turbinate region and reducing superimposition of the mandibles. Radiographs should be evaluated for increased soft tissue opacity of the nasal cavity or frontal sinuses, bony lysis, destruction of the normal turbinate pattern, new bone formation, and foreign bodies (Figs. 28.39 and 28.40). Early nasal tumors are often difficult to recognize radiographically because of their similarity to inflammatory changes. Bone destruction usually suggests neoplasia, although severe fungal or bacterial infection may be responsible. Increased soft tissue opacity may occur in both neoplastic and inflammatory diseases. Extension into the frontal sinus or the contralateral nasal cavity and destruction of the hard palate indicate an aggressive process. Increased soft tissue opacity in the frontal sinus without bony erosion should not be interpreted as neoplastic extension into the frontal sinus because obstruction of outflow secondary to a nasal tumor often results in fluid accumulation there. Destruction of the cribriform plate may indicate extension into the brain and a poor prognosis.

CT and MRI are superior to radiographs for evaluation of the extent of nasal disease in animals with nasal tumors, both for prognosis and for planning of radiation therapy. However, no clinically relevant benefit is gained by using one modality over the other: CT scans provide good anatomic detail of bony tissues (Fig. 28.41), and contrast-enhanced scans can give detail regarding soft tissue abnormalities. Dogs with unilateral intranasal involvement without bone destruction beyond the turbinates have long median survival times; involvement of the cribriform plate has a poor prognosis

MRI scans may be better for evaluating soft tissue structures and finding small amounts of fluid. In dogs with a nasal mass effect, evidence of cribriform plate erosion, vomer bone lysis, paranasal bone destruction, sphenoid sinus invasion, or nasopharyngeal invasion is helpful in distinguishing neoplasia from nonneoplastic masses. CT and MRI are also useful in patients

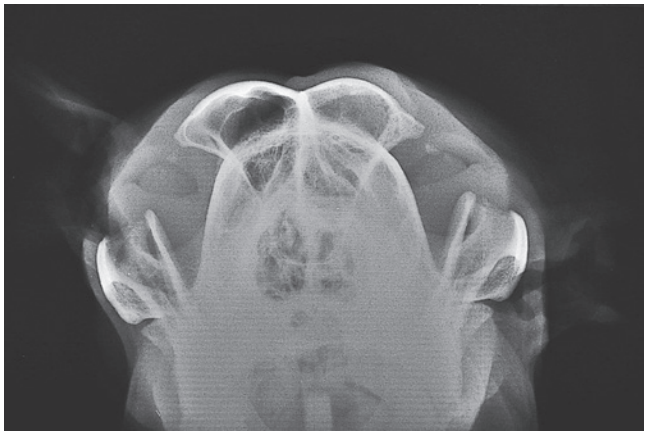


FIG. 28.40 Skull radiograph of an 8-year-old dog with a nasal adenocarcinoma. Note increased density and loss of turbinate detail in the left nasal cavity *(right side of image).*



FIG. 28.39 Frontal sinus view of the dog in Fig. 28.39. Note increased density of the frontal sinus on the right. This may represent neoplasia or fluid accumulation that occurred secondary to obstruction of drainage caused by the nasal disease.



FIG. 28.41 Axial noncontrast computed tomography image of the midaspect of the nasal cavity in a dog with neoplasia. Note the turbinate destruction in both nasal cavities and extension of the tumor mass through the hard palate *(arrow).*

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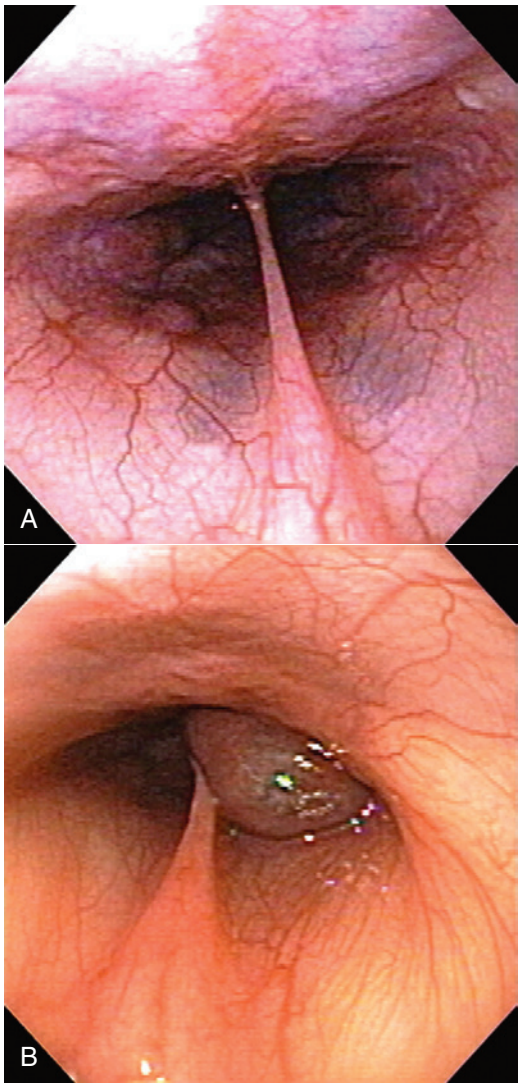


FIG. 28.42 Endoscopic view of (A) normal choana and (B) one with a nasal tumor protruding. The tumor was a carcinoma that was diagnosed with endoscopic biopsy.

with minimal neurologic signs but with evidence of destruction of the rostral portion of the calvarium.

Endoscopy of the Choana and Nares

Endoscopy for possible nasal tumors includes examination of the choana and the anterior nares (Fig. 28.42A). Although a relatively small percentage of nasal tumors extend caudally out into the nasopharynx, this is a useful endoscopic procedure because it is exceedingly easy and quick (i.e., <2 minutes) to do. If the owners have decided that they will not attempt surgery or radiation if a neoplasm is diagnosed from specimens obtained by endoscopy, then this technique is best performed before imaging is done because a positive diagnosis here will negate the need for more expensive imaging techniques.

The patient must be under a deep plane of anesthesia because this technique will strongly stimulate the gag reflex. A strong mouth gag is mandatory. *With the patient in sternal recumbency and the tongue pulled forward, advance the tip of a relatively small-diameter flexible scope a centimeter or more past the caudal edge of the soft palate. Then maximally flex the tip up (i.e., at least 180 degrees) so that the tip is above the soft palate and is looking cranially.* The choana should be seen easily. *If necessary, push the scope caudally or cranially to obtain a better view. Use the smallest-diameter scope; this works well because using a smaller-diameter scope lessens stimulation of the gag reflex and minimizes trauma (and subsequent bleeding) as the tip of the scope rubs against the dorsal pharynx while it is flexed. If a mass is seen protruding from the choana (see Fig. 28.42B), carefully advance biopsy forceps through the scope and take a biopsy of the mass under direct vision. Be careful lest forcing the biopsy forceps through the maximally flexed channel ruptures it. If necessary, preplace the biopsy forceps before flexing the scope up and over the soft palate. Alternatively, perform brush cytology of the mass (but biopsy is usually preferred).*

The mass rarely represents a tumor that has pushed up normal epithelium over it, and one cannot make a definitive diagnosis. However, most such masses can be definitively identified. Finding a mass that is not fungal (e.g., cryptococcosis) is strongly suggestive of neoplasia. Biopsies of these masses are sometimes associated with hemorrhage, and a cuffed endotracheal tube is mandatory. Bleeding typically stops within 5 minutes, but the back of the pharynx should be manually cleaned out before the endotracheal tube is removed. Rare tumors (e.g., intravascular lymphoma) can bleed so severely as to be life-threatening.

Rhinoscopy

Rhinoscopy sometimes locates nasal tumors, but many patients have substantial nasal discharge and/or bleeding that makes it difficult to see the mass. Rhinoscopy is appropriate because it is an excellent method to diagnose nasal aspergillosis, a disease that closely mimics nasal malignancy. The patient must be under a deep plane of anesthesia or the procedure may cause sneezing, which may traumatize the nose, causing hemorrhage that further obscures visualization. This procedure is more difficult and time-consuming than examination of the choana; therefore it should be done after imaging studies.

Flexible and rigid scopes may be used. Rigid cystoscopes with outer cannulas allow one to flush away debris with cold saline during the procedure, thereby improving visualization. One may attempt to do the same through the biopsy channel of a flexible scope.

Use care and methodically examine all passages. If the tip of the scope is pushed against the mucosa, hemorrhage typically results. *If a mass is seen, take a biopsy. More often one cannot see a mass; therefore use imaging to help you blindly place punch-type biopsy forceps as close as possible to the lesion. Open the jaws and advance the forceps until resistance is felt (take care not to perforate the cribriform plate), and then close the jaws and retrieve the sample.*

This technique is often successful, and sampling during rhinoscopy provides a definitive diagnosis in most patients. Keep in mind that biopsies may cause profuse hemorrhage, and a cuffed endotracheal tube is mandatory. *If the hemorrhage is worrisome, insert a nasal tampon into the affected nostril for 10 to 15 minutes. Be sure that hemorrhage has stopped before waking the patient, and clean out the pharynx before removing the endotracheal tube.*

Laboratory Findings

Laboratory abnormalities are uncommon. In rare cases, severe epistaxis may cause anemia and hypoalbuminemia. White cell counts are seldom increased, even with a secondary bacterial infection. Thrombocytopenia or prolonged partial thromboplastin time may suggest a coagulopathy (especially in patients with

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bleeding from venipuncture sites, ecchymoses, petechiation, melena, hematuria, and/or retinal hemorrhage). Profuse epistaxis can cause thrombocytopenia, so care must be taken in deciding cause and effect. Cats should be evaluated for feline leukemia virus and feline immunodeficiency virus infections. Imprint or brush cytologic studies of nasal lesions may help differentiate inflammation from tumor and assist in identification of the tumor type. Cytology of nasal exudate seldom helps because most animals have neutrophils present caused by secondary bacterial infections. However, *Cryptococcus* is occasionally identified. Finding fungal hyphae in nasal exudate is not diagnostic of aspergillosis because they are occasionally found in normal dogs. Fecal flotation may detect ova from nasal parasites. Serology may rule out rickettsial causes of thrombocytopenia and certain mycoses (e.g., cryptococcosis and possibly aspergillosis). Culturing the nose is seldom helpful because an abundant normal flora is present (e.g., *E. coli*, *Streptococcus* spp., *Pasteurella* spp.), and most bacterial rhinitis occurs secondary to other causes (e.g., nasal tumor, aspergillosis, foreign body).

DIFFERENTIAL DIAGNOSIS

Nasal tumors usually cause unilateral epistaxis or nasal discharge, although some tumors only cause relatively mild crusting. However, given time, bilateral discharge typically develops. Bacterial or fungal rhinitis typically causes unilateral discharge initially; however, these disorders are often not diagnosed until the discharge has become bilateral. Common nasal fungal diseases (e.g., aspergillosis and penicilliosis in dogs; cryptococcosis in cats) may be diagnosed by identifying fungal plaques and hyphae and/or yeast in tissues, or by finding diagnostic titers. Foreign bodies, oronasal fistulae, tooth root abscesses, and bleeding diatheses may be associated with unilateral nasal discharge. Nasal obstruction may be caused by foreign bodies, nasopharyngeal polyps, or nasopharyngeal or caudal choanal stenosis. Nasal mites (e.g., *Pneumonyssoides caninum*) may cause rhinitis, sneezing, reverse sneeze, and an impaired sense of smell. They are diagnosed during rhinoscopy by the observation of 1- to 1.5-mm, light-yellow mites in the nares or the choana, or when the mites exit the nostrils during anesthesia. Other (rare) nasal parasites that cause rhinitis include nasal nematodes (e.g., *Capillaria aerophila, Eucoleus boehmi* [1.5–4 cm long]) and the arthropod *Linguatula serrata*. Other organisms that may cause epistaxis include *Cuterebra* and *Leishmania* (systemic protozoan). Hemostatic defects, ehrlichiosis, immune-mediated thrombocytopenia, lymphocytic-plasmacytic rhinitis, multiple myeloma, systemic hypertension, polycythemia vera, and hyperviscosity syndrome may cause epistaxis or nasal discharge and should be ruled out. Although rare, nasopharyngeal stenosis and choanal atresia cause nasal obstruction that mimics neoplasia. Facial deformity, which is sometimes seen with tumor, should be differentiated from a draining tract associated with a nasal dermoid sinus cyst.

MEDICAL MANAGEMENT

Therapy for nasal tumors is directed at control of local disease. Reported treatment options include surgical debulking, surgical debulking combined with radiation therapy, radiation therapy alone, iridium implants, chemotherapy, immunotherapy, cryosurgery, and photodynamic therapy with laser ablation. Radiotherapy appears to be the most effective treatment for nasal carcinomas. Most studies have investigated orthovoltage irradiation (125–400 keV), although other studies have reported the use of megavoltage x-irradiation (>1 keV), cobalt radiation, and intensity-modulated radiation therapy. The optimum protocol, dosage, and method of delivery have not been determined. The use of cone-based stereotactic radiosurgery for canine sinonasal tumors appears promising, as it is currently associated with an overall median survival time of 8.5 months.[28]

Radiotherapy of nasal tumors in cats appears as effective as in dogs, but with a lower level of acute side effects. Palliative hypofractionated radiotherapy was recently associated with an overall median survival of 432 days. Acute complications were common, but manageable and acceptable to owners.[29]

Whether radiation therapy should be combined with surgical debulking is controversial. One reason for doing so is to improve the clinical status of the dog before radiation therapy. Prior surgery may reduce dyspnea from nasal cavity obstruction, nasal discharge, and epistaxis during radiation therapy.

Cryosurgery or immunotherapy has not appreciably prolonged the survival times of dogs with nasal tumors. Adjunctive therapy with chemotherapy (e.g., cisplatin) in combination with radiation therapy may improve median survival times. Administration of piroxicam may palliate some dogs with inoperable nasal tumors, but one must watch for gastric erosions and/or ulcers.

SURGICAL TREATMENT

Surgery as the sole treatment for dogs with nasal tumors has not prolonged survival time. The poor response of dogs with nasal tumors to surgery is due to the advanced nature of most tumors at the time of diagnosis, a propensity for these tumors to invade bones that are inaccessible or that cannot be surgically removed, and lack of appreciable encapsulation; each of these makes it almost impossible to completely remove the tumor. However, surgery may palliate clinical signs in some dogs by alleviating obstruction and epistaxis. Surgery may be of value in patients with recurrence following radiation therapy. Permanent tracheostomy (see p. 843) may benefit some dogs that have severe respiratory difficulties and in which other treatment options are not feasible.

Cats have poor tolerance of rhinotomy for intranasal tumors; surgery alone is associated with a fast rate of tumor recurrence. Surgical resection of squamous cell carcinoma of the nasal planum offers the greatest possibility of cure.

Preoperative Management

Evaluate coagulation parameters and cross match animals before surgery in anticipation of the need for a blood transfusion. Anemic animals may benefit from preoperative blood transfusion (see discussion under Preoperative Management) and should be preoxygenated. Perioperative antibiotics may be given at induction of anesthesia and continued for 12 hours after surgery, but they are generally unnecessary and may inhibit bacterial growth from tissue obtained at surgery.

Anesthesia

Selected anesthetic protocols for animals undergoing nasal surgery are provided on p. 834. Biopsy and rhinotomy are performed under general anesthesia. A cuffed endotracheal tube is mandatory to prevent aspiration of blood or fluids into the airway. Blood, colloids, and/or hypertonic saline should be available in case severe hemorrhage occurs (see Chapter 4). Evaluation of blood pressure, ECG, and pulse oximetry during surgery is warranted,

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and acepromazine and alpha-2 agonists should be avoided. Before extubation, the mouth and the oropharyngeal region should be suctioned to remove blood and secretions to the greatest extent possible. It is helpful to recover the patient in a quiet environment, with the patient in sternal recumbency and the head and neck extended. The head should be forward and, if possible, below the level of the thorax. The animal should be extubated with the cuff slightly inflated to help remove any blood and mucus sitting above the cuff. If the patient is coughing and gagging after extubation, be prepared to suction the mouth and the oropharynx again to clear the airway of blood and secretions.

Surgical Anatomy

See p. 837.

Positioning

For a dorsal approach to the nasal cavity, the animal is positioned in sternal recumbency with a rolled towel under the neck. The entire head and nasal area should be clipped and prepared for aseptic surgery. For a ventral approach to the nasal cavity, the animal is positioned in dorsal recumbency with the mouth tied open maximally (see Fig. 28.5). For an oral approach to the rostral nasal cavity, the animal is positioned in lateral recumbency. The oral cavity is flushed with sterile saline, and the palate is swabbed with dilute Betadine (povidone-iodine) or chlorhexidine solution.

SURGICAL TECHNIQUE

Nasal tumors may be diagnosed by blind biopsy with alligator forceps, by anterior rhinoscopy with biopsy, or by endoscopic examination of choanae and a biopsy of protruding masses.

Biopsy

Endoscopic biopsies were described previously under Diagnostic Imaging and Endoscopy of the Choana and Nares (see pp. 874–876).

Transnostril Core Biopsies

Transnostril core biopsies may be obtained by using the outer protective shield of a Sovereign catheter with the end cut off at a sharp angle, or an alligator forceps. To prevent inadvertent penetration of the cribriform plate, the catheter or forceps should be measured with the tip placed at the medial canthus of the eye; these instruments should not be advanced past this point. This technique is traumatic and is seldom done anymore.

Attach the catheter (with the metal stylet removed) to a 12-mL syringe. Discern the location of the lesion from the radiographs, and advance the catheter through the tumor several times while applying negative pressure to the syringe. After withdrawing the catheter from the naris, remove the barrel of the syringe and add a small amount of air to the syringe. Use this air to propel the tissue sample forcefully from the syringe hub onto a microscopic slide or into a formalin-filled container. Repeat sampling at various angles until sufficient tissue is obtained. When taking a biopsy of masses in the caudal nasal cavity, be sure to adjust the length of the catheter so as to prevent perforation of the cribriform plate.

Nasal Flushes

Nasal flushes may be performed by using the same catheter as described for transnostril core biopsies. Hemorrhage may occur postprocedure but is generally mild and transient. *To prevent inadvertent entry into the calvarium in patients with bony lysis of the cribriform plate, measure the distance from the medial canthus of the eye to the external naris, and mark the catheter to correspond to this length. Place gauze sponges above the soft palate and below the external nares to collect fluid and tissues dislodged during flushing. Attach a 35-mL syringe to the catheter and vigorously flush 150 to 300 mL of saline into the nasal cavity as fast and as hard as possible. Evaluate the gauze sponges for the presence of tissue and debris. Examine the tissues cytologically and save samples for microbiological examination, including fungal and bacterial cultures. If sufficient quantities are obtained, place samples in formalin for histopathologic examination.*

Advanced Imaging-Guided Biopsy

Advanced imaging, such as CT, can be used to help guide the location of nasal biopsies; however, no advantage was shown over rhinoscopy-guided or blind biopsy techniques in a 2014 study.[30] Repeated biopsies are often required for definitive diagnosis. Recently, the use of a frameless CT-guided stereotactic biopsy system was described for sampling the nasal and sinus passages of five dogs.[31] This system allowed for definitive diagnosis in these dogs that was not attainable by other biopsy methods.

Rhinotomy

When the aforementioned techniques do not allow diagnosis, surgical exploration and biopsy may be necessary. Generally in such cases, diagnostic and therapeutic procedures (rhinotomy and debulking) are combined. Intraoperative cytologic or frozen section examination of tissues (or both) is helpful. Although rhinotomy may not appreciably extend the life of patients with nasal tumors, it often makes them more comfortable. Some surgeons prefer to perform temporary carotid artery ligation before rhinotomy to lessen hemorrhage (see p. 334 for technique). Most nasal tumors are approached using a dorsal rhinotomy because greater exposure of the sinuses and the caudal nasal cavity is possible (see p. 840). Ventral rhinotomy through the hard or soft palate or both is preferred for some lesions (see p. 840).

Nasal Planum Resection

AP This procedure is performed most commonly on cats with squamous cell carcinoma of the nasal planum. All or a portion of the nasal planum is excised. The procedure may be combined with rostral maxillectomy if the tumor invades or originates from the oral cavity (see p. 335). The basic technique is described here; other reconstructive techniques may be added to improve cosmesis.

> **NOTE** It is wise to show owners pictures of animals that have had a similar procedure performed, so they may fully appreciate its cosmetic effect. Not all owners will be accepting of this procedure (Fig. 28.43).

Position the animal in dorsal recumbency. Make an incision in the skin an acceptable distance from the lesion, continuing around or through the nasal planum (Fig. 28.44A). Continue the incision through the nasal cartilages, bone, and turbinates. Control hemorrhage and inspect the rostral aspect of the nasal cavity for tumor invasion. If necessary, reconstruct the lip by creating a labial advancement flap from one or both sides of the defect. Appose the gingival and buccal mucosa with 3-0 to 4-0 monofilament absorbable suture. Anatomically align and suture the labial margin, then appose

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FIG. 28.43 Appearance of a cat following bilateral rostral maxillectomy and nasal planum resection with advancement of a full-thickness labial flap to reconstruct the lip.

the labial muscles and skin. Place a purse-string suture in the skin around the nasal opening and tighten to approximate the size of the defect (Fig. 28.44B). Alternatively, create holes in the nasal bones surrounding the opening with a pin and pin chuck or drill and, while using these holes, suture the skin to the bone.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Vacuum suction devices and suction tips are extremely helpful during rhinotomy. Without suctioning devices, hemorrhage is often severe enough to preclude visualization of abnormal tissues. An oscillating saw is preferred if a bone flap is to be removed, otherwise a Steinmann pin and rongeurs may be used. Sterile gauze packing (e.g., Plain Nu-Gauze packing strip, 2 inches × 3.5 yards) may be used to pack the nose after completion of the procedure.

POSTOPERATIVE CARE AND ASSESSMENT

The airway should be suctioned to remove blood and fluid before extubation, and patients should be recovered with their heads

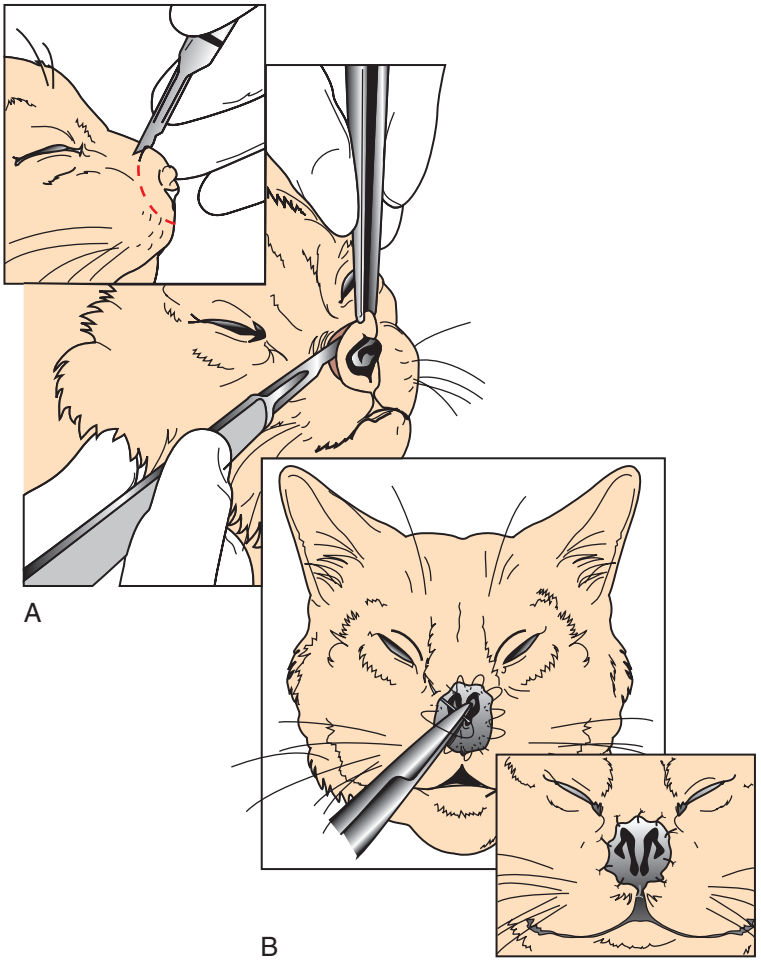


FIG. 28.44 Nasal planum resection. (A) Make an incision around the nasal planum through the skin, nasal cartilages, and turbinates. (B) Appose the skin to the nasal defect with a purse-string suture.

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down to reduce aspiration of blood. These animals should be closely monitored for epistaxis after surgery or biopsy.

The hematocrit should be evaluated during and after surgery, and transfusions should be given if the packed cell volume is below 20% (see Preoperative Management, p. 877). Patients should be prevented from banging their heads on the cage during recovery. Analgesics (e.g., an opioid, lidocaine, or ketamine constant-rate infusion) may be administered if they appear to be excited or in pain during recovery (see Table 13.1). Acepromazine (0.025–1 mg/kg IV, IM, SC up to a *maximum* of 2 mg in dogs and 1 mg in cats, total dose) may be given if the patient is normovolemic, is not hemorrhaging, and has been given adequate analgesics (see Chapter 13). Alternatively, in animals that have dysphoric recoveries, low dose dexmedetomidine (1–2 µg/kg IV) will provide sedation and analgesia. Neurologic function should be assessed postoperatively.

SC air may accumulate after a dorsal approach to the nasal cavity if the bone flap is not replaced adequately, or if an adequate stoma is not left in the SC tissues and skin for air to exit. Generally, if an adequate stoma is left, SC air accumulation is not a problem. The stoma contracts and heals within 5 to 10 days. Soft food should be fed for several days after the ventral approach and the animal prevented from chewing on hard objects for 3 to 4 weeks until the palate incision is healed.

PROGNOSIS

The prognosis for dogs with nasal tumors is generally poor. In patients not treated and in those treated with surgery, chemotherapy, immunotherapy, or cryosurgery, the mean survival time is generally 3 to 6 months. Improvement in this survival period has been accomplished with radiation therapy combined with or without surgical debulking (see previous discussion), with mean reported survival times of 8 to 25 months. Animals with lymph node or pulmonary metastasis have shortened median survival times. The prognosis for carcinoma is better than that for sarcoma, and adenocarcinoma appears to have the best overall prognosis. It is unlikely that therapy will result in cure in most dogs, and more successful local control may lead to increased detection of metastasis. Conversely, the prognosis for cats with lymphoid neoplasia of the nasal cavity appears good.

NASAL ASPERGILLOSIS

DEFINITIONS

Nasal aspergillosis (**sinonasal aspergillosis**) is characterized by infection of the nasal cavity and often the frontal sinus by large colonies of fungal hyphae. Masses formed by the fungal hyphae are sometimes called **aspergillomas. Trephination** is the removal of a circular piece of bone, especially of the skull.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Nasal aspergillosis is a relatively common disease in dogs in some areas and clinically may mimic nasal neoplasia (Fig. 28.45). It is the second most common cause of chronic nasal discharge in dogs, following nasal neoplasia. Nasal aspergillosis should not be confused with systemic aspergillosis; the latter has a very poor prognosis. The nasal form usually remains confined to the nasal cavity or the paranasal sinus, where it causes marked turbinate destruction. Invasion of the cranial vault may also occur. Nasal aspergillosis usually occurs without concomitant malignant or immunosuppressive disease.

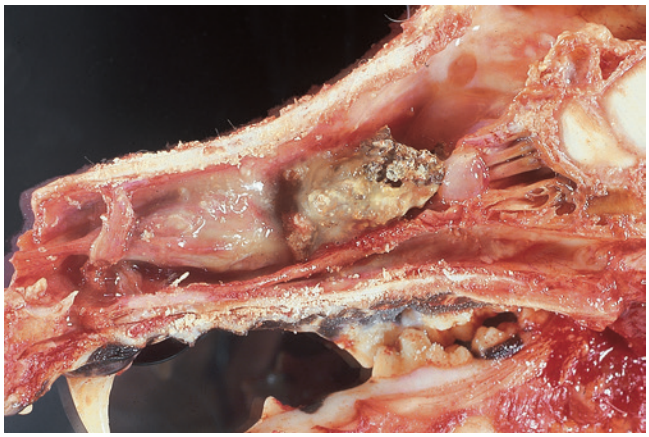


FIG. 28.45 Necropsy specimen of a dog with nasal aspergillosis. Note the large aspergilloma and associated destruction of turbinates.

DIAGNOSIS

Clinical Presentation

Signalment

Dogs with nasal aspergillosis tend to be younger (1–7 years) than dogs with nasal neoplasia, but overlap occurs. Aspergillosis is rare in brachycephalic breeds and cats. Male and female dogs appear to be equally affected, but several studies have noted a male predisposition.

History

Affected dogs are typically brought in for treatment of chronic nasal discharge, often with epistaxis. Owners may also note ulceration of the external nares.

Physical Examination Findings

Dogs may have erosion of the nasal planum associated with chronic nasal discharge. The nasal discharge may be unilateral or bilateral and may be sanguinopurulent, mucopurulent, or hemorrhagic. Although most dogs have profuse nasal discharge, a few have relatively sparse discharge. Other clinical signs include sneezing, reverse sneezing, epistaxis, depression, and decreased appetite.

Diagnostic Imaging

Affected dogs usually have obvious turbinate destruction plus areas of increased radiolucency, mimicking what may be seen in neoplasia. Frontal sinus osteomyelitis is commonly noted. CT and MRI are better than nasal radiographs at determining the extent of disease. CT images are best for detecting cortical bone lesions, whereas MRI is more useful for differentiating between thickened mucosa and secretions or fungal colonies.

Rhinoscopy

At rhinoscopy, cavernous areas caused by marked destruction of turbinates are often identified, and samples are obtained for cytology, histopathology, and culture. In addition, rhinoscopy

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provides a route for endoscopically guided debridement (see p. 876). Obvious mats of fungal hyphae (i.e., "fuzzy" plaques) or aspergillomas (e.g., look like masses of inspissated mucus, but may have discoloration caused by fungi) may be evident. Because aspergillosis causes marked turbinate destruction, much more space is usually present in the nose, typically making endoscopic diagnosis of aspergillosis relatively easy; however, rhinoscopy alone cannot define the extent of the disease process.

Sinuscopy

Trephination (see p. 882) and sinuscopy of the frontal sinus may help diagnose nasal aspergillosis as it is possible for some dogs to have fungal plaques just in the sinus alone.

Laboratory Findings

False-positive and false-negative results occur with cytology, culture, and serology. Therefore it is generally recommended that at least two of the following criteria are met: (1) radiographic features typical of fungal rhinitis (see previous discussion); (2) fungal plaques on the nasal or sinus mucosa; (3) *Aspergillus* or *Penicillium* organisms on culture, cytologic, or histologic examination; and (4) positive agar gel immunodiffusion, counterimmunoelectrophoresis, enzyme-linked immunosorbent assay, or polymerase chain reaction. Different serologic tests vary as to their sensitivity and specificity. Noticing fungal plaques during rhinoscopy in a dog with severe nasal turbinate destruction is essentially diagnostic. Cytologic examination of exudate or fungal mats collected by brush samples or squash-prep of mucosal biopsy samples during direct rhinoscopic visualization is accurate for diagnosing nasal aspergillosis; nasal discharge samples and blind swabs have poor diagnostic value.

NOTE The finding of a few hyphae or growing *Aspergillus* organisms from a nasal flush does not reliably indicate that the dog has clinical aspergillosis. A small number of hyphae may be found in dogs with clinical signs not associated with aspergillosis.

DIFFERENTIAL DIAGNOSIS

Tumors, allergic rhinitis, nasal foreign bodies, lymphocytic-plasmacytic rhinitis, and tooth root abscesses are other causes of chronic nasal discharge that must be differentiated from nasal aspergillosis. Most of these dogs will have secondary bacterial rhinitis, but primary bacterial rhinitis appears uncommon in dogs.

MEDICAL MANAGEMENT

Oral administration of azole antifungal agents is effective in only 43% to 70% of cases, requires months of therapy, and is costly. Conversely, clotrimazole can be infused into the nasal passages and frontal sinuses of dogs through endoscopically placed catheters. This method is associated with an 80% to 90% success rate; however, it is time-consuming and may have significant complications (including death) if performed incorrectly. In many patients, a single application is curative, but some dogs require repeated therapy. CT is necessary to ensure an intact cribriform plate before such therapy. Rhinoscopic debridement of fungal plaques followed by infusion of enilconazole is described as effective in some animals. A small number of dogs with severe or recurrent nasal aspergillosis have been successfully treated with rhinotomy (see p. 839) and surgical debridement of nasal plaques followed by infusion of enilconazole.

NOTE Infusion procedures should be performed with care because leakage of clotrimazole into the lungs can cause fatal pneumonitis.

SURGICAL TREATMENT

Rhinotomy with resection of affected turbinates and associated soft tissues alone is seldom indicated and does not cure aspergillosis. Rhinostomas have been created to allow debridement and repeated topical treatment over many weeks; this treatment is traumatic to the animal, costly, and labor intensive. As previously noted, some dogs with nasal aspergillosis have been successfully treated with rhinotomy, debridement, and infusion of an antifungal agent, but the number is small.

Canine nasal aspergillosis is treated most appropriately by topical clotrimazole (Lotrimin). Although placement of tubes through the dorsal aspect of the nasal bones has been described for instillation of antifungal drugs in the past, instillation through tubes placed through the oral cavity and nares has proved effective and eliminates the need for surgical removal or trephination (see p. 882) of the nasal bone. Placement through the oral cavity and nares is described later. In cases where only the frontal sinus is involved, trephination (see p. 882) of the frontal bone for debridement and topical treatment of the sinus have been recently advocated and have led to favorable outcomes with significant reduction of time under general anesthesia.

Preoperative Management

Complications and death may occur if the cribriform plate is not intact; therefore a CT scan should be done before clotrimazole therapy is administered.

Anesthesia

The dog is placed under general anesthetic, and the cuff of the endotracheal tube is checked to make sure it is fully inflated; this prevents leakage of the infused solution into the trachea.

Surgical Anatomy

The surgical anatomy of the nasal cavity is described on p. 837.

SURGICAL TECHNIQUE

Technique for Administering Clotrimazole

With the dog in lateral or dorsal recumbency, place a Foley catheter (24 Fr) into the mouth in such a way that the tip of the catheter lies dorsal to the soft palate (Fig. 28.46). This process may be aided by grasping the catheter tip with a pair of right-angle forceps or long-handled needle holders so that the catheter tip is directed rostrally. *If necessary, place a mouth gag and pull the tongue rostrally to improve visualization during placement of the catheter. Advance the catheter until the balloon is dorsal to the junction of the hard and soft palates. Inflate the balloon of the Foley catheter to occlude the nasopharynx. Palpate the balloon through the soft palate to confirm its position just caudal to the hard palate. Place moistened laparotomy sponges in the pharynx to prevent the catheter from migrating caudally and to absorb any infusion that might escape around the balloon. Remove the mouth gag and advance a polypropylene infusion catheter (10 Fr) through each nostril. Beginning dorsomedially, advance each catheter into*

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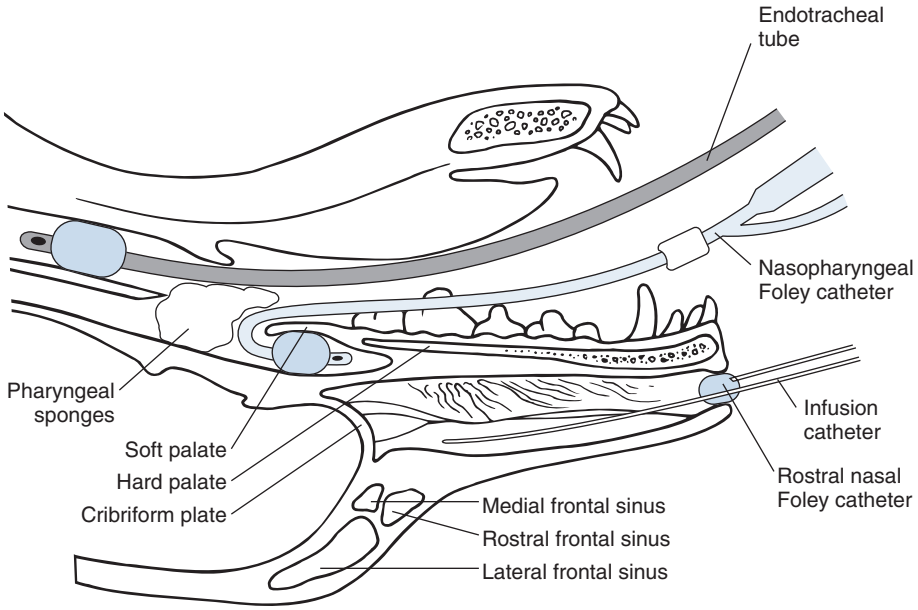


FIG. 28.46 For placement of tubes for nasal instillation of clotrimazole, place a Foley catheter (24 Fr) into the mouth so that the tip of the catheter lies dorsal to the soft palate. Place moistened laparotomy sponges in the pharynx. Remove the mouth gag and advance a polypropylene infusion catheter (10 Fr) through each nostril. Then insert a Foley catheter (12 Fr) into each nostril, and inflate the balloons so they lie just caudal to and occlude the nostrils.

the dorsal nasal meatus to the level of the medial canthus of the ipsilateral palpebral fissure. Then insert a Foley catheter (12 Fr) into each nostril and inflate the balloons so that they lie just caudal to and occlude the nostrils. With the dog in dorsal recumbency, place an additional laparotomy sponge just caudal to the upper incisors between the endotracheal tube and the incisive papilla to control leakage of clotrimazole through the incisive ducts.

Evenly divide 100 mL of a polyethylene glycol–based 1% clotrimazole solution between two 60-mL syringes. Infuse the clotrimazole slowly over 1 hour (50 mL per infusion catheter). Maintain the polypropylene catheters in a horizontal position, parallel to the table, throughout the infusion. Rotate the dog's head to ensure drug contact with all nasal surfaces. After 1 hour, place the dog in sternal recumbency and remove the catheters and sponges, allowing the clotrimazole to drain rostrally. Suction the pharynx and the proximal esophagus, and allow the dog to recover from the anesthesia.

Trephination

Place the dog in sternal recumbency. Landmarks for trephination into the frontal sinus include the zygomatic process of the frontal bone laterally, the midline of the skull medially, and the orbital rim ventrally. *Make a 1-cm skin incision in the center of the triangle bordered by these landmarks. Use a Michel trephine (Fig. 28.47) or a large Steinmann pin (e.g., 5/32 inch [0.4 cm]) to enter the frontal sinus. Pass an endoscope through the trephine hole to visualize the fungal plaques and begin debridement. When the procedure is complete, leave the bony defect open and close the skin incision routinely.*



FIG. 28.47 Michel trephine.

POSTOPERATIVE CARE AND ASSESSMENT

After clotrimazole treatment, the animal should be observed carefully for seizure or respiratory difficulty. Seizures and sometimes death occur if the cribriform plate has been eroded. Pneumonia may occur if clotrimazole is aspirated. Some dogs with advanced disease may require several 1-hour infusions. In most dogs, nasal pain and discharge resolve rapidly, and the nasal erosion heals quickly. Failure to cure the disease is a problem with some antifungal agents. Some animals with severe rhinitis may develop caudal choanal stenosis.

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PROGNOSIS

Although fungal disease can be eradicated, some dogs continue to have clinical signs associated with loss of normal turbinate structure. Owners should be warned that mild to moderate nasal discharge might continue in these dogs despite improvement in clinical signs. Bacterial rhinitis may occur in up to 25% of dogs after treatment, but it typically responds to appropriate antibiotic therapy. Relapse of aspergillosis does not appear to be a common problem.

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29

Surgery of the Lower Respiratory System: Lungs and Thoracic Wall



GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Thoracotomy is surgical incision of the chest wall; it may be performed by incising between the ribs (**intercostal** or **lateral thoracotomy**) or by splitting the sternum (**median sternotomy**). Pulmonary **lobectomy** is removal of a lung lobe (**complete**) or a portion of a lung lobe (**partial**). **Pneumonectomy** is removal of all lung tissue on one side of the thoracic cavity.

PREOPERATIVE MANAGEMENT

Animals with traumatic lesions that impair respiration (e.g., flail chest) or those with acute respiratory impairment (i.e., ruptured bulla or ruptured pulmonary abscess) often require emergency stabilization (e.g., stabilization of rib segments, thoracentesis, and oxygen therapy) before surgery. Equipment for thoracentesis and thoracostomy tube placement should be readily available, and clinicians should be familiar with these techniques (see pp. 920–924). The thorax is one of the most common regions injured following blunt trauma. Approximately three-quarters of animals having blunt trauma will have thoracic injuries, and approximately half will have concurrent abdominal and thoracic injuries.

NOTE Carefully auscultate the thorax of all trauma patients. Many will have pulmonary contusions, and finding abnormal sounds on thoracic auscultation typically correlates well with finding radiographic signs of thoracic trauma.

With large neoplastic lesions, positioning the animal in sternal recumbency or in lateral recumbency with the affected side down while providing oxygen supplementation (i.e., nasal insufflation or oxygen cage) is often beneficial (see Box 4.4). Blood gas analysis or evaluation with pulse oximetry is warranted preoperatively in patients undergoing thoracic surgery to detect and define the severity of respiratory impairment. Unexplained abnormalities should be investigated because ventilatory impairment caused by nonsurgically correctable disease (i.e., diffuse micrometastasis) is occasionally identified. If possible, significant anemia should be corrected before surgery (see p. 27).

ANESTHESIA

Respiratory patients should be managed with extreme care until intubation has been accomplished and ventilation can be assisted. Premedication with any drug that causes hypoventilation is contraindicated (see Table 30.3). Additionally, every attempt should be made to minimize stress to the patient prior to induction. Oxygen support, even while placing an intravenous catheter, may be necessary. Pulse oximetry with a sensor that can be secured to the tail can be especially helpful with perioperative monitoring of patients in respiratory distress. Prior to induction, the anesthesia provider should be prepared with airway devices, anesthesia machine, and monitors, as well as induction and emergency drugs. Preoxygenating the patient for 3 to 5 minutes before induction (see p. 29) should be followed with a rapid induction and intubation. Intubation of a bronchus rather than the trachea may be disastrous in compromised animals; therefore both sides of the chest cavity should be auscultated and the endotracheal tube palpated in the thoracic inlet to ensure proper placement. Confirmation should be made with the presence of end-tidal CO_2 ($EtCO_2$).

Animals with open chest cavities (including those with diaphragmatic hernias) require intermittent positive pressure ventilation. $EtCO_2$ is the measurement of CO_2 in exhaled respiratory gases. $EtCO_2$ is measured by capnography and serves as an estimate of arterial CO_2. Thus capnography is a noninvasive method of measuring systemic metabolism, cardiac output, pulmonary perfusion, and ventilation; its measurement may be helpful in identifying potentially life-threatening situations (e.g., hypoventilation, airway obstruction, hypotension, ventilator malfunctions, or esophageal intubation). The normal arterial CO_2 in an awake and healthy animal is 35 to 45 mm Hg. If ventilation and perfusion are well matched, $EtCO_2$ is generally 2 to 5 mm Hg less than that of the arterial partial pressure of CO_2 ($PaCO_2$).

If possible, positive pressure ventilation should be performed with lower tidal volumes, lower peak ventilation pressures, and higher respiratory rates. This will decrease leakage of air through damaged alveoli if pneumothorax is present. However, if the animal has a diaphragmatic hernia or pleural effusion, tidal volumes may be kept low but peak airway pressures may need to be higher to adequately ventilate the patient. If external pressures asserted on the lung tissue are completely compressing alveoli, bronchioles, and bronchi, it may take higher pressures to ventilate until the thorax is opened. When possible, however, smaller tidal volumes, higher respiratory rates, and lower peak airway pressures should be used. Dogs and cats with respiratory insufficiency should be maintained with inhalation anesthetics (e.g., isoflurane or sevoflurane; Table 29.1). Inhalation anesthesia is advantageous because it allows rapid recovery and more precise control of anesthetic depth than does maintaining anesthesia with longer-acting intravenous anesthetics. Nitrous oxide should not be used in patients with pneumothorax or diaphragmatic hernias

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ABSTRACT

Surgery of the lungs involves partial or complete lobectomy due to neoplasia, consolidated abscessation, trauma, or torsion. Trauma or neoplastic invasion may require thoracic wall resection or, in the case of flail chest, stabilization. Access to the thoracic cavity can be achieved through minimally invasive methods such as keyhole incisions or thoracoscopy to perform diagnostic procedures (e.g., aspirate, biopsy) or accomplish lung lobectomy for small, peripheral pulmonary lesions. Otherwise, more extensive evaluation of the lungs and thoracic cavity involves intercostal thoracotomy or median sternotomy.

KEYWORDS

lungs
thoracic wall
thoracotomy
sternotomy
lobectomy
thoracoscopy
flail chest
pulmonary
neoplasia
pulmonary abscess
lung lobe torsion
pectus excavatum

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TABLE 29.1 Anesthetic Considerations for Thoracotomy

Preoperative Considerations

Associated conditions	• May be septic if purulent effusion with dehydration, hypotension, electrolyte abnormalities, and abnormal glucose • If older patient, may have other comorbidities • Adrenal suppression may be present in the critically ill patient • Anemia
Bloodwork	• HCT • Electrolytes • BUN • Cr • TP • +/– Lactate • +/– BG • +/– Blood gas
Physical exam	• May be markedly stressed and extremely dyspneic • Absent pulmonary sounds depending on etiology • May have muffled heart sounds • Hypotension, dehydration, tachycardia, or bradycardia in the critically ill patient
Other diagnostics	• Thoracocentesis if pleural effusion • Blood pressure • ECG • SpO_2 • Radiographs (thoracic and +/– abdominal) • +/– Ultrasound • +/– CT

Premedications	• Oxygen with flow-by or face mask
	• Avoid sedatives in depressed or dyspneic patients
	• In patients that have minimal respiratory compromise, give the following if anxious:
	  • Midazolam (0.2 mg/kg IV, IM), *or*
	  • Diazepam (0.2 mg/kg IV), *or*
	  • Buprenorphine (0.005–0.02 mg/kg IV, IM)
	  • Butorphanol (0.2–0.4 mg/kg IV, IM)
	• Prior to induction, avoid all respiratory depressants, including but not limited to, opioids, xylazine, medetomidine, dexmedetomidine; if hypotensive, avoid acepromazine, xylazine, medetomidine, dexmedetomidine.
	• After a benzodiazepine is given but before induction, clip hair from thorax; this can usually be done with patient standing or in lateral recumbency; do not place patient in ventrodorsal position until after intubation.

Intraoperative Considerations

Induction	• Preoxygenate for 3–5 min with face mask or flow-by oxygen
	• If normal cardiac function, give:
	  • Propofol (4–8 mg/kg IV); administer rapidly to expedite intubation and ventilation, *or*
	  • Alfaxalone (2–5 mg/kg IV)
	• If decreased cardiac function, give:
	  • Etomidate (0.5–1.5 mg/kg IV); if possible, avoid its use in critically ill patients. Give a benzodiazepine before etomidate to avoid myoclonic activity.

Maintenance	• Isoflurane or sevoflurane, *plus*
	  • Fentanyl (2–10 µg/kg IV PRN in dogs; 1–4 µg/kg IV PRN in cats) for short-term pain relief, *plus*
	  • Fentanyl CRI (1–5 µg IV loading dose, then 2–30 µg/kg/h IV), *or*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Oxymorphone (0.1–0.2 mg/kg IV), *or*
	  • Buprenorphine[b] (0.005–0.02 mg/kg IV PRN) *plus*
	  • Ketamine (low dose) (0.5–1 mg/kg IV), *or*
	  • Ketamine CRI (0.5 mg/kg IV loading dose, then 10 µg/kg/min IV)
	• If hypotensive (to keep MAP 60–80 mm Hg), give phenylephrine, ephedrine, dopamine, norepinephrine, or fluids as needed
	• Avoid nitrous oxide
	• Maintain SpO_2 >95%
	• Ventilation parameters:
	  • Small tidal volumes: 6 mL/kg
	  • Respiratory rates: 10–30
	  • Peak airway pressures: <12–15 mm Hg if able to maintain adequate tidal volumes
	• If unable to meet above parameters, higher peak pressures may need to be used initially; as intrathoracic pressure is reduced, drop peak airway pressures and tidal volumes accordingly; slowly expand the lungs while manually ventilating and visually observing lung expansion. Consider PEEP.

Fluid needs	• 5–20 mL/kg/h to replace evaporative losses plus 3× EBL; consider colloids for EBL replacement in 1:1 ratio

Continued

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TABLE 29.1 Anesthetic Considerations for Thoracotomy—cont'd

Monitoring	• BP • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature • +/– Arterial line • U/O

Blocks	• Epidural:
	  • Morphine (0.1 mg/kg preservative free), *or*
	  • Buprenorphine (0.003–0.005 mg/kg diluted in saline), *plus*
	  • Local anesthetics (see Chapter 13)
	• Intercostal nerve blocks with local anesthetics (see Box 30.2), *plus*
	• Incisional block with local anesthetic

Postoperative Considerations	
Analgesia	• Fentanyl CRI (1–10 µg/kg IV loading dose, then 2–20 µg/kg/h IV), *or* • Morphine[c] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.1–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h in cats) if no hypotension, *or* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats), *or* • Oxymorphone (0.1–0.2 mg/kg IV, IM q2–4h), *or* • Hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs), *or* • Buprenorphine[b] (0.005–0.02 mg/kg IV, IM q4–8h or 0.01–0.02 mg/kg OTM q6–12h in cats), *plus* • +/– Ketamine CRI (2 µg/kg/min IV; if no previous loading dose, give 0.5 mg/kg IV before CRI) • Avoid NSAIDs in elderly, hypotensive, and critically ill patients
Monitoring	• SpO_2 • Blood pressure • ECG • HR • Respiratory rate • Temperature • U/O • Chest tubes
Bloodwork	• HCT • TP • Electrolytes • Blood gas • +/– Albumin • +/– BG
Estimated pain score	Severe

[a]Monitor for hyperthermia in cats.
[b]Buprenorphine is a better analgesic than morphine in cats.
[c]Give slowly to prevent histamine release.
BG, Blood glucose; *BP,* blood pressure; *BUN,* blood urea nitrogen; *Cr,* creatinine; *CRI,* constant-rate infusion; *CT,* computed tomography; *EBL,* estimated blood loss; *ECG,* electrocardiogram; *$ETCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *MAP,* mean arterial pressure; *NSAID,* nonsteroidal antiinflammatory drug; *OTM,* oral transmucosal; *PEEP,* positive end-expiratory pressure; *PRN,* as needed; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein; *U/O,* urine output.

because it rapidly diffuses into air-filled spaces (e.g., pleural cavity or gas-filled organs) causing further lung compression or organ enlargement. In addition, nitrous oxide is comparatively less soluble in plasma than are oxygen and other inhalation anesthetics; therefore it rapidly diffuses into the alveoli when it is discontinued, resulting in diffusion hypoxia if the nitrous oxide is not turned off 5 to 10 minutes prior to extubation. Once surgery is completed, extubation should not be rushed. Make sure the patient is awake and not overly sedated, respirations are adequate, and the patient is comfortable prior to extubation.

For specific anesthetic recommendations for animals with flail chest or pectus excavatum, see pp. 899 and 910, respectively.

NOTE Do not use mask or chamber induction in animals with respiratory dysfunction. Use anesthetics that allow for rapid intubation and control of the patient's airway.

Thoracoscopy is best done without insufflation; simply establishing a pneumothorax is sufficient for almost all cases. Should intrathoracic insufflation be required for thoracoscopy, it may have profound effects on cardiopulmonary parameters. If insufflation pressures exceed central venous pressures, venous blood flow return to the heart decreases, causing a significant drop in cardiac output and systemic blood pressure. If the heart rate also decreases, this can be an ominous sign of cardiovascular collapse. Therefore insufflation-assisted thoracoscopy should be done with care using the lowest inspiratory pressure possible. Use of 5 cm H_2O of positive end-expiratory pressure (PEEP) increases the arterial partial pressure of oxygen (PaO_2) by decreasing shunt fraction and dead space in dogs with one lung ventilation, but it does not affect cardiac output. Therefore PEEP is recommended with one lung ventilation.

Thoracotomy procedures often cause substantial pain, and multimodal postoperative analgesic therapy is indicated (see also Chapters 13 and 30). Performing intercostal nerve blocks prior

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to extubation can provide significant analgesia in the immediate postoperative period (see Box 30.2). Injectable analgesics may also be used (e.g., hydromorphone, butorphanol, or buprenorphine; see Tables 13.2 and 13.3). Although opioids are respiratory depressants, the analgesic effects of these drugs often outweigh the negative respiratory effects. If hypoventilation occurs after administration of opioids, oxygen should be given by nasal insufflation (see Chapter 4). Epidural administration of morphine is an effective analgesic in patients undergoing thoracic procedures and should be considered in any patient undergoing a thoracotomy (see Table 13.5). There is a delay of the onset of thoracic analgesia of approximately 6 to 8 hours. This coincides with the wearing off of the intercostal nerve blocks. When used together, nerve blocks and epidural morphine can provide substantial pain relief for the first 12 to 24 hours postoperatively. Epidural administration of morphine alone does not usually cause significant changes in cardiorespiratory measurements, but these patients should be monitored carefully for 24 hours for a delayed respiratory depression, especially when intravenous (IV) opioids are also administered.

NOTE If a morphine epidural has been administered perioperatively, butorphanol and buprenorphine should not be administered because of their antagonistic properties.

Reexpansion pulmonary edema (RPE), although uncommon, may develop in animals after chronically collapsed lung lobes are reexpanded and is not associated with cardiac disease. In sheep, goats, and rats, RPE can occur after lungs have been collapsed for 3 days or more. It is possible for RPE to occur in any type of chronically collapsed lung that can be reexpanded. Therefore lung protective ventilator strategies are recommended for any lung reexpansion surgeries (e.g., diaphragmatic hernia, pectus excavatum, pleural effusion, pneumothorax, large pulmonary neoplasia). RPE is characterized by fluid within the alveoli, interstitial edema, and thickened basement membranes. It is believed that RPE is a permeability pulmonary edema associated with the injury of pulmonary microvessels.

There are two major causes of RPE. One is the abnormality of the pulmonary microvessels caused by the chronic collapse. The microvasculature becomes thickened, less flexible, and more susceptible to injury. The second is mechanical stress caused by the abrupt reexpansion. For years, barotrauma had been discussed as the primary cause of this mechanical stress. However, more recent research into ventilation-induced lung injury and acute respiratory distress syndrome has revealed that there are multiple aspects to lung injury with barotrauma playing less of a role than initially believed. The mechanical stress of volutrauma (the overdistension of alveoli) may occur whenever chronically collapsed lungs are reexpanded. When a given tidal volume is delivered, it goes to the areas of the lung that are most compliant, which is generally not the area that is diseased or collapsed. If normal tidal volumes are used, these healthy and compliant regions become overly distended with subsequent shearing of alveolar walls and destruction of pulmonary microvasculature. This overdistention and initiation of an inflammatory cascade may have severe systemic consequences. Another mechanical stress, atelectrauma, occurs with repeated opening and closing of the collapsed alveoli. These unstable alveoli may undergo opening and closing with each respiratory cycle. This rapid and repetitive wall stress can also destroy pulmonary microvessels, contributing to the pulmonary edema seen with reexpansion of collapsed lungs.

To avoid RPE, there are two general concepts to consider. The first is slow recruitment of collapsed lung by using PEEP. A PEEP of 5 to 10 helps to prevent recollapsing of alveoli and the associated atelectrauma. The second strategy is to be very careful not to over distend healthy lungs. This may be challenging because most veterinary anesthesia machines do not have measured tidal volume displays. Therefore careful observation of the lungs within the surgical field is imperative. Use estimated tidal volumes of less than 6 mL/kg, being careful not to over inflate normal lung tissue. Do not attempt to rapidly recruit collapsed lung because this will only damage healthy, compliant lung. To achieve adequate oxygenation, lung volumes will need to be small and respiratory rates more frequent. Do not administer "sighs," as they will almost certainly cause overdistention. Once the chest tube is in place and the thoracic closure is being completed, do not distend the lungs to remove the air from the pleural space. Slowly correct the pneumothorax by incrementally removing small portions over several hours. The goal is to gradually reduce the pneumothorax to avoid further lung trauma, rather than rapid correction. Although not easy to do, avoiding RPE is much easier than treating it. Glucocorticoids are of little value in treating RPE once it has occurred. In severe RPE cases, prolonged ventilation using low tidal volumes, higher respiratory rates, and PEEP is usually necessary. Other therapies targeting oxygen-derived free radicals may be on the horizon, but currently the best treatment of RPE is careful avoidance.

Obstruction of blood flow in the pulmonary vasculature by a thrombus or an embolus formed in the systemic venous system or right side of the heart is *pulmonary thromboembolism* (PTE). It may occur as a complication of a number of diseases (e.g., neoplasia, cardiac disease, sepsis, immune-mediated hemolytic anemia, hyperadrenocorticism, heparin-induced thrombocytopenia, and protein-losing nephropathy or enteropathy). It is difficult to diagnose antemortem because of the lack of specific signs. If it happens while the animal is under anesthesia, a rapid drop in $EtCO_2$ may occur. This is followed by a significant drop in blood pressure, a rise in heart rate, and a decline in hemoglobin saturation with oxygen (SpO_2). Clinical signs associated with feline PTE include lethargy, anorexia, weight loss, and difficulty breathing. In dogs, vomiting, melena, fever, labored breathing, and lethargy have been reported. Risk factors for PTE include administration of corticosteroids, chemotherapeutic agents, or blood; indwelling catheters; and recent surgery. PTE should be suspected in animals with thoracic radiographic changes suggestive of uneven distribution of blood flow between lung lobes or patchy interstitial densities; pulmonary perfusion scintigraphy or computed tomography (CT) angiography may help confirm the diagnosis. Treatment of PTE includes anticoagulant and thrombolytic therapies plus standard hemodynamic and respiratory support (Box 29.1). Heparin acts primarily to limit the conversion of fibrinogen to fibrin by accelerating the action of antithrombin III in inhibiting activated coagulation factors (II, IX, X, XI, and XII). Unfractionated heparin (UFH) has been most commonly used because of its widespread availability and low cost. It should be dosed to prolong the activated partial thromboplastin time (aPTT) 1.5 to 2 times that of baseline. Fractionated or low-molecular-weight heparin (LMWH; e.g., enoxaparin, dalteparin) differs from UFH in that it more specifically acts on factor X and is likely more effective. It also requires different monitoring than UFH and is more expensive. Prolongation of the aPTT

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BOX 29.1 Drugs Used in Medical Management of Pulmonary Thromboembolism

Unfractionated Heparin
Dogs: Start with 200–400 U/kg SC q6h[a]
Cats: Start with 300–500 U/kg SC q8h[a]

Fractionated Heparin—Dalteparin (Fragmin)[b]
Dogs: 100–150 U/kg SC q8h
Cats: 100–150 U/kg SC q4h or 180 U/kg SC q6h

Fractionated Heparin—Enoxaparin (Lovenox, Xaparin, Clexane)[b]
Dogs: 0.8 mg/kg SC q6h
Cats: 1.0 mg/kg SC q12h up to 1.25 mg/kg SC q6h

Aspirin
Dogs: 0.5–2 mg/kg PO q24h
Cats: 20 mg/cat PO q3d

Clopidogrel (Plavix)
Dogs: Loading dose of 10 mg/kg then 2–3 mg/kg PO q24h
Cats: 18.75 mg/cat q24h

Streptokinase[c]
90,000 U given IV over 30–60 min, then 45,000 U/h CRI

Tissue Plasminogen Activator
Dogs: 1 mg/kg IV over 15 min q 60–80 min until resolved
Cats: 1 mg/kg IV up to 6 mg total dose; give 10% of dose as a bolus with remainder as a CRI over 60 min

[a]Dose to be adjusted based on aPTT results (goal is to prolong it to 1.5–2 times baseline).
[b]These are doses currently thought to be appropriate; however, as of this writing, there has been minimal research published on the use of this product in diseased animals.
[c]There is minimal experience with this drug in veterinary medicine, and serious side effects are possible. Its use is not recommended except by internists with experience and training with this drug.
aPTT, Activated partial thromboplastin time; *IV,* intravenous; *PO,* oral; *PT,* prothrombin time; *SC,* subcutaneous.

BOX 29.2 Calculation of International Normalized Ratio (INR) for Warfarin Therapy[a]

$$INR = \left(\frac{PT_{patient}}{PT_{control}}\right)^{ISI}$$

- The international standardization index (ISI) is provided by the manufacturer.
- ISI is a measure of the responsiveness of a specific thromboplastic compared to the reference population.
- INR should be assessed daily until a therapeutic range is achieved.[b]
- INR is then checked at least twice weekly for 1 to 2 weeks, then less frequently depending on the stability of results.

[a]See www.easycalculation.com/medical/inr.php.
[b]In humans, the normal range for INR is 0.8–1.2. The therapeutic range for anticoagulation is 2–3. Values <0.5 indicate high risk for clot formation; values >3.5 indicate a high risk for bleeding.
PT, Prothrombin time.

does not occur at therapeutic doses of LMWH, but patients may be monitored by following clinical signs and assessing for anti-Xa activity. Low-dose aspirin administration to inhibit platelet activity may be considered, but care must be taken to ensure that the aspirin does not cause renal or gastrointestinal complications, especially in patients receiving steroids or other nephrotoxic drugs.

Warfarin prevents the formation of vitamin K-dependent coagulation factors (II, VII, IX, and X). It also inhibits production of protein C. Warfarin therapy is monitored using prothrombin time (PT) and the calculated international normalization ratio (INR). The traditional end point of warfarin therapy is a PT of 1.5 to 2 times that of baseline; however, due to variations in the PT assay, PT standardization is best achieved using INR (Box 29.2). The therapeutic range of warfarin is an INR of 2.0 to 3.0. Due to the narrow therapeutic window and high incidence of adverse effects, use of warfarin in veterinary medicine is not routinely recommended.

Newer thromboprophylactic agents include the platelet inhibitor clopidogrel (Plavix) and direct factor Xa inhibitors apixaban (Eliquis) and rivaroxaban (Xarelto). Scientific information regarding the use of these drugs in veterinary medicine is limited, although they are frequently prescribed in dogs and cats at risk for thromboembolism (e.g., immune-mediated hemolytic anemia, feline hypertrophic cardiomyopathy).

ANTIBIOTICS

Animals with underlying pulmonary disease or trauma (i.e., pulmonary contusions) are at increased risk of developing pulmonary infections. These patients should be monitored carefully, and prophylactic antibiotics (e.g., cefazolin, 22 mg/kg IV or amoxicillin/sulbactam [Unasyn], 30 mg/kg IV at induction; repeat once or twice at 90-minute to 2-hour intervals) should be provided, or therapeutic antibiotics should be initiated at the earliest sign of infection (i.e., leukocytosis and/or fever). Until culture and antimicrobial susceptibility test results return, ampicillin, clindamycin, or enrofloxacin are other rational choices for treatment of suspected infectious lower respiratory tract diseases of dogs.

Appropriate use of prophylactic antibiotics depends on the length of surgery, the type of surgery being performed, the animal's immune status, and the underlying disease process. Debilitated animals undergoing thoracotomy for removal of large neoplastic lesions (which may contain focal areas of necrosis) are likely to benefit from prophylactic antibiotic therapy. Prophylactic antibiotics should be given intravenously at induction of anesthesia and generally discontinued immediately after surgery or within 12 to 24 hours (see Chapter 9).

SURGICAL ANATOMY

The thoracic cavities of dogs and cats are compressed laterally; therefore the greatest dimension is dorsoventral. The ribs, sternum, and vertebral column form the thoracic skeleton. The sternum is composed of eight unpaired bones and forms the floor of the thorax (Fig. 29.1). The first and last sternebrae are known as the *manubrium* and *xiphoid*, respectively. There are usually 13 pairs of ribs. The 10th, 11th, and 12th ribs do not articulate with the sternum but instead form the costal arch bilaterally. The cartilaginous portion of the 13th rib terminates free in the musculature.

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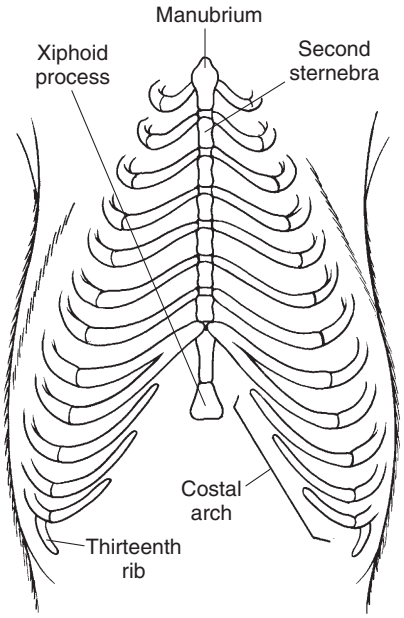


FIG. 29.1 Sternal anatomy.

TABLE 29.2 Recommended Intercostal Spaces for Thoracotomy[a]

	Left	Right
Heart	4, 5	4, 5
PDA	4 (5)	
PRAA	4	
Pulmonic valve	4	
Lungs	4–6	4–6
Cranial lobe	4, 5	4, 5
Middle lobe		5
Caudal lobe	5 (6)	5 (6)
Esophagus		
Cranial	3, 4	
Caudal	7–9	7–9
Cranial vena cava	(4)	4
Caudal vena cava	(6–7)	6–7

[a]Numbers in parentheses indicate alternative surgical sites.
PDA, Patent ductus arteriosus; *PRAA,* persistent right aortic arch.

The space between the ribs, known as the *intercostal space*, is generally two to three times as wide as the adjacent ribs. Blood supply to the thoracic wall is provided by the intercostal arteries, which lie caudal to the adjacent rib in conjunction with a satellite vein and nerve. A typical intercostal nerve begins where the dorsal branch of the thoracic nerve divides and runs distally among the fibers of the internal intercostal muscle. In most intercostal spaces, intercostal vessels and nerves are covered medially only by the pleura.

The muscles of the thorax not only serve a structural function but are also important in respiration. The deepest muscles of the thoracic wall are the intercostal muscles. The fibers of the *external intercostal muscle* arise on the caudal border of each rib and run caudoventrally to the cranial border of the next rib. This muscle is important primarily in inspiration. The *internal intercostal muscles,* on the other hand, run from the cranial border of one rib to the caudal border of the preceding rib, primarily functioning to aid expiration. Other inspiratory muscles are the scalenus, serratus dorsalis cranialis, levatores costarum, and diaphragm. Additional expiratory muscles include the rectus abdominis, external abdominal oblique, internal abdominal oblique, transversus abdominis, serratus dorsalis caudalis, transversus costarum, and iliocostalis.

The lungs of dogs and cats have deep fissures that create distinct lobes, which allow the lungs to alter their shape in response to alterations in the shape of the thoracic cavity from diaphragmatic movement or flexion or extension of the spine. These fissures also allow individual lobes to be isolated and removed without compromising the integrity of the surrounding lobes. The left lung is divided into a cranial lobe, with a cranial and caudal part, and a caudal lobe (Fig. 29.2). The right lung is larger than the left and is divided into cranial, middle, caudal, and accessory lobes (see Fig. 29.2). The *cardiac notch* is a small area overlying the heart where lung tissue is not interposed between the heart and body wall. It is usually located at the ventral aspect of the fourth intercostal space and is larger on the right side.

The pulmonary arteries carry nonaerated blood from the right ventricle of the heart to the lungs, and the pulmonary veins return aerated blood from the lungs to the left atrium. The left pulmonary artery lies cranial to the left bronchus, whereas the left pulmonary veins are ventral to it. On the right side, the pulmonary artery lies dorsal and slightly caudal to the right bronchus, and the pulmonary veins lie craniodorsal and ventral to it.

SURGICAL TECHNIQUE

Thoracotomy

Thoracotomy may be performed by incising between the ribs or by splitting the sternum. The approach used depends on the exposure needed and underlying disease process. Regardless of the type of thoracotomy performed, a large area should be prepared for aseptic surgery to allow extension of the incision if needed. Depending on which left lobe is affected, a left lateral thoracotomy at the fourth, fifth, or sixth intercostal space provides adequate exposure for lobectomy (Table 29.2). A left fourth intercostal space thoracotomy allows exposure of the right ventricular outflow tract, main pulmonary artery, and ductus arteriosus. Bilateral removal of the pericardial sac can be difficult from this approach. A right intercostal thoracotomy provides exposure of the right side of the heart (auricle, atrium, and ventricle), cranial and caudal vena cava, right lung lobes, and azygos vein. Median sternotomy affords exposure to both sides of the thoracic cavity. Bilateral, partial lobectomy is easily performed from a median sternotomy; however, complete lobectomy can be challenging. The caudal vena cava, main pulmonary artery, and both sides of the pericardial sac can be isolated and manipulated through this approach.

NOTE Be sure to count surgical sponges at the start of the surgical procedure and before closure of the thorax. This helps to ensure that you do not leave a sponge in the thoracic cavity.

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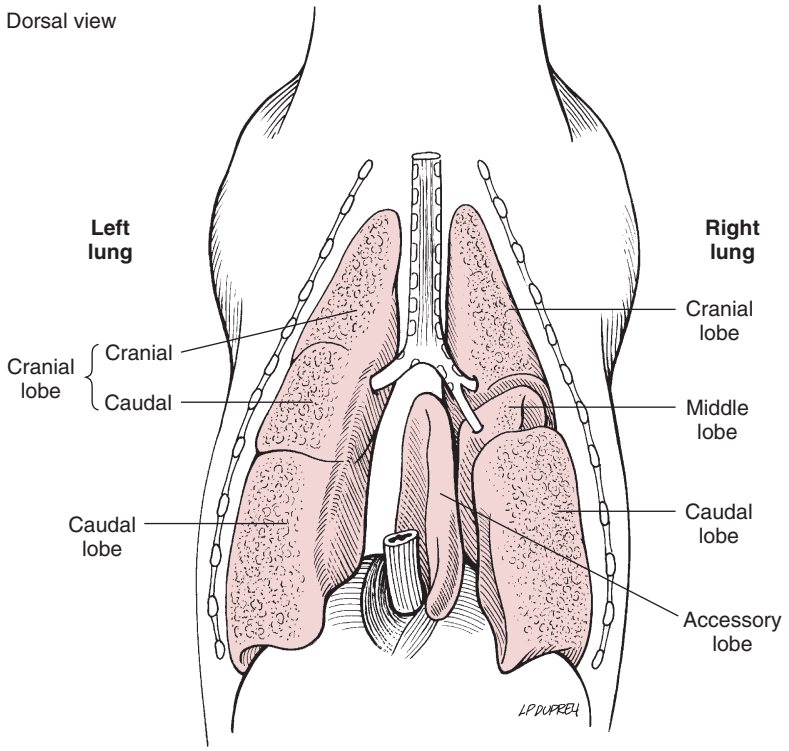


FIG. 29.2 Subdivisions of the canine and feline lung lobes.

Intercostal Thoracotomy

With the dog in lateral recumbency, select the site for incision (Video 29.1). Locate the approximate intercostal space, and sharply incise the skin, subcutaneous tissue, and cutaneous trunci muscle. The incision should extend from just below the vertebral bodies to near the sternum. Deepen the incision through the latissimus dorsi muscle with scissors (Fig. 29.3A), then palpate the first rib by placing a hand cranially under the latissimus dorsi muscle. Count back from the first rib to verify the correct intercostal space.

The ribs cranial to an intercostal incision are more easily retracted than the caudal ribs; therefore choose the more caudal space if you must choose between two adjacent intercostal spaces. Transect the scalenus and pectoral muscles with scissors perpendicular to their fibers, then separate the muscle fibers of the serratus ventralis muscle at the selected intercostal space (Fig. 29.3B). Near the costochondral junction, place one scissor blade under the external intercostal muscle fibers, and push the scissors dorsally in the center of the intercostal space to incise the muscle (Fig. 29.3C). Incise the internal intercostal muscle similarly. Notify the anesthetist that you are about to enter the thoracic cavity and, after identifying the lungs and pleura, use closed scissors or a blunt instrument to penetrate the pleura. This allows air to enter the thorax, causing the lungs to collapse away from the body wall. *Extend the incision dorsally and ventrally to achieve the desired exposure. Identify and avoid incising the internal thoracic vessels, as they course subpleurally near the sternum. Moisten laparotomy sponges and place them on the exposed edges of the chest incision. Use a Finochietto retractor to spread the ribs (Figs. 29.3D and 29.4).* If further exposure is necessary, a rib adjacent to the incision can be removed; however, this is seldom required.

Place a thoracostomy tube through an intercostal space one to two spaces caudal to the incision prior to closing the thorax. Alternatively, remove residual air from the thoracic cavity using an over-the-needle catheter following rib apposition and muscular closure.

Close the thoracotomy by preplacing four to eight sutures of heavy monofilament absorbable or nonabsorbable suture (3-0 to No. 2, depending on the animal's size) around the ribs adjacent to the incision (Fig. 29.5A and Video 29.2). Approximate the ribs with a rib approximator or have an assistant cross two sutures to appose the ribs (Fig. 29.5B), then tie the remaining sutures. Tie all the sutures before you remove the rib approximator. Suture the serratus ventralis, scalenus, and pectoralis muscles in a continuous pattern with absorbable suture material. Appose the edges of the latissimus dorsi muscle similarly. Remove residual air from the thoracic cavity using the preplaced thoracostomy tube or an over-the-needle catheter (see pp. 920–924). Close the subcutaneous tissue and skin in a routine fashion.

A muscle-sparing technique may also be performed in cats and small dogs. Here, rather than incising the latissimus dorsi muscle, the muscle is sharply separated along its ventral fascial attachments and elevated. Puppies may be more willing to ambulate shortly after thoracotomy if the latissimus dorsi muscle has not been incised. A 2015 study in 20 dogs found the muscle-sparing technique was associated with a less painful recovery during the first 7 days after surgery when compared to the traditional technique.[1] In addition, the muscle-sparing technique did not compromise exposure of thoracic viscera.

Transcostal sutures have also been reported for closure of intercostal thoracotomies; small holes are drilled in the caudal rib through which the suture is passed to avoid entrapment of the intercostal nerve against the rib. This closure method appears to result in markedly less postoperative pain; however, it is time consuming and may be associated with rib fracture in smaller animals.

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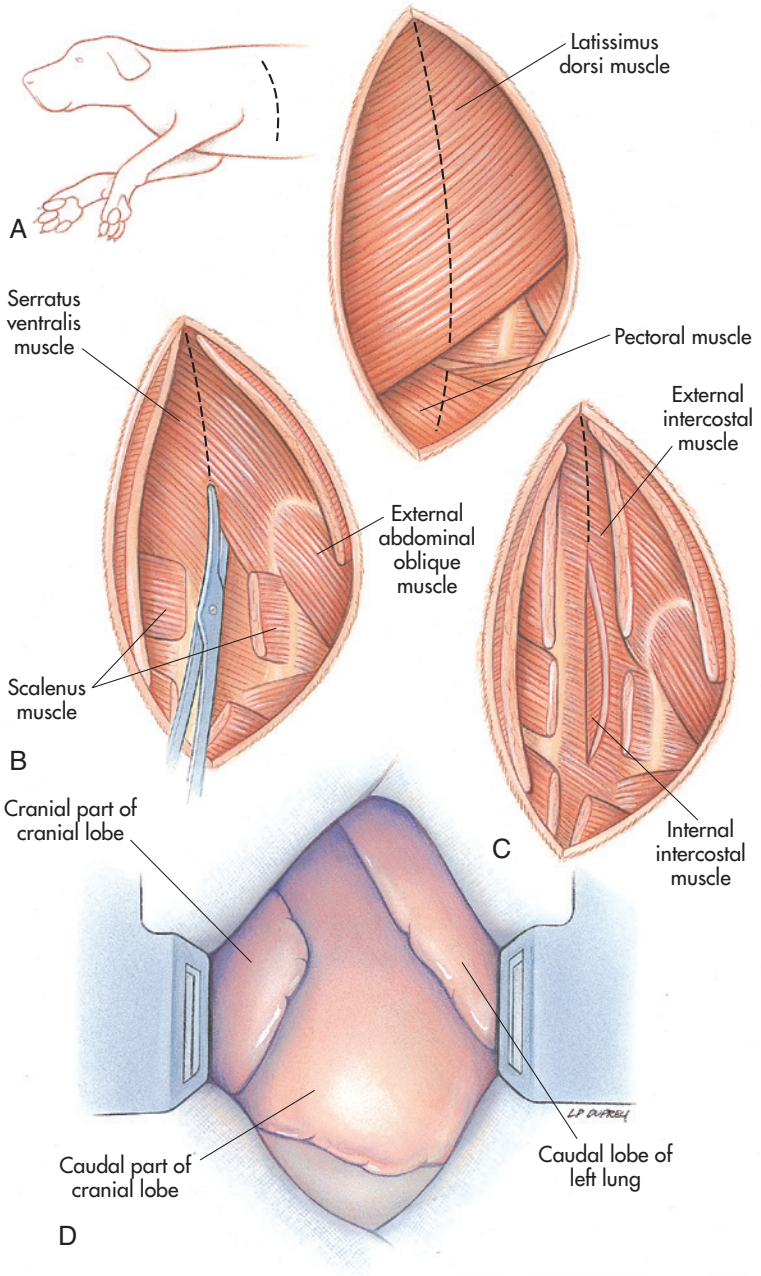


FIG. 29.3 Intercostal thoracotomy. (A) Sharply incise the skin, subcutaneous tissues, and cutaneous trunci muscle. Deepen the incision through the latissimus dorsi muscle with scissors. (B) and (C) Transect the scalenus, pectoral, serratus ventralis, and intercostal muscles. (D) Use a Finochietto retractor to spread the ribs.

Median Sternotomy

When performing median sternotomy, two or three sternebrae should be left intact cranially or caudally (depending on where the lesion is located) to reduce postoperative pain and prevent delayed healing caused by sternebral shifting. If exposure of the lungs or heart is necessary (i.e., in dogs with spontaneous pneumothorax or for pericardiectomy), the sternotomy should extend from the xiphoid cartilage cranially to the second or third sternebra. If exposure of the cranial mediastinum is desired, the sternotomy should extend from the manubrium caudally to the sixth or seventh sternebra.

In large dogs, sternotomies closed with wire may be more stable as healing is associated with chondral or osteochondral bridging. However, recent cadaveric studies have found sternotomy closures with suture (polydioxanone or nylon) are mechanically comparable to wire.[2,3] Single or double-twist figure-eight patterns centered between two sternebrae are associated with least displacement during biomechanical loading, whereas double-loop cerclage wiring should be avoided due to high failure rate under load.

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With the dog in dorsal recumbency, incise the skin on the midline over the sternum. Expose the sternum by a combination of sharp incision and blunt dissection of the overlying musculature. Transect the sternebrae longitudinally on the midline with a sternal saw (Fig. 29.6), bone saw (Fig. 29.7), or chisel and osteotome. A sternal saw has a guide that sits under the sternum, making it much easier to cut the sternum without damaging the heart or lungs underneath. *In young animals, heavy straight scissors may be adequate; however, avoid crushing the bone.* Splitting the sternebrae on the midline facilitates closure. *If using a bone saw or chisel, take extra precaution to ensure that the underlying lung and heart are not damaged while completing the sternotomy.* Once the xiphoid is split, a malleable (ribbon) retractor can be placed in the thorax underneath the sternum to protect the underlying viscera. *Place moistened laparotomy sponges on the incised edges of the sternebrae and retract the edges with a Finochietto rib retractor. If a thoracostomy tube is to be placed, do so before closing the sternotomy. Do not exit the tube from between the sternebrae; exit it from between the ribs or through the diaphragm. Close the sternotomy with wires (dogs larger than approximately 15 kg) or heavy suture (cats and dogs smaller than approximately 15 kg) placed around*

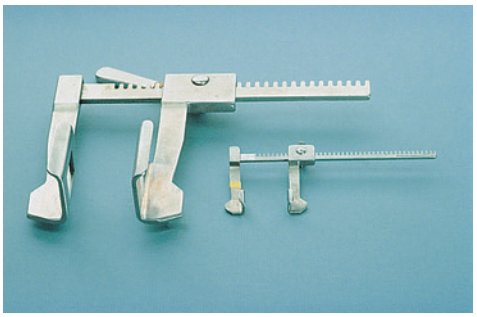


FIG. 29.4 Finochietto retractors.



FIG. 29.6 Sternal saw for performing a median sternotomy.

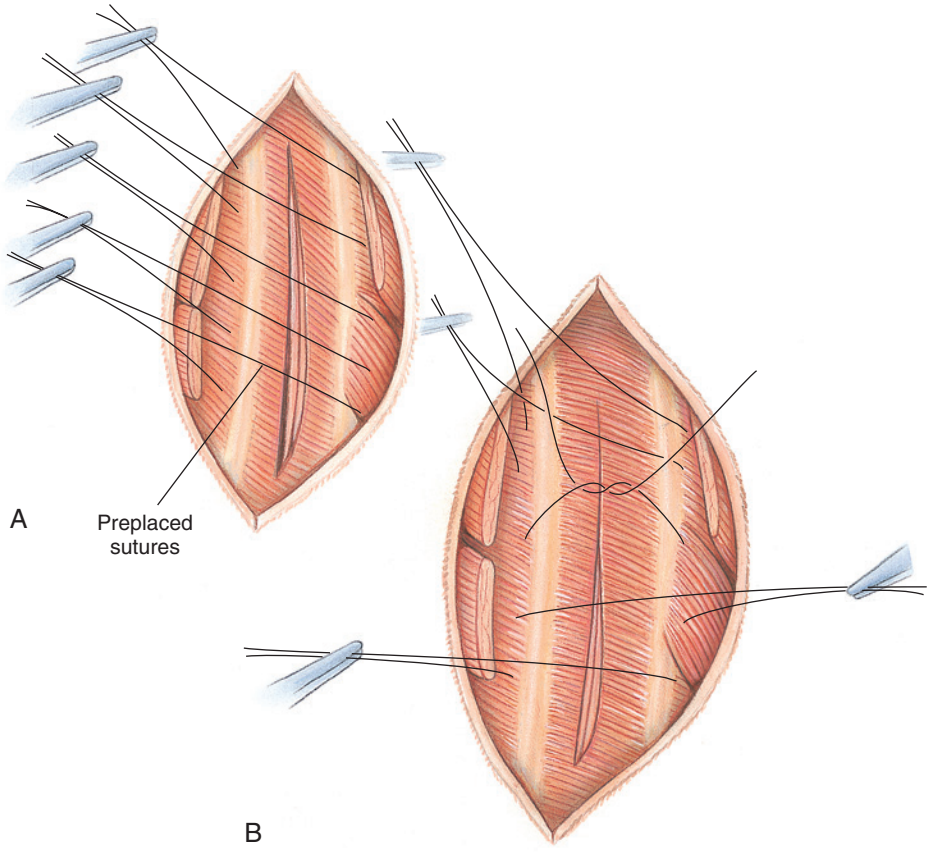


FIG. 29.5 (A) Close the thoracotomy by preplacing four to eight heavy monofilament sutures around the ribs adjacent to the incision. (B) Approximate the ribs with a towel clamp or rib approximator or have an assistant cross two sutures to appose the ribs. Tie the remaining sutures.

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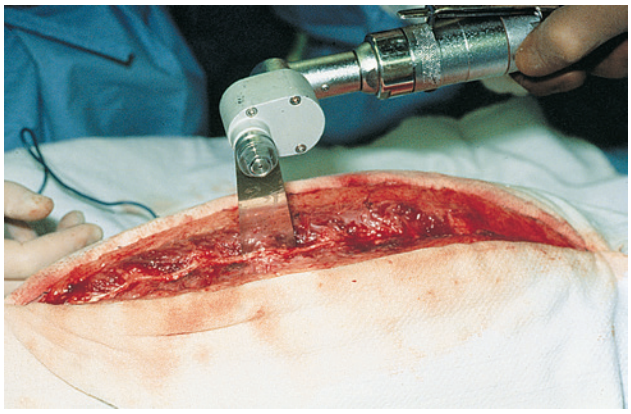
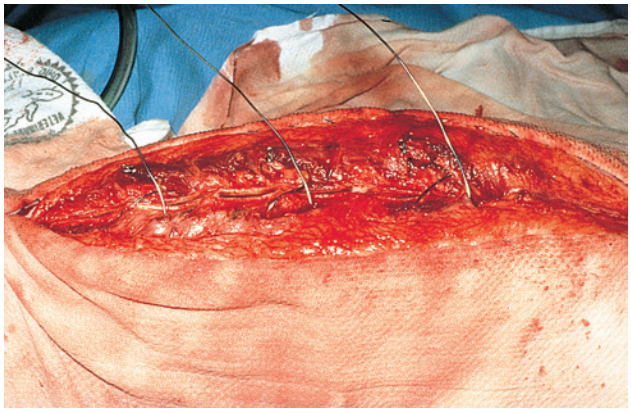


FIG. 29.7 When performing a median sternotomy, transect the sternebrae longitudinally on the midline with a bone saw, chisel and osteotome, or bone cutters.



FIG, 29.8 Depending on the animal's size, close a sternotomy with wires or heavy suture placed around the sternebrae.

the sternebrae in a figure-eight pattern (Fig. 29.8). Suture the subcutaneous tissue in a simple continuous pattern with absorbable suture. Remove residual air from the thoracic cavity and close the skin routinely.

Thoracoscopy

Video-assisted thoracic surgery or thoracoscopy is a minimally invasive technique for diagnostic and therapeutic procedures of the thoracic cavity. Thoracoscopy is performed in either dorsal recumbency with the camera portal placed in a paraxiphoid position or lateral recumbency with camera and instrument portals placed through intercostal spaces. To improve visualization and operative working space, one-lung ventilation or mainstem bronchial blockade can be performed (see Anesthesia on p. 886). Procedures that can be performed by thoracoscopy include, but are not limited to, exploratory thoracotomy (e.g., pyothorax); biopsies of lung, mediastinum, or pleura; pericardiectomy; lung lobectomy; correction of vascular ring anomalies; thymoma removal; and thoracic duct ligation. Thoracoscopic-assisted pulmonary surgery has been described for partial and complete lung lobectomy that does not require one-lung ventilation.[4] A minithoracotomy is performed using direct thoracoscopic visualization to allow exteriorization and resection of the affected lung. Thoracoscopy is associated with less postoperative pain, fewer wound complications, decreased recovery time, shorter hospitalization periods, and more rapid return to function. See also Chapter 14.

Lung Biopsy

Fine-Needle Aspiration

This technique is best suited for nodular lesions that are close to the thoracic wall or diffuse pulmonary infiltrates. Fine-needle aspiration may be performed blindly or be guided by ultrasound or fluoroscopy. Using ultrasound or fluoroscopy to direct the aspiration substantially increases the chances of accurate needle placement in focal lesions; it can be easy to miss such lesions when determining needle placement from radiographs. Sometimes air in the lungs makes pulmonary lesions difficult to detect on ultrasound. In the case of nodules deep in the pulmonary parenchyma, one may anesthetize the patient and place it under a fluoroscopic unit so that respirations can be controlled and the needle accurately placed. Radiographs may help determine the optimal site for needle placement if ultrasound or fluoroscopy is not available. In addition, CT-guided aspirates can be performed if this modality is available. In the case of diffuse lesions, it is usually best to aspirate the larger caudal lobes to stay away from the heart and major vessels. There is always the risk of pneumothorax after such a procedure. Despite the risk being small, patients have died from complications of aspirates, and clinicians must be prepared to place a thoracostomy tube and/or do a thoracotomy to stop air leakage. *After identifying the appropriate spot to puncture the thoracic wall, clip and prep the area.* If the patient will remain still, no sedation or anesthetic is required; however, anesthesia is generally recommended to ensure that the animal does not move during the procedure. For superficial lesions, typically use a 25-gauge hypodermic needle; for deep lesions, a 25-gauge spinal needle with a stylet is preferred. *Once the needle is believed to be in the correct spot, apply several bursts of 5 to 8 mL of negative pressure, and then withdraw the needle without maintaining the negative pressure. If blood is seen entering the hub, stop the procedure and withdraw the needle.* Blood is undesirable because it dilutes the sample and can make cytologic analysis more difficult. *Perform the procedure quickly so that respiratory movement does not cause pulmonary laceration. After performing the procedure, watch the patient carefully for the next 30 to 60 minutes to ensure that pneumothorax does not cause clinical compromise.*

Keyhole Biopsy

Identify the appropriate intercostal space, and make a 3- to 7-cm keyhole intercostal thoracotomy approach (see the previous discussion) to expose the lung. Place a small Finochietto rib retractor into the space to gain exposure to the thoracic cavity. Exteriorize the lung lobe and obtain a lung biopsy using an encircling ligature or thoracoabdominal (TA) stapler (see p. 894). Inspect the stump that remains after biopsy for evidence of hemorrhage and evaluate it for air leakage by filling the thoracic cavity with warm sterile saline solution and examining for air bubbles. Evacuate the fluid using suction. Place a thoracostomy tube if necessary, or close the thorax routinely (see previous discussion) and aspirate air from the thoracic cavity with a needle or catheter (see pp. 920–924).

Thoracoscopic Biopsy

A paraxiphoid or substernal observation port is usually the best choice for this procedure, although a lateral port may work as

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well. Examine the lung lobes thoroughly for abnormalities. These biopsies are usually done on the tips or margins of lung lobes. After identifying the site to be biopsied, there are two basic techniques (see below).

Intrathoracic Biopsy

Position a ligation loop of a commercially available ligature (e.g., 18-inch [46 cm], Endoloop ligature; 21-inch [53 cm] Surgitie Ligating Loop) in the intrapleural space and pass a forceps through the ligation loop. Alternatively, use a modified Roeder knot. Grasp the margin of the affected portion of the lung, and pull the lung tissue into the ligation loop. Tighten the ligature around the base of the biopsy specimen or portion of lung to be removed. Once the ligature is secure, transect the pulmonary tissue (leaving the ligature on the lung). For larger biopsies, use a thoracoscopic stapler (e.g., EndoGIA 45-2.5). Conflicting evidence exists about the safety of vessel-sealing devices (e.g., Ligasure) for lung biopsy. A small cadaveric study found inconsistent sealing of lung tissue, as well as leakage at lower median airway pressure when compared to other methods.[5] However, an experimental study in healthy dogs did not have any complications with air leakage from biopsy sites.[6] Further investigation is required to evaluate vessel-sealing devices for biopsy of diseased lung.

Directed Keyhole Biopsy

Determine the optimal site for making a 1-inch (2.5 cm) incision into the chest, typically two to three intercostal spaces cranial to where the tip of the lung extends during inspiration. After making the incision, insert a Babcock forceps, grasp the tip of the lung lobe from which a biopsy will be taken, and pull it out of the thorax. Place a ligature around the tip and resect it, leaving the ligature on the lung. Check the site for leakage, and then place the lung back in the chest.

Surgical Biopsy

Perform an intercostal thoracotomy and identify the affected lung lobe. Palpate each lung lobe for additional nodules and take a biopsy of the hilar lymph node for staging purposes. Perform a partial lobectomy (see the later discussion) if the tumor is located at the peripheral margin of the lobe; otherwise, perform a complete lobectomy. Submit excised tissue for cytologic and histologic examination. If the lesion is cavitary, or if there is evidence of preexisting pyothorax, submit cultures of the mass. Place a thoracostomy tube before closing the thorax if there is evidence of infection or if pneumothorax or hemorrhage seems likely postoperatively. Remove residual air from the pleural space after closure.

Partial Lobectomy

Partial lobectomy may be performed to remove a focal lesion involving the peripheral one-half to two-thirds of the lung lobe or for biopsy. Partial lobectomy may be performed through a lateral fourth or fifth space intercostal thoracotomy or median sternotomy.

Identify the lung tissue to be removed, and place a pair of crushing forceps across the lobe proximal to the lesion (Fig. 29.9A). Place a continuous, overlapping pattern of absorbable suture (2-0 to 4-0) 4 to 6 mm proximal to the forceps (see Fig. 29.9B). If necessary, place a second row of sutures in a similar manner. Excise the lung between the suture line(s) and forceps, leaving a 2- to 3-mm margin of tissue distal to the sutures (see Fig. 29.9C). Oversew the lung in a simple continuous pattern with absorbable suture (3-0 to 5-0; see Fig. 29.9D). Replace the lung in the thoracic cavity and fill the thoracic cavity with warmed sterile saline solution. Inflate the lungs and check the bronchus for air leaks. Remove the fluid before closing the thorax.

Partial lobectomy may also be performed with stapling devices (e.g., TA stapler; Fig. 29.10). The stapling equipment comes in various sizes, which produce staple lines 30-, 55-, or 90-mm long. Select the staple size based on the width of the lung so that the staple line extends across the entire width of the lung to be removed but not beyond the edges. If air leakage or hemorrhage are noted, place a simple continuous pattern of absorbable suture along the lung margin. The stapling devices compress tissue to a thickness of 1.0 mm (2.5-mm staples; 30-mm length only), 1.5 mm (3.5-mm staples), or 2 mm (4.8-mm staples). Avoid stapling excessively thick or fibrotic lung because this may result in large air leakage or hemorrhage. Check the lung for leaks and close as described previously.

Complete Lobectomy

Complete lobectomy is best performed through a lateral thoracotomy. If the lung contains a large amount of purulent material, prevent excessive fluid from draining into the proximal bronchi and trachea by clamping the bronchus near the hilus before manipulating the lobe. Similarly, torsed lung lobes should be removed without untwisting the pedicle (which would release necrotic material trapped in the lung) (see p. 906). Dogs can survive acute loss of up to 50% of their lung volume; however, transient respiratory acidosis and exercise intolerance may occur. Major respiratory complications from pneumonectomy were recently documented in dogs and cats and consisted of persistent pleural effusion, oxygen dependence, persistent tachypnea, and coughing.[7] A separate study documented estimated median survival time in dogs following pneumonectomy as 1868 days.[8] Pneumonectomy causes compensatory changes in the contralateral lung and myocardium, even in normal dogs. Although residual lung volume, vital capacity, and maximal breathing capacity substantially decrease initially following pneumonectomy, residual lung volume increases significantly after 3 months.

Identify the affected lobe or lobes and isolate them from the remaining lobes with moistened sponges (laparotomy or 4 × 4s, depending on the animal's size). Identify the vasculature and bronchus to the lobe (Fig. 29.11A). Using blunt dissection, isolate the pulmonary artery supplying the affected lobe and pass a ligature of nonabsorbable or absorbable suture (2-0 or 3-0) around the proximal end of the vessel. Do not compromise the lumen of the parent vessel from which this vessel arises. Place a second ligature in a similar fashion distal to the site where the vessel is to be transected. A transfixing suture may be placed between these sutures proximal to the transection site to prevent the first suture from being inadvertently dislodged. *Transect the artery between the distal two ligatures. Ligate the pulmonary vein in a similar fashion. Identify the main bronchus supplying the lobe and clamp it with a pair of Satinsky or crushing forceps proximal and distal to the selected transection site (Fig. 29.11B). Sever the bronchus between the clamps and remove the lung. Suture the bronchus proximal to the remaining clamp in a continuous horizontal mattress pattern (Fig. 29.11C) or, in cats and small dogs, place a transfixing ligature around the bronchus. Before removing the clamp, secure a suture in the bronchus distal to the clamp. After removing the clamp, oversew the end of the bronchus in a simple continuous suture pattern (Fig. 29.11D). Fill the thoracic cavity with warmed sterile saline solution. Inflate the lungs and check the bronchus for air leaks. Before closure, check lungs that have been "packed off" to make sure they reinflate and are not*

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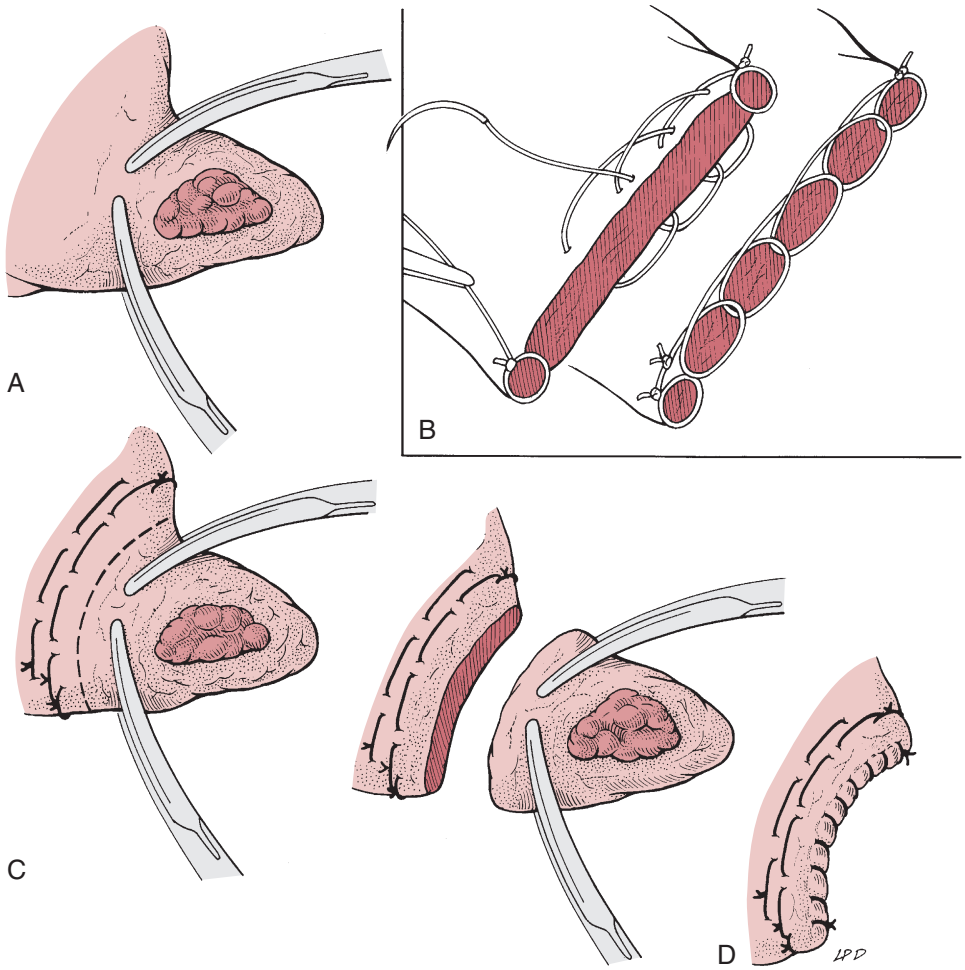


FIG. 29.9 Partial lobectomy may be performed through an intercostal thoracotomy or a median sternotomy. (A) Identify the lung tissue to be removed and place a pair of crushing forceps proximal to the lesion. (B) Place a continuous, overlapping suture pattern proximal to the forceps. (C) Excise the lung between the suture lines and clamps. (D) Oversew the lung in a simple continuous pattern with absorbable suture.

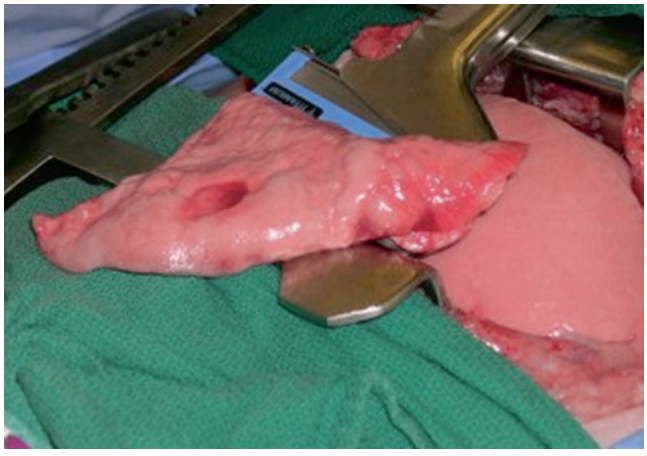


FIG. 29.10 Intraoperative image of a partial lobectomy using a thoracoabdominal (TA) stapler.

twisted. Remove the fluid, and close the thorax as described previously.

A Miller's knot (see Fig. 26.4) or transfixation suture can be used successfully to ligate the vessels and bronchus in many animals. Stapling devices (e.g., 30-mm TA stapler, white [2.5-mm staples] cartridge) may also be used for complete lobectomy, but make sure the bronchus and vessels are adequately ligated by the staples.

HEALING OF THE LUNGS AND STERNUM

After multiple lobectomies or partial lobectomy of several lobes, expansion of the remaining lung may occur in an attempt to restore normal lung volume; therefore exercise intolerance may decline in some animals with time after pneumonectomy. The healing of median sternotomies has been a matter of concern; however, these incisions heal readily and without complication

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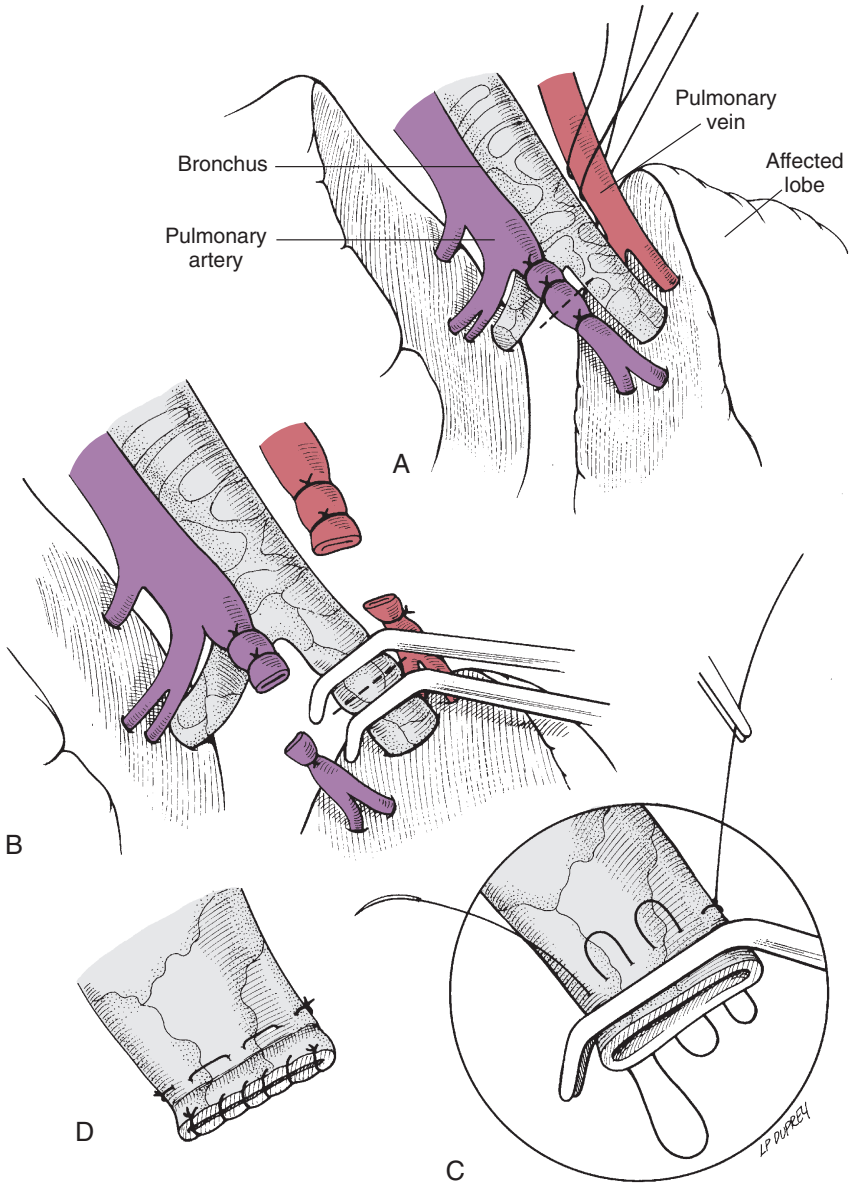


FIG. 29.11 Complete lobectomy. (A) Ligate and transect the vasculature to the affected lobe. (B) Clamp the main bronchus with a pair of Satinsky or crushing forceps; sever the bronchus between the clamps and remove the lung. (C) Suture the bronchus in a continuous horizontal mattress pattern. (D) Oversew the end in a simple continuous suture pattern.

even in animals with pyothorax if several sternebrae are left intact and if the closure is performed properly.

SURGICAL MATERIALS AND SPECIAL INSTRUMENTS

Absorbable or nonabsorbable suture material can be used for complete lobectomy; however, braided, multifilament, nonabsorbable suture (e.g., silk) should be avoided if infection is present. Finochietto rib retractors, Satinsky clamps (for clamping the bronchus), and right-angled forceps (such as Mixter forceps [also known as gallbladder, gall duct forceps, or thoracic forceps]) are useful when performing thoracic surgery (Fig. 29.12). A sternal saw or bone saw is recommended for median sternotomy, particularly in medium or large dogs. Vacuum suction devices facilitate removal of the fluid placed

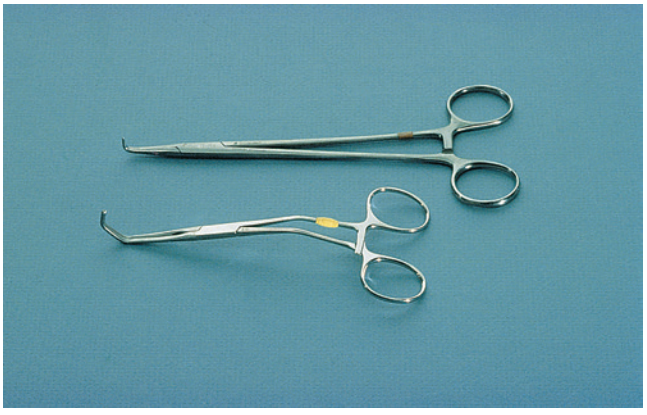


FIG. 29.12 Right-angled forceps *(top)* and Satinsky clamp *(bottom)*.

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in the thorax to identify air leaks. TA staplers are also useful for lobectomies.

POSTOPERATIVE CARE AND ASSESSMENT

Respiration should be monitored closely once the animal begins ventilating on its own. If respiratory excursions are inadequate, the thorax should be evaluated to verify that residual air was removed after thoracic closure. If there is any doubt, thoracic radiographs should be examined for pneumothorax (see p. 939). Blood gas analysis can help evaluate the adequacy of ventilation; hypoxic animals should receive oxygen by nasal insufflation or oxygen cage (see Chapter 4). Animals with severe or progressive hypoxemia should be evaluated for pulmonary edema. Inadequate ventilation may be due to pain in some animals. Median sternotomy may cause decreased ventilation compared with that seen with intercostal thoracotomy. Multimodal analgesia is needed in all patients undergoing thoracotomy procedures (see p. 886). Hypothermia is common after thoracic surgery; warm water bottles and circulating water or warm air blankets should be used to rewarm these patients.

NOTE If the animal hypoventilates postoperatively, thoracic radiographs are indicated to rule out pneumothorax, hemothorax, and pulmonary edema.

COMPLICATIONS

Postoperative complications are reported to occur in 39% of thoracic surgery cases, mostly consisting of wound complications and problems with the thoracic drain. The incidence of pyothorax following thoracic surgery was found to be 6.5% in a study of 232 dogs with a mortality rate of 67%.[9] Subcutaneous fluid accumulation at the ventral aspect of the thoracotomy incision occasionally occurs but can be avoided by carefully closing the distal musculature (i.e., serratus ventralis and pectoralis muscles). The major complication of partial or complete lobectomy is air leakage or hemorrhage (or both). Minor air leaks usually seal, but massive air leaks or severe hemorrhage require reoperation. With median sternotomy, adequate closure and leaving several sternebrae intact prevents delayed healing or nonunion of the sternebrae; however, wound complications are still common. Postoperatively, lameness associated with pain and severing of the latissimus dorsi muscle can occur but usually resolves within 1 to 2 days.

NOTE Monitor these animals closely in the early postoperative period for pneumothorax, hemothorax, or both.

SPECIAL AGE CONSIDERATIONS

Uptake of inhalation agents in pediatric patients (i.e., <12 weeks of age) may be more rapid than in adults, and the level of anesthesia fluctuates more readily in these patients; therefore extra care should be used when anesthetizing them. Young animals are particularly prone to hypothermia when the chest cavity is opened. Temperature regulation, blood glucose requirements, and fluid and electrolyte replacement should be aggressively managed in these patients. Geriatric patients with compromised pulmonary function and/or decreased cardiovascular capacity may also have an abnormal uptake of inhalant anesthetics.

SPECIFIC DISEASES

THORACIC WALL TRAUMA

DEFINITION

Flail chest occurs when several ribs on both sides of the point of impact are fractured, such that the fractured segment moves paradoxically with respiration.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Thoracic wall injury may be due to blunt trauma (e.g., motor vehicle accidents or being kicked by a horse) or penetrating trauma. The most common causes of penetrating injuries of the thorax in dogs are bite wounds and gunshot injuries. Both blunt and penetrating trauma may cause extensive soft tissue damage of the thoracic wall (Fig. 29.13). Although soft tissue damage is rarely the cause of major morbidity or mortality, it may be the only external evidence of severe thoracic trauma in some animals. Pain associated with muscular tears may cause altered respiration because the animal is unwilling to breathe deeply. Unless associated with pulmonary parenchymal damage, hypoxia resulting from ventilatory alterations seldom occurs with chest wall trauma.

Subcutaneous emphysema may occur with both blunt and penetrating trauma but is usually insignificant. This occurs when air is forced into subcutaneous tissue and dissects along muscular

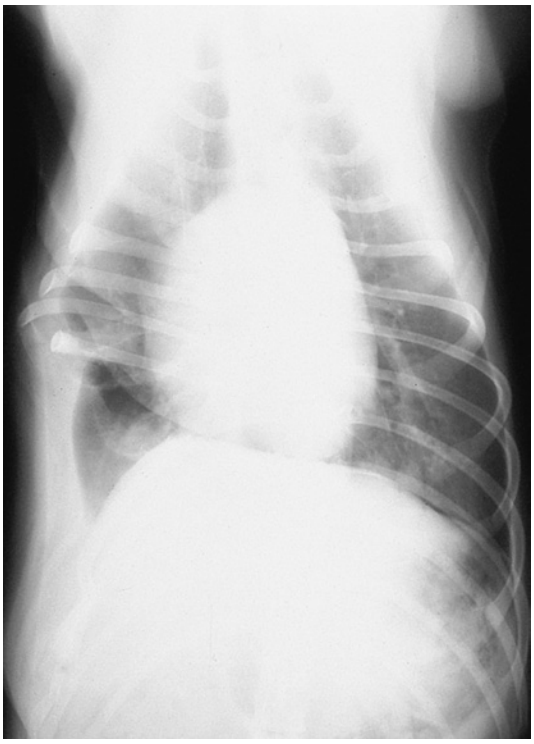


FIG. 29.13 Thoracic radiograph of a dog that was kicked by a horse. Note the large defect between the ribs on the right side of the thorax and the extrapleural sign created by the chest wall trauma.

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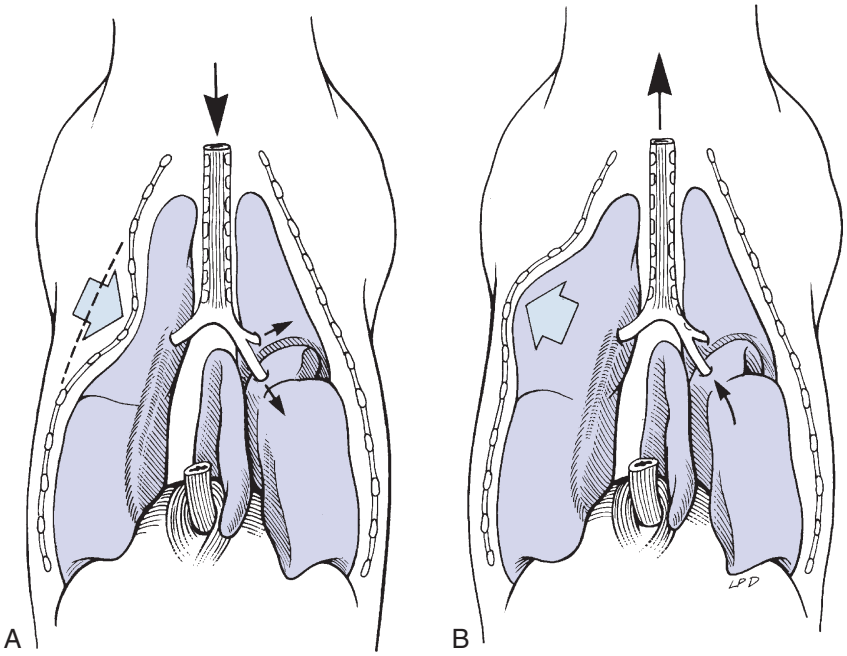


FIG. 29.14 With flail chest, paradoxical movement of the chest wall occurs during respiration because of intrapleural pressure changes; the fractured segment moves (A) inward during inspiration and (B) outward during expiration.

and fascial planes. The air may reach the subcutaneous tissue through a disruption of the pleura and intercostal muscles, by direct communication with an external wound, or as an extension of mediastinal emphysema. Treatment of subcutaneous air should be directed at its cause. Similarly, isolated rib fractures are seldom associated with major morbidity. Occasionally, rib fractures produce sharp fragments that may injure a major vessel or lacerate the lung. Rib fractures may interfere with ventilation if the animal splints the thorax in an attempt to reduce pain by reducing motion of the fragments.

Flail chest occurs when several ribs on both sides of the point of impact are fractured such that the intervening rib segments lose their continuity with the remainder of the thorax (Fig. 29.14). Paradoxical movement of the thoracic wall occurs during respiration as a result of intrapleural pressure changes; the fractured segment moves inward during inspiration and outward during expiration. Respiratory abnormalities in patients with flail chest may be severe and may include decreased vital capacity, reduced functional residual capacity, hypoxemia, decreased compliance, increased airway resistance, and increased work to breathe. These abnormal respiratory parameters were once thought to be caused primarily by the movement of the flail segment; however, it is now believed that the underlying lung damage and hypoventilation from thoracic pain are more important factors in the development of respiratory insufficiency.

DIAGNOSIS

Clinical Presentation

Signalment

Thoracic wall trauma may occur in dogs or cats of any age but is most common in young animals prone to trauma. Thoracic bite wounds are more often found in small-breed dogs.

History

A history of trauma may or may not be obtained. The animal may be presented for evaluation of respiratory distress, reluctance to move because of pain, depression, lethargy, and/or anorexia.

Physical Examination Findings

There may be little or no external evidence of thoracic trauma. Examine the animal closely for small penetrating wounds and areas of subcutaneous emphysema. Flail chest is diagnosed by observing paradoxical movement of a chest wall segment. Animals in severe respiratory distress may be cyanotic.

Animals with thoracic trauma should be examined for delayed-onset cardiac arrhythmias. Cardiac arrhythmias, particularly premature ventricular contractions and ventricular tachycardia, may occur after either blunt or penetrating thoracic trauma. These arrhythmias may not begin until 12 to 72 hours after the trauma and may be associated with myocardial contusion, myocardial ischemia that occurs secondary to shock, or neurogenic injuries that result in sympathetic overstimulation. Cardiac contusions are frequently overlooked in injured patients because (1) attention is directed toward visually obvious injuries, (2) there is no external evidence of thoracic trauma, or (3) there is no evidence of thoracic trauma at the initial examination. Cardiac function, therefore, should be evaluated frequently in most trauma patients.

Diagnostic Imaging

Thoracic radiographs are recommended in all animals suffering blunt or penetrating trauma. A 2015 study sustaining thoracic bite trauma found 22% of dogs and cats with normal respiratory patterns had lesions apparent on thoracic radiographs.[10] Thoracic radiographs should be carefully evaluated for pulmonary

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contusions, pneumothorax (see p. 936), pleural effusion (see p. 938), and diaphragmatic herniation (see p. 927). Rib fractures are easily missed on thoracic radiographs if careful attention is not paid to the rib contour, particularly if the fractured segment is minimally displaced. Orthogonal radiographic views should be evaluated. Evidence of other bony trauma should be sought by carefully examining the vertebrae, scapulae, and proximal forelimbs. Full-body CT can be considered instead of multiple radiographs if multiple injuries are suspected.

NOTE Evaluate thoracic radiographs of dyspneic animals carefully to differentiate intraparenchymal damage (i.e., contusion) from pneumothorax.

Laboratory Findings

Laboratory findings are nonspecific. Blood gas analysis may show hypoxemia and respiratory acidosis (resulting from hypoventilation) or alkalosis (resulting from hyperventilation).

DIFFERENTIAL DIAGNOSIS

Rib fractures may occur secondary to neoplasia or infectious processes; however, these lesions are generally accompanied by lysis or proliferation of the adjacent bone.

MEDICAL MANAGEMENT

Most animals with thoracic wall trauma can be stabilized without surgery. Antibiotic therapy is indicated in patients with notable pulmonary contusion or hemorrhage. Concurrent pneumothorax should be identified and treated (see p. 933). If the animal is dyspneic, supplemental oxygen should be provided. With flail chest, the rib segment can initially be immobilized by positioning the patient with the affected side down. Mechanical ventilation rather than surgery is the treatment of choice in human beings with pulmonary contusions and flail chest; however, long-term mechanical ventilation may not be possible or practical in many veterinary patients. Stabilization may prevent further damage to intrathoracic structures, improve pulmonary ventilation, and reduce pain associated with movement of fragments; however, its benefit is uncertain (discussed later in the Prognosis section).

SURGICAL TREATMENT

Surgical exploration of thoracic bite wounds is recommended in order to debride devitalized tissue and reduce the degree of contamination. Negative pressure wound therapy was recently described for successful management of a perforating thoracic bite wound in a dog.[11]

Indications for exploratory thoracotomy include penetrating injury, more than three radiographic lesions, or both.[10] Rib fractures seldom require surgical treatment; however, multiple rib fractures may cause a defect in thoracic wall continuity (i.e., concavity) that warrants surgical repair. Open stabilization of rib fractures may be indicated if concurrent intrathoracic trauma requires surgery. Flail chest may be managed by placing an external splint over the thorax to stabilize the fractured segment (see the later discussion).

Preoperative Management

If possible, animals with pulmonary contusions should be in stable condition before surgical repair of rib fractures. Shock treatment (i.e., fluids and antibiotics, with or without corticosteroids) should be initiated if necessary. Oxygen supplementation may be beneficial (see Chapter 4), and antibiotics are indicated if pulmonary contusions or hemorrhage is present. If a flail segment is present, placing the animal with the affected side down may be beneficial.

Anesthesia

A splint may be applied to the flail segment of some animals using an intercostal nerve block (see p. 918) rather than general anesthetic. See the anesthetic recommendations on p. 884 for animals with respiratory disease. If general anesthesia is required, refer to Table 30.3 for the anesthetic recommendation for the acute trauma patient.

Positioning

For rib fractures and flail chest, the lateral thorax encompassing the fractured ribs is clipped and prepared aseptically.

SURGICAL TECHNIQUE

Rib Fractures

Place a small intramuscular pin through the proximal fragment and into the marrow canal. Reduce the fracture, and drive the pin into the distal fragment. Exit the pin through the cortex, and bend the ends slightly to help prevent migration. As an alternative, use cerclage wires or cross pins.

Flail Chest

Secure the affected ribs to a sheet of plastic splinting material (e.g., Orthoplast) that has been molded to conform to the thoracic wall (Fig. 29.15). Using a Steinmann pin, place holes in the splinting material large enough to pass the selected suture (see the later discussion) through. Place sutures circumferentially around the affected ribs. Pass the suture ends through the predrilled holes and tie securely. As an alternative, aluminum rods, tongue depressors, or Lubra plates may be substituted for the plastic splinting material.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

For application of a splint in animals with flail chest, use large monofilament suture (2-0 to No. 2, depending on the animal's size) with an attached large, curved needle. A Steinmann pin and pin chuck are also needed. For rib fracture repair, small intramedullary pins and cerclage wire are necessary.

POSTOPERATIVE CARE AND ASSESSMENT

Animals with thoracic trauma should be monitored closely in the postoperative period for hypoventilation and pneumothorax (see p. 933). Analgesics are warranted in these animals (see p. 886).

PROGNOSIS

The prognosis for animals with thoracic wall trauma generally depends on the amount of concurrent pulmonary or cardiac trauma, and the development of postoperative wound

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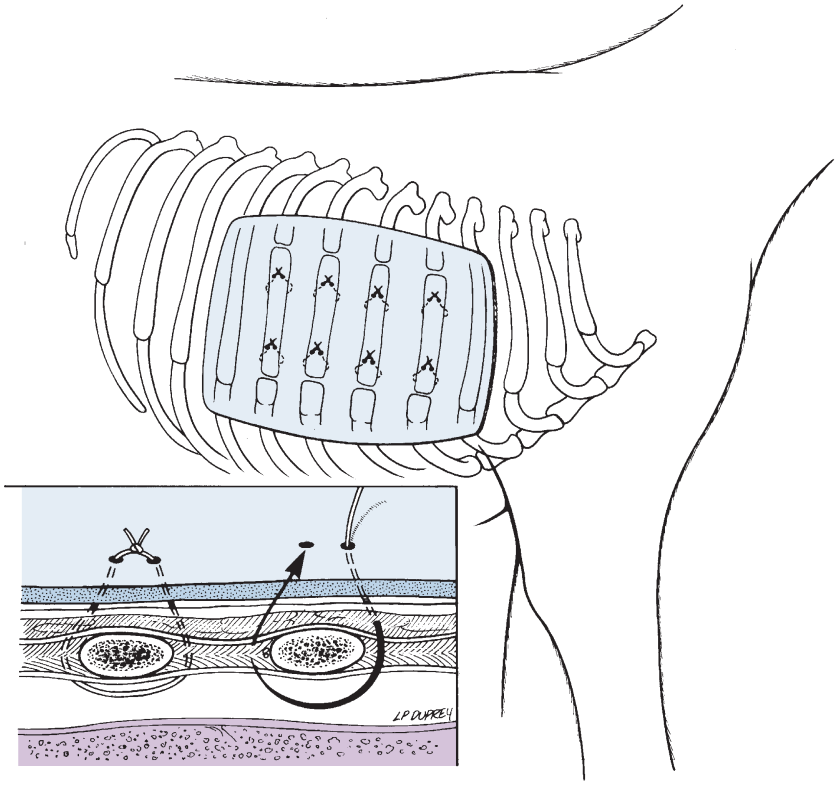


FIG. 29.15 To repair flail chest, secure the affected ribs to a sheet of plastic splinting material. Place sutures circumferentially around the affected ribs and through holes in the splinting material.

complications; a mortality rate of 15% in dogs and cats with thoracic bite wounds has been reported.[10] Most rib fractures heal without surgery, and there is no difference in outcomes whether a flail segment was stabilized or not stabilized. Cats with rib fractures secondary to trauma generally have a good outcome, but the prognosis is worse if flail chest, pleural effusion, or diaphragmatic hernias are present.

PULMONARY NEOPLASIA

DEFINITION

Primary pulmonary neoplasms originate in pulmonary tissue and may arise as a solitary mass or in rare cases may be multicentric.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Primary pulmonary neoplasia is less common than metastatic neoplasia in dogs and cats. The diaphragmatic lobes are most frequently involved, with the right lung lobes more often affected than the left. Specific anatomic localization of tumor origin is not always possible, and more than one tumor type may be present; therefore classification of primary lung tumors is usually based on the predominant histologic pattern. Adenocarcinoma is the most common histologic type found in dogs and cats; squamous cell carcinoma and anaplastic carcinomas are less common. Primary pulmonary tumors of connective tissue origin (e.g., osteosarcoma, fibrosarcoma, and hemangiosarcoma) are rare. Although most pulmonary tumors are malignant, benign tumors (e.g., papillary adenoma, bronchial adenoma, fibroma, myxochondroma, and plasmacytoma) occur. Pulmonary neoplasms are highly aggressive and tend to metastasize early. Most anaplastic carcinomas and squamous cell carcinomas have metastasized at the time of diagnosis, whereas approximately half of adenocarcinomas have done so. Metastasis is often to the lung itself or to regional lymph nodes or both.

Metastatic pulmonary neoplasia is an important differential diagnosis for nodular lung disease. Tumors with a high likelihood of resulting in pulmonary metastasis include mammary carcinoma, thyroid carcinoma, hemangiosarcoma, osteosarcoma, transitional cell carcinoma, squamous cell carcinoma, and oral and digital melanoma.

Feline lung-digit syndrome is an unusual pattern of metastasis in cats with primary bronchial or bronchioalveolar adenocarcinoma. Metastases are found on the distal phalanges of multiple digits and on multiple limbs. Weight-bearing digits are thought to be most commonly affected.

DIAGNOSIS

Clinical Presentation

Signalment

The average age of dogs and cats with primary lung tumors is over 10 years. Anaplastic carcinomas tend to occur at a slightly younger age (8–9 years) than adenocarcinomas. There does not seem to be a gender or breed predilection, although boxers may be overrepresented. Mean age of presentation in cats is 12 years, with no apparent sex or breed predisposition.

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History

Nearly 25% of dogs with pulmonary neoplasia are asymptomatic at the time of diagnosis (i.e., pulmonary neoplasia is an incidental finding when thoracic radiographs are evaluated for an unrelated problem). If clinical signs are present, the owner may report that they have been apparent for weeks to months.

Physical Examination Findings

The most common clinical finding in dogs with primary pulmonary neoplasia is a nonproductive cough; other signs include hemoptysis, fever, lethargy, exercise intolerance, weight loss, dysphagia, and anorexia. Lameness may be associated with metastasis to bone or skeletal muscle or with development of hypertrophic osteopathy or in feline lung-digit syndrome. Hyporexia, weight loss, lethargy, dyspnea, and cough are common clinical signs in cats with primary lung tumors; respiratory signs may be present in as few as one-third of affected cats.

Diagnostic Imaging

Thoracic radiographs should be obtained in animals suspected of having pulmonary neoplasia (Fig. 29.16). The most common finding with primary pulmonary neoplasia in dogs is a solitary nodular density in the periphery of a dorsocaudal lung lobe (Fig. 29.17). Multiple miliary lesions are less common (Fig. 29.18). The radiographic pattern may be classified as solitary nodular, multiple nodular, or disseminated-infiltrative. Multiple discrete lesions within a single lobe or multiple lobes usually represent metastatic neoplasia rather than multicentric primary neoplasia. Feline bronchoalveolar carcinoma may appear as a mixed bronchoalveolar pattern, an ill-defined alveolar mass, or a mass with cavitation. Bronchial disease is typically seen in affected cats (e.g., bronchointerstitial pattern, peribronchial cuffing, or bronchiectasis) and may represent airway metastasis. Because radiographic signs of pulmonary neoplasia in cats are not specific (many inflammatory diseases will cause similar changes), lung fine-needle aspirates may be the most helpful diagnostic tool.

NOTE Because of the lack of respiratory signs in many animals with lung tumors, take thoracic radiographs when presented with a cat or dog with chronic, unexplained clinical signs.

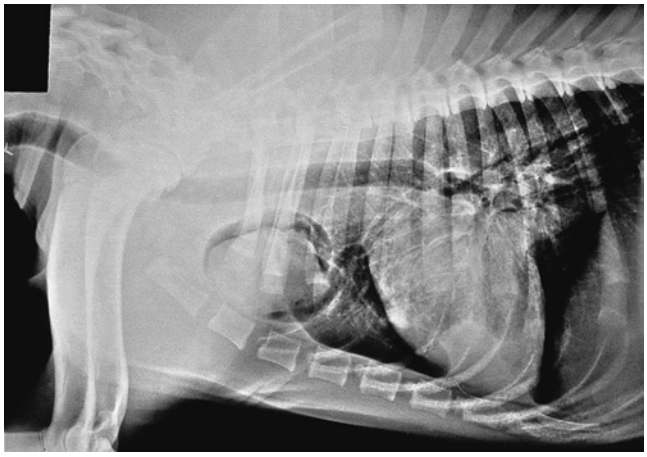


FIG. 29.16 Thoracic radiograph of a 2-year-old German shepherd dog with a bronchogenic cyst in the left cranial lung lobe. Surgical excision was performed through a median sternotomy approach.

Thoracic evaluation should include a three-view radiographic study (opposite lateral views and an orthogonal view). Lung lesions may go undetected in recumbent lateral radiographs when the affected lung is dependent because of the recumbent atelectasis that occurs. Thoracic radiographs are relatively insensitive indicators of pulmonary neoplasia because nodules must be at approximately 0.5 to 1 cm in diameter to be reliably recognized. Radiographs should also be evaluated for sternal or hilar lymphadenopathy and/or pleural effusion. It may be difficult to differentiate metastatic pulmonary neoplasia from pulmonary metastasis of a primary pulmonary tumor. Compared with primary lesions, metastatic tumors are generally smaller and more well circumscribed and are usually located in the peripheral

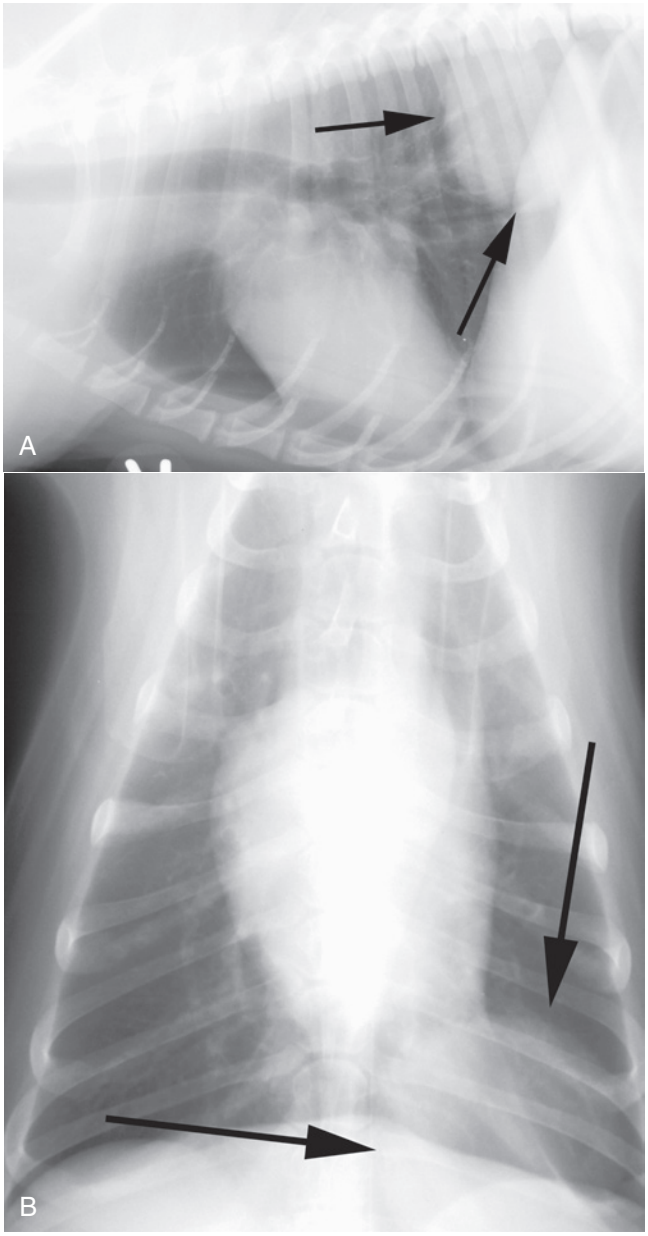


FIG. 29.17 (A) Right lateral and (B) ventrodorsal radiographs of a dog with a large mass in the left caudal lung lobe. Note the well-defined soft tissue mass *(arrows)*. The primary differential is a primary pulmonary neoplasm.

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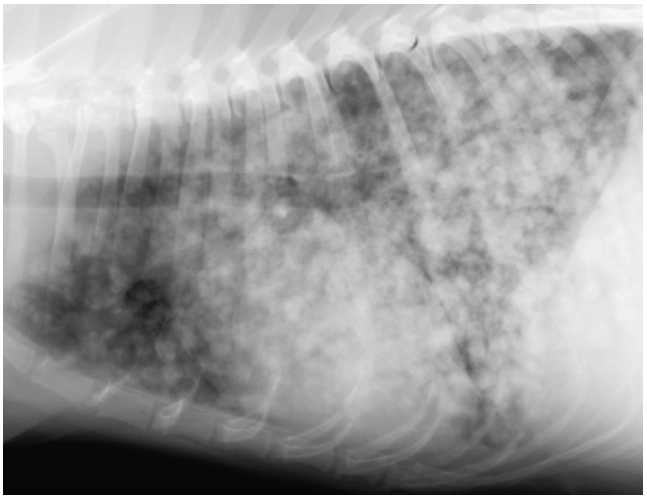


FIG. 29.18 Thoracic radiograph of a 7-year-old Siberian husky with metastatic neoplasia. Notice the miliary nodular pattern in the lung. The other major differential for this pattern is fungal pneumonia.

or middle portions of the lung. Multiple nodules associated with primary lung tumors often consist of one large mass and smaller secondary nodules. When multiple nodules are metastases, there are usually several large masses and a variety of smaller lesions. Contrast-enhanced CT is the most sensitive means for detecting pulmonary lesions. CT was more sensitive than three-view radiography in detecting pulmonary nodules, particularly in large and giant breeds.[12] In cats, additional foci of pulmonary disease compatible with metastatic disease was found in 53% cats receiving thoracic CT, whereas pleural effusion was found in 30%.[13]

NOTE To improve the likelihood of diagnosing primary or metastatic pulmonary neoplasia, take a ventrodorsal and both lateral recumbent views or perform CT. Always assess radiographs of animals with pulmonary neoplasia for multiple masses, lymphadenopathy, and/or pleural effusion.

Ultrasound- and CT-guided aspirates of thoracic masses may be performed (see p. 893).

Diagnoses obtained by fine-needle aspiration (ultrasound-guided and blind) cytopathology accurately reflect the diagnosis obtained on histopathologic examination. CT-guided intrathoracic fine-needle aspiration and core biopsies are also diagnostically accurate. Complications include pneumothorax and pulmonary hemorrhage that uncommonly require treatment. Use of fine-gauge (25- or 27-gauge) needles may reduce complications.

Endoscopic ultrasonography with the tip of the endoscope in the esophagus may be useful in evaluating radiographically detected intrathoracic lesions that do not lend themselves to routine ultrasonographic evaluation because of their location and superimposition of gas-filled structures. Ultrasound-guided tissue biopsies can be performed during the examination.

Laboratory Findings

Laboratory abnormalities are nonspecific but may include nonregenerative anemia, leukocytosis, and hypercalcemia.

DIFFERENTIAL DIAGNOSIS

Pulmonary neoplasia must be differentiated from abscesses or granulomas (e.g., fungal and heartworm). Samples can be collected for cytologic examination by surgical biopsy, percutaneous fine-needle aspiration, transtracheal lavage, thoracoscopy, and/or bronchoscopy. Fine-needle aspiration cytology is a useful, noninvasive diagnostic tool if the needle can be directed into the nodule. Thoracoscopic lung biopsy can often obtain biopsy specimens in a safe and minimally invasive manner, depending on the location of the lesion.

NOTE Distinguishing severe fungal disease from miliary neoplasia may be difficult radiographically.

MEDICAL MANAGEMENT

Surgical removal of primary pulmonary neoplasia is the treatment of choice in small animal patients. Chemotherapy is routinely used for some pulmonary neoplasms in human beings; adjunctive chemotherapy may be of benefit in veterinary patients. Refer to an oncology text for information regarding chemotherapy for pulmonary neoplasia.

SURGICAL TREATMENT

Wide surgical resection is the treatment of choice for solitary nodules or multiple masses involving a single lobe if there is no evidence of distant metastasis or extrapleural involvement. Surgical resection is occasionally indicated for lung metastasis of a distant primary tumor (e.g., limb osteosarcoma). An intercostal thoracotomy is preferred over median sternotomy because it provides adequate exposure for lobectomy and lymph node biopsy (see p. 890). Partial lobectomy should be performed only when the tumor is located at the periphery of the lung lobe; otherwise, total lobectomy should be performed. Thoracoscopy can help determine whether pulmonary metastasis is present before a thoracotomy, particularly if the presence of metastasis is an important factor in determining whether resection of the pulmonary mass should be performed. Successful thoracoscopic or thoracoscopic-assisted lung lobectomy for primary lung tumors has been described.[4,14,15] When compared to open thoracotomy, the only significant difference was longer surgical time with thoracoscopy, whereas completeness of excision, hospitalization time, complication rate, short-term survival, and median survival times were similar.[15]

Preoperative Management

Preoperative management of patients with pulmonary neoplasia and dyspnea is similar to that for other animals with respiratory diseases (see p. 884). If the mass is large, positioning the animal with the affected side down may improve ventilation.

Anesthesia

See the anesthetic management of patients with respiratory abnormalities on p. 884.

Surgical Anatomy

See the anatomic description of the lung lobes on p. 889.

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Positioning

Because most neoplasms are removed through an intercostal thoracotomy, the lateral thorax should be prepared for aseptic surgery.

NOTE Be careful how dogs with large pulmonary masses are positioned during induction and preparation. Dogs may not be able to ventilate if placed in lateral recumbency with the normal (unaffected) lung down because the weight of the mass may prevent lung inflation. The surgical clip may need to be performed with these patients in ventral or dorsal recumbency.

SURGICAL TECHNIQUE

Thoracoscopic Biopsy or Partial Lobectomy

The technique will depend on the size and location of the mass. *Examine the lung lobes thoroughly for abnormalities. For small nodules at the periphery of a lobe, remove them as described previously for lung biopsy (p. 894).*

For larger masses requiring partial lobectomies, use thoracoscopy to determine the optimal position for making a minithoracotomy (keyhole) incision (thoracoscopic-assisted partial lobectomy). The size of the incision will depend on the size of the lesion and the lung lobe to be exteriorized. *Grasp the affected lung lobe with Babcock forceps, and pull it out of the chest until a stapling device (e.g., TA stapler, TA-30 or TA-55) can be placed between the mass and the hilus. Staple and resect the affected portion. Check the incision site for leakage, and then reposition the lung in the chest.*

Thoracoscopic Lobectomy

Lungs with lesions located near the periphery may be removed thoracoscopically. Large lesions or masses located near the pulmonary hilus are challenging to remove by this technique because of the impaired visualization and difficult manipulation of the affected lung. One-lung ventilation is recommended to optimize the operative working space.

Position the animal in oblique lateral recumbency to improve visualization of the hilus. Place an endoscope portal and two to three operative portals using triangulation (see p. 893). Manipulate the affected lung with atraumatic forceps, and perform careful sharp dissection to expose the hilus. Insert an endoscopic stapler (EndoGIA, 45–65 mm in length; 3.5 mm cartridge) perpendicularly across the hilus. After firing the stapler, place the resected lung in a retrieval bag to avoid seeding thoracic wall tissues upon removal. Extend one of the operative portals to allow retrieval of the lung. Biopsy or remove any enlarged hilar lymph nodes. Observe the staple line for any bleeding or leakage of air. Place a thoracostomy tube and close all portal sites routinely.

Surgical Partial or Complete Lobectomy

See p. 894.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Avoid nonabsorbable braided suture (e.g., silk) if there is evidence of infection (see also p. 896).

POSTOPERATIVE CARE AND ASSESSMENT

The animal should be monitored postoperatively for dyspnea. Oxygen should be available. Postoperative analgesics should be given (see p. 886). Evaluation of ventilation by analysis of blood gas parameters or pulse oximetry is useful. Sudden respiratory distress may be due to hemorrhage or pneumothorax.

COMPLICATIONS

The major complications of lung lobectomy for tumor removal in dogs are hemorrhage and pneumothorax (see p. 933). The rate of conversion from thoracoscopy to thoracotomy ranges from 9% to 23%.

PROGNOSIS

The prognosis is favorable for dogs with well-differentiated, nonmetastasized, primary lung tumors that do not have associated clinical signs. Dogs with tumors in the lung periphery or near the base of a lung have better survival times than those in which the tumor involves an entire lobe. The most important prognostic factor related to survival in dogs after surgery is whether lymph node metastasis has occurred. Two-year survival rate following lung lobectomy in dogs is approximately 50%.[14]

The prognosis for most cats with primary lung tumors is often poor because of the advanced nature of the disease at the time of diagnosis and the tumors' aggressive metastatic behavior. In one study, overall median survival time in cats is 156 days; lymph node involvement is also prognostic (65 days with lymph node enlargement versus 498 days without lymph node enlargement). Cats with low or intermediate-grade tumors had a median survival of 730 days compared to 105 days for cats with high-grade tumors.[16] In a separate study, median survival time was only 11 days with the following factors correlating with reduced survival time: clinical signs at the time of diagnosis, pleural effusion, stage M1, and poorly differentiated tumor type.[17] Cats with no clinical signs on presentation had a median survival time of 578 days.

PULMONARY ABCESSES

DEFINITION

A **pulmonary abscess** is a localized collection of pus that often causes cavitation in the lung.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Pulmonary abscesses are rare but may occur as a complication of foreign bodies, neoplasia, bacterial pneumonia, aspiration pneumonia, fungal infections, or parasites. Abscesses secondary to neoplasia may be sterile or infected. The most common organisms cultured from abscesses associated with necrotizing pneumonia in dogs are *Escherichia coli, Pseudomonas* spp., and *Klebsiella* spp. Rupture of pulmonary abscesses may result in pyothorax (see p. 947), pneumothorax (see p. 933), or both. In some parts of the country, pulmonary abscesses often occur secondary to inhalation or thoracic penetration of plant material (e.g., foxtails) that migrate through the lung.

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DIAGNOSIS

Clinical Presentation

Signalment

Pulmonary abscesses may occur in dogs or cats of any age, breed, or gender.

History

The animal may be brought in for treatment of a persistent low-grade fever, varying degrees of respiratory distress, weight loss, lethargy, and/or anemia. The duration of illness may vary from hours to days or even weeks. Rupture of the abscess often causes pneumothorax and dyspnea.

Physical Examination Findings

Physical examination findings vary depending on whether pneumothorax or pleural effusion (or both) is present (see pp. 935 and 937). Most animals are febrile, and inspiratory crackles may be heard over the mass.

Diagnostic Imaging

Pulmonary abscesses generally appear as nodular or cavitary radiopaque lesions on thoracic films. The walls of the abscess are usually poorly defined. If pleural effusion is present, thoracentesis may be necessary before a definitive diagnosis can be made. Ultrasound evaluation of thoracic masses may help differentiate noncavitary from cavitary lesions if an appropriate acoustic window is available. CT imaging may also be beneficial to differentiate pulmonary abscess from neoplasia (Fig. 29.19).

Laboratory Findings

The leukogram may be normal or have leukocytosis with or without a degenerative left shift. If the infection is chronic, a nonregenerative anemia may be present.

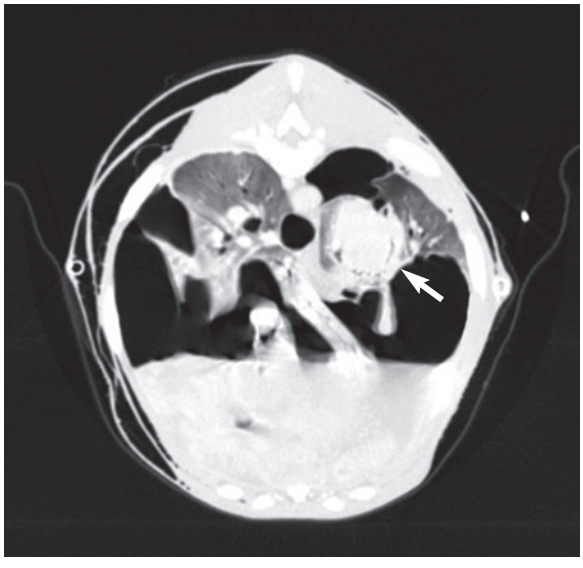


FIG. 29.19 Isolated transverse thoracic CT image from a 5-year-old German shorthaired pointer with a pulmonary abscess of the left caudal lung lobe *(arrow),* severe pneumothorax, and pleural effusion.

DIFFERENTIAL DIAGNOSIS

Abscesses should be differentiated from other nodular or cavitary pulmonary lesions (e.g., granulomas, *Paragonimus* infection, and neoplasia) by cytologic and/or histologic examination of samples obtained by fine-needle aspiration or surgery. Preoperative aspiration of the mass may help distinguish between these lesions and provide samples for culture; however, care should be taken to prevent causing pyothorax. Ultrasound is often useful in locating the appropriate site for aspiration. Some nonneoplastic lesions can be managed without surgery (e.g., *Paragonimus* infection); however, a definitive diagnosis may require surgical biopsy.

MEDICAL MANAGEMENT

Initial therapy is aimed at stabilizing dyspneic animals. Thoracentesis should be performed if pleural fluid or air is present. A broad-spectrum antibiotic with a good anaerobic spectrum should be chosen (see p. 949) and later modified, based on culture and susceptibility testing. Antibiotic therapy should be continued for 3 to 6 weeks. If pyothorax is present, thoracostomy tubes should be placed and the thorax lavaged (see Pyothorax on p. 947). Some animals respond to medical management and the abscess resolves. Surgical intervention is warranted if no improvement is seen in the clinical signs or lung expansion or if pleural fluid is loculated and does not resolve after several days.

SURGICAL TREATMENT

A solitary pulmonary abscess that does not resolve with medical therapy is best managed by partial or complete lobectomy of the diseased lung performed through an intercostal thoracotomy (see p. 890). A median sternotomy approach (see p. 891) is preferred if multiple opacities are present involving both sides of the thorax or if pleural effusion is present.

Preoperative Management

If the animal is dyspneic, thoracentesis should be performed before surgery. Antibiotic therapy should be initiated after the mass or pleural space (or both) have been cultured, if not before.

Anesthesia

Refer to comments and anesthetic protocols for animals with respiratory dysfunction on p. 884. See also specific comments regarding the anesthetic management of animals with pneumothorax on p. 937.

Surgical Anatomy

The surgical anatomy of the thorax is described on p. 888.

Positioning

See pp. 890 and 892 for positioning of animals for intercostal or median thoracotomy.

SURGICAL TECHNIQUE

Identify the diseased lobe before handling it, and clamp the pedicle to prevent drainage of purulent material into dependent lung lobes. Perform a complete or partial lobectomy, depending on the size and location of the abscess (see p. 894). Submit the lung for bacterial or fungal cultures (or both) and for histologic examination. Explore

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the remainder of the chest cavity for the presence of foreign matter. Palpate all the lung lobes that can be reached to identify other pulmonary lesions. Free remaining lung lobes of adhesions so that all lobes are movable, and remove loculated areas of exudate. Remove sheets of fibrin that cover the lung lobes. Place a chest tube before thoracic closure.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Braided, multifilament, nonabsorbable suture (e.g., silk) should be avoided if infection is present.

POSTOPERATIVE CARE AND ASSESSMENT

Appropriate antibiotics should be continued for at least 3 to 6 weeks if infection is present. Pyothorax should be treated with thoracic lavage (see p. 949). Postoperative analgesics should be provided (see p. 886).

PROGNOSIS

The prognosis for animals with pulmonary abscesses depends on the underlying cause. With appropriate management, the prognosis for animals with abscesses associated with nonneoplastic disease is good.

LUNG LOBE TORSION

DEFINITION

Lung lobe torsion (LLT) is a rotation of the lung lobe along its long axis, with twisting of the bronchus and pulmonary vessels at the hilus.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Any mechanism that increases mobility of a lung lobe seems to favor torsion (Box 29.3). Partial collapse of the lung with pulmonary disease or trauma frees it from its normal spatial relationships with the thoracic wall, mediastinum, and adjacent lung lobes. This may enhance mobility. Pleural effusion or pneumothorax, along with subsequent atelectasis of lung lobes, can allow increased movement of a lobe, predisposing to torsion. Although LLT has been reported to cause chylothorax in dogs, it may be chylothorax that caused LLT. LLT has been reported secondary to previous thoracic surgery in which lung lobes are manipulated and remain partly collapsed after thoracic closure. LLT can occur in any lobe but has been reported most commonly in the right middle lung lobe in large-breed dogs and the left cranial lobe in small dogs.

BOX 29.3 Possible Causes of Lung Lobe Torsion

Atelectasis associated with the following:
- Pneumonia
- Trauma
- Pneumothorax
- Pleural effusion
- Spontaneous
- Surgical manipulation
- Not replacing the lobe in its proper relationship after thoracic surgery

LLT causes venous congestion of the affected lobe; however, the arteries remain at least partly patent, allowing blood to enter. As fluid and blood enter the alveoli, lung consolidation occurs and the lobe becomes dark colored and firm, similar in shade to the liver. The shape of the affected lobe is often altered, and it may appear displaced from its normal location in the thorax radiographically. Pleural fluid usually accumulates because of continued venous congestion.

DIAGNOSIS

Clinical Presentation

Signalment

Deep-chested, large-breed dogs, particularly Afghan hounds, are more commonly affected; however, LLT often occurs in small and toy-breed dogs. Specifically, young male pugs appear to be predisposed to LLT. LLT in Afghan hounds may be associated with chylothorax (see p. 941). In other large-breed dogs and pugs, LLT frequently occurs spontaneously without previous history of disease or trauma. LLT in other small breeds is often secondary to primary pleural effusion, thoracic surgery, or trauma. LLT is rare in cats. Middle-aged dogs are more commonly affected, but LLT may occur in animals of any age. Recently, seven dogs younger than 12 months of age (presenting most commonly for dyspnea and lethargy) were found to have LLT.[19]

History

Dogs with LLT present for coughing and hemoptysis. Some animals may be anorectic and depressed. With significant pleural effusion, dogs may present in severe respiratory distress. There may be a previous history of pneumothorax, pneumonia, and/or trauma.

Physical Examination Findings

Pleural effusion is consistently present in animals with LLT; therefore findings often include muffled heart and lung sounds. Other findings may include depression, anorexia, coughing, fever, dyspnea, hemoptysis, hematemesis, and/or vomiting.

Diagnostic Imaging

Thoracic radiographic changes vary depending on the volume of pleural fluid, the presence or absence of preexisting disease, and the duration of the torsion. The most consistent finding is pleural effusion accompanied by an opacified lung lobe. Initially, air bronchograms are present in the torsed lobe and may be seen running in an abnormal direction. Air bronchograms eventually disappear as fluid and blood fill the bronchial lumen. The presence of a noninflated, radiopaque lung lobe that persists after removal of pleural fluid should increase suspicion for LLT (Fig. 29.20). Small, dispersed air bubbles are often seen within the affected lobe (vesicular lung pattern). The lobar bronchi may be difficult to see but if located, often appear irregular, focally narrowed or blunted, or displaced. Mediastinal shift, curved and dorsally displaced trachea, and axial rotation of the carina are also commonly seen. Positional radiographs using horizontal beam radiographs (lateral decubitus or upright ventrodorsal) are often helpful. Pleural fluid that occurs secondary to LLT may persist around the affected lobe rather than fall to the dependent side. Failure of the lobe to reinflate in the "up" or nondependent hemithorax is a nonspecific indication of LLT. Thoracic ultrasound typically finds the affected lung to be large and hypoechoic with centrally located reverberating foci consistent with gas. Often

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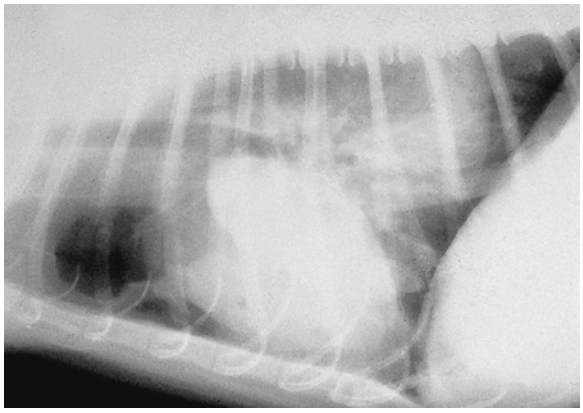


FIG. 29.20 Lateral thoracic radiograph of a dog with a torsion of the right middle lung lobe. Pleural fluid was removed before this radiograph was obtained. Notice the soft tissue mass overlying the cardiac silhouette.

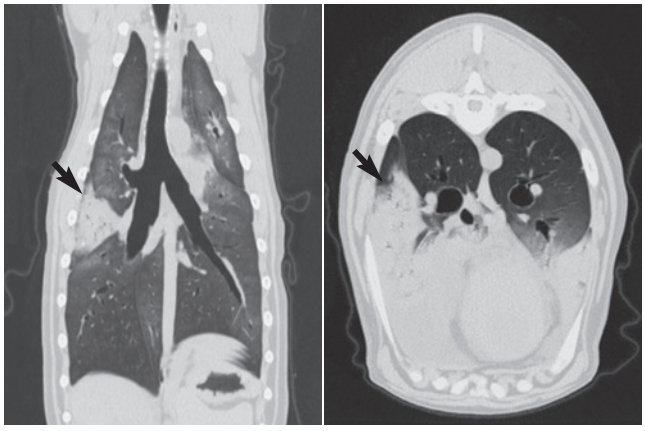


FIG. 29.21 Isolated coronal and transverse thoracic computed tomography images of 3-year-old mixed-breed dog with a lung lobe torsion of the right middle lung lobe *(arrows).*

the findings on thoracic radiographs and ultrasound are nonspecific, and other diagnostics are indicated prior to surgery.

Computed Tomography

CT findings in dogs with LTT include pleural effusion and an abruptly ending bronchus, as well as enlargement, consolidation, and emphysema of the affected lobe (Fig. 29.21). Rotated lung lobes do not enhance with intravenous contrast medium administration, whereas the nearby collapsed or aerated lungs do. Reconstruction of CT images to generate an internal view of the trachea and bronchi (virtual bronchoscopy) can help make a more definitive diagnosis of LTT.

Bronchoscopy

Bronchoscopy typically reveals a bronchus that is occluded and appears to be twisted. Sometimes the tissue at the site appears edematous. There may or may not be blood in the bronchi (Fig. 29.22).

NOTE Lung lobe torsion usually causes massive pleural effusion. A torsed lung may not be seen on radiographs until the pleural fluid is removed.

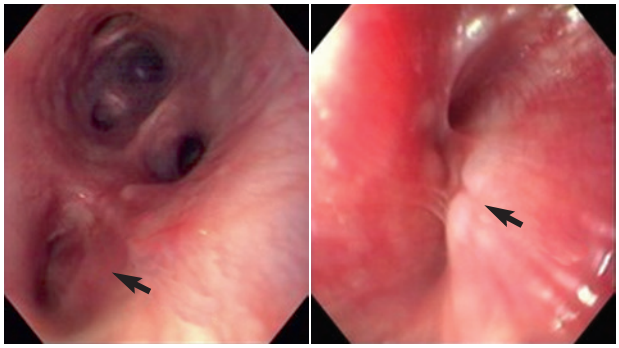


FIG. 29.22 Bronchoscopy of a 7-year-old mixed-breed dog with a torsed lung lobe. *Arrows* point toward the twisted and occluded bronchus.

Laboratory Findings

Laboratory findings with LLT are variable. Fluid analysis may reveal a sterile, inflammatory effusion or chyle, or the fluid may be bloody. Pleural effusion of any etiology, however, can initiate a secondary LLT, making the results of pleural fluid analysis variable and confusing. The appearance of blood in a previously nonhemorrhagic pleural fluid may indicate LLT. An inflammatory leukogram may be present; however, these changes may reflect the initial disease process rather than the LLT.

DIFFERENTIAL DIAGNOSIS

Pneumonia, pulmonary thromboembolism, contusion, neoplasia, atelectasis, hemothorax, diaphragmatic hernia, and pyothorax can mimic radiographic changes seen with LLT. Demonstration of LLT at surgery provides the definitive diagnosis.

MEDICAL MANAGEMENT

Initial therapy is aimed at stabilizing the animal's condition and alleviating respiratory distress before surgery. Thoracentesis should remove pleural fluid (see p. 920), but persistent or massive pleural effusion may require a chest tube (see p. 922). Oxygen therapy given by oxygen cage or nasal insufflation benefits some animals. Underlying diseases, such as pneumonia, should be identified and treated with appropriate antibiotic therapy. Intravenous fluid therapy is beneficial before and during surgery to maintain hydration.

NOTE Spontaneous resolution of lung lobe torsion is extremely uncommon. This is a surgical condition.

SURGICAL TREATMENT

Spontaneous correction of a torsed lung lobe is uncommon because of swelling of the lobe and rapid formation of adhesions. The treatment of choice for LLT is lobectomy of the affected lobe. Unless LLT is diagnosed quickly (i.e., immediately after a surgical procedure), damage to the pulmonary parenchyma is generally severe enough that attempts to salvage the lobe are not warranted. Recurrence has been reported after surgical correction where lobectomy was not performed.

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Preoperative Management

Prophylactic antibiotics are warranted in animals with LLT. Pleural effusion should be removed before induction of anesthesia in animals that have compromised ventilation.

Anesthesia

See p. 884 for anesthetic management of animals with respiratory abnormalities.

Positioning

The affected lateral thorax should be prepared for an intercostal thoracotomy (see p. 890).

SURGICAL TECHNIQUE

Before attempting to derotate the affected pedicle, clamp it with a noncrushing forceps to prevent release of toxins into the bloodstream or fluids into the dependent lobes. Untwisting the lobe before its removal may facilitate identification of the vascular structures and bronchus for ligation; however, in many cases the lobe cannot be easily returned to its normal position because of extensive adhesions. A transfixion suture or Miller's knot can often be used in such cases to ligate the vessels and bronchus. *Alternatively, use a surgical stapler (TA stapler) to remove the lobe. Check the remaining lobes for position and normal expansion. Culture the pulmonary parenchyma after removal of the lobe. Submit excised tissue for histologic examination to help determine underlying causes (e.g., pneumonia or neoplasia). Place a thoracostomy tube before closing the thoracic cavity.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Avoid braided, multifilament suture because of the risk of infection. Large clamps, such as Satinsky clamps (see p. 896), are useful for clamping the bronchus.

POSTOPERATIVE CARE AND ASSESSMENT

Antibiotics should be continued if there is evidence of infection, and postoperative analgesics should be provided (see p. 886). The thoracostomy tube should be removed when the effusion diminishes to less than 2.2 mL/kg per day of body weight (see p. 924). Oxygen therapy may be warranted in some patients in the postoperative period, particularly if there is underlying lung disease, such as pneumonia. If dyspnea remains after surgery, thoracic radiographs are indicated to rule out recurrence of LLT.

PROGNOSIS

The prognosis is good for most animals with LLT if surgery is performed. Pleural effusion usually resolves within a few days of surgery unless the animal has concurrent chylothorax.

PECTUS EXCAVATUM

DEFINITIONS

Pectus excavatum (PE) is a deformity of the sternum and costocartilage that results in a dorsal to ventral narrowing of the thorax. **Pectus carinatum** is a protrusion of the sternum, which occurs much less frequently than PE. Synonyms for pectus excavatum include *funnel chest, chondrosternal depression, chonechondrosternon, koilosternia,* and *trichterbrust*.

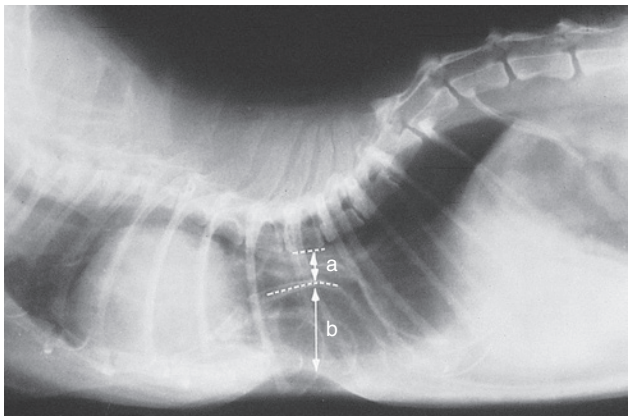


FIG. 29.23 Lateral thoracic radiograph of a cat with severe pectus excavatum (PE). The amount of depression is subjectively assessed based on the minimum distance between the vertebral column and the dorsal aspect of the sternum *(a)* or the depth of the concavity *(b)*. Note that in severe PE, costocartilage is also deformed. (From Fossum TW, et al. Pectus excavatum in eight dogs and six cats. *J Am Anim Hosp Assoc.* 1989;25:595.)

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The cause or causes of PE in animals are unknown (Fig. 29.23). Theories proposed include shortening of the central tendon of the diaphragm, intrauterine pressure abnormalities, and congenital deficiency of the musculature in the cranial portion of the diaphragm. Abnormal respiratory gradients appear to play a role in the development of this disease in some animals because brachycephalic dogs are most commonly affected, many of which have concurrent hypoplastic tracheas. Pectus excavatum may be associated with swimmer's syndrome, which is a poorly characterized disease of neonatal dogs in which the limbs tend to splay laterally, impairing ambulation. Abnormalities of the joints of the limbs and the long bones may also occur.

Patients with PE may have abnormalities of both respiratory and cardiovascular function. Circulatory disorders in animals with PE may occur as a result of abnormal cardiac positioning, which causes kinking of the large veins and disturbance of venous return; compression of the heart, predisposing to arrhythmias (particularly the auricles); restriction of ventricular capacity; and diminished respiratory reserve. Cardiac abnormalities are common (see the Differential Diagnosis section presented later).

DIAGNOSIS

Clinical Presentation

Signalment

Pectus excavatum is a congenital abnormality in dogs and cats. In symptomatic animals, clinical signs are usually present at birth or shortly thereafter. PE may occur in any breed, but brachycephalic dogs appear to be predisposed. A gender predisposition has not been identified.

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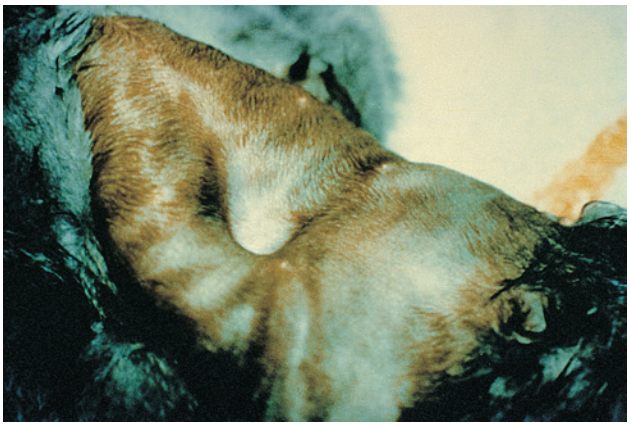


FIG. 29.24 Pectus excavatum in a cat. The head is pointed to the left, and a large depression is evident in the caudal sternum. (From Fossum TW, et al. Pectus excavatum in eight dogs and six cats. *J Am Anim Hosp Assoc.* 1989;25:595.)

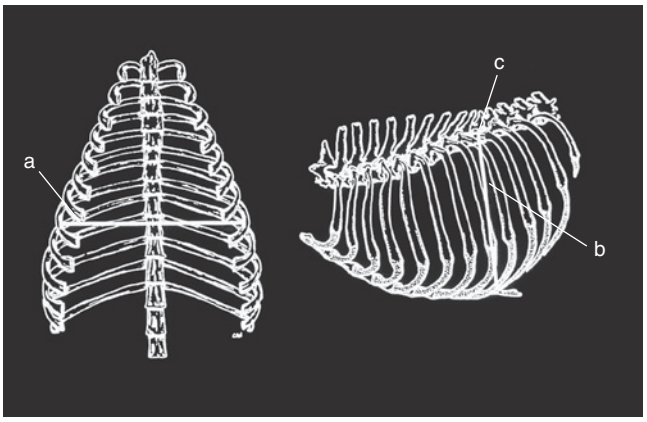


FIG. 29.25 The frontosagittal index is the ratio between the width of the chest at the 10th thoracic vertebra *(a)* and the distance between the center of the ventral surface of the 10th thoracic vertebral body and the nearest point on the sternum *(b)*. The vertebral index is the ratio between the distance from the center of the dorsal surface of the 10th vertebral body to the nearest point on the sternum (*b* and *c*) and the dorsoventral diameter of the vertebral body at the same level *(c)*. (From Fossum TW, et al. Pectus excavatum in eight dogs and six cats. *J Am Anim Hosp Assoc.* 1989;25:595.)

History

Many animals with PE are asymptomatic; however, the defect is often palpable, and this may prompt owners to seek veterinary care despite lack of clinical signs (Fig. 29.24). Symptomatic animals may be presented for evaluation of exercise intolerance, weight loss, hyperpnea, recurrent pulmonary infections, cyanosis, vomiting, persistent and productive coughing, inappetence, and/or mild episodes of upper respiratory disease. A correlation between the severity of clinical signs and the severity of anatomic or physiologic abnormalities has not been observed.

NOTE Although the cause of PE is uncertain, multiple animals in some litters have been affected. Do not breed affected animals; neuter them.

Physical Examination Findings

The sternal deformity is usually palpable. Other physical examination findings may include cardiac murmurs and harsh lung sounds. Dyspnea is variable, but rapid, shallow respirations may be noted.

NOTE Do not assume that cardiac murmurs in animals with PE are due to heart disease. They may be due to abnormal positioning of the heart because of the sternal deformity.

Diagnostic Imaging

Thoracic radiographs show abnormal elevation of the sternum in the caudal thorax. Objective assessment of the deformity may be determined by measuring the frontosagittal and vertebral indices on thoracic radiographs (Box 29.4). The frontosagittal index is calculated by taking the ratio of the width of the chest at the 10th thoracic vertebra, measured on a dorsoventral or ventrodorsal radiograph, and the distance between the center of the ventral surface of the 10th thoracic vertebra and the nearest point on the sternum (Fig. 29.25). The vertebral index is calculated as the ratio of the distance between the center of the dorsal surface of the selected vertebral body to the nearest point on the sternum and the dorsoventral diameter of the center of the same vertebral body (see Fig. 29.25). The severity of PE may be characterized as mild, moderate, or severe based on the frontosagittal and vertebral indices (Table 29.3). This determination may aid in the objective assessment of improvement of thoracic diameters after surgery.

BOX 29.4 Normal Frontosagittal and Vertebral Indexes

Frontosagittal

Nonbrachycephalic dogs: 0.8–1.4
Brachycephalic dogs: 1–1.5
Cats: 0.7–1.3

Vertebral

Nonbrachycephalic dogs: 11.8–19.6
Brachycephalic dogs: 12.5–16.5
Cats: 12.6–18.8

TABLE 29.3 Characterization of Pectus Excavatum in Dogs and Cats Based on Frontosagittal (FS) and Vertebral (Vert) Indices

	INDEX	
PE	FS	Vert
Mild	≤2	>9
Moderate	2–3	6–8.99
Severe	>3	<6

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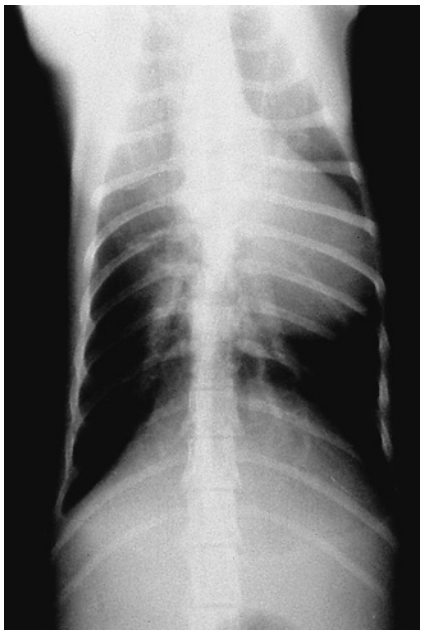


FIG. 29.26 Thoracic radiograph of a dog with pectus excavatum. Note the displacement of the heart caused by the sternal abnormality. (From Fossum TW, et al. Pectus excavatum in eight dogs and six cats. *J Am Anim Hosp Assoc.* 1989;25:595.)

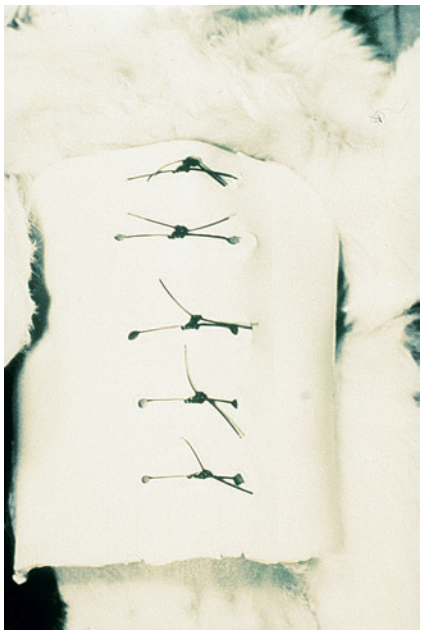


FIG. 29.27 Application of an external splint to the ventral aspect of the thorax in a young dog with pectus excavatum. (From Fossum TW, et al. Pectus excavatum in eight dogs and six cats. *J Am Anim Hosp Assoc.* 1989;25:595.)

Thoracic radiographs should be evaluated for the evidence of concurrent abnormalities (i.e., tracheal hypoplasia, cardiac abnormalities, and pneumonia). Most animals with PE have abnormally positioned hearts (Fig. 29.26), which may cause the heart to appear enlarged radiographically; thus true cardiac enlargement cannot always be distinguished from apparent enlargement as a result of abnormal heart position.

Laboratory Findings

Laboratory abnormalities are uncommon.

DIFFERENTIAL DIAGNOSIS

Diagnosis of PE is straightforward; however, associated abnormalities may be more difficult to diagnose. Cardiac murmurs are common in patients with PE and appear to be associated with the cardiac malpositioning. These murmurs often disappear after surgical correction of the defect or a change in the patient's position. Systolic murmurs in some patients appear to be related to kinking of the pulmonary artery or to exaggeration of the artery's normal vibrations caused by its proximity to the chest wall. Animals with PE and innocent systolic murmurs must be differentiated from those that have underlying cardiac defects, such as pulmonic stenosis or atrial septal defects.

MEDICAL MANAGEMENT

Animals with merely a flat chest may contour to a normal or near normal configuration without surgical intervention. However, owners should be encouraged to regularly perform medial-to-lateral compression of the chest on these young animals. Animals with severe elevation of the sternum will not benefit from this technique or from splintage that simply provides medial-to-lateral compression and does not correct the malpositioned sternum.

Other medical management includes treatment of respiratory tract infections and, if the animal is severely dyspneic, oxygen therapy.

SURGICAL TREATMENT

Application of an external splint to the ventral aspect of the thorax is the most common technique used to correct this defect in animals (Fig. 29.27). Definitive treatment of PE using external splintage is possible because of the young age of affected patients at the time of diagnosis. The costal cartilages and sternum are pliable in these young animals, and the thorax can be reshaped by applying traction to the sternum using sutures that are placed around the sternum and through a rigid splint. Soft tissues that might be abnormal and play a role in the development of this deformity are probably stretched or torn when the sternum is pulled ventrally. Circular, V-shaped, and U-shaped splints that can be contoured to the shape of a normal thorax have been described. Whether surgical correction of the defect should be performed in asymptomatic patients with moderate or severe PE is unknown. Symptomatic patients that do not have associated cardiac abnormalities will benefit from surgery. Repair using an intrasternal pin and external splinting has been reported in dogs, as well as internal splinting using a bone plate applied to the ventral side of the sternum for correction in a young cat with noncompliant sternum.

Preoperative Management

Respiratory infections should be treated before surgery. If the animal is severely dyspneic, oxygen should be provided by nasal insufflation, flow-by, or an oxygen cage until surgery is performed. Prophylactic antibiotic therapy may be given; however, antibiotics are unlikely to prevent skin infections around the splint. Intrathoracic infection secondary to surgery is uncommon.

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Anesthesia

Anesthetic management in these young animals should include attention to the airway, ventilation, body temperature, and blood glucose concentration. Animals should be intubated; ventilation should be assisted or controlled; a high fraction of inspired oxygen should be used; intravenously administered fluids should be warm; and fluids should contain glucose if serum glucose concentrations cannot be monitored (Table 29.4). In addition, the animal should be insulated from the cool surgical environment. The splint should be formed and fitted before anesthesia to reduce the duration of anesthesia. Do not use nitrous oxide in these patients because of the risk of pneumothorax. Postoperative analgesia should be provided in puppies and kittens with fentanyl, hydromorphone, or buprenorphine (see Table 29.4). Do not use chamber or mask induction if the animal is dyspneic.

Surgical Anatomy

The surgical anatomy of the thorax is described on p. 888.

Positioning

The patient is placed in dorsal recumbency, and the ventral thorax is prepared for aseptic surgery.

TABLE 29.4 Anesthetic Considerations for Repair of Pectus Excavatum

Preoperative Considerations	
Associated conditions	• May have other comorbidities
Bloodwork	• HCT • BG • Electrolytes • BUN • Cr • TP
Physical exam	• May be markedly stressed and extremely dyspneic; may have cardiac murmurs; possible orthopedic abnormalities
Other diagnostics	• Blood pressure • ECG • Radiographs (thoracic) • +/– Cardiac ultrasound

Premedications	• Oxygen with flow-by or face mask
	• Avoid sedatives in depressed or dyspneic patients
	• In patients that have minimal respiratory compromise give the following:
	  • Midazolam (0.2 mg/kg IV, IM), *or*
	  • Diazepam (0.2 mg/kg IV)
	Before induction, avoid all respiratory depressants including but not limited to opioids, xylazine, medetomidine, dexmedetomidine; avoid acepromazine, xylazine, medetomidine, and dexmedetomidine if hypotensive.

Intraoperative Considerations	

Induction	• Preoxygenate for 3–5 min with face mask or flow-by oxygen
	• If normal cardiac function, give:
	  • Propofol (4–8 mg/kg IV); administer rapidly to expedite intubation and ventilation
	  • Alfaxalone (2–5 mg/kg IV)
	• If marked cardiac compromise, give:
	  • Etomidate (0.5–1.5 mg/kg IV); give a benzodiazepine before etomidate to avoid myoclonic activity

Maintenance	• Isoflurane or sevoflurane, *plus*
	  • Fentanyl (2–10 µg/kg IV PRN in dogs; 1–4 µg/kg IV PRN in cats) for short-term pain relief, *plus*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Oxymorphone (0.1–0.2 mg/kg IV), *or*
	  • Morphine[b] (0.1–1 mg/kg IV PRN in dogs; 0.05–0.2 mg/kg IV PRN in cats), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV PRN), *plus*
	  • Ketamine (low dose) (0.5–1 mg/kg IV)
	• Avoid nitrous oxide
	• Ventilation parameters:
	  • SpO_2 >95%
	  • Small tidal volumes <6 mL/kg
	  • Respiratory rates: 10–30
	  • Peak airway pressures <12–15 mm Hg if able to maintain adequate tidal volumes
	  • Make sure to maintain small tidal volumes to avoid pulmonary trauma
	• If unable to meet above parameters, consider PEEP

Fluid needs	• 5-20 mL/kg/h to replace evaporative losses plus 3× EBL; consider colloids for EBL replacement in 1:1 ratio
Monitoring	• BP • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature • U/O

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TABLE 29.4 Anesthetic Considerations for Repair of Pectus Excavatum—cont'd

Postoperative Considerations

Analgesia	• Morphine[b] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.05–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h in cats), *or* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats), *or* • Oxymorphone (0.1–0.2 mg/kg IV, IM q2–4h), *or* • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM or 0.01–0.02 mg/kg OTM in cats) • NSAIDs contraindicated in patients age <3 weeks[d]
Monitoring	• SpO_2 • Temperature • HR • Respiratory rate • U/O
Bloodwork	• HCT • TP • BG, possibly serial in the very young
Estimated pain score	Moderate

[a]Monitor for hyperthermia in cats.
[b]Give slowly to prevent histamine release.
[c]Buprenorphine is a better analgesic than morphine in cats.
[d]The safety of most NSAIDs has not been evaluated in very young canine patients. Prior to using an individual NSAID in pediatric patients less than 6 months of age, check on age recommendations of that NSAID.
BG, Blood glucose; *BP,* blood pressure; *BUN,* blood urea nitrogen; *Cr,* creatinine; *EBL,* estimated blood loss; *$ETCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *MAP,* mean arterial pressure; *NSAID,* nonsteroidal antiinflammatory drug; *OTM,* oral transmucosal; *PEEP,* positive end-expiratory pressure; *PRN,* as needed; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein; *U/O,* urine output.

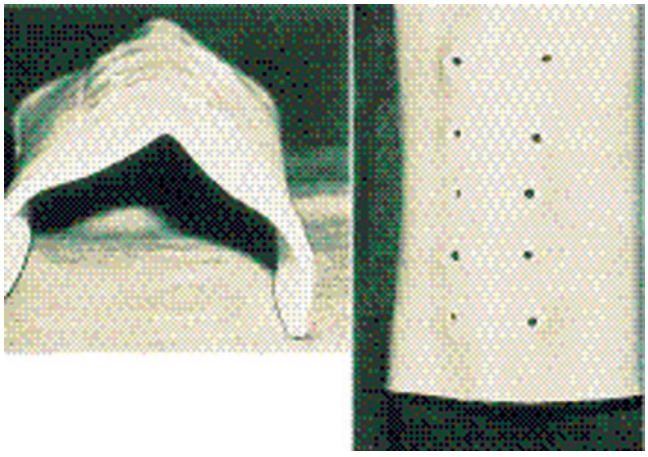


FIG. 29.28 External splint for correction of pectus excavatum. Place two parallel rows of four to six holes in the splint with a small Steinmann pin. (From Fossum TW, Boudrieau RJ, Hobson P, Rudy RL. Surgical correction of pectus excavatum using external splintage in two dogs and a cat. *J Am Vet Med Assoc.* 1989;195:91.)

SURGICAL TECHNIQUE

Fashion a rectangular piece of moldable splinting material into a U shape (Fig. 29.28), and mold it to fit the ventral aspect of the thorax. Apply a small amount of adhesive padding to the cranial border and inner surface of the splint or, as an alternative, pad the splint with cast padding after it has been positioned. Place two parallel rows of four to six holes in the splint with a small Steinmann pin (see Fig. 29.28). Position the holes so that the distance between adjacent holes is slightly greater than the width of the sternum. Pass the selected suture (see the discussion offered later in the Suture Materials and Special Instruments section) around the sternum by maneuvering the needle blindly off the lateral edge of the sternum. As an alternative, pass the needle around the sternebra at a 45-degree angle to incorporate the costocartilage and possibly reduce the chance of the suture pulling through the soft sternebral bone (Fig. 29.29). Sutures must be placed around the sternum and not subcutaneously. Additionally, sutures must be placed in the area of the greatest concavity. If the sutures are placed proximal to the area with the greatest depression, the sternum cannot be pulled into a normal position, resulting in less than optimal correction of the defect. *Keep the needle as close as possible to the dorsal aspect of the sternum to prevent piercing the heart or lungs. Leave the suture ends long and tag them. When all sutures have been placed, pass the ends through the predrilled holes in the splint and tie them securely on its ventral aspect (see Fig. 29.28). Two sutures may be placed and tied to themselves and then these sutures tied together so that the splint can be adjusted without replacement of sutures or use of anesthesia.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

A taper-point needle is recommended; if suture material with a large, swaged-on needle is not available, a large, eyed, taper-point needle should be selected (to prevent bending and possible breakage as it is passed around the sternum). Large (i.e., No. 0 to No. 2) monofilament absorbable or nonabsorbable suture material is recommended (e.g., polydioxanone [PDS], polyglyconate [Maxon], or nylon suture).

POSTOPERATIVE CARE AND ASSESSMENT

The animal should be evaluated in the early postoperative period for intrathoracic hemorrhage because piercing of the heart, lung, or internal thoracic vessels as the needle is passed around the sternum is possible. Positioning the animal in dorsal recumbency, paying close attention to the phase of respiration, and keeping the needle as close to the sternum as possible help prevent such complications. The splint should be left in place 10 to 21 days if possible, although less time may also be effective.

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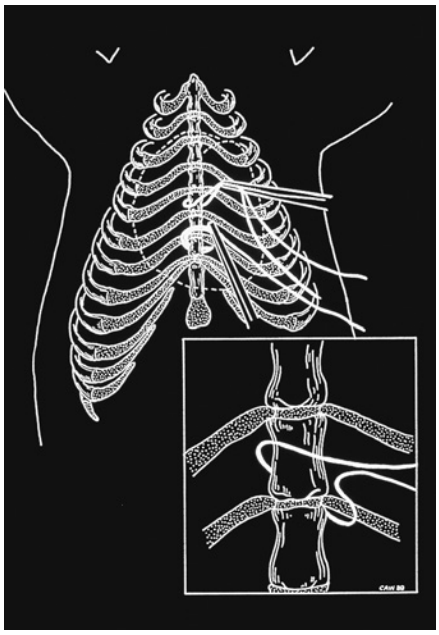


FIG. 29.29 Sutures placed around the sternebrae may be horizontal to the long axis or at a 45-degree angle. (From Fossum TW, Boudrieau RJ, Hobson P, Rudy RL. Surgical correction of pectus excavatum using external splintage in two dogs and a cat. *J Am Vet Med Assoc.* 1989;195:91.)

COMPLICATIONS

Suture abscesses, mild superficial dermatitis, and skin abrasions are common, but these are usually minor and heal quickly after removal of the splint. Adequate padding of the splint may help prevent abrasions. Fatal reexpansion pulmonary edema (see p. 887) has been reported in a kitten after correction of PE.

PROGNOSIS

The prognosis is excellent for animals without underlying disease in which surgery is performed at a young age. Older animals with a less pliable sternum may not respond as favorably to external splintage. Partial sternectomy may benefit such animals (see p. 913).

THORACIC WALL NEOPLASIA

DEFINITIONS

Thoracic wall neoplasia may arise from the ribs, musculature, or pleura. **Chondrosarcomas** are tumors that arise from cartilage, whereas **osteosarcomas** arise from bone.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Primary tumors of the rib have a high metastatic rate and are uncommon in dogs or cats. Osteosarcomas are the most common neoplasm of the canine rib, followed by chondrosarcomas; the costochondral junction is the usual site of origin of these tumors. Hemangiosarcoma and fibrosarcoma have also been reported. Metastatic and primary tumors of the sternum have rarely been reported in dogs. Primary sternal tumors of dogs include chondrosarcoma and osteosarcoma.

DIAGNOSIS

Clinical Presentation

Signalment

Primary rib tumors generally develop in young and middle-aged dogs. Rib tumors should be considered as a possible differential diagnosis for masses involving the thoracic wall, even in young dogs.

History

Animals with rib tumors may be brought in because of dyspnea or a painless thoracic wall mass. Animals with sternal neoplasia are often presented for evaluation of a palpable sternal mass.

Physical Examination Findings

Most rib tumors cause a localized swelling of the thoracic wall; however, pleural effusion without evidence of a thoracic mass occasionally occurs in dogs with small primary rib tumors and metastatic pulmonary lesions. Other clinical signs of rib tumors are weight loss and dyspnea. Sternal tumors usually cause a localized swelling but may be associated with dyspnea if they metastasize to the lungs.

Diagnostic Imaging

Rib tumors are generally expansile masses that cause bone destruction and proliferation. They typically cause an extrapleural sign (protrusion of an infectious, neoplastic, or traumatic lesion medially from the thoracic wall causing a broad-based intrathoracic mass effect); moreover, the majority of the mass is often within the thoracic cavity. Sternal tumors may also produce lysis of sternebrae and adjacent ribs. Thoracic radiographs of animals with neoplasia of the ribs or sternum should be evaluated for pulmonary metastasis, lymph node involvement, and/or pleural effusion. Contrast-enhanced thoracic CT is likely to be of more value for staging and surgical planning.

Laboratory Findings

Laboratory findings are nonspecific. Blood gas analysis may show hypoxemia and respiratory acidosis or alkalosis (because of hyperventilation). Increased activity of serum alkaline phosphatase is associated with decreased survival in dogs with osteosarcoma of flat bones, including rib and sternum.[18]

DIFFERENTIAL DIAGNOSIS

Neoplasia of the sternum or ribs should be differentiated from osteomyelitis, fungal infections, or abscesses based on cytologic or histologic findings. A tentative diagnosis of the cell type can usually be made by fine-needle aspiration of the mass. Definitive diagnosis usually requires histologic examination of a biopsy specimen. Although pleural effusion is common in dogs with rib tumors, neoplastic cells are rarely found in the fluid.

MEDICAL MANAGEMENT

Medical management of animals with rib or sternal tumors (i.e., thoracentesis if pleural effusion exists and oxygen therapy for dyspnea) is generally palliative only. Pleuroperitoneal shunts are used in human beings with pleural effusion caused by terminal

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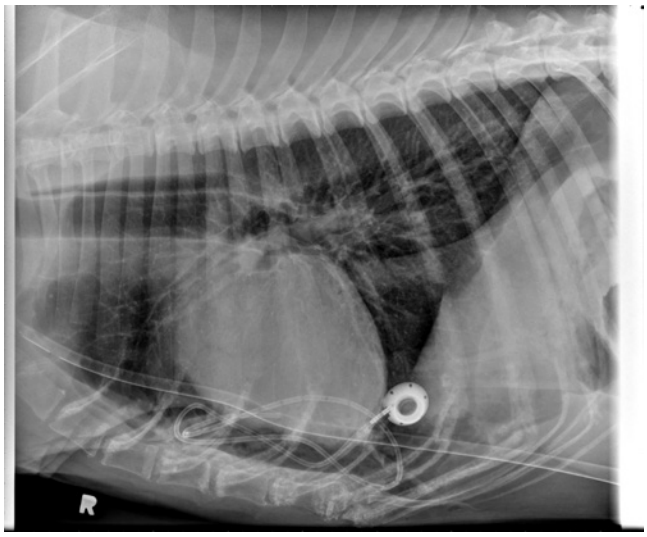


FIG. 29.30 Lateral thoracic radiograph of a 9-year-old German shepherd dog with neoplastic pleural effusion and a unilateral Pleuralport (Norfolk Veterinary Products).



FIG. 29.31 Huber needle access to a Pleuralport (Norfolk Veterinary Products) in a 6-year-old English bulldog with chronic pleural effusion.

neoplasia; however, such use has not been reported in dogs or cats. Instead, a Pleuralport (Norfolk Veterinary Products) can be placed to manage pleural effusion and provide access for intracavitary chemotherapy (Fig. 29.30). The device consists of a multifenestrated radiopaque silicone thoracostomy tube that is connected to a drainage hub that is placed outside the thorax in the subcutaneous tissue. The hub is accessed percutaneously using a Huber point (noncoring) needle to allow for repeated thoracic drainage (Fig. 29.31). Reported advantages of this device include increased comfort, decreased risk of infection, decreased risk of complications associated with repeated thoracocentesis, decreased risk of accidental removal, and potential management of thoracic effusion in a home environment. The device is placed using a keyhole thoracotomy.

SURGICAL TREATMENT

Surgical resection of thoracic wall tumors is the treatment of choice. Full-thickness or en bloc resection of three or more ribs for thoracic wall neoplasia requires surgical reconstruction to reestablish thoracic wall continuity. Removal of more than six ribs is generally not recommended. With tumors of the caudal thorax, advancement of the diaphragm cranial to the resected ribs reduces the need for rigid fixation of the thoracic wall. Synthetic mesh may be used to fill other defects. A possible complication of polypropylene mesh is fistula formation secondary to adhesions; use of omentum or well-vascularized autologous muscle (latissimus dorsi or deep pectoral muscle) over the implant may reduce postoperative complications. Resection of large tumors may require a muscle and skin composite flap to close the resultant defect. A latissimus dorsi myocutaneous flap has been shown to create an airtight closure following chondrosarcoma resection in five dogs. Partial or complete sternectomy may be curative in dogs with primary sternal neoplasia. Although temporary instability of the thorax may occur after large sternal resections, this does not appear to cause any permanent or significant respiratory dysfunction.

Preoperative Management

Thoracentesis should be performed before induction of anesthesia in dogs with pleural effusion associated with thoracic wall neoplasia.

Anesthesia

See anesthesia recommendations for animals with respiratory disease on p. 884.

Positioning

With thoracic wall or sternal neoplasia, a generous area surrounding the tumor should be prepared for aseptic surgery.

SURGICAL TECHNIQUE

En Bloc Resection of Thoracic Wall Neoplasia

Remove the thoracic wall containing the neoplasm and a margin of normal tissue, leaving a square or rectangular defect. Cut a piece of polypropylene mesh slightly larger than the defect. Fold over the edges of the mesh, and suture the double thickness of mesh to the pleural side of the defect (Fig. 29.32). Draw the mesh tightly across the defect when suturing it to prevent it from moving paradoxically with respiration. If more than four or five ribs are removed, support the ribs with plastic spinal plates or rib grafts. Mobilize and advance the thoracic wall musculature (Fig. 29.33) over the defect or, if there is insufficient muscle, exteriorize an omental pedicle flap through a paracostal abdominal approach and tunnel it subcutaneously to the defect. As an alternative, exteriorize the omental flap through the diaphragm. Place the omental flap over the mesh and suture skin over the defect.

For caudal rib tumors, advancement of the diaphragm may be done after en bloc resection of the mass and surrounding tissue. Synthetic reconstruction of the rib cage is rarely necessary.

Partial Sternectomy

Partial sternectomy should be considered only for relatively small, localized sternal neoplasms that do not appear to have intrathoracic involvement.

Sternectomy has been used successfully for extensive sternal osteomyelitis. The entire sternum can be removed in small animals. *Incise through the skin overlying the neoplasm (if skin involvement is suspected, resect the skin). Identify the rib articulations on the sternum. Use rongeurs to remove the affected sternebrae and ribs. If possible, remove one sternebra caudal and one cranial*

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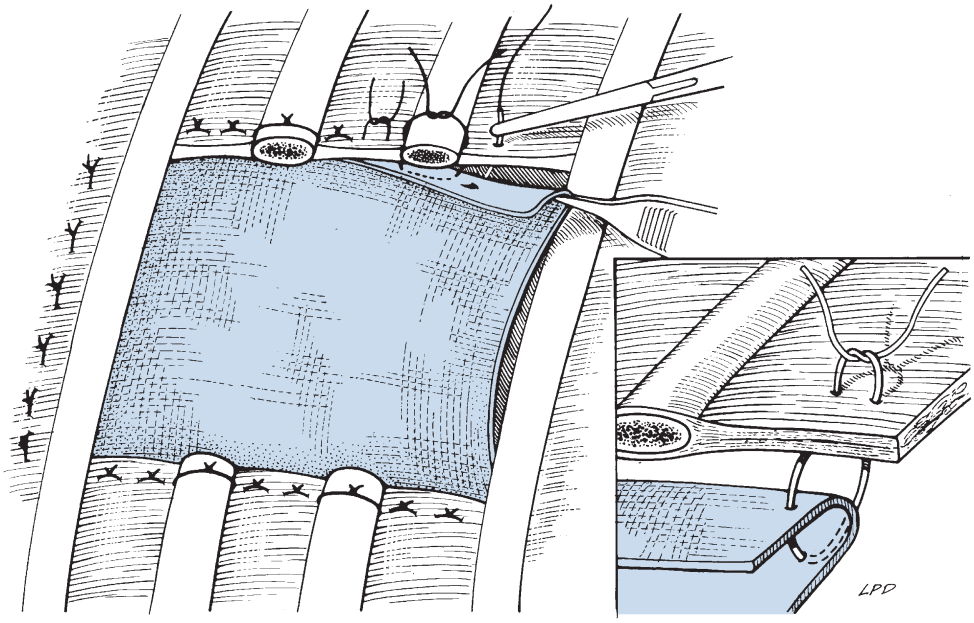


FIG. 29.32 Some thoracic tumors may be removed by en bloc resection of the thoracic wall. Remove the thoracic wall containing the neoplasm and a margin of normal tissue. Fold the edges of a piece of mesh over and suture the double thickness of mesh to the pleural side of the defect.

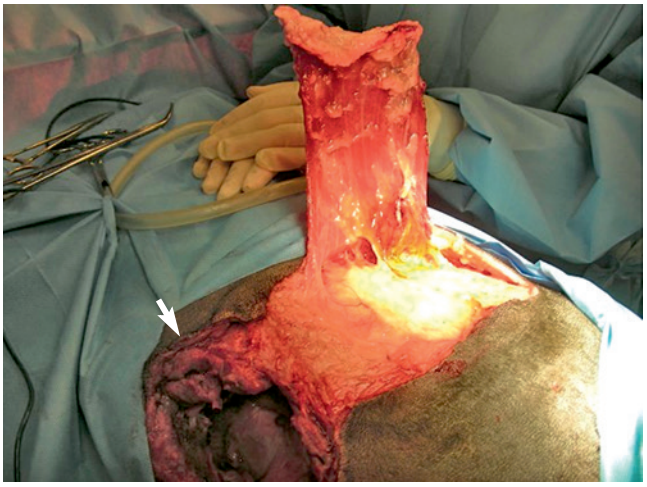


FIG. 29.33 Intraoperative image of elevation of the latissimus dorsi muscle for reconstruction of the thoracic wall defect following partial sternectomy *(arrow)*.

to the lesion. Assess the thoracic cavity for involvement. Avoid lacerating the internal thoracic arteries; ligate them if necessary. Appose the ribs and intercostal muscles with a large (e.g., No. 1) monofilament suture in an interrupted or horizontal mattress pattern. Use a simple continuous suture pattern to appose remnants of the rectus abdominis muscle over the junction of the rib ends. Minimize dead space by apposing the skin and underlying tissue with walking sutures. Place a thoracostomy tube and evacuate air from the thoracic cavity. Place a light support wrap over the thorax to protect the incision and thoracostomy tube.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Reconstruction of thoracic wall defects should be done with monofilament nonabsorbable suture (e.g., polypropylene or nylon). Polypropylene (Marlex) mesh may be used for thoracic wall reconstruction.

POSTOPERATIVE CARE AND ASSESSMENT

Animals with surgically created thoracic wall defects should be monitored closely in the postoperative period for hypoventilation or the development of pneumothorax (or both). Analgesic therapy is warranted in these animals (see Chapter 13; for dosages of opioids, see Tables 13.2 and 13.3). See p. 897 for additional comments on the postoperative management of animals with respiratory disorders.

COMPLICATIONS

Postoperative complications associated with thoracic wall and sternal reconstruction include seroma formation, wound infection, pleural effusion, peripheral edema, and muscle flap failure. Complications are more likely to occur with sternal reconstruction and thoracic wall defects reconstructed with prosthetic mesh.

PROGNOSIS

The prognosis for dogs with rib osteosarcoma is guarded, as a median survival of nearly 300 days has been reported following wide surgical resection and adjunctive chemotherapy. In contrast, dogs with primary rib chondrosarcoma have a good prognosis with surgery alone. Too few sternal tumors have been reported to define the prognosis in affected animals.

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30

Surgery of the Lower Respiratory System: Pleural Cavity and Diaphragm



GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

The **pleurae** are the serous membranes that cover the lungs and line the thoracic cavity, completely enclosing a potential space known as the **pleural cavity.** The **parietal pleurae** are the portions of the pleura that line the walls of the thoracic cavity, whereas the **visceral, or pulmonary, pleura** invests the lungs and lines their fissures, completely separating the different lobes. **Thoracocentesis** (or **thoracentesis**) is a surgical puncture of the thoracic wall to remove air (**pneumothorax**) or fluid (**pleural effusion**) from the pleural space. **Pleurodesis** is the creation of adhesions between the visceral and parietal pleurae by instilling irritating agents into the pleural cavity or mechanically damaging the pleura at surgery.

PREOPERATIVE CONCERNS

Respiratory function should be carefully monitored in patients with pleural cavity or diaphragmatic abnormalities. Qualitative assessments of respiratory function include monitoring the respiratory rate and pattern plus capillary refill time and color (Table 30.1 and Box 30.1). Animals with pleural cavity disease usually have a restrictive respiratory pattern (i.e., rapid, shallow respirations). Arterial blood gas analysis can augment qualitative information about the effectiveness of ventilation and gas exchange (Table 30.2). Pulse oximetry noninvasively measures hemoglobin saturation of blood and thus indirectly provides quantitative information about oxygenation. Cardiovascular parameters (e.g., heart rate and rhythm) should also be evaluated (see Table 30.1). An electrocardiogram (ECG) should be performed in all trauma patients to detect arrhythmias resulting from traumatic myocarditis. Intravenous fluids should be provided to dehydrated animals or those drinking insufficient fluids to maintain hydration. Care should be taken to avoid causing overhydration and pulmonary edema, which further compromise respiration. Monitoring central venous pressure may be useful in some patients.

Severely dyspneic animals strongly suspected of having pneumothorax or pleural effusion should have thoracentesis (Fig. 30.1) before radiographs are made. Removal of even small amounts of pleural effusion or air may significantly improve ventilation, allowing safer manipulation of the patient for radiographic procedures. If the patient is not severely dyspneic, it is safer to first obtain a dorsoventral thoracic radiograph lest lung laceration during thoracocentesis causes pneumothorax and worsens the dyspnea. Most dyspneic animals allow thoracentesis with minimal restraint; general anesthetics are contraindicated. The animal should be allowed to remain in sternal recumbency, and if they will tolerate it, oxygen should be provided by a face mask, flow-by, or nasal insufflation (see Chapter 4). A negative tap does not rule out pleural effusion or pneumothorax (e.g., the fluid may be in pockets, there may be exudate plugging the needle, or the needle may be too short). If there is no evidence of third space disease or if removal of fluid or air from the chest does not help alleviate dyspnea, then underlying pulmonary disease (e.g., pneumonia, pulmonary edema, pulmonary contusions, pulmonary neoplasia) or severe fibrosing pleuritis should be suspected (discussed later). Providing nasal oxygen or placing the animal in an oxygen cage may be beneficial while treatment of the pulmonary disease is initiated (see p. 29).

NOTE Do not attempt to place thoracostomy tubes or take radiographs in animals with known pleural effusion that are extremely dyspneic—perform thoracentesis first!

Thoracostomy tube placement should not be attempted in animals with severe respiratory distress. Generally, stabilization and improved ventilation can first be accomplished by removing some pleural air or fluid by means of needle thoracentesis. In critically ill patients, large-bore or trocar thoracostomy tubes rarely can be placed without the use of general anesthesia; local anesthesia (i.e., local anesthetic infiltration or an intercostal nerve block) may be sufficient (Box 30.2). However, this should be avoided if at all possible because animals with pleural cavity disease benefit from intermittent positive-pressure ventilation (IPPV) and oxygen supplementation during tube placement. Control of the animal's airway (by means of endotracheal intubation and positive pressure ventilation) and oxygen therapy should be achieved rapidly (discussed later). The use of small-bore (e.g., 14-gauge) wire-guided chest drains placed using a modified Seldinger technique may also be considered, and these catheters may be placed in some animals with sedation only. For preoperative concerns of patients with diaphragmatic herniation, see p. 928.

NOTE Preoxygenation of dogs with oxygen versus room air for 3 minutes significantly increases the time to desaturation in healthy dogs, as well as those with respiratory compromise. Thus, if possible, preoxygenate animals with pleural cavity disease prior to anesthesia or sedation.

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ABSTRACT

Animals presenting with diseases of the thoracic cavity are often severely dyspneic and may require prompt, aggressive therapy to reduce mortality. Common causes of thoracic disease include pneumothorax and pleural effusion (e.g., chylohtroax, pyothorax), diaphragmatic hernia, and mediastinal masses.

KEYWORDS

thoracocentesis
thoracostomy tube placement
traumatic pneumothorax
spontaneous pneumothorax
chylothorax
pyothorax
traumatic diaphragmatic hernia
peritoneopericardial diaphragmatic hernia
myasthenia gravis
thymoma

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TABLE 30.1 Normal Heart Rate and Respiratory Rate in Conscious Dogs and Cats

	HR (beats/min)	RR (breaths/min)
Dog	70–140	20–40
Cat	145–200	20–40

HR, Heart rate; *RR,* respiratory rate.

BOX 30.1 Normal Capillary Refill Time

<1–2 seconds

TABLE 30.2 Normal pH and Arterial Blood Gas Values on Room Air

	Value	Range
pH	7.4	(7.35–7.45)
PaO_2	95 mm Hg	(80–110)
PvO_2	40 mm Hg	(35–45)
$PaCO_2$	40 mm Hg	(35–45)
$PvCO_2$	45 mm Hg	(40–48)
HCO_3^-	24 mEq/L	(22–27)

PaO_2, Partial pressure of arterial oxygen; *PvO_2,* venous oxygen concentration; *$PaCO_2$,* carbon dioxide partial pressure; *$PvCO_2$,* venous carbon dioxide concentration; *HCO_3^-,* bicarbonate.

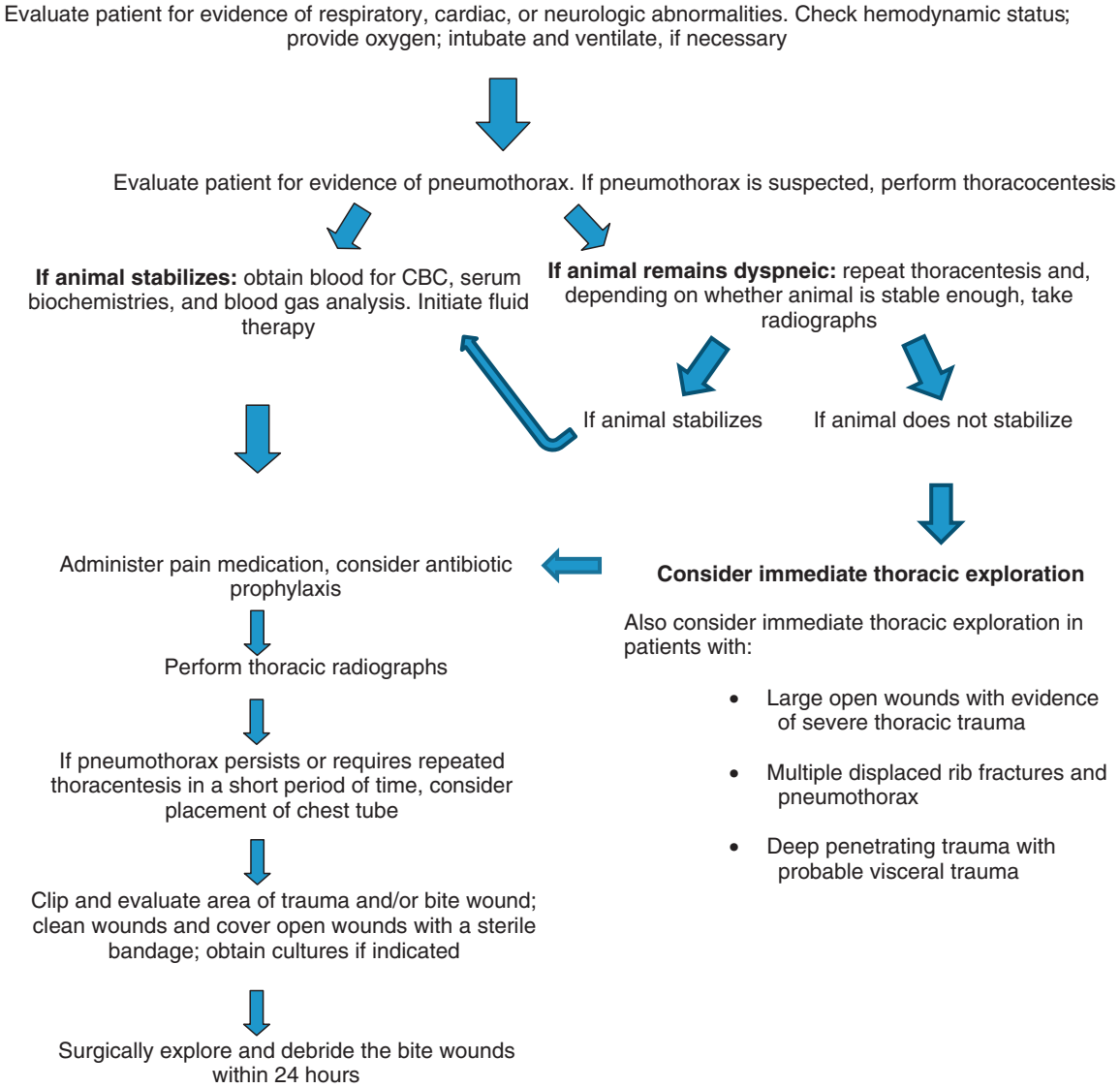


FIG. 30.1 Algorithm for management of dyspneic animals with thoracic bite wounds. *CBC,* Complete blood cell count.

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BOX 30.2 Key Points for Placing Intercostal Blocks and Incisional Blocks

- Determine maximum dose of bupivacaine to be used at 2 mg/kg over 6 to 8 h.
- Dilute in saline or lactated Ringer's solution to create a 0.125% solution and divide solution approximately in half (for 0.5% [5 mg/mL] bupivacaine, add 3 mL saline for each milliliter of local anesthetic).
- For intercostal analgesia, selectively block the intercostal nerves supplying the thoracotomy incision site by blocking two nerves cranial to the incision site and two nerves caudal to the incision, as well as the nerve at the incision. Use a 22-gauge needle and block the five nerves caudal to the ribs as dorsally as possible (see p. 152).
- Take the remainder of the bupivacaine, and with a 22-gauge needle, inject subcutaneously along either side of the incision, running along the entire incision line.
- If bupivacaine is placed down the thoracostomy tube, do so with care and while the animal is asleep. Any amount of bupivacaine placed into the thoracostomy tube should be considered part of the total maximum bupivacaine dose. For each mL of 0.5% bupivacaine, dilute it into 5 to 10 mL of saline. Then inject this solution into the thoracostomy tube. Roll the animal so the thoracostomy tube is down, wait for 2 to 3 min and roll the animal onto its back. Wait another 2 to 3 min and roll the animal onto its other side, if necessary. Wait another 2 to 3 min and remove any remaining fluid. Then wake up the animal and extubate. **There is no benefit in placing local anesthetic into the thoracostomy tube if there is blood or purulent discharge in the pleural cavity.**
- Splash blocks are of little value and are a waste of valuable local anesthetic.

Most structures of the thorax can be visualized on computed tomography (CT) images at the soft tissue setting. CT allows for three-dimensional reconstruction and elimination of the superimposition of overlying structures, thus permitting a more precise characterization of abnormalities before surgical intervention than is obtained radiographically. For example, CT evaluation of tracheobronchial lymph nodes in dogs with lymph node pathology often correlates with metastasis or severe granulomatous lymphadenitis, and contrast-enhanced CT may be used to diagnose pulmonary thromboembolism.

ANESTHETIC CONSIDERATIONS

Respiratory patients should be managed with extreme care until they have been intubated and ventilation can be assisted. Premedication with any drug that causes hypoventilation is contraindicated (Table 30.3). Additionally, every attempt should be made to minimize stress to the patient prior to induction. Oxygen support, even while placing an intravenous catheter, may be necessary. Pulse oximetry with a sensor that can be secured to the tail can be especially helpful with perioperative monitoring of the respiratory distressed patient. Prior to induction, the anesthesia provider should be prepared with airway devices, anesthesia machine, and monitors, as well as induction and emergency drugs. Preoxygenating the patient for 3 to 5 minutes prior to induction (see p. 29) should be followed with a rapid induction and intubation. If possible, positive pressure ventilation should be performed with lower tidal volumes, lower peak ventilation pressures, and higher respiratory rates. This will decrease leakage of air through damaged alveoli if pneumothorax is present. However, if the diagnosis is diaphragmatic hernia or pleural effusion, tidal volumes may be kept low, but peak airway pressures may need to be higher to accomplish adequate ventilation. If external pressures asserted on the lung tissue are completely compressing alveoli, bronchioles, and bronchi, it may take higher pressures to ventilate until the thorax is surgically opened. When possible, however, smaller tidal volumes, higher respiratory rates, and lower peak airway pressures should be used. Dogs and cats with respiratory insufficiency should be maintained with inhalation anesthetics (e.g., isoflurane or sevoflurane) (see Table 30.3). Analgesia needs to be provided to decrease the amount of inhalant required, as high concentrations of inhalants can lead to hypotension. Inhalation anesthesia is advantageous because it allows rapid recovery and more precise control of anesthetic depth than does maintaining anesthesia with longer-acting intravenous anesthetics. Nitrous oxide should not be used in patients with pneumothorax or diaphragmatic hernias because it rapidly diffuses into air-filled spaces (i.e., pleural cavity or gas-filled organs), causing further lung compression or organ enlargement. In addition, nitrous oxide is comparatively less soluble in plasma than oxygen and other inhalation anesthetics; therefore it rapidly diffuses into the alveoli when it is discontinued, resulting in diffusion hypoxia if the nitrous oxide is not turned off 5 to 10 minutes before extubation. Once the surgery has been completed, extubation should not be rushed. Make sure the patient is awake and not overly sedated, respirations are adequate, and the patient is comfortable prior to extubation. For specific anesthetic recommendations for animals with pneumothorax and diaphragmatic hernias, see pp, 937 and 928, respectively. See also Chapter 29.

NOTE Do not use mask or chamber induction in patients with respiratory distress; intubate as rapidly as possible using injectable anesthetic agents.

ANTIBIOTICS

Needle thoracocentesis performed with proper aseptic technique is unlikely to induce infection in patients with normal immune function; therefore prophylactic antibiotics are not indicated. Prophylactic antibiotics in patients with thoracostomy tubes are of dubious benefit. Studies in humans have not shown lower infection rates when patients with thoracostomy tubes are given prophylactic antibiotics. However, thoracostomy tubes must be maintained and handled with appropriate precautions (e.g., sterile gloves and syringes, chest bandages) to reduce the potential for iatrogenic contamination. Gram-negative bacteria and anaerobes are common isolates in animals with respiratory disease. A 2016 study of normal dogs evaluated with CT and fluid analysis demonstrated development of pyothorax in six of eight dogs between 4 and 6 days after placement of a thoracostomy tube.[1] Therapy for pyothorax should be based on culture and sensitivity test results, if possible, because unpredictable antibiotic sensitivity is common with the microorganisms commonly encountered with this condition. For specific antibiotic recommendations for patients with pyothorax, see p. 948.

SURGICAL ANATOMY

Each pleural cavity is only a potential space unless air or fluid collects between the parietal and visceral pleurae, preventing normal lung expansion. In a normal animal, only a capillary film of fluid exists to moisten the mesothelial cells that line its pleural surface. Therefore, except for this capillary fluid, the

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TABLE 30.3 Anesthetic Considerations for Thoracotomy of the Acute Trauma Patient

Preoperative Considerations

Associated conditions	• Anemia • Dehydration • Electrolyte abnormalities • Hypotension • Dysrhythmias; often ventricular • Presence of other trauma (e.g., pelvic, abdominal, thoracic)
Bloodwork	• HCT • Electrolytes • BUN • Cr • TP • Albumin • +/– Blood gas
Physical exam	• May be dehydrated, in shock, hypotensive, tachycardic or bradycardic, and hypothermic • Absence of pulmonary sounds in the dorsal lateral thorax with auscultation if pneumothorax; absence of pulmonary sounds in the ventral thorax and muffled heart sounds if hemothorax; the abdomen may be painful if the animal has a uroabdomen or hemoabdomen
Other diagnostics	• Blood pressure • ECG • Radiographs (thoracic and abdominal) • +/– CT • Ultrasound • Thoracocentesis • SpO_2

Premedications	• Oxygen with flow-by or face mask
	• Give shock dose of fluids, if necessary to stabilize. Reconsider need for surgery if patient is unstable.
	• Give blood transfusion if HCT <20% in dogs, <18% in cats (see pp. 27 and 33)
	• Avoid sedatives in depressed or dyspneic patients
	• Administer gastrointestinal protectant of choice
	• In patients that are have minimal respiratory compromise, give
	  • Midazolam (0.1–0.2 mg/kg IV, IM), *or*
	  • Diazepam (0.1–0.2 mg/kg IV)
	• Before induction, avoid all respiratory depressants, including but not limited to opioids, xylazine, medetomidine, and dexmedetomidine; if hypotensive, avoid acepromazine, xylazine, medetomidine, and dexmedetomidine; if tachycardic, avoid atropine, glycopyrrolate, and induction doses of ketamine.
	• After a benzodiazepine is given but before induction, clip hair from surgical area; this can usually be done with patient standing or in lateral recumbency; do not place patient in ventrodorsal position until after intubation.

Intraoperative Considerations

Induction	• Preoxygenate for 3–5 min with face mask or flow-by oxygen
	• If dehydrated, give:
	  • Etomidate (0.5–1.5 mg/kg IV); give after administering a benzodiazepine to prevent myoclonus
	• If normal hydration, give:
	  • Propofol (2–6 mg/kg IV), *or*
	  • Alfaxalone (2–5 mg/kg IV)
	• If dyspneic, administer rapidly to expedite intubation and ventilation

Maintenance	• Isoflurane or sevoflurane, *plus*
	  • Fentanyl (2–10 µg/kg IV PRN in dogs; 1–4 µg/kg IV PRN in cats) for short-term pain relief, *plus*
	  • Fentanyl CRI (1–5 µg/kg IV loading dose, then 2–30 µg/kg/h IV), *or*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Oxymorphone (0.05–0.2 mg/kg IV, IM), *or*
	  • Buprenorphine[b] (0.005–0.02 mg/kg IV PRN), *plus*
	  • Ketamine (low dose) (0.5–1 mg/kg IV), *or*
	  • Ketamine CRI (0.5 mg/kg IV loading dose, then 10 µg/kg/min IV)
	• If hypotensive (to keep MAP 60–80 mm Hg), give phenylephrine, ephedrine, dopamine, norepinephrine, vasopressin, or fluids as needed
	• Have surgeon ready to drape and make incision when positioning patient ventrodorsally
	• Ventilation parameters:
	  • SpO_2 >95%
	  • Small tidal volumes: 5–7 mL/kg
	  • Respiratory rates: 10–30
	  • Peak airway pressures: <12–15 mm Hg if able to achieve adequate tidal volumes
	• Slowly expand the lungs while manually ventilating and visually observing the lung expansion.
	• Consider PEEP

Fluid needs	• 5–20 mL/kg/h to replace evaporative losses plus 3 × EBL; consider colloids or blood for EBL replacement in 1:1 ratio

Continued

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TABLE 30.3 Anesthetic Considerations for Thoracotomy of the Acute Trauma Patient—cont'd

Monitoring	• BP • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature • +/– Arterial line • U/O

Blocks	Epidural:
	• Morphine (0.1 mg/kg preservative free), *or*
	• Buprenorphine (0.003–0.005 mg/kg diluted in saline), *plus*
	• Local anesthetics (see Box 30.2 and Chapter 13, p. 144):
	  • Intercostal nerve blocks with local anesthetics, *plus*
	  • Incisional block with local anesthetic
	• Bupivacaine diluted in saline may be placed down the thoracostomy tube but should only be done when the patient is asleep and is still intubated

Postoperative Considerations	
Analgesia	• Fentanyl CRI (1–10 µg/kg IV loading dose, then 2–20 µg/kg/h IV), *or* • Morphine[c] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.05–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h in cats) if no hypotension, *or* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats), *or* • Hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs), *or* • Oxymorphone (0.1–0.2 mg/kg IV, IM), *or* • Buprenorphine[b] (0.005–0.02 mg/kg IV, IM q4–8h or 0.01–0.02 mg/kg OTM q6–12h in cats), *plus* • +/– Ketamine CRI (2 µg/kg/min IV; if no previous loading dose, give 0.5 mg/kg IV before CRI)
Monitoring	• SpO_2 • Blood pressure • ECG • HR • Respiratory rate • Temperature • U/O • Thoracostomy tubes
Bloodwork	• HCT • TP • Electrolytes • Blood gas • +/– Albumin
Estimated pain score	Severe

[a]Monitor for hyperthermia in cats.
[b]Buprenorphine is a better analgesic than morphine in cats.
[c]Give slowly to prevent histamine release.
BP, Blood pressure; *BUN,* blood urea nitrogen; *Cr,* creatinine; *CRI,* constant-rate infusion; *CT,* computed tomography; *EBL,* estimated blood loss; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *MAP,* mean arterial pressure; *OTM,* oral transmucosal; *PEEP,* positive end-expiratory pressure; *PRN,* as needed; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein; *U/O,* urine output.

visceral pleura is in contact with the pleural lining of the thoracic wall. The pleurae of dogs contain smooth muscle fibers and a network of elastic fibers and are more delicate than in other domestic animals. The subserosa is composed of collagen and elastic fibers, which in the visceral pleura communicate with underlying lung. Fluid secreted into the pleural cavity is normally reabsorbed by lymphatics underlying the parietal pleura. Thickening of the pleura (i.e., fibrosing pleuritis) may prevent reabsorption of fluid, resulting in pleural effusion.

Fibers of the diaphragm arise from attachments on the ventral surface of the lumbar vertebrae, ribs, and sternum and radiate toward the tendinous center (Fig. 30.2). The diaphragm is composed of a central tendinous portion and an outer muscular portion. The costal part of the diaphragm attaches to the internal surface of the last few ribs, and the central portion extends cranially into the thoracic cavity.

SURGICAL TECHNIQUES

Treatment of pleural cavity disease varies, depending on the underlying etiology. For traumatic pneumothorax (see p. 933), intermittent needle thoracentesis may be sufficient in some animals to prevent dyspnea while the lung heals, but thoracostomy tubes are occasionally required. With some types of pleural effusion (i.e., pyothorax; see p. 947), tube thoracentesis and thoracic lavage are optimal in the primary treatment of many affected animals.

Needle Thoracentesis

Needle thoracentesis is performed with a small-gauge butterfly needle (No. 19 to No. 23) attached to a three-way stopcock and syringe, or an over-the-needle catheter attached to an extension

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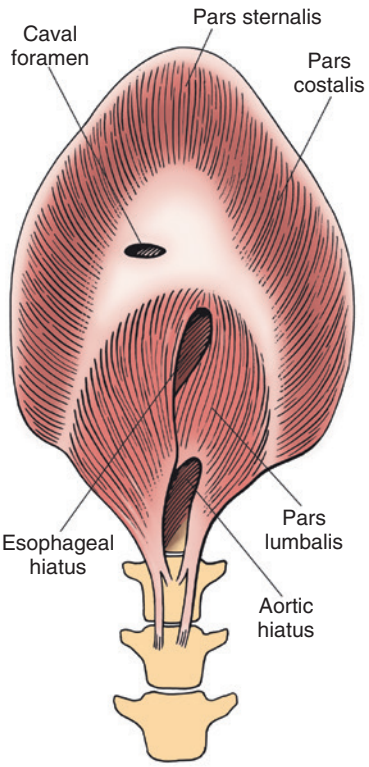


FIG. 30.2 Anatomy of the diaphragm.

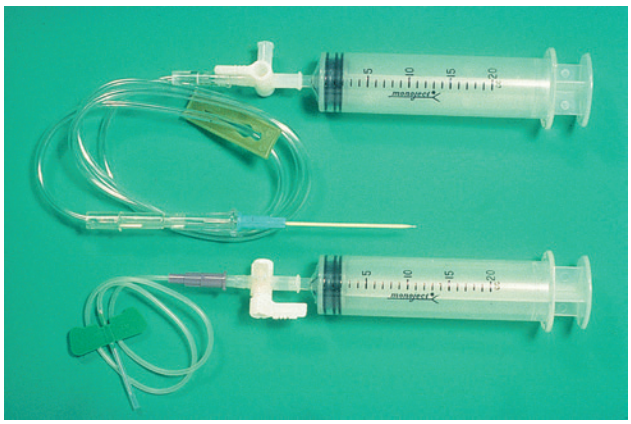


FIG. 30.3 A small-gauge butterfly needle *(bottom)* or an over-the-needle catheter attached to extension tubing *(top),* and a three-way stopcock and syringe are used for needle thoracentesis.

tubing, three-way stopcock, and syringe (Fig. 30.3). Be sure the needle is long enough to penetrate to the pleural space in large or obese animals. In such animals, a catheter rather than a butterfly needle may be needed. The appropriate site for thoracentesis should be selected based on the physical examination or, if available, radiographs or ultrasound. The mediastinum in dogs and cats is thin and permeable to fluids, and aspiration of one side of the thorax typically drains the contralateral hemithorax adequately. With some diseases, particularly chylothorax and pyothorax, unilateral effusions may occur as a result of thickening of the mediastinum associated with chronic inflammation (Fig. 30.4).



FIG. 30.4 Thoracic radiograph of a cat with unilateral pleural effusion that occurred secondary to chronic chylothorax. Notice the retraction of the left lung (right side of the image) away from the thoracic wall.

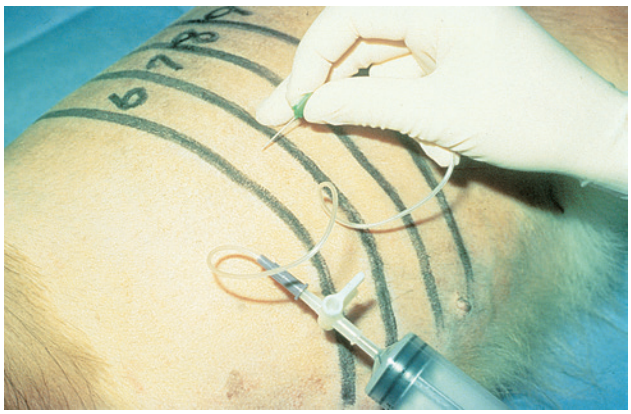


FIG. 30.5 Thoracentesis is performed at the sixth, seventh, or eighth intercostal space, near the level of the costochondral junction.

Unless there is a reason to go elsewhere, perform thoracentesis at the sixth, seventh, or eighth intercostal space, near the level of the costochondral junction (Fig. 30.5). Clip the selected site and perform a local anesthetic block if needed (this is rarely the case). Aseptically prepare the site, and introduce the needle into the middle of the selected intercostal space. Carefully avoid large vessels associated with the posterior aspect of the rib margins. Advance the needle into the pleural space. Aspirate fluid while the needle is being advanced to allow prompt recognition of the appropriate depth of needle placement. If you feel the heart beating or lungs rubbing across the tip of the needle, withdraw the needle and reassess the situation. With the bevel of the needle facing inward, orient the needle against the rib cage to prevent damage to the lung surface. Gently aspirate fluid and place 5-mL samples in an ethylenediaminetetraacetic acid (EDTA) tube and a clot tube for a cell count and biochemical parameters, respectively. Also, make six to eight

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direct smears for cytologic evaluation. Submit samples for aerobic and anaerobic cultures.

Thoracostomy Tube Placement

Incorrectly placed or improperly managed thoracostomy tubes are extremely dangerous. If precautions are taken to ensure that the animal cannot remove the tube prematurely or to prevent the animal from chewing on the tube, a pneumothorax should not occur. Thoracostomy tubes simplify the management of some animals with pleural effusion or pneumothorax. Determine which side to place the thoracostomy tube by evaluating the radiographs. Occasionally, bilateral thoracostomy tubes may be necessary; however, most dogs and cats have a mediastinum permeable to fluid or air, allowing drainage of both hemithoraces through a single tube. The exception to this may be in chylothorax or pyothorax (discussed previously).

Equipment needed for a tube thoracostomy include a thoracostomy tube, an apparatus to connect the tube to a syringe or to a continuous suction bottle, and a device to collect the drained material (syringe or collecting bottle). Large-bore commercially available tubes are usually made of polyvinyl chloride or silicone rubber and are less reactive than red rubber feeding tubes. Commercial tubes come with a metal stylet that simplifies tube placement but may increase the risk of perforating lung tissue compared with red rubber feeding tubes. The red rubber feeding tubes are usually inserted using a large hemostat or Carmalt clamp. Commercial thoracostomy tubes come in various sizes ranging from 14 to 40 Fr. Both large and small-bore thoracostomy tubes have been advocated in the veterinary literature. When using large-bore tubes, the size of the thoracostomy tube should generally approximate the diameter of the mainstem bronchus; smaller tubes may be adequate for removing air, whereas larger tubes may be required with more viscous effusions (Box 30.3). If a commercial tube is used, attach it by means of a tube adaptor (e.g., five-in-one connector, Christmas tree adapter) to either a three-way stopcock or the tubing from a continuous suction device. The ends of red rubber feeding tubes can be cut to accommodate a three-way stopcock; attaching these tubes to a continuous suction device is generally not recommended because of their tendency to collapse (Box 30.4). Small tubes (14 and 12 gauge), such as the MILA catheter, are placed using a guide wire with sedation and a local block (see later). Advantages of these tubes over larger tubes may be fewer insertional and infectious complications when compared with larger tubes. Potential complications of thoracostomy tube placement (large or small bore) include pneumothorax, kinking or malpositioning of the tube, pyothorax, hemorrhage from intercostal vessels, leakage around the catheter, and damage to or premature removal of the tube by the animal. Pneumothorax, kinking, and malpositioning are the most common complications associated with the use of small-bore catheters.

A subcutaneous vascular access port (VAP) attached to intrathoracic drain tubing is an effective means of removing pleural effusion in dogs where chronic removal (months to years) may be necessary. The VAP may be attached to a regular thoracostomy tube or to a Jackson-Pratt drain tube (Fig. 30.6). Pleural access ports, such as the PleuralPort device, have been reported in the management of dogs with recurrent spontaneous pneumothorax and pleural effusion.[2,3] In a study of 10 animals with pleural effusion in which PleuralPorts were used (nine had chylous effusion; one dog had pleural carcinomatosis), four animals developed complications (obstruction in three animals, pneumothorax in one cat).[3] Excluding one cat that developed pneumothorax and was euthanized, median duration of port function was 20 days (range, 1–391 days), and median duration of port implantation

BOX 30.3 Guidelines for Estimating Trocar and Large-Bore Thoracostomy Tube Size

Cats and Dogs <7 kg
14–16 Fr

Dogs 7–15 kg
18–22 Fr

Dogs 16–30 kg
22–28 Fr

Dogs >30 kg
28–36 Fr

BOX 30.4 Key Points When Placing Trocar and Large-Bore Thoracostomy Tubes

- When placing additional holes in commercial tubes, make sure that the last hole is through the radiopaque line.
- Start the thoracostomy tube dorsally rather than midthorax to minimize fluid or air leakage around the tube.
- Firmly grasp the tube 1–2 cm above the body wall when inserting the tube.
- Clamp the tube before removing the stylet (trocar) to prevent pneumothorax.
- Securely fasten all connectors to the tube to prevent inadvertent dislodgment.

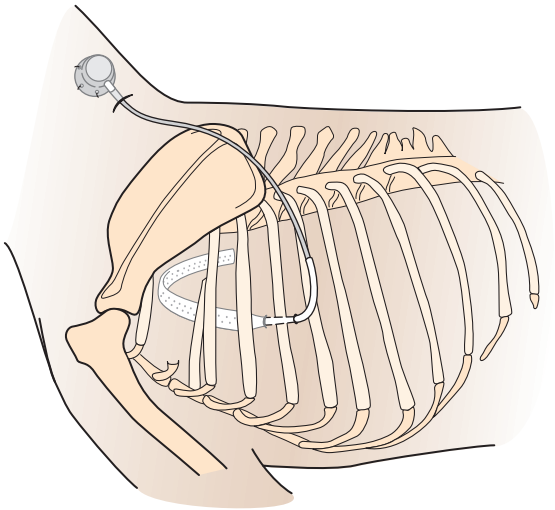


FIG. 30.6 Placement of a vascular access port and Jackson-Pratt drain for treatment of pleural effusion in a dog (views from the left side). Notice the cervical subcutaneous location of the port and the intrathoracic location of the Jackson-Pratt drain. (From Cahalane AK, Flanders JA, Steffey MA, Rassnick KM. Use of vascular access ports with intrathoracic drains for treatment of pleural effusion in three dogs. *J Am Vet Med Assoc.* 2007; 230(4):527–531.)

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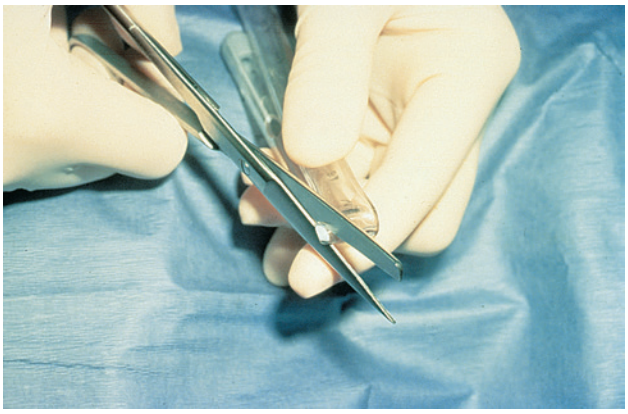


FIG. 30.7 For thoracostomy tube placement, make additional holes in the tube by bending it and removing a notch with a pair of sterile scissors.

was 391 days (range, 6–723 days). No significant port migration, irritation, or infection of the device was reported in this study.

Trocar and Large-Bore Thoracostomy Tubes

Clip and prepare the lateral thorax for aseptic surgery. To allow sufficient drainage, place additional holes in the tube by bending the tube and removing a notch with a pair of sterile scissors (Fig. 30.7). Make sure that the holes are no larger than one-third the circumference of the tube. Be certain that all the holes will easily fit within the thoracic cavity. If using a commercial tube with a radiopaque line, place the last hole through the line to allow identification of its position on a thoracic radiograph. Make a small skin incision in the dorsal one-third of the lateral thoracic wall at the level of the 10th or 11th intercostal space. Advance the tube subcutaneously in a cranioventral direction for three to four intercostal spaces, and introduce the tube through the muscle and pleura using the stylet or a large hemostat. When using a trocar tube, firmly grasp the tube 1 to 2 cm from the body wall with one hand while using the other hand to "pop" the tube through the intercostal musculature and pleura (Fig. 30.8). This prevents the tube from being inadvertently pushed farther into the thorax than anticipated, thereby damaging the lung or other thoracic structures. *Feed the tube in a cranioventral direction to a predetermined point; before completely removing the trocar, clamp the tube with a hemostat. For added safety when the chest cavity is not being suctioned, clamp the tube where it exits the body wall with a hemostat or tube clamp device (Fig. 30.9).* The latter device is preferred as it will not damage the tube and is more comfortable for the patient than a hemostat. *Place a purse-string suture in the skin around the tube (do not enter the lumen of the tube), and leave both ends of the suture long. Use this suture to make a Chinese finger-trap or Roman sandal suture (Fig. 30.10). Connect the thoracostomy tube to a three-way stopcock to increase the ease of thoracic drainage. Use a tube adapter or female Luer-Lok (with small tubes) between the tube and the three-way stopcock to ensure an air-tight seal. Use suture to secure the tube to the connecting devices so that they are not inadvertently dislodged, resulting in a pneumothorax (Fig. 30.11). Verify appropriate placement of the chest drain radiographically (Fig. 30.12) before covering it with a loose bandage.* In selected cases (e.g., multiple adhesions, loculated fluid), it may be advantageous to place the thoracostomy tube under thoracoscopic guidance.

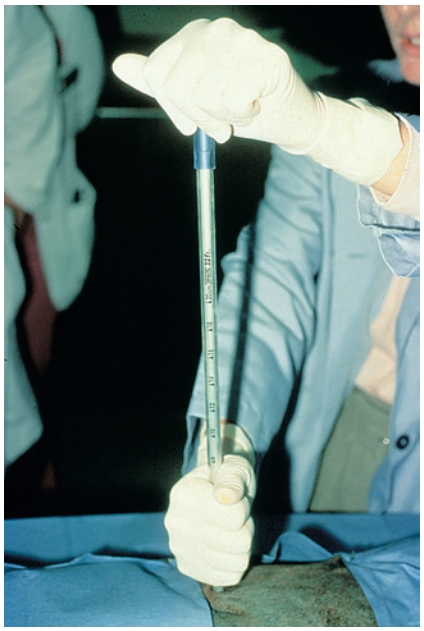


FIG. 30.8 When using a trocar tube, firmly grasp the tube 2 to 4 cm from the body wall with one hand while using the other hand to pop the tube through the intercostal musculature and pleura. Be sure that the hand holding the tube has a firm grasp on the tube; failing to do so may result in the tube inadvertently being pushed too far into the thoracic cavity and puncturing the lungs or heart.

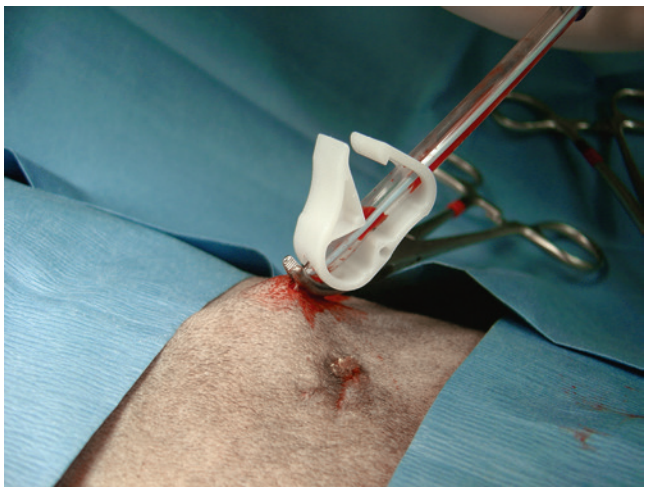


FIG. 30.9 For added safety when the thoracic cavity is not being suctioned, clamp the tube where it exits the body wall with a hemostat or tube clamp (preferred).

Small-Bore Wire-Guided Thoracostomy Tubes

Clip and prepare the lateral thorax for aseptic surgery. Make a small skin incision in the dorsal one-third of the lateral thoracic wall at the level of the 9th or 10th intercostal space (Fig. 30.13A). Tunnel the introducer catheter to the seventh or eighth intercostal space and insert it at the cranial aspect of the rib into the thoracic cavity (Fig. 30.13B–C and Video 30.1). Advance the introducer into the thorax over the stylet and thread a J-wire through the catheter (Video 30.2). Advance the wire in a cranioventral direction until resistance is encountered (Fig. 30.13D). Remove the catheter over

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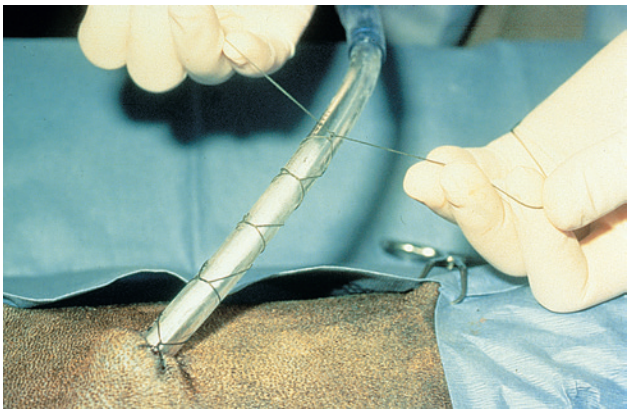


FIG. 30.10 Secure the tube with a Chinese finger-trap or Roman sandal suture.

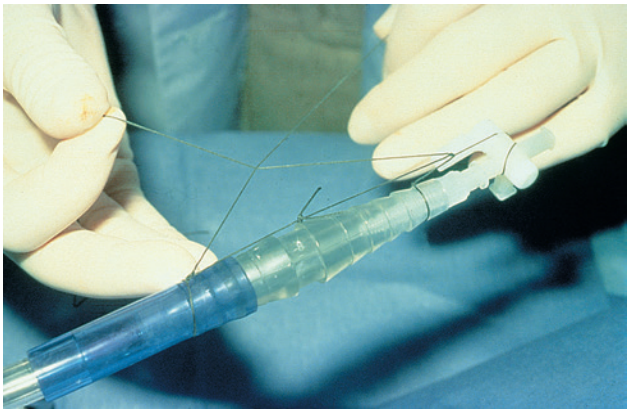


FIG. 30.11 Use suture to secure the tube to the connecting devices so that they are not inadvertently dislodged.

the guide wire, leaving the guide wire in place. Insert a small-bore catheter into the thoracic cavity over the guide wire (Video 30.3). Gently aspirate the drain to assess accurate placement. Secure the drain to the skin through the suture holes on the catheter. If the tube cannot be fully inserted into the thoracic cavity (e.g., in cats or small dogs), secure the external portion of the tube to the skin using a Chinese finger-trap suture (see Fig. 30.10).

If using a VAP, the VAP-drain combination may be placed via a regular intercostal thoracotomy at the time of surgical exploration of the thoracic cavity or via a small mini-thoracotomy (see Fig. 30.6).

Drainage may be either intermittent or continuous. Generally, intermittent pleural drainage is adequate; however, in some situations continuous suction is preferable (e.g., large volumes of air, pleurodesis). Heimlich valves should be used only in medium to large dogs because small dogs and cats may not develop sufficient expiratory pressure for effective drainage. In addition, these valves are prone to malfunction if fluid is aspirated into the apparatus. "Milking" or "stripping" of thoracostomy tubes to prevent obstruction of the tube by clots has been recommended in the veterinary literature; however, these techniques generate high intrapleural pressures and may cause pulmonary damage.

Thoracostomy Tube Removal

Because the tube is a foreign body, it will cause fluid to be produced in most animals (generally 2.2 mL/kg of body weight per day). With pleural effusion, tubes can generally be removed when the volume of fluid is compatible with that which can be managed by intermittent thoracentesis. The tube can be removed in patients with pneumothorax once negative pressure has been achieved for 12 to 24 hours. *Culture the end of the tube after removal if the tube has been in place for several days or if the animal shows signs of infection. Close the skin incision with one or two simple interrupted sutures.*

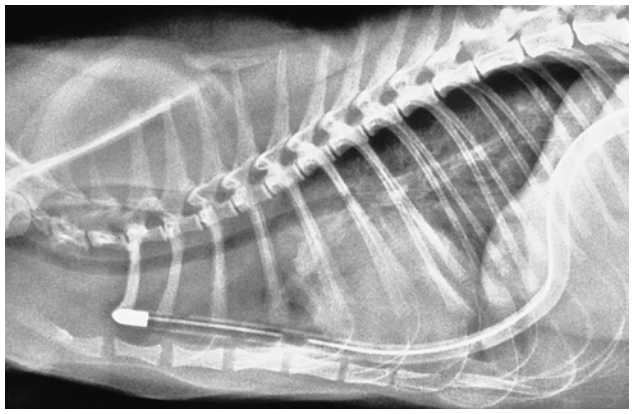


FIG. 30.12 Verify appropriate placement of the thoracostomy tube radiographically before covering it with a loose bandage.

Continuous Thoracic Suction

If fluid accumulates so quickly that intermittent drainage is not practical, continuous suction may be used. It is also commonly used after open-heart surgery to monitor thoracic bleeding. Two-bottle and three-bottle systems and commercial suction units are available for veterinary use and are economical and simple to use. A continuous 10- to 15-cm negative pressure on the thorax effectively aspirates pneumothorax, increasing the likelihood of spontaneous sealing of large pulmonary defects. Slightly higher pressures may be necessary (up to 20 cm H_2O) when viscous fluid is being drained. *Connect the thoracostomy tube to a commercial continuous suction device (Fig. 30.14 and Video 30.4). If a commercial continuous suction device is not available, use a three-bottle system. Connect the thoracostomy tube to a bottle that serves as an underwater seal (filled with 2–3 cm of sterile water), which in turn is connected to a suction bottle (also partly filled with water) attached to a suction device (Fig. 30.15). Vary the amount of suction by raising or lowering the level of water in the suction bottle.*

A rigid plastic vent tube open to room air allows air to be aspirated into the bottle as the vacuum is applied. A third bottle interposed between the thoracostomy tube and the underwater seal bottle collects fluid and prevents the level from rising in the underwater seal bottle as fluid is drained from the chest. The third bottle is unnecessary in animals with pneumothorax.

HEALING OF THE PLEURA

Healing or damaged pleurae is prone to adhesion formation in some species; however, dogs and cats seem resistant to chemical pleurodesis. They may have greater pleural fibrinolytic capacity than other species (e.g., rabbits or humans). Fibrosing pleuritis has been reported in dogs and cats secondary to prolonged exudative or blood-stained effusions. In animals with fibrosis,

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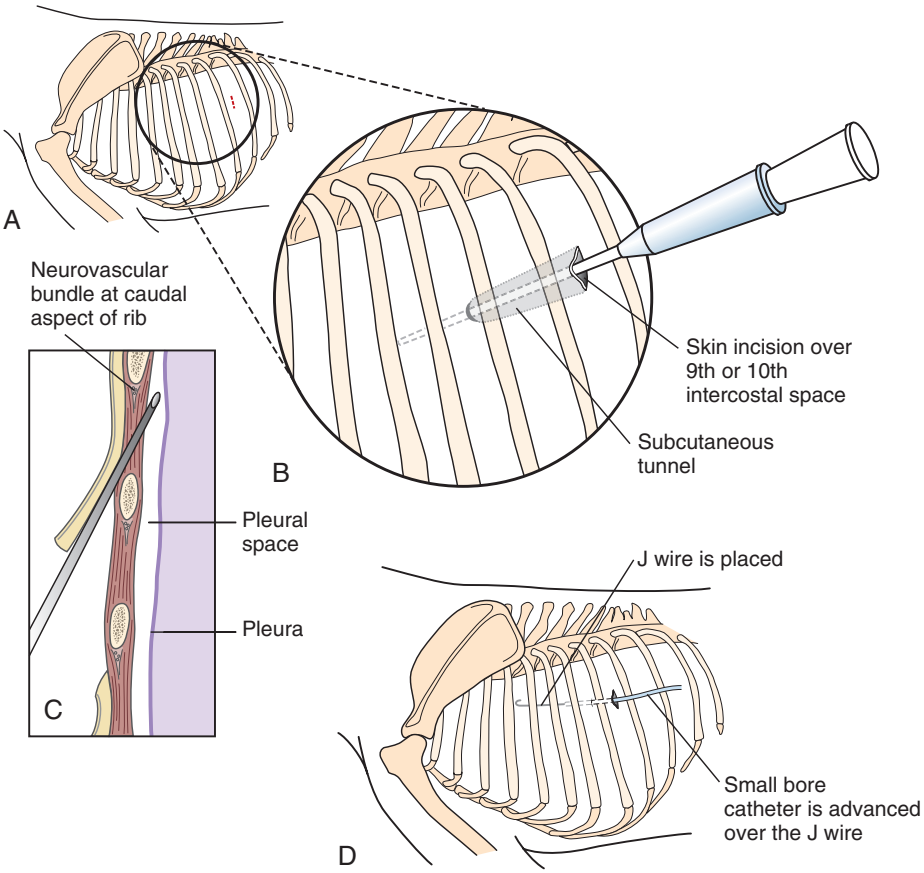


FIG. 30.13 Placement of a small-bore guide wire catheter for thoracic drainage.



FIG. 30.14 Commercial continuous suction device.

the pleura is thickened by diffuse, fibrous tissue that restricts normal pulmonary expansion (the lungs do not adhere to the body wall in these patients; Fig. 30.16). Exudates are characterized by a high rate of fibrin formation and degradation because chronic inflammation induces changes in the morphologic features of mesothelial cells that cause increased permeability, desquamation of mesothelial cells, and triggering of both pathways of the coagulation cascade. The desquamated mesothelial cells have also been shown to produce type III collagen in cell culture, promoting fibrosis. Additionally, the chronic presence of pleural fluid might lead to impaired fibrin degradation.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Trocar thoracostomy tubes (e.g., Teleflex, Redax, or Argyle thoracic trocar catheters) and continuous suction devices (e.g., Pleur-Evac or Thora-Seal chest drainage systems) (see Fig. 30.14) may be purchased from several commercial sources. Tubing clamps, tube adaptors, and small-bore wire-guided catheters may be purchased from commercial sources.

POSTOPERATIVE CARE AND ASSESSMENT

If dyspnea persists after needle thoracentesis or thoracostomy tube placement, oxygen therapy (nasal insufflation or oxygen cage) may be beneficial (see Chapter 4). Thoracic radiographs should be taken to assess fluid or air removal and/or to evaluate the position of the thoracostomy tube. Animals with thoracostomy tubes should be monitored continually to prevent iatrogenic dislodgment or damage to the tube or connectors, resulting in pneumothorax (discussed below under Complications). Care should be exercised when handling tubes to prevent thoracic contamination. Thoracostomy tubes should be aspirated gently so that pulmonary tissue is not suctioned into the tube drainage ports.

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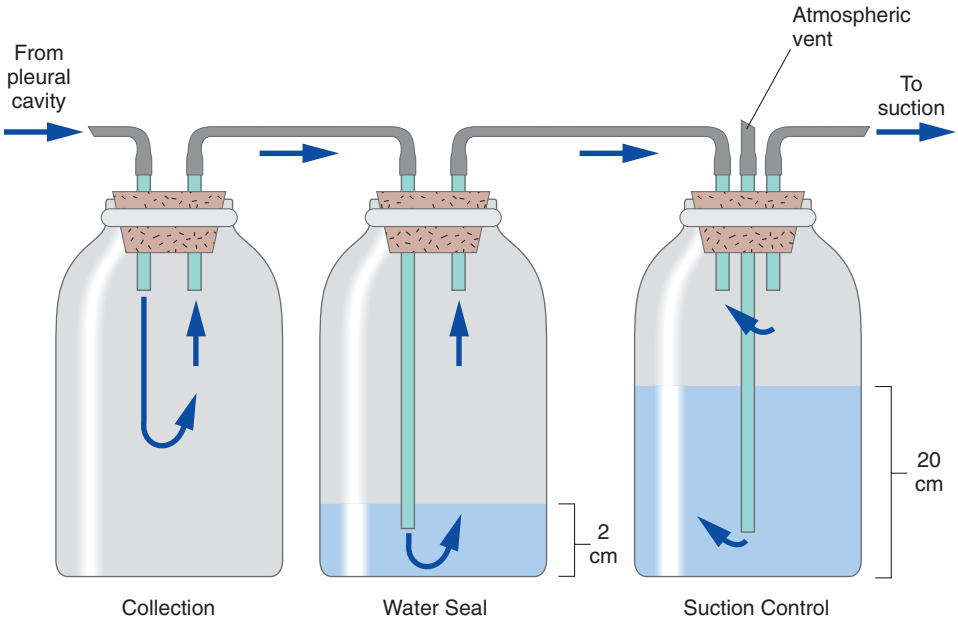


FIG. 30.15 Three-bottle system for continuous pleural drainage.

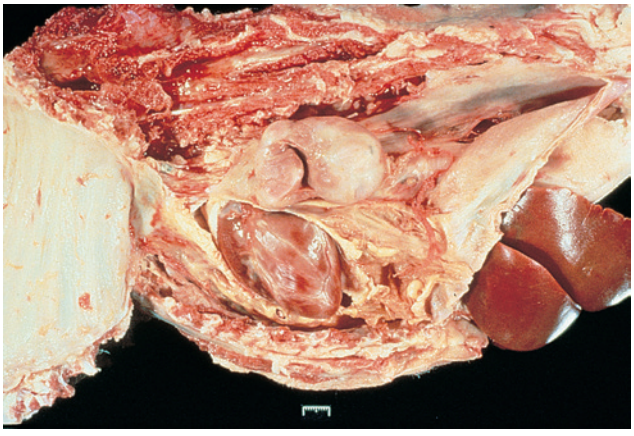


FIG. 30.16 Necropsy specimen of a dog with chronic chylothorax and severe fibrosing pleuritis. Note the small, consolidated lungs and the thickened pleura.

COMPLICATIONS

Although lung penetration and damage are possible with needle thoracentesis, risk is minimal if proper technique is used. The major complication associated with thoracostomy tubes is pneumothorax caused by damage to the tube by the patient (e.g., biting or scratching) or loosening of the connections of the tube to the adapters. Risk of these complications can be minimized by placing a clamp close to the tube's exit site (see Fig. 30.9), by securing the tube to the adapters (see Fig. 30.11), and by proper bandaging of the chest and tube. Constant surveillance of animals with a thoracostomy tube is recommended. Other complications associated with thoracostomy tube placement are rare but include lung perforation, empyema, laceration of an intercostal vessel, and pulmonary injury caused by aspiration of a portion of lung into a tube drainage port. Risk of lung perforation is related to the type of tube placed and underlying pleuropulmonary disease. If needle thoracentesis or ultrasonography suggests that the fluid is severely loculated or that extensive adhesions are present, surgical or thoracoscopic placement of the thoracostomy tube may be advisable.

SPECIAL AGE CONSIDERATIONS

Surgical correction of respiratory abnormalities in young animals requires special attention to their anesthetic requirements (see p. 722). Diaphragmatic hernia repair is commonly performed in young animals because they are prone to trauma that produces such lesions. Peritoneopericardial diaphragmatic hernias (PPDHs) are usually diagnosed at a young age (<1 year), and concurrent cardiac abnormalities may be present, complicating the anesthetic management of these patients (see p. 931). Geriatric animals may have severe, concurrent underlying pulmonary or cardiac conditions complicating management of pleural cavity disease, and careful monitoring is necessary.

SPECIFIC DISEASES

TRAUMATIC DIAPHRAGMATIC HERNIA

DEFINITION

A **diaphragmatic hernia** occurs when the continuity of the diaphragm is disrupted such that abdominal organs can migrate into the thoracic cavity.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Diaphragmatic hernias are commonly recognized by small animal clinicians and may be congenital or occur secondary to trauma. Congenital pleuroperitoneal hernias are seldom diagnosed in small animals because many affected animals die at birth or shortly thereafter (discussed later under Peritoneopericardial

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Diaphragmatic Hernia). Most diaphragmatic hernias in dogs and cats are caused by trauma, particularly motor vehicle accidents. The abrupt increase in intraabdominal pressure accompanying forceful blows to the abdominal wall causes the lungs to rapidly deflate (if the glottis is open), producing a large pleuroperitoneal pressure gradient. Alternately, the pressure gradient that occurs between the thorax and the abdomen may cause the diaphragm to tear. The tears occur at the weakest points of the diaphragm, generally the muscular portions. Location and size of the tear or tears depend on the position of the animal at the time of impact and location of viscera. Traumatic diaphragmatic hernias are often associated with significant respiratory embarrassment; however, chronic diaphragmatic hernias in asymptomatic animals are not uncommon. Diaphragmatic hernias may also occur in animals with connective tissue disorders or other diseases where structural integrity of the diaphragm or its attachments is compromised.

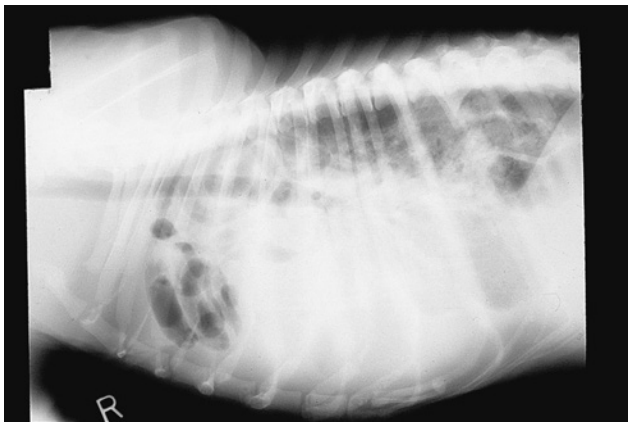


FIG. 30.17 Lateral thoracic radiograph of a dog with a diaphragmatic hernia. Note the air-filled intestinal loops in the thoracic cavity.

DIAGNOSIS

Clinical Presentation

Signalment

There is no breed predisposition for traumatic diaphragmatic hernias. Young males have historically been thought to be more commonly affected; however, more recent studies have not identified a sex predilection.

History

Duration of diaphragmatic hernias may range from a few hours to years. Many (15%–25%) are diagnosed weeks after the injury. The animals may be presented in shock acutely after the trauma (discussed later), or the hernia may be an incidental finding. Animals sustaining trauma often suffer from associated injuries (e.g., fractures). With a chronic diaphragmatic hernia, clinical signs are most often referable to either respiratory (e.g., dyspnea, exercise intolerance) or gastrointestinal systems (e.g., anorexia, vomiting, diarrhea, weight loss, pain after ingestion of food); or they may be nonspecific (e.g., depression). Many animals with chronic hernias are not dyspneic at the time of diagnosis.

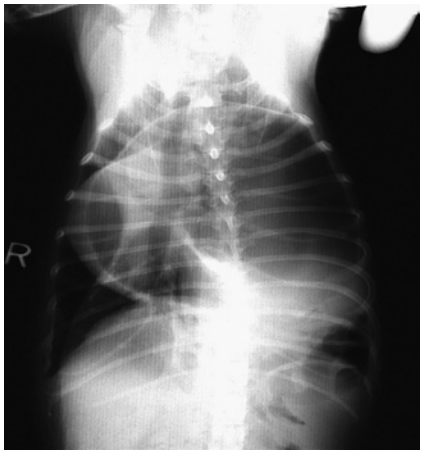


FIG. 30.18 Dorsoventral thoracic radiograph of a dog with a dilated, herniated stomach.

Physical Examination Findings

Animals with recent traumatic diaphragmatic hernias frequently are in shock when presented for treatment; therefore clinical signs may include pale or cyanotic mucous membranes, tachypnea, tachycardia, and/or oliguria. Cardiac arrhythmias are common and associated with significant morbidity. Other clinical signs depend on which organs have herniated and may be attributed to gastrointestinal, respiratory, or cardiovascular systems. The liver is the most commonly herniated organ, a condition often associated with hydrothorax caused by entrapment and venous occlusion; however, any organ can be herniated, including the kidney.

Diagnostic Imaging

Definitive diagnosis of pleuroperitoneal diaphragmatic hernia is usually made by radiography or ultrasonography. If significant pleural effusion is present, thoracentesis may be necessary for diagnostic radiographs. Radiographic signs of diaphragmatic hernia may include loss of the diaphragmatic line, loss of the cardiac silhouette, dorsal or lateral displacement of lung fields, presence of gas or a barium-filled stomach or intestines in the thoracic cavity, pleural effusion, and/or failure to observe the stomach or liver in the abdomen (Figs. 30.17 and 30.18). It may be difficult to diagnose diaphragmatic hernias radiographically if only a small portion of the liver is herniated. Ultrasound examination of the diaphragmatic silhouette may help when herniation is not obvious radiographically (i.e., hepatic herniation, pleural effusion). Ultrasonography accurately demonstrates diaphragmatic hernia in the majority of patients. Ultrasonography may be particularly difficult if severe pulmonary contusions are present (which make the lungs appear ultrasonographically similar to the liver), if only the omentum is herniated, or if adhesions between the liver and lung are present. Care should be taken not to mistake a normal mirror-image artifact (usually seen as apparent liver parenchyma on the thoracic side of the diaphragmatic line) for herniated liver. If ultrasound is not diagnostic, *positive contrast celiography* has been recommended; however, contrast CT is now more commonly performed.

Laboratory Findings

Specific laboratory abnormalities are uncommon. Serum alanine aminotransferase and serum alkaline phosphatase values may be elevated in cases of liver herniation.

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DIFFERENTIAL DIAGNOSIS

Any disorder causing respiratory abnormalities (e.g., pleural effusion, pneumothorax, pneumonia) should be a differential diagnosis for traumatic diaphragmatic hernia. The concurrent presence of pleural effusion in many animals with liver herniation may make diagnosis of diaphragmatic hernia difficult (discussed previously).

MEDICAL MANAGEMENT

If the animal is dyspneic, oxygen should be provided by face mask, nasal insufflation, or oxygen cage (see Chapter 4). Positioning the animal in sternal recumbency with the forelimbs elevated may help ventilation. If moderate or severe pleural effusion is present, thoracentesis (see p. 920) should be performed. Fluid therapy and antibiotics should be given if the animal is in shock.

SURGICAL TREATMENT

Chronic diaphragmatic hernias may have a higher mortality than acute diaphragmatic hernias; however, the prognosis with both groups is good to excellent with surgery (discussed later under Prognosis). If pulmonary contusions are severe, surgical repair of diaphragmatic hernias should be delayed until the patient's condition has been stabilized; however, herniorrhaphy should not be delayed unnecessarily. Animals with gastric herniation should be evaluated carefully for gastric distention and should be operated on as soon as they can safely be anesthetized, because acute gastric distention within the thorax may cause rapid, fatal respiratory impairment. Rarely, large diaphragmatic defects require a muscle transposition (e.g., rectus abdominis muscle pedicle flap) or insertion of mesh.[4]

NOTE Do not delay surgery unnecessarily in animals in stable condition. If the stomach has herniated into the thoracic cavity, perform surgery as soon as possible (i.e., on an emergency basis).

NOTE Be prepared to perform organ resection (e.g., partial lobectomy, intestinal resection and anastomosis) if the hernia is chronic. Be aware that reexpansion pulmonary edema may occur in patients with chronic diaphragmatic hernias, but it is rare.

Preoperative Management

Prophylactic antibiotics should be given 30 minutes before making the surgical incision in animals with hepatic herniation. Toxins may be released into circulation in cases of hepatic strangulation or vascular compromise. Premedicating such patients with steroids may be beneficial. An ECG should be performed on all trauma patients before surgery.

Anesthesia

Because of the animal's already compromised ventilation, drugs with minimal respiratory depressant effects should be used (Table 30.4). Premedication in severely distressed patients may not be advisable. Patients that have minimal dyspnea can be given a benzodiazepine (e.g., midazolam, diazepam). Supplementing oxygen before induction improves myocardial oxygenation, and preoxygenation for 3 to 5 minutes allows for a safer induction. Chamber or mask induction should be avoided in animals with diaphragmatic hernia (see p. 918). Gentle and quiet handling of the animal may allow surgical clipping prior to induction, minimizing the delay from time of induction to the surgical incision.

NOTE Have all airway devices, anesthesia monitoring equipment, anesthesia machines, and emergency and induction drugs ready and immediately available prior to the induction of anesthesia in these patients.

Injectable anesthetics allowing rapid intubation are preferred (see also p. 884). Inhalation anesthetics should be used for maintenance of anesthesia; analgesia needs to be provided to avoid high inhalant levels that can lead to hypotension. IPPV should be performed, and high inspiratory pressures should be avoided if possible. However, once the animal is placed in a dorsal position causing a shift of abdominal organs into the thorax, higher pressures may be needed to accomplish ventilation. It is important to understand that with severe compression of the lungs, the alveoli are collapsed and the bronchioles and bronchi also may be compressed. In this situation, the airway structures experiencing the higher ventilator pressures are primarily the bronchial tree. Once the thorax is opened, allowing room for pulmonary expansion, peak airway pressures should immediately be decreased and the lungs slowly expanded. This slow expansion helps to reduce the chance of reexpansion pulmonary edema (RPE; see p. 887). Nitrous oxide is contraindicated in patients with diaphragmatic hernia (see p. 918). Drugs such as methylprednisolone may be beneficial for preventing RPE in animals with chronic diaphragmatic hernia. Adequate pain management is especially important in respiratory compromised patients. Postoperatively, these patients need to be able to take slow deep breaths; however, a patient in pain will take rapid and shallow breaths. Epidural, intercostal nerve blocks, incisional local block, and intravenous opioids may be used in combination to minimize pain (see Box 30.2 and Table 30.3).

Positioning

The animal is placed in dorsal recumbency for a midline abdominal incision. The entire abdomen and caudal one-half to two-thirds of the thoracic cavity should be prepared for aseptic surgery. Because acute ventilatory compromise may occur during positioning, these animals should be carefully monitored.

NOTE Be sure to prepare an adequate area so that the incision can be extended into the caudal sternum for thoracic access if necessary.

SURGICAL TECHNIQUE

Make a ventral midline abdominal incision; if greater exposure is needed, extend the incision cranially through the sternum (Video 30.5). Replace the abdominal organs in the abdominal cavity (if necessary, enlarge the diaphragmatic defect). If adhesions are present, dissect the tissues gently from the thoracic structures to prevent pneumothorax or bleeding. With chronic hernias, debride the edge of the defect before closure. Close the diaphragmatic defect in a simple continuous suture pattern. If the diaphragm is avulsed

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TABLE 30.4 Anesthetic Considerations for the Diaphragmatic Hernia Patient

Preoperative Considerations

Associated conditions	• May be very young with other congenital defects • If older patient, may have evidence of old trauma • If recent trauma, see Table 30.3
Bloodwork	• HCT • Electrolytes • BUN • Cr • TP • +/– Blood gas • +/– BG
Physical exam	• Absence of pulmonary sounds ventrally with auscultation; may hear bowel sounds in the thorax; usually unilateral unless mediastinal/pericardial involvement then may be bilateral; muffled heart sounds
Other diagnostics	• Blood pressure • ECG • Radiographs (thoracic +/– abdominal) • +/– CT • Ultrasound • Perform thoracocentesis with care to avoid puncturing abdominal contents

Premedications	• Oxygen with flow-by or face mask
	• Avoid sedatives in depressed or dyspneic patients
	• In patients that are have minimal respiratory compromise, give:
	  • Midazolam (0.1–0.2 mg/kg IV, IM), *or*
	  • Diazepam (0.1–0.2 mg/kg IV)
	• Before induction, avoid all respiratory depressants, including but not limited to opioids, xylazine, medetomidine, and dexmedetomidine; avoid acepromazine, xylazine, medetomidine, and dexmedetomidine if the patient is hypotensive.
	• After a benzodiazepine is given but before induction, clip hair from abdomen and thorax; this can usually be done with patient standing or in lateral recumbency; do not place patient in ventrodorsal position until after intubation.

Intraoperative Considerations

Induction	• Preoxygenate for 3–5 min with face mask or flow-by oxygen
	• If normal cardiac function, give:
	  • Propofol (2–4 mg/kg IV) if sedated, *or*
	  • Propofol (4–8 mg/kg IV) if unsedated, *or*
	  • Alfaxalone (2–5 mg/kg IV)
	• If marked cardiac compromise, give:
	  • Etomidate (0.5–1.5 mg/kg IV); give after administering a benzodiazepine to avoid myoclonus

Maintenance	• Isoflurane or sevoflurane, *plus*
	  • Fentanyl (2–10 µg/kg IV PRN in dogs; 1–4 µg/kg IV PRN in cats) for short-term pain relief, *plus*
	  • Fentanyl CRI (1–5 µg/kg IV loading dose, then 2–30 µg/kg/h IV), *or*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Morphine[b] (0.1–1 mg/kg IV PRN in dogs; 0.1–0.2 mg/kg IV PRN in cats) if not hypotensive, *or*
	  • Oxymorphone (0.05–0.2 mg/kg IV, IM), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV PRN), *plus*
	  • Ketamine (low-dose 0.5–1 mg/kg IV), *or*
	  • Ketamine CRI (0.5 mg/kg IV loading dose, then 10 µg/kg/min IV)
	• If hypotensive (to keep MAP 60–80 mm Hg), give phenylephrine, ephedrine, dopamine, norepinephrine, vasopressin, or fluids as needed
	• Have surgeon ready to drape and make incision when positioning patient ventrodorsally
	• Ventilation parameters:
	  • SpO_2 >95%
	  • Small tidal volumes: 5–7 mL/kg
	  • Respiratory rates: 10–30
	  • Peak airway pressures: <12–15 mm Hg if possible
	• If unable to meet above parameters, higher peak pressures may need to be used initially; as abdominal organs are being removed from the thorax, drop peak airway pressures and tidal volumes immediately; slowly expand the lungs while manually ventilating and visually observing lung expansion. Consider positive end expiratory pressure.

Fluid needs	• 5–20 mL/kg/h to replace evaporative losses plus 3 × EBL; consider colloids for EBL replacement in 1:1 ratio

Continued

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TABLE 30.4 Anesthetic Considerations for the Diaphragmatic Hernia Patient—cont'd

Monitoring	• BP • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature • +/– Arterial line • U/O
Blocks	Epidural: • Morphine (0.1 mg/kg preservative free), *or* • Buprenorphine (0.003–0.005 mg/kg diluted in saline), *plus* Local anesthetics (see Box 30.2 and Chapter 13, p. 144): • Intercostal nerve blocks with local anesthetics if thoracotomy performed and thoracostomy tube placed, *plus* • Incisional block with local anesthetic
Postoperative Considerations	
Analgesia	• Fentanyl CRI (1–10 µg/kg IV loading dose, then 2–20 µg/kg/h IV), *or* • Morphine[b] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.1–0.2 mg/kg IV *or* 0.1–0.5 mg/kg IM q1–4h in cats) if not hypotensive, *or* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats), *or* • Hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs), *or* • Oxymorphone (0.1–0.2 mg/kg IV, IM), *or* • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM q4–8h or 0.01–0.02 mg/kg OTM q6–12h in cats), *plus* • +/– Ketamine CRI (2 µg/kg/min IV); if loading dose not used, give 0.5 mg/kg IV before CRI
Monitoring	• SpO_2 • Blood pressure • ECG • HR • Respiratory rate • Temperature • U/O • Thoracostomy tubes
Bloodwork	• +/– BG • HCT • TP • Electrolytes • Blood gas • +/– Albumin
Estimated pain score	Severe

[a]Monitor for hyperthermia in cats.
[b]Give slowly to prevent histamine release.
[c]Buprenorphine is a better analgesic than morphine in cats.
BG, Blood glucose; *BP,* blood pressure; *BUN,* blood urea nitrogen; *Cr,* creatinine; *CRI,* constant-rate infusion; *CT,* computed tomography; *EBL,* estimated blood loss; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *MAP,* mean arterial pressure; *OTM,* oral transmucosal; *PRN,* as needed; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein; *U/O,* urine output.

from the ribs, incorporate a rib in the continuous suture for added strength (Fig. 30.19). Remove air from the pleural cavity after closing the defect. If continued pneumothorax or effusion is likely, place a thoracostomy tube (see p. 922). Explore the entire abdominal cavity for associated injury (i.e., compromise of the vasculature to the intestine or splenic, renal, or bladder trauma), and repair any defects.

NOTE If the diaphragmatic defect is particularly large, synthetic material such as Silastic sheeting can be used to close it; however, this is seldom necessary.

An abdominal flap graft has been reported for repair of chronic diaphragmatic hernia in dogs. The graft is obtained from the peritoneum and transverse abdominal muscle caudal to the diaphragm. The graft is elevated, placed over the defect, and sutured to the diaphragm.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

To close the diaphragm, use either a nonabsorbable (e.g., polypropylene) or an absorbable (e.g., polydioxanone, polyglyconate) suture.

POSTOPERATIVE CARE AND ASSESSMENT

Patients should be monitored postoperatively for hypoventilation, and oxygen should be provided if necessary. RPE is a possible complication associated with rapid lung reexpansion after repair of a diaphragmatic hernia (see p. 887). Postoperative analgesics

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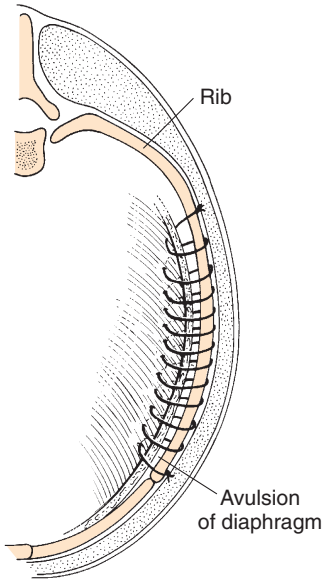


FIG. 30.19 To repair a diaphragm avulsed from the thoracic wall, incorporate a rib in the suture line.

should be provided (see Chapter 13; for dosages of opioids, see Table 13.2).

COMPLICATIONS

The most common complication after surgical repair of diaphragmatic hernias is pneumothorax, especially if the hernia is chronic and adhesions are present. RPE may occur in lungs that have been chronically collapsed (discussed previously).

PROGNOSIS

The prognosis is generally excellent if the animal survives the early postoperative period (i.e., 12–24 hours), and recurrence is uncommon with proper technique. In a 2017 study of 96 animals (79 dogs, 17 cats) with acute traumatic diaphragmatic hernias, 79.2% and 83.3% of the dogs and cats, respectively, survived to discharge.[5] Of the chronic cases, 100% of the cats ($n = 5$) and 80.6% ($n = 25$) of the dogs survived to discharge. Interestingly, an association between the time of the trauma and when surgery was performed did not appear to affect survival, nor was the time between trauma and admission or admission and surgery associated with survival. Increased duration of anesthesia and increased length of time for the surgical procedure were associated with increased mortality. Animals with concurrent injuries were also less likely to survive. Perioperative oxygen dependence was associated with increased mortality.

PERITONEOPERICARDIAL DIAPHRAGMATIC HERNIA

DEFINITION

A **PPDH** occurs when a congenital communication exists between the abdomen and the pericardial sac. Synonyms include *pericardial diaphragmatic hernia* and *congenital hernia*. A **true diaphragmatic hernia** is a congenital diaphragmatic malformation that is distinct from PPDH and also from herniation of abdominal contents into the pleural cavity. In a true diaphragmatic hernia there is an incomplete diaphragmatic tear in which a direct communication between the pleural and peritoneal cavities is prevented by an intact serosa on the thoracic surface of the diaphragm.

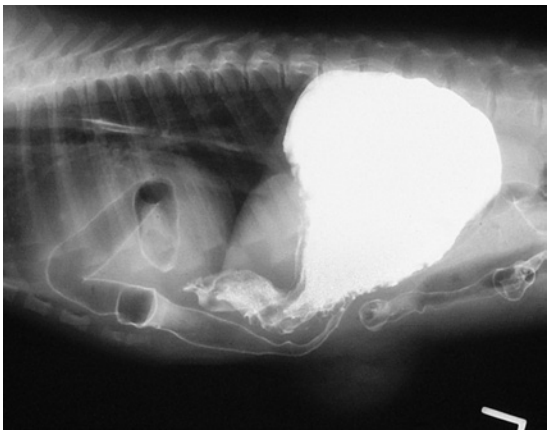


FIG. 30.20 Lateral thoracic radiograph of a dog with a peritoneopericardial diaphragmatic hernia. Note the cranial abdominal hernia.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

PPDHs are less commonly recognized by small animal clinicians than traumatic diaphragmatic hernias. Although PPDHs are often associated with respiratory embarrassment, asymptomatic PPDH is common. PPDH may occur as a result of trauma in humans (in whom the diaphragm forms one wall of the pericardial sac); however, these hernias are always congenital in dogs and cats because no direct communication exists between the pericardial and peritoneal cavities after birth. The most widely accepted theory regarding the embryogenesis of this defect is that the hernia occurs because of faulty development or prenatal injury of the septum transversum. This could be a result of a teratogen, genetic defect, or prenatal injury. True diaphragmatic hernias, although rare, have been reported in dogs.

Cardiac abnormalities and sternal deformities often occur concomitantly with PPDH. The combination of congenital cranial abdominal wall, caudal sternal, diaphragmatic, and pericardial defects has been reported in dogs, often associated with ventricular septal defects or other intracardiac defects (Fig. 30.20). It is not known if this condition is heritable; however, several breed predispositions have been recognized (discussed later). Polycystic kidneys have been reported in association with PPDH in cats.

NOTE If you identify a cranial abdominal wall defect in a young animal, consider that it may also have PPDH, a congenital cardiac abnormality, or both.

DIAGNOSIS

Clinical Presentation

Signalment

Although PPDH is congenital, it is not uncommon for the diagnosis to be made when the animal is middle-aged or older

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because clinical signs vary and may be intermittent. Weimaraners and cocker spaniels may be at increased risk. Domestic longhair and Himalayan cats may be predisposed. PPDH is one of the most common congenital cardiac defects diagnosed in cats 2 years of age and older. There is no sex predisposition in cats or dogs.

History

The clinical signs may be referable to the gastrointestinal, cardiac, or respiratory systems and include anorexia, depression, vomiting, diarrhea, weight loss, wheezing, dyspnea, exercise intolerance, and/or pain after eating, or the animal may be asymptomatic. Tachypnea and dyspnea are the most common presenting clinical signs in cats, whereas gastrointestinal signs are more prevalent in dogs. Neurologic signs may occur as a result of hepatoencephalopathy. PPDH is often an incidental finding in both dogs and cats.[6]

Physical Examination Findings

Physical examination findings in animals with PPDH may include muffled heart sounds, ascites, murmurs caused either by displacement of the heart by visceral organs or by intracardiac defects, and concurrent ventral abdominal wall defects. The most commonly herniated organ is the liver, and associated pericardial effusion is common.

Diagnostic Imaging

A tentative diagnosis of PPDH may be made based on the history, clinical signs, and physical examination, but radiography, ultrasonography, or CT is essential for a definitive presurgical diagnosis. Box 30.5 lists the radiographic signs of PPDH (Fig. 30.21). Contrast studies (i.e., nonselective angiogram, barium contrast study) are rarely needed and should be undertaken only if a definitive diagnosis cannot be made on survey films (Fig. 30.22), or with ultrasound or advanced imaging. A distinct curvilinear radiopacity has been identified between the cardiac silhouette and the diaphragm on a lateral thoracic radiograph in cats with PPDH. This radiographic finding has been called the *dorsal peritoneopericardial mesothelial remnant*; however, it is not always apparent on radiographs of affected cats. Ultrasonography is useful because there is often discontinuity of the diaphragmatic outline and, more importantly, abdominal organs may be visualized in the pericardial sac. Hepatic herniation is usually evident. Echocardiography should be performed in animals with murmurs.

Laboratory Findings

Specific laboratory abnormalities are uncommon.

BOX 30.5 Radiographic Signs of Peritoneopericardial Diaphragmatic Hernia

- Enlarged cardiac silhouette
- Dorsal elevation of the trachea
- Overlap of the heart and diaphragmatic borders
- Discontinuity of the diaphragm
- Gas-filled structures in the pericardial sac
- Sternal defects
- Dorsal peritoneopericardial mesothelial remnant

DIFFERENTIAL DIAGNOSIS

The most common differential diagnoses for PPDH are pericardial effusion and cardiomegaly. Ultrasound and echocardiography are useful for distinguishing these abnormalities from PPDH.

MEDICAL MANAGEMENT

If the animal is dyspneic, oxygen should be provided by face mask, nasal insufflation, or an oxygen cage (see Chapter 4). Positioning the animal in sternal recumbency with the forelimbs elevated may help ventilation.

SURGICAL TREATMENT

Surgical repair should be performed as early as possible to reduce the likelihood of adhesions and to maximize the potential of having pliable skin, muscles, sternum, and rib cage, which facilitates closure of large defects. Early correction of PPDH may prevent acute decompensation and the possible development of

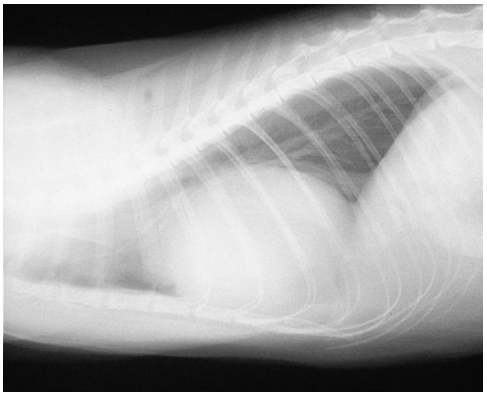


FIG. 30.21 Lateral thoracic radiograph of a cat with a peritoneopericardial diaphragmatic hernia. Note the large, globoid appearance of the cardiac silhouette.

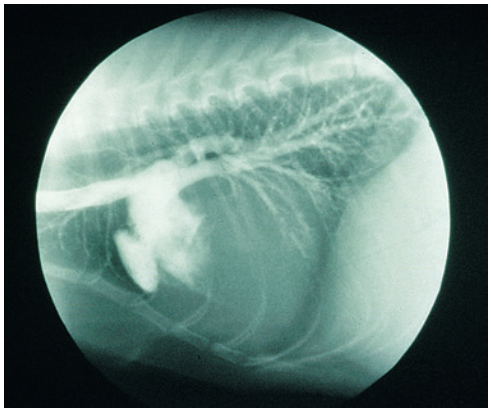


FIG. 30.22 Nonselective angiogram in a cat with a peritoneopericardial diaphragmatic hernia. Note the size of the heart in comparison with the cardiac silhouette. The liver is within the pericardial sac. (Courtesy M. Miller, VetMed, Phoenix AZ.)

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acute postoperative pulmonary edema (see p. 887). If the hernia is not diagnosed until the animal is older, conservative or surgical management may be used; however, owner satisfaction may be higher in operated animals than in animals managed conservatively. Some animals that are initially managed medically may have progression of clinical signs, necessitating surgical intervention or resulting in death.

Although supplementation with prosthetic materials, muscle flaps, autogenous grafts, or tissue-engineered materials is seldom necessary in dogs or cats, small intestinal submucosa (SIS) has been evaluated experimentally for repair of congenital diaphragmatic hernias where closure is difficult. Eight-ply SIS repair of diaphragmatic defects appears to be superior to four-ply SIS, as the former is more durable and persists for a longer period. Organ adherence will occur and appears to be necessary for neovascularization of the SIS composite.

Preoperative Management

Prophylactic antibiotics should be given 30 minutes prior to making the surgical incision in animals with hepatic herniation. In animals with hepatic strangulation or vascular compromise, repositioning of the liver into the abdominal cavity may cause a massive release of toxins into the bloodstream; premedicating such patients with steroids may be beneficial.

Anesthesia

See anesthesia under Traumatic Diaphragmatic Hernia (p. 928).

Positioning

The animal is placed in dorsal recumbency for a midline abdominal incision. The entire abdomen and caudal two-thirds of the thoracic cavity should be prepared for aseptic surgery.

SURGICAL TECHNIQUE

Make a ventral midline abdominal incision (Video 30.6). If greater exposure is needed, extend the incision cranially through the sternum. Enlarge the diaphragmatic defect if necessary, and replace the abdominal organs in the abdominal cavity. If adhesions are present, gently dissect the tissues from the thoracic structures, resecting or debriding necrotic tissue as necessary. Debride the edges of the defect and close in a simple continuous suture pattern. Do not close the pericardial sac. Remove air from the pericardial sac or pleural cavity or both after closing the defect. If continued pneumothorax or effusion is likely, insert a thoracostomy tube (see p. 922). Repair concomitant sternal or abdominal wall defects.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

To close the diaphragm, use either nonabsorbable (e.g., polypropylene) or absorbable (e.g., polydioxanone or polyglyconate) suture.

POSTOPERATIVE CARE AND ASSESSMENT

These patients should be monitored postoperatively for hypoventilation, and oxygen should be provided if necessary. RPE is a possible complication associated with rapid lung reexpansion after diaphragmatic hernia repair (see p. 887). Patients with PPDH may also have pulmonary hypoplasia, which contributes to the development of high intrapleural pressures and RPE. Postoperative analgesics should be provided (see Chapter 13 for dosages of opioids; also see Table 13.2). Transient hyperthermia may occur in cats following PPDH repair.

PROGNOSIS

If the animal survives the early postoperative period (i.e., 12–24 hours), the prognosis is excellent, and recurrence is uncommon with proper technique. The postoperative mortality rates of dogs with PPDH is approximately 10%; in cats, it may be less than 5%. Precise, controlled ventilation by use of a mechanical ventilator is recommended during anesthesia for animals undergoing surgical correction of PPDH. The prognosis is worse in patients with PPDH that have concurrent cardiac abnormalities. Although complications are rare in animals with PPDH, a pericardial cyst and constrictive pericarditis have each been reported in a cat after repair of a PPDH.[7,8]

PNEUMOTHORAX

DEFINITIONS

Pneumothorax is an accumulation of air or gas in the pleural space. **Traumatic pneumothorax** may be classified as open or closed. An **open pneumothorax** is one in which there is free communication between the pleural space and the external environment. With a **closed pneumothorax**, air accumulates because of leakage from the pulmonary parenchyma, bronchial tree, or esophagus. A **tension pneumothorax** occurs when a flap of tissue acts as a one-way valve so that there is a continuous influx of air into the pleural cavity on inspiration that does not return to the lung on expiration. **Spontaneous pneumothorax** occurs as a result of air leakage from the lung but without trauma as a precipitating cause. **Cysts** are closed cavities or sacs lined by epithelium that are usually filled with fluid or semisolid material. **Bullae** are nonepithelialized cavities produced by disruption of intraalveolar septa (Fig. 30.23 and Video 30.7). A **bleb** is a localized collection of air contained within the visceral pleura (see Fig. 30.23). For nontraumatic pneumothorax, the terms *spontaneous pneumothorax* and *idiopathic pneumothorax* have been used interchangeably.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Traumatic pneumothorax is the most common type of pneumothorax in dogs. It most often occurs as a result of blunt trauma (e.g., vehicular accidents, being kicked) that causes parenchymal pulmonary damage to the lung and a closed pneumothorax. When the thorax is forcefully compressed against a closed glottis, the lung or bronchial tree may rupture. In other cases, pulmonary parenchyma may be torn as a result of shearing forces on the lung. Pulmonary trauma occasionally results in the formation of subpleural blebs, similar to those seen with spontaneous pneumothorax (discussed later; Fig. 30.24). Open pneumothorax is less common but is often caused by trauma (e.g., gunshot, bite or stab wounds, lacerations that occur secondary to rib fractures). Immediate thoracic exploration of patients with severe thoracic trauma and pneumothorax may be warranted (see Fig. 30.1). Some penetrating injuries are called *sucking chest wounds* because large defects in the thorax wall allow an influx of air into the pleural space when the animal inspires. These large, open chest wounds may allow enough air to enter the pleural

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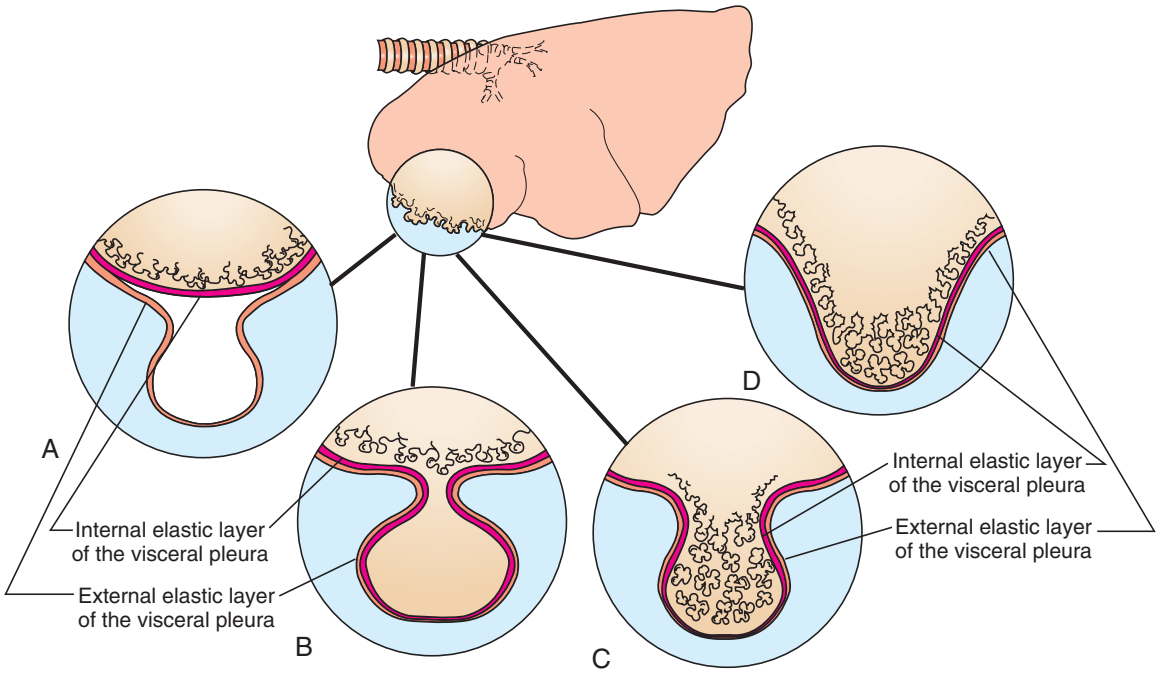


FIG. 30.23 Typical location and different forms of pulmonary blebs and bullae *(inset).* (A) Pulmonary bleb. (B) A thin-walled bulla with a narrow connection to the pulmonary parenchyma. (C) A subpleural bulla that is connected to the lung by a stalk of emphysematous tissue. (D) A large bulla that extends deep into the pulmonary parenchyma. (Modified from Lipscomb VJ, Hardie RJ, Dubielzig RR. Spontaneous pneumothorax caused by pulmonary blebs and bullae in 12 dogs. *J Am Anim Hosp Assoc.* 2003;39[5]:435–445.)

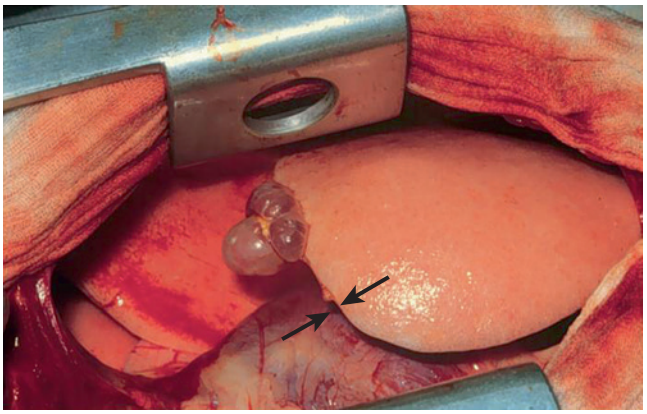


FIG. 30.24 Ruptured *(arrows)* and intact subpleural blebs in a dog with spontaneous pneumothorax.



FIG. 30.25 Lateral thoracic radiograph of a cat with pneumomediastinum and subcutaneous emphysema associated with tracheal rupture.

space to cause lung collapse and markedly reduced ventilation. Atmospheric and intrapleural pressures equilibrate rapidly through the defect, interfering with the normal mechanical function of the thoracic bellows, which normally provides the necessary pressure gradient for air exchange. Pneumomediastinum may be associated with pneumothorax or tracheal, bronchial, or esophageal defects, or it may be due to subcutaneous air migration along fascial planes at the thoracic inlet.

Tracheal rupture may occur because of trauma (see Chapter 28). The primary signs of tracheal rupture are pneumomediastinum and subcutaneous emphysema; pneumothorax is less pronounced (Fig. 30.25). **Pneumomediastinum** in cats is most commonly associated with overinflation of the endotracheal tube cuff[1]; however, it may also be associated with tracheal damage due to puncture by a stylet during intubation. Other causes of pneumomediastinum in cats include trauma and tracheal foreign bodies. Barotrauma associated with overinflation of the lung, particularly in animals with severe lung pathology, is more likely

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to lead to pneumothorax than pneumomediastinum. Tracheoscopy is rarely necessary to document/localize tracheal rupture. It is important to distinguish tracheal rupture without pneumothorax from that causing pneumothorax. Tracheal rupture without pneumothorax is best treated by cage rest; tracheoscopy is unnecessary and often only makes the tear worse. Tracheal rupture with pneumothorax, in which the clinician is planning on surgery, may benefit from first performing tracheoscopy to localize the lesion(s). However, it can be difficult to find the site of rupture endoscopically or even at surgery. Fortunately, surgery is rarely indicated as most lesions heal spontaneously with medical management (e.g., cage rest and oxygen supplementation).

Spontaneous pneumothorax occurs in previously healthy animals without antecedent trauma and may be primary (without evidence of underlying pulmonary disease) or secondary (with underlying disease such as pneumonia, pulmonary abscess, neoplasia, chronic granulomatous infection, or pulmonary parasitic infection, such as *Paragonimus* spp.). Based on the histologic appearance of the pulmonary lesion, both cysts and bullae have been reported in dogs. Primary spontaneous pneumothorax in dogs may be due to rupture of subpleural blebs; remaining lung tissue may appear normal. These blebs are most commonly located in the apices of the lungs. Overexpression of matrix metalloproteinases and an imbalance between proteinases and antiproteinases has been suggested as a possible cause in people.

Secondary spontaneous pneumothorax is more common in dogs than the primary form. In these animals, the subpleural blebs are associated with diffuse emphysema or other pulmonary lesions. Volume strain from expansive pressure in the lung has been shown to increase disproportionately at the apex as height increases. Most affected humans are cigarette smokers, suggesting that the underlying pulmonary disease could be a result of interference with the normal function of α_1-proteinase inhibitor in inhibiting elastase. It is believed that α_1-proteinase inhibitor is inactivated in people who smoke, allowing increased elastase-induced destruction of pulmonary parenchyma.

NOTE Be sure to differentiate traumatic and spontaneous pneumothorax, because the former usually responds to medical management, whereas the latter typically requires surgery.

Secondary spontaneous pneumothorax in cats is frequently associated with underlying lung pathology, particularly asthma; however, in some cats other conditions associated with pulmonary pathology (e.g., pulmonary neoplasia, heartworm infection, pulmonary abscesses, lungworm infection) may be found.[9] Clinical presentation of cats with asthma-associated spontaneous pneumothorax and that due to other causes is not different and supportive medical management is most appropriate, except in rare cases with focal congenital abnormalities that may benefit from surgical intervention.[9]

Pneumothorax may occur as a result of lung lobectomy. If a partial lobectomy (see p. 894) has been done and the staple line or sutured area is leaking, a fleece-bound fibrin sealant (TachoSil) may be applied over the area of leakage and has been shown to result in a reduction of air leakage when compared with standard techniques. It consists of a collagen sponge, made from horse tendons, coated with human fibrinogen and thrombin. Recent reports suggest that it is safe and efficacious in dogs when applied to experimental pleural leaks.[10]

DIAGNOSIS

Clinical Presentation

Signalment

Traumatic pneumothorax is most common in young dogs because they are more likely to be hit by cars or to suffer other trauma resulting in pulmonary damage. For similar reasons, males may be more commonly affected than females. Traumatic pneumothorax is less common in cats. Spontaneous pneumothorax usually occurs in large and deep-chested breeds; however, it may occur in small dogs. Purebred dogs, particularly Siberian huskies, may be more commonly affected than mixed breed dogs. Dogs of any age may develop spontaneous pneumothorax. Male and female dogs appear to be equally affected. Cats with asthma-associated secondary spontaneous pneumothorax are most commonly domestic shorthair cats, and in one study the median age was 8 years old (range, 7 weeks–17 years) with no sex predilection.[9]

History

Pneumothorax as a result of trauma usually causes acute dyspnea. Trauma is often not reported, making differentiation between traumatic and spontaneous pneumothorax difficult. Although the history of dogs with spontaneous pneumothorax varies depending on underlying etiology, most animals have acute onset of dyspnea. Some dogs may have nonspecific signs (e.g., lethargy, anorexia, depression, coughing, exercise intolerance), and respiratory signs may not be noted for several days until the pneumothorax worsens. Occasionally a chronic cough or fever may be noted. Recurrence of dyspnea in an animal previously treated for pneumothorax suggests spontaneous rather than traumatic pneumothorax.

Physical Examination Findings

Animals with moderate to severe pneumothorax typically have bilateral disease and an acute onset of severe dyspnea. Other evidence of trauma (e.g., rib fractures, limb fractures, traumatic myocarditis, pulmonary contusions) may be evident in animals with trauma-induced pneumothorax. Most animals with pneumothorax show a restrictive respiratory pattern (i.e., rapid, shallow respirations). If hypoventilation causes hypoxemia, they may appear cyanotic, and the heart and lung sounds are often muffled dorsally. Dogs are initially able to tolerate massive pneumothorax by increasing their chest expansion. However, respiration becomes ineffectual in animals with tension pneumothorax as the thorax becomes barrel shaped and fixed in maximal extension. This condition is life-threatening. Subcutaneous emphysema is occasionally noted in animals with pneumomediastinum and pneumothorax. Dull respiratory sounds, especially in the dorsal thorax, may be evident on auscultation. Air may migrate from the mediastinal space to the thoracic inlet and be noticeable under the skin over the neck and trunk.

The most common signs in cats presenting with pneumomediastinum are tachypnea, increased respiratory effort, and subcutaneous emphysema. In one study of 45 cats with pneumomediastinum, concurrent pneumothorax was identified in 21 of 45 (47%) cats, pleural effusion in 10 of 45 (22%), and pneumoretroperitoneum in 21 of 45 (47%).[11]

NOTE Tension pneumothorax is a life-threatening condition. It must be recognized and treated promptly.

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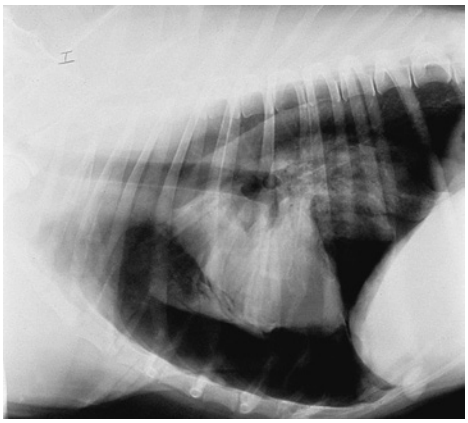


FIG. 30.26 Lateral thoracic radiograph of a dog with spontaneous pneumothorax. Note the apparent elevation of the heart from the sternum.

Diagnostic Imaging

Thoracic radiographs should be delayed until after thoracentesis in severely dyspneic animals (see p. 938) in which pneumothorax is strongly suspected. Pneumothorax usually occurs bilaterally in animals because air diffuses through the thin mediastinum. Pneumothorax causes large, air-filled spaces in the pleural cavity. On a recumbent lateral thoracic radiograph, the lungs collapse and retract from the thoracic wall, and the heart usually appears to be elevated from the sternum (Fig. 30.26). This apparent elevation of the heart is not noticeable on a standing lateral radiograph. Partly collapsed (atelectatic) lung lobes appear radiopaque compared with the air-filled pleural space. The vascular pattern does not extend to the thoracic wall as the lungs collapse. This may be particularly noticeable in the caudal thorax on a ventrodorsal view.

Radiographs should be carefully evaluated for underlying pulmonary disease (e.g., abscess, neoplasia) or associated trauma (e.g., rib fractures, pulmonary contusion). Pulmonary blebs found in some animals with spontaneous pneumothorax are seldom visible radiographically, although CT is more sensitive for finding these lesions. This is probably because the large blebs have ruptured, causing the pneumothorax. CT may be useful in identifying bullae. If imaging does not identify the lesion, surgical or thoracoscopic identification of bullae is indicated. Air-filled bullae may be incidental findings on thoracic radiographs of some animals. Pneumomediastinum is characterized by the ability to visualize thoracic structures (e.g., aorta, thoracic trachea, vena cava, esophagus) that are not usually apparent on thoracic radiographs. Improved imaging techniques for bullae would be helpful in surgical planning of affected animals; however, in a 2013 study the sensitivity and positive predictive value of CT for bulla detection was low.[12] In this study, CT was determined to be an ineffective preoperative diagnostic technique in dogs with spontaneous pneumothorax caused by bulla rupture because lesions were missed or incorrectly diagnosed in many animals unless they were large. False-positive diagnoses are also common.

NOTE Remember that bullae may not be seen in dogs with pneumothorax because they have often already ruptured. Additionally, air in the thoracic cavity may make other (nonruptured) bullae difficult to visualize.

Laboratory Findings

Specific laboratory abnormalities are uncommon in animals with pneumothorax; however, blood gas derangements may occur.

DIFFERENTIAL DIAGNOSIS

Any abnormality causing respiratory distress (e.g., diaphragmatic hernia, pleural effusion, pulmonary edema) is a differential diagnosis for pneumothorax. Because the management of animals with primary and spontaneous pneumothorax differs, once the animal's condition has been stabilized, the cause of the pneumothorax should be determined.

MEDICAL MANAGEMENT

Medical management of pneumothorax consists of initially relieving dyspnea by thoracentesis (see p. 920). If the pleural air accumulates quickly or cannot be managed effectively with needle thoracentesis, a thoracostomy tube should be placed (see p. 922). Tube thoracostomy is typically required in animals with spontaneous pneumothorax. Intermittent or continuous pleural drainage may be used, depending on the speed with which air accumulates. Continuous drainage may allow quicker resolution of pneumothorax in animals with large, traumatic defects. Providing an enriched oxygen environment may be beneficial, particularly in animals with concurrent pulmonary trauma (e.g., pulmonary contusion or hemorrhage). Providing analgesics to animals with fractured ribs or severe soft tissue damage may improve ventilation (see p. 886). Blood pleurodesis (5–10 mL/kg of whole blood) can be considered in dogs with persistent pneumothorax that have failed conservative or surgical management (Videos 30.8 and 30.9). In a 2014 study the procedure was successful in seven of eight dogs with persistent pneumothorax, although several procedures were required in some dogs.[13] The blood should be collected aseptically and injected into the thorax without additives in aliquots of 25 to 50 mL. Surgical intervention is seldom required in animals with traumatic pneumothorax; however, severe pulmonary parenchymal damage with a large rupture leading to pneumothorax and pleural effusion has been reported in two dogs after a tornado.[14] Both dogs underwent surgery for removal of the affected lung. Thoracentesis should be performed as necessary to prevent dyspnea while the pulmonary lesion heals, usually within 3 to 5 days. Recurrence is uncommon. Conversely, animals with spontaneous pneumothorax commonly have recurrent pneumothorax if surgery is not performed.

An open chest wound should be covered immediately with any readily available material (see Fig. 30.1). A sterile occlusive dressing should be applied as soon as possible and intrapleural air evacuated by thoracentesis or tube thoracostomy.

SURGICAL TREATMENT

Surgical therapy of animals with traumatic pneumothorax is seldom necessary (discussed previously). However, nonsurgical management of spontaneous pneumothorax usually results in an unsatisfactory outcome. Thoracoscopic treatment of bullous emphysema has been reported in dogs. *Mechanical pleurodesis* of the lungs (discussed later) may reduce the recurrence of pneumothorax in animals that undergo surgery for spontaneous pneumothorax. Mechanical pleurodesis damages the pleura, causing it to thicken.

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Preoperative Management

An ECG and thoracentesis should be performed before the induction of anesthesia. Preoxygenating these animals is often beneficial (see p. 916). Perioperative antibiotics are seldom warranted and may prevent culturing of bacteria from infected pulmonary tissue during surgery.

Anesthesia

Care should be used when anesthetizing and ventilating animals with pneumothorax and/or pulmonary bullae. IPPV may rupture intact bullae or accelerate air leakage from the damaged lung or bronchial tree. For these reasons, the inspiratory pressure should not exceed 10 to 12 cm H_2O in these animals until the chest cavity is opened. The adequacy of ventilatory pressures should then be reevaluated. Because IPPV may induce a tension pneumothorax, immediate treatment of this condition (e.g., needle thoracentesis, thoracostomy tube placement) may be necessary and should be anticipated. The use of nitrous oxide is contraindicated in patients with pneumothorax. See p. 884 for selected anesthetic protocols for use in animals with respiratory dysfunction.

Surgical Anatomy

See the discussion of the surgical anatomy of the pleural space on p. 918 and the anatomic description of the lungs in dogs and cats on p. 889.

Positioning

See intercostal thoracotomy, p. 890, or median sternotomy, p. 891.

SURGICAL TECHNIQUE

If an underlying pulmonary lesion is readily identified (i.e., pulmonary abscess or neoplasia) and can be localized to one hemithorax, an intercostal thoracotomy (see p. 890) allows a lobectomy to be performed more readily than from a median sternotomy approach. However, dogs with spontaneous pneumothorax usually have diffuse, bilateral pulmonary disease with multiple bullae. A median sternotomy allows visualization of all lung lobes and partial resection of any diseased lobes (see p. 891). Mechanical pleurodesis might be of benefit in dogs with spontaneous pneumothorax to reduce recurrence (discussed later).

Identify and remove diseased lung. If the source of the pleural air is not evident, fill the chest with warmed, sterile saline and look for air bubbles when the anesthetist ventilates the animal. If multiple partial lobectomies are necessary, use an automatic stapling device to reduce operative time. Perform pleural abrasion (mechanical pleurodesis) using a dry gauze sponge. Gently abrade the entire surface of the lung. Before closure, fill the thoracic cavity with warmed fluid and look for air bubbles when the animal is ventilated to ensure that no other air leaks are present. Place a thoracostomy tube and remove residual air before recovering the animal.

NOTE In animals with an open pneumothorax, definitive closure of large thoracic wall defects may require mobilization of adjacent muscles to provide an air-tight closure.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

In animals with spontaneous pneumothorax (in which multiple pulmonary bullae may be present), stapling devices allow partial lobectomies to be performed rapidly (see p. 894). Continuous suction devices are available commercially, or three-bottle systems can be made (see p. 924).

POSTOPERATIVE CARE AND ASSESSMENT

The animal should be observed postoperatively for pain or hypoventilation or both. Nasal insufflation is beneficial in most patients but is especially indicated in those with diffuse underlying pulmonary diseases or those in which significant portions of the lung were resected. Analgesic therapy should be utilized for all animals undergoing thoracotomy (see p. 886).

PROGNOSIS

With appropriate monitoring and care, the prognosis is excellent for animals with traumatic pneumothorax in which therapy is initiated before extreme dyspnea or respiratory arrest. Most dogs with spontaneous pneumothorax that are treated with needle thoracentesis alone or thoracostomy tubes will have continued air leakage or recurrence of pneumothorax, whereas the majority of dogs undergoing thoracotomy and lobectomy (with or without mechanical pleurodesis) for spontaneous pneumothorax do not have recurrences. Mortality is generally higher in dogs with spontaneous pneumothorax that are managed conservatively (vs. with surgery) in the author's experience. The prognosis for cats with pneumomediastinum with supportive care is good to excellent.

PLEURAL EFFUSION

DEFINITIONS

Pleural effusion refers to excessive fluid in the potential space between the **visceral pleura** of the lung and the **parietal pleura** of the thorax wall.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Fluid may accumulate in the pleural space because of decreased oncotic pressure (e.g., pure transudate caused by hypoalbuminemia [serum albumin concentration usually less than 1.6 g/dL]), increased hydrostatic pressure (e.g., modified transudate caused by cardiac disease, diaphragmatic hernia, tumor, lung lobe torsion), increased permeability (e.g., infection, tumor, lung lobe torsion), or hemorrhage (e.g., coagulopathy, tumor, trauma). Chylothorax (see p. 941) and pyothorax (see p. 947) are discussed separately from effusion.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, breed, or gender of dog or cat may be affected.

History

The history of affected animals typically depends on the underlying cause. Most owners note that the animal has exercise intolerance if not overt respiratory distress.

Physical Examination Findings

The most common physical examination finding in animals with pleural effusion is dyspnea. The dyspnea may be marked by a

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forceful inspiration with delayed expiration, making the animal appear to be holding its breath. This respiratory pattern is particularly noticeable in cats. Increased bronchovesicular sounds may be heard dorsally. Lung sounds may be absent ventrally (usually bilaterally, but occasionally unilaterally). Other physical examination findings in affected dogs may include muffled heart sounds and weight loss.

Diagnostic Imaging

If the animal is not severely dyspneic, thoracic radiographs should be obtained to confirm the diagnosis of pleural fluid. Taking dorsoventral (rather than ventrodorsal views) and "standing lateral" radiographic views, minimizing handling, and supplementing oxygen by face mask during the radiographic procedures help prevent further compromise of respiration. If the animal is not dyspneic and only small amounts of fluid are suspected, ventrodorsal and expiratory views may help delineate the effusion. Radiographic signs associated with pleural effusion include soft tissue opacity causing a silhouette sign with the heart and diaphragm, visualization of soft tissue interlobar fissure lines, rounding of lung margins at the costophrenic angles, widening of the mediastinum, separation of the lung borders from the thoracic wall by a soft tissue opacity, and scalloping of the lung margins at the sternal border (Figs. 30.27 and 30.28). The presence of pleural fluid often prevents satisfactory radiographic evaluation of the thoracic cavity. Adequate visualization of the entire thorax is necessary to rule out cranial mediastinal masses (e.g., lymphoma, thymoma); therefore radiographs should be repeated after removal of most of the pleural fluid. When available, ultrasound should be utilized to evaluate the mediastinum prior to removal of the pleural fluid, as the fluid serves as an acoustic window. Ultrasonography may be used to evaluate cardiac function, valvular lesions and function, congenital cardiac abnormalities, the presence of pericardial effusion, and mediastinal masses.

NOTE Delay thoracic radiographs until after thoracentesis in animals with pleural effusion that are severely dyspneic.

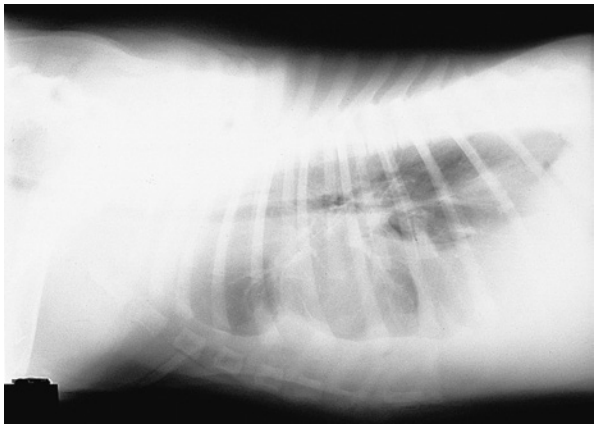


FIG. 30.27 Lateral thoracic radiograph of a dog with pleural effusion. Note the scalloped appearance of the ventral aspect of the lungs along the sternum.

Thoracoscopy

Thoracoscopy may be used to help determine the etiology of pleural effusion.

Laboratory Findings

Pleural fluid should be placed in a clot tube for biochemical studies and an EDTA tube for cytologic examination, and some should be saved in a sterile syringe for culture. It is important to analyze the fluid for protein concentration, nucleated cell count, and differential nucleated cell count. Pleural fluid may be cultured for mesothelioma cells by research laboratories.

The serum albumin and electrolyte concentrations should be determined. Severe hypoalbuminemia (≤1.6 g/dL) may cause pleural effusion, but sequestration of albumin in pleural effusion (or any such third space disorder) may cause minor hypoalbuminemia (usually >2 g/dL). Frequent withdrawal of large volumes of pleural fluid may cause hyponatremia and/or hyperkalemia, as well as lower the serum albumin concentration.

DIFFERENTIAL DIAGNOSIS

Any cause of respiratory distress or coughing should be considered a differential diagnosis.

MEDICAL MANAGEMENT

For medical management of chylothorax and pyothorax, see pp. 943 and 949, respectively. Management of animals with lung lobe torsion is described on pp. 905 to 907. For the medical management of the various other disorders causing pleural effusion, refer to a medical textbook. If the animal is severely hyperkalemic, symptomatic therapy with potassium-free fluids may be necessary until the effusion is controlled.

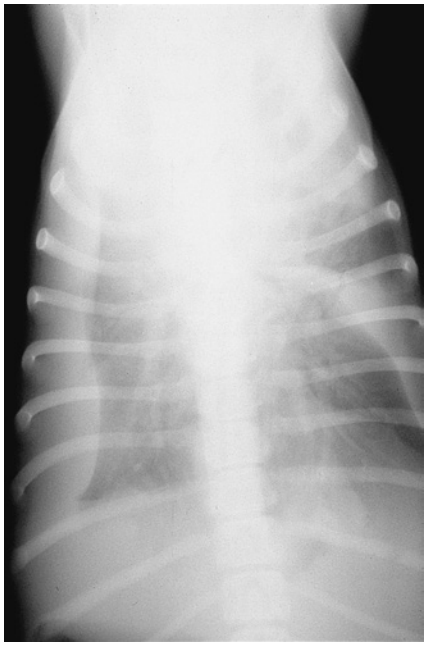


FIG. 30.28 Dorsoventral thoracic radiograph of a dog with pleural effusion. Note the interlobar fissure lines, the rounding of the lung margins at the costophrenic angles and the separation of the lung margins from the thoracic wall.

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SURGICAL TREATMENT

If the cause of modified transudates or exudates cannot be found with routine laboratory testing, imaging, and/or thoracoscopy, then exploratory thoracotomy may be necessary. If the patient has a bicavitary effusion (especially thorax and abdomen) that is not due to hypoalbuminemia, then cardiac disease, neoplasia, and infectious diseases are the major differential diagnoses.

Preoperative Management

The preoperative management of animals undergoing thoracotomy is presented on p. 884.

Anesthesia

See p. 884 for selected anesthetic protocols for animals with respiratory disease. If the animal has moderate or severe pleural fluid, the chest should be tapped before the induction of anesthesia. Anesthetic recommendations for patients with pleural effusion undergoing a thoracotomy are provided in Table 30.5.

Surgical Anatomy

The surgical anatomy of the thoracic cavity is presented on p. 888.

Positioning

See pp. 890 and 892.

SURGICAL TECHNIQUE

Thoracotomy may be performed using a median sternotomy (see p. 891) if there is no discernible lesion and the surgeon wishes to examine the entire thorax, or using a lateral thoracotomy (see p. 890) if a lesion has been identified and lung lobectomy is anticipated.

POSTOPERATIVE CARE AND ASSESSMENT

See p. 897 for the postoperative care of patients that have undergone thoracotomy.

TABLE 30.5 Anesthetic Considerations for Thoracotomy of the Pleural Effusion Patient

Preoperative Considerations	
Associated conditions	• May be septic if purulent effusion with dehydration, hypotension, electrolyte abnormalities, and abnormal glucose • If older patient, may have other comorbidities • Adrenal suppression may be present in the critically ill patient • +/– Anemia
Bloodwork	• HCT • Electrolytes • BUN • Cr • TP • Blood gas, if available • +/– Lactate • +/– BG
Physical exam	• May be markedly stressed and extremely dyspneic, absent pulmonary sounds ventrally, either unilateral or bilateral; muffled heart sounds; hypotension in the critically ill patient
Other diagnostics	• Thoracocentesis prior to further diagnostics • Blood pressure • ECG • Radiographs (thoracic +/– abdominal) • +/– Ultrasound • +/– CT

Premedications	• Oxygen with flow-by or face mask
	• Avoid sedatives in depressed or dyspneic patients
	• In patients that are have minimal respiratory compromise, give:
	  • Midazolam (0.2 mg/kg IV, IM), *or*
	  • Diazepam (0.2 mg/kg IV)
	• Before induction, avoid all respiratory depressants, including but not limited to opioids, xylazine, medetomidine, and dexmedetomidine; avoid acepromazine, xylazine, medetomidine, and dexmedetomidine if patient is hypotensive.
	• After a benzodiazepine is given but before induction, clip hair from abdomen and thorax; this can usually be done with patient standing or in lateral recumbency; do not place patient in ventrodorsal position until after intubation.

Intraoperative Considerations	

Induction	• Preoxygenate for 3–5 min with face mask or flow-by oxygen
	• If normal cardiac function, give:
	  • Propofol (2–4 mg/kg IV) if sedated, *or*
	  • Propofol (4–8 mg/kg IV) if unsedated
	  • Alfaxalone (2–5 mg/kg IV)
	• If decreased cardiac function, give:
	  • Etomidate (0.5–1.5 mg/kg IV); if possible avoid its use in critically ill patients

Continued

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TABLE 30.5 Anesthetic Considerations for Thoracotomy of the Pleural Effusion Patient—cont'd

Maintenance	• Isoflurane or sevoflurane, *plus*
	  • Fentanyl (2–10 µg/kg IV PRN in dogs; 1–4 µg/kg IV PRN in cats) for short-term pain relief, *plus*
	  • Fentanyl CRI (1–5 µg/kg IV loading dose, then 2–30 µg/kg/h IV), *or*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Oxymorphone (0.05–0.2 mg/kg IV PRN), *or*
	  • Buprenorphine[b] (0.005–0.02 mg/kg IV PRN) *plus*
	  • Ketamine (low-dose)(0.5–1 mg/kg IV once), *or*
	  • Ketamine CRI (0.5 mg/kg IV loading dose, then 10 µg/kg/min IV)
	• If hypotensive (to keep MAP 60–80 mm Hg) give phenylephrine, ephedrine, dopamine, norepinephrine, vasopressin, or fluids as needed
	• Have surgeon ready to drape and make incision when positioning patient ventrodorsally
	• Ventilation parameters:
	  • SpO_2 >95%
	  • Small tidal volumes: 5–7 mL/kg
	  • Respiratory rates: 10–30
	  • Peak airway pressures: <12–15 mm Hg if possible
	  • Slowly expand the lungs while manually ventilating and visually observing the lung expansion
	• If unable to meet above parameters, higher peak pressures may need to be used initially; as intrathoracic pressure is reduced, drop peak airway pressures and tidal volumes accordingly; slowly expand the lungs while manually ventilating and visually observing lung expansion. Consider positive end expiratory pressure.

Fluid needs	• 5–20 mL/kg/h to replace evaporative losses plus 3 × EBL; consider colloids for EBL replacement in 1:1 ratio
Monitoring	• BP • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature • +/– Arterial line • U/O

Blocks	• Epidural:
	  • Morphine (0.1 mg/kg preservative free), *or*
	  • Buprenorphine (0.003–0.005 mg/kg diluted in saline), *plus*
	Local anesthetics (see Box 30.2 and p. 144):
	• Intercostal nerve blocks with local anesthetics, *plus*
	  • Incisional block with local anesthetic

Postoperative Considerations

Analgesia	• Fentanyl CRI (1–10 µg/kg IV loading dose, then 2–20 µg/kg/h IV), *or* • Morphine[c] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.1–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h in cats), *or* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats), *or* • Hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs), *or* • Oxymorphone (0.1–0.2 mg/kg IV, IM), *or* • Buprenorphine[b] (0.005–0.02 mg/kg IV), IM q4–8h or 0.01–0.02 mg/kg OTM q6–12h in cats) *plus* • +/– Ketamine CRI (2 µg/kg/min IV); if no previous loading dose, give 0.5 mg/kg IV before CRI NOTE: Avoid NSAIDs in hypotensive, elderly, and critically ill patients
Monitoring	• SpO_2 • Blood pressure • ECG • HR • Respiratory rate • Temperature • U/O • Thoracostomy tubes
Bloodwork	• HCT • TP • Electrolytes • Blood gas • +/– Albumin • +/– Lactate • +/– BG
Estimated pain score	Severe

[a]Monitor for hyperthermia in cats.
[b]Buprenorphine is a better analgesic than morphine in cats.
[c]Give slowly to prevent histamine release.
BG, Blood glucose; *BP,* blood pressure; *BUN,* blood urea nitrogen; *Cr,* creatinine; *CRI,* constant-rate infusion; *CT,* computed tomography; *EBL,* estimated blood loss; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *MAP,* mean arterial pressure; *NSAID,* nonsteroidal antiinflammatory drug; *OTM,* oral transmucosal; *PRN,* as needed; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein; *U/O,* urine output.

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PROGNOSIS

The prognosis for animals with pleural effusion depends on the underlying cause. See pp. 907, 947, and 950 for the prognosis in animals with lung lobe torsion, chylothorax, and pyothorax, respectively.

CHYLOTHORAX

DEFINITIONS

Chyle is the term used to denote lymphatic fluid arising from the intestine and therefore containing a high quantity of fat. **Chylothorax** is a collection of chyle in the pleural space. Chylothorax is termed **idiopathic** when an underlying cause cannot be identified.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

In most animals, abnormal flow or pressures in the thoracic duct (TD) are thought to lead to exudation of chyle from intact but dilated thoracic lymphatic vessels (a condition known as *thoracic lymphangiectasia*; Fig. 30.29). These dilated lymphatic vessels may form in response to increased lymphatic flow (caused by increased hepatic lymph formation), decreased lymphatic drainage into the venous system as a result of high venous pressures, or both factors acting simultaneously to increase lymph flow and reduce drainage. Any disease or process that increases systemic venous pressures (e.g., right heart failure, mediastinal neoplasia, cranial vena cava thrombi, ligation of the left brachycephalic vein, or granulomas) may cause chylothorax (Box 30.6). Trauma is an uncommonly recognized cause of chylothorax in dogs and cats because the TD heals rapidly after injury, and the effusion resolves within 1 to 2 weeks without treatment.

Possible causes/associations of chylothorax include anterior mediastinal masses (e.g., mediastinal lymphoma, thymoma), heart disease (e.g., cardiomyopathy, pericardial effusion, heartworm infection, foreign objects, tetralogy of Fallot, tricuspid dysplasia, double-chambered right ventricle, or cor triatriatum dexter), fungal granulomas, venous thrombi, PPDHs causing intrapericardial compression of the heart, and congenital abnormalities of the TD. It may occur in association with diffuse lymphatic abnormalities, including intestinal lymphangiectasia, generalized lymphangiectasia with subcutaneous chyle leakage, and lymphedema[15] (see p. 634). The underlying etiology is undetermined in most animals (e.g., idiopathic chylothorax) despite extensive diagnostic workups. Because the treatment of this disease varies considerably depending on the underlying etiology, it is imperative that clinicians identify concurrent disease processes before instituting definitive therapy.

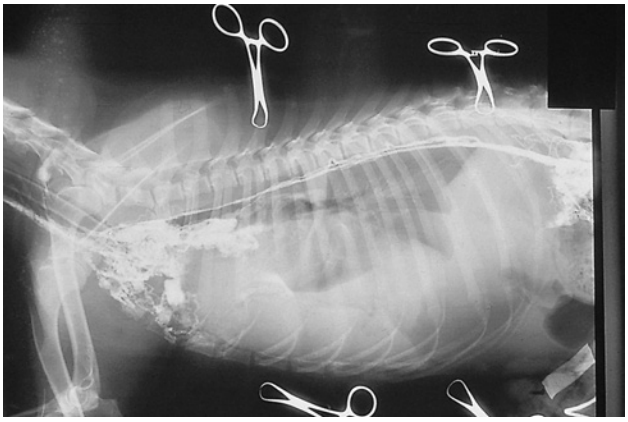


FIG. 30.29 Thoracic lymphangiectasia in a dog with idiopathic chylothorax. Note the dilated, tortuous lymphatics in the cranial mediastinum.

DIAGNOSIS

Clinical Presentation

Signalment

Any breed of dog or cat may be affected; however, for several years a breed predisposition has been suspected in the Afghan hound. It has been suggested that the shiba inu breed may also be predisposed. Among cats, oriental breeds (e.g., Siamese and Himalayan) appear to have an increased prevalence. Chylothorax may affect animals of any age; however, older cats may be more likely than young cats to develop it. This finding was believed to indicate an association between chylothorax and neoplasia. Afghan hounds appear to develop this disease in middle age, but affected shiba inus have been less than 1 year old. A gender predisposition has not been identified.

History

Coughing is often the first (and occasionally the only) abnormality until the animal becomes dyspneic. Many owners report that coughing began months before presenting the animal for care; therefore animals that cough and do not respond to standard treatment of nonspecific respiratory problems should be evaluated for chylothorax. Coughing may be due to irritation caused by the effusion or may be related to the underlying disease process (e.g., cardiomyopathy, thoracic neoplasia).

NOTE Coughing may be the only clinical sign in animals with pleural effusion. Therefore thoracic radiographs are warranted in any animal with a chronic, nonresponsive cough.

Physical Examination Findings

Most animals with chylothorax have a normal body temperature unless they are extremely excited or severely depressed. Additional

BOX 30.6 Abnormalities Associated With Chylothorax in Dogs and Cats

Cardiomyopathy
Mediastinal lymphosarcoma or thymoma
Cranial vena cava thrombi
Heartworm disease
Fungal granulomas
Pericardial effusion/heart base tumors
Foreign objects
Tetralogy of Fallot
Tricuspid dysplasia
Cor triatriatum dexter
Congenital thoracic duct abnormalities
Lymphangioleiomyomatosis
Peritoneopericardial diaphragmatic hernia

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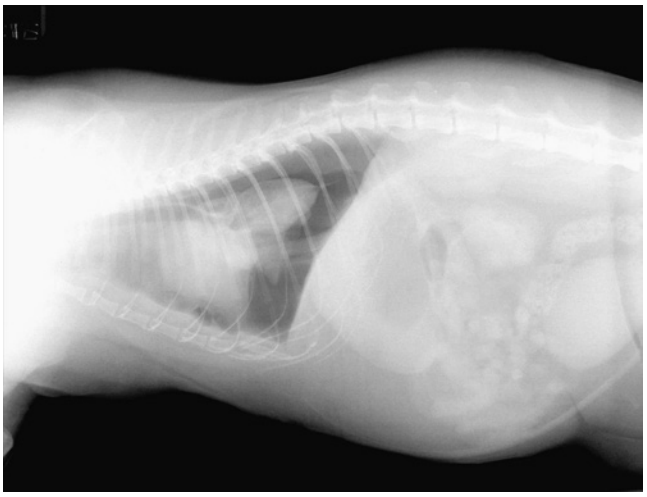


FIG. 30.30 Lateral radiograph of a cat with fibrosing pleuritis secondary to chronic chylothorax. Note the rounded margins of the lung lobes.

findings may include muffled heart sounds, depression, anorexia, weight loss, pale mucous membranes, arrhythmias, murmurs, and pericardial effusion.

Diagnostic Imaging

The radiographic signs of pleural effusion are provided on p. 938. Animals that have collapsed lung lobes that do not appear to reexpand after removal of chyle or other pleural fluid should be suspected of having underlying pulmonary parenchymal or pleural disease, such as fibrosing pleuritis (Fig. 30.30). Although the etiology of the fibrosis is unknown, it apparently can occur subsequent to any prolonged exudative or blood-stained effusion. Diagnosis of fibrosing pleuritis is difficult. The atelectatic lobes may be confused with metastatic or primary pulmonary neoplasia, lung lobe torsion, or hilar lymphadenopathy. Radiographic evidence of pulmonary parenchyma that fails to reexpand after removal of pleural fluid should be considered possible evidence of atelectasis with associated fibrosis. Fibrosing pleuritis should also be considered in animals with persistent dyspnea in the face of minimal pleural fluid.

NOTE Cats with fibrosing pleuritis are often mistaken as having postoperative pleural fluid based on radiographs. Perform an ultrasound exam on these cats before performing thoracentesis to ensure that fluid is present; thoracentesis in these cats often causes severe pneumothorax!

CT lymphangiography may be able to quantify branches of the TD more accurately than standard radiographic lymphangiography; however, its value in animals undergoing TD ligation remains to be substantiated. Injection of 60 mg/kg of iohexol into the popliteal lymph node of dogs enabled quality CT without extranodal leakage. Injection of contrast into a mesenteric lymph node may be done percutaneously with ultrasound guidance, via a small paracostal incision, or with laparoscopy. Recently, imaging of the TD was reported using CT after the injection of an iodine contrast agent into the subcutaneous tissue surrounding the anus.[16]

TABLE 30.6 Characteristics of Chylous Effusions

Characteristic	Cats	Dogs
Color	White or pink (occasionally red)	Same
Clarity	Opaque, remains opaque when centrifuged	Same
SG	1.019–1.050	1.022–1.037
TP	2.6–10.3	2.5–6.2
WBC	Average = 7987	Average = 6167

SG, Specific gravity; *TP,* total protein (g/dL); *WBC,* total white blood cells/μL.

BOX 30.7 Other Characteristics of Chylous Effusions

Triglyceride content higher than that of serum
Cholesterol content less than or equal to that of serum
Chylomicrons present
Predominant cell type may be the lymphocyte or neutrophil
Sudanophilic fat globules present[a]
Clears with ether[a]

[a]These tests are seldom performed.

Laboratory Findings

Fluid recovered by thoracentesis should be placed in an EDTA tube for cytologic examination. Placing the fluid in an EDTA tube rather than a clot tube allows cell counts to be performed. Although chylous effusions are routinely classified as exudates, the physical characteristics of the fluid may be consistent with a modified transudate (Table 30.6). The color varies depending on the dietary fat content and the presence of concurrent hemorrhage. The protein content is variable and often inaccurate because of interference with the refractive index by the high lipid content of the fluid. The total nucleated cell count is usually below 10,000/μL and consists primarily of small lymphocytes or neutrophils with smaller numbers of lipid-laden macrophages.

Chronic chylous effusions may contain low numbers of small lymphocytes because of the body's inability to compensate for continued lymphocyte loss. Nondegenerative neutrophils may predominate with prolonged loss of lymphocytes or if multiple therapeutic thoracenteses have induced inflammation. Degenerative neutrophils and sepsis are uncommon findings because of the bacteriostatic effect of fatty acids but can occur iatrogenically as a result of repeated aspiration. To help determine if a pleural effusion is truly chylous, several tests have been recommended, including comparison of fluid and serum triglyceride levels, Sudan III staining for lipid droplets, and the ether clearance test. The most diagnostic test is comparison of serum and fluid triglyceride levels (Box 30.7). Chylous effusions have a higher triglyceride concentration than simultaneously collected serum.

DIFFERENTIAL DIAGNOSIS

Once pleural effusion has been identified, differential diagnoses include diseases that cause exudative pleural effusion, such as pyothorax. Although chylous effusions usually have a characteristic appearance, the physical characteristics of chylous effusions and

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other exudative effusions may be similar. In addition, the appearance and cell populations of chylous effusions can be altered by diet and chronicity.

Pseudochylous effusion is a term that has been misused in the veterinary literature to describe effusions that look like chyle but with which a ruptured TD is not found. Given the known causes of chylothorax in dogs and cats, this term should be reserved for effusions in which the pleural fluid cholesterol is greater than the serum cholesterol concentration and the pleural fluid triglyceride is less than or equal to the serum triglyceride. Pseudochylous effusions are extremely rare in veterinary patients but may be associated with tuberculosis.

MEDICAL MANAGEMENT

If an underlying disease is diagnosed, it should be treated and the chylous effusion managed by intermittent thoracocentesis. If the underlying disease is effectively treated, the effusion often resolves; however, complete resolution may take several months. Surgical intervention should be considered only in animals with idiopathic chylothorax or those that do not respond to medical management.[17] Thoracostomy tubes should be placed only in animals suspected of having traumatic chylothorax (very rare) with rapid fluid accumulation, or occasionally after surgery. Electrolytes should be monitored; hyponatremia and hyperkalemia can occur in dogs with chylothorax undergoing multiple thoracentesis. A low-fat diet may reduce the amount of fat in the effusion, which may improve the animal's ability to resorb fluid from the thoracic cavity.

NOTE Animals with traumatic chylothorax heal and resolve the effusion within a few weeks; however, traumatic chylothorax is extremely rare.

Commercial low-fat diets are preferable to homemade diets; however, if commercial diets are refused, homemade diets are a reasonable alternative (Tables 30.7 and 30.8; the fat content of these diets usually ranges from 1.7–2.5 g fat/100 kcal). Medium-chain triglycerides (once thought to be absorbed directly into the portal system, bypassing the TD) are transported via the TD in dogs; therefore they are no longer considered useful. It is unlikely that dietary therapy alone will cure this disease, but it may help in the management of animals with chronic chylothorax. Clients should be informed that with the idiopathic form of this disease, the only surgical treatment that is likely to stop the effusion is TD ligation. However, the condition may resolve spontaneously in some animals after several weeks or months of medical management.

NOTE Do not expect low-fat diets to cure chylothorax; however, a lower-fat chyle may be easier to resorb from the pleural space.

Benzopyrone drugs have been used for the treatment of lymphedema in humans for years. Whether these drugs might be effective in reducing pleural effusion in animals with chylothorax is unknown; however, preliminary findings suggest that some animals treated with rutin (Box 30.8) have complete resolution of effusion 2 months after the initiation of therapy. Whether the effusion resolves spontaneously in these animals or is associated with the drug therapy is unknown.

TABLE 30.7 Canine Homemade Low-Fat Diet[a]

Ingredient	Amount
Cooked white rice	2⅔ cups
Stewed chicken	⅓ lb (150 g)
Dicalcium phosphate[b]	1¼ teaspoons
GNC Ca-Mg (250 mg calcium, 155 magnesium/tablet)[c]	2 tablets
Morton Lite Salt	1 teaspoon
Zinc (50 mg zinc/tablet)[d]	½ tablet
Pet Tab	1 tablet
Radiant Valley Natural Selenium (100 µg Se/tablet)[e]	1 tablet
GNC Copper (2 mg copper/tablet)[c]	1 tablet

Directions: Cook rice without salt. Boil chicken and skim off fat. Crush tablets to a fine powder. Combine all ingredients and mix well. Refrigerate unused portions.
[a]Calculations based on the average published nutrient content of each ingredient indicate that this diet meets or exceeds the nutrient requirements for maintenance for adult dogs published by the Association of American Feed Control Officials. This recipe makes approximately 1.5 lb (680 g) of food that contains 910 kcals of metabolizable energy.
[b]Dicalcium phosphate: 18.5% phosphorus, 22% to 24% calcium, available at farm supply and feed stores.
[c]Available at General Nutrition Centers (GNCs).
[d]Available at most supermarkets.
[e]Available at many supermarkets or health food stores.

TABLE 30.8 Feline Homemade Low-Fat Diet[a]

Ingredient	Amount
Cooked white rice	3⅔ cups
Stewed chicken	½ lb (227 g)
Dicalcium phosphate[b]	1½ teaspoons
GNC Ca-Mg (600 mg calcium/tablet)[c]	1½ tablets
Morton Lite Salt	1 teaspoon
Taurine tablets (500 mg taurine/tablet)[d]	3 tablets
Zinc (50 mg zinc/tablet)[e]	½ tablet
Feline Pet Tab	3 tablets
Radiant Valley Natural Selenium (100 µg Se/tablet)[e]	½ tablet
Nature Made Balanced B-50 Complex[e]	½ tablet
GNC Choline (250 mg choline/tablet)[c]	1 tablet

Directions: Cook rice without salt. Boil chicken and skim off fat. Crush tablets to a fine powder. Combine all ingredients and mix well. Refrigerate unused portions.
[a]Calculations based on the average published nutrient content of each ingredient indicate that this diet meets or exceeds the nutrient requirements for maintenance for adult cats published by the Association of American Feed Control Officials. This recipe makes approximately 2¼ pounds of food that contains 1293 kcals of metabolizable energy.
[b]Dicalcium phosphate: 18.5% phosphorus, 22% to 24% calcium, available at farm supply and feed stores.
[c]Available at General Nutrition Centers (GNCs).
[d]Taurine tablets can be purchased at most health food stores and cooperatives as 500-mg and 1000-mg tablets.
[e]Available at most supermarkets.

BOX 30.8 Benzopyrone for Treatment of Chylothorax[a]

Rutin[b] 50–100 mg/kg, given orally q8h

[a]Efficacy is unproven at this time; clinical studies are needed.
[b]Obtained at health food stores.

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Somatostatin is a naturally occurring substance that has an extremely short half-life. It inhibits gastric, pancreatic, and biliary secretions (i.e., glucagon, insulin, gastric acid, amylase, lipase, and trypsin) and prolongs gastrointestinal transit time, decreases jejunal secretion, and stimulates gastrointestinal water absorption. In recent years, analogs of somatostatin have been used to successfully treat traumatic or postoperative chylothorax in humans. In these patients, reduced gastrointestinal secretions may aid healing of the TD by decreasing TD lymphatic flows. It has also been reported to cause early decreased drainage and early fistula closure in dogs with experimental transection of the TD. The mechanism by which nontraumatic chylothorax may benefit from this treatment is unclear; however, resolution of pleural fluid (chyle and postoperative serosanguinous effusion) in both dogs and cats has occurred after the administration of octreotide. Octreotide (Sandostatin; 10 µg/kg subcutaneously three times a day for 2–3 weeks) is a synthetic analog of somatostatin that has a prolonged half-life and minimal side effects.[18,19] Soft stools that resolve after withdrawal of the drug may occur. Prolonged treatment should be discouraged because people treated for longer than 4 weeks are at risk for gallstones.

SURGICAL TREATMENT

Surgical intervention is warranted in animals that do not have underlying disease and in which medical management has become impractical or is ineffective. Surgical options include TD ligation plus pericardectomy (with or without mesenteric lymphangiography), passive pleuroperitoneal shunting, active pleuroperitoneal or pleurovenous shunting, pericardiectomy, omental drainage, TD glue embolization, cisterna chyli ablation, and pleurodesis. Only TD ligation, pericardiectomy, mesenteric lymphangiography, cisterna chyli ablation, and active pleuroperitoneal shunting are recommended by the author and are described here. The mechanism by which TD ligation is purported to work is that abdominal lymphaticovenous anastomoses form after TD ligation, which allows transport of chyle to the venous system. Chyle bypasses the TD, and the effusion resolves. Properly performed, TD ligation resolves effusion in over 80% of dogs and cats. Continued production of a nonchylous effusion (from pulmonary lymphatics) may occur in some animals after surgery.

Mesenteric lymphangiography may be particularly difficult to perform in cats. Although mesenteric lymphangiography is not essential, catheterization of a mesenteric lymphatic and injection of methylene blue makes identification of the TD and its branches much easier. TD ligation has been performed using thoracoscopy.[20,21] TD glue embolization has been reported for the management of two dogs with recurrent chylothorax, but its value has not been substantiated.[22] Cisterna chyli ablation via laparotomy or thoracoscopically via an abdominal or transdiaphragmatic approach may be of benefit in animals with chylothorax.[23,24]

Many animals with chylothorax have either a thickened pericardium or thick tissue overlying the pericardium. Chronic irritation from chyle is thought to cause this thickening. It is believed that the thickened tissue might elevate systemic venous pressures, and these abnormal venous pressures may impede drainage of chyle into the cranial vena cava while increasing lymphatic flow through the TD. When the pericardium or overlying tissues are thickened or abnormal in animals with derangements in lymphatic flow (be it chylothorax or continued serosanguinous flow after TD ligation), pericardiectomy may act to lower right-sided venous pressures. Such normalization of venous pressures might be sufficient to allow the animal to route lymphatic fluid through normal channels. Pericardiectomy may be performed alone or in conjunction with TD ligation in a number of animals; in most of these animals, the effusion has resolved. Therefore pericardiectomy is recommended in conjunction with TD ligation.

En bloc ligation of the TD has been suggested as an alternative to dissection of the duct and can be done after a right lateral thoracotomy or thoracoscopically.[20,21] For this technique, a path is dissected across the mediastinum immediately dorsal to the aorta and below the ventral aspect of the thoracic vertebrae and hypaxial muscles. The mediastinum is incised adjacent and parallel to the dorsolateral wall of the caudal thoracic aorta for a length of 1 to 2 cm. If the TD is visualized thoracoscopically, it can be ligated with clips. Otherwise multiple, large clips are applied to the tissues in mass, or silk ligatures are passed through the mediastinum immediately dorsal to the aorta and back through the mediastinum immediately ventral to the thoracic vertebrae. Care should be taken to ensure that ducts that are lateral or even ventral to the aorta are ligated if present.[22] A single paracostal approach has been reported to provide excellent exposure of cisterna chyli, caudal TD, and intestinal lymphatics, eliminating the need for an intercostal thoracotomy and repositioning during combined TD ligation and cisterna chyli ablation.[25]

NOTE Most failures of thoracic duct ligation are technical failures: complete ligation of the thoracic duct is not accomplished. This is particularly common in animals with severe restrictive pleuritis and in cats where the duct(s) is more difficult to visualize because of its small size.

Preoperative Management

Food is withheld 12 hours before surgery. Cream and/or oil may be fed before surgery (i.e., given every hour until induction of anesthesia, starting 3–4 hours before surgery) to help the clinician to visualize lymphatics. Methylene blue may be injected into a lymph node at surgery if absorption of the cream or oil is not sufficient.

Anesthesia

See Table 30.5 for anesthetic recommendations for patients with pleural effusion undergoing thoracotomy.

Surgical Anatomy

The TD is the cranial continuation of the cisterna chyli and generally is said to begin between the crura of the diaphragm (Fig. 30.31). In cats the TD lies between the aorta and azygous vein on the left side of the mediastinum. In dogs it lies slightly on the right side of the mediastinum until it reaches the fifth or sixth vertebra and then crosses to the left side. The TD terminates in the venous system of the neck (left external jugular vein or jugulosubclavian angle).

Positioning

If a thoracic approach to the TD is used (discussed later), the left side (cats) or right side (dogs) of the thorax and abdomen is prepared for aseptic surgery. If a transdiaphragmatic approach is used, the cranial abdomen and caudal thorax are prepped.

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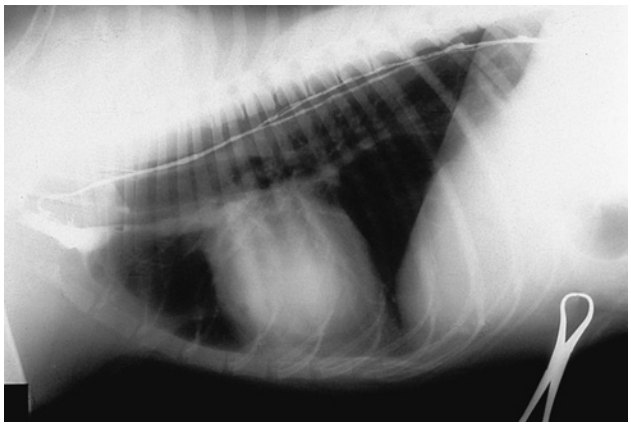


FIG. 30.31 Mesenteric lymphangiogram of a normal dog. Note the multiple branches of the thoracic duct.

SURGICAL TECHNIQUES

Mesenteric Lymphangiography

For a thoracic approach, make a paracostal incision (or for a transdiaphragmatic approach, make a cranial midline abdominal incision), exteriorize the cecum, and locate an adjacent lymph node (Video 30.10).

> **NOTE** The thoracic duct is difficult to approach transdiaphragmatically in deep-chested dogs. This approach may be used in small dogs and cats but should be avoided in larger dogs. An intercostal approach to the thoracic duct is preferred in medium and large breeds and can be used in any animal.

If necessary, inject a small amount (i.e., 0.5–1 mL of diluted methylene blue (i.e., 1 part methylene blue plus 5–10 parts saline) into the lymph node to increase lymphatic visualization. Avoid repeated doses of methylene blue because of the risk of inducing a Heinz body anemia or renal failure. Find a lymphatic near the node to catheterize by gently dissecting the mesentery (Video 30.11). Cannulate the lymphatic with a 20- to 25-gauge over-the-needle catheter, and attach a three-way catheter and extension tubing (filled with heparinized saline) to the catheter with a suture (3-0 silk). Place an additional suture around the extension tubing and through a segment of intestine to prevent dislodgment of the catheter. Either inject a small amount of dilute methylene blue directly into the catheter (Video 30.12), or if a contrast radiograph is preferred, inject 1 mL/kg of a dilute (diluted 50% with sterile saline) water-soluble contrast agent (e.g., Renovist) into the catheter and take a lateral thoracic radiograph as the last milliliter is flushed into the catheter. Use the lymphangiogram to help identify the number and location of branches of the TD that need to be ligated (Fig. 30.31). If desired, repeat the lymphangiogram or methylene blue injection after TD ligation (discussed later) to identify branches that were not occluded.

> **NOTE** The author seldom performs contrast lymphangiography, but catheterization of a mesenteric lymphatic for injection of methylene blue is extremely helpful for localizing the ducts and ensuring their complete ligation.

Embolization of the TD with cyanoacrylate injected through a mesenteric lymphatic catheter has been reported in dogs. Advantages of TD embolization are that direct visualization of the TD is not required, which eliminates the need for a thoracotomy or diaphragmatic incision. Disadvantages of this procedure are the same as those for mesenteric lymphangiography and TD ligation (i.e., not all TD branches may fill with the cyanoacrylate mixture, and collateralization may occur past the obstruction).

Thoracic Duct Ligation

Perform an intercostal thoracotomy (right side for dogs, left side for cats) at the 8th, 9th, or 10th intercostal space, or make an incision in the diaphragm (see the previous Note). Locate the TD(s), and use hemostatic clips and/or silk suture (2-0 or 3-0) to ligate it (discussed later). Visualization of the TD is greatly aided by injecting methylene blue into the lymphatic catheter (discussed previously). *Perform a subtotal pericardectomy by reaching forward under the rib cage.*

Thoracoscopic Ligation of the Thoracic Duct

Clip the entire thorax from the manubrium to the level of the second lumbar vertebra. Position the animal in sternal recumbency, tilted slightly to the left, and aseptically prepare and drape the right hemithorax in dogs (left hemithorax in cats). Establish three thoracoscopic portals for introduction of surgical instruments. Make a 1- to 2-cm skin incision midthorax in the 10th intercostal space for thoracoscope portal placement by bluntly dissecting tissues with a Kelly forceps. After creating a pneumothorax, place a 5-mm blunt obturator/flexible portal assembly (e.g., Endopath TT012) into the chest. Introduce a 5-mm outer diameter, 301 rigid telescope through the portal. After examination of the right hemithorax, use the thoracoscope to facilitate placement of two additional portals. Insert a 5-mm trocar/portal assembly (e.g., Endopath 355) in the dorsal third of an intercostal space for passage of surgical instruments directly perpendicular to the aorta, avoiding the intercostal arteries that originate dorsally. Then insert a 15-mm blunt obturator/flexible portal assembly (e.g., Flexipath FP015) in the dorsal third of a caudal intercostal space for instrument passage and hemostatic clip application (e.g., 10 mm Microline clip applier) perpendicular to the aorta (directly across, from one hemithorax to the other, avoiding the intercostal arteries). After establishing the three thoracoscopic portals, place a straight grasping forceps through the caudal portal and use it to retract the mediastinal pleura laterally toward the thoracoscope. Pass microscissors through the cranial portal and use them to incise the mediastinal pleura longitudinally, ventrolateral to the aorta, establishing a plane of dissection ventrally and proceeding dorsally along the lateral and dorsal aspect of the aorta into the contralateral hemithorax. Ventral retraction of the aorta allows entry into the contralateral hemithorax ventral to contralateral branches of the TD. Bilateral thoracoscopy can increase the chances of adequate ventral dissection in the contralateral hemothorax to include ventrally located branches of the TD. *Then, incise the mediastinal pleura ventral to the sympathetic chain similarly to establish the dorsal plane of dissection into the contralateral hemithorax and bluntly dissect until the contralateral hemithorax is entered, often by visualization of the movement of the lungs during ventilation. Use straight grasping forceps to retract and stabilize the mediastinum during these incisions.* Significant ventral retraction of the aorta using closed, curved forceps may be required to aid dissection ventrolateral to the aorta and thus ventral to the TD bilaterally. *Use curved dissecting forceps to dissect across the mediastinum just dorsal to the aorta, and then dissect ventrolateral to include aberrant branches of the TD. Then, dissect dorsal to the TD in a plane of dissection ventral to the sympathetic*

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trunk. Once the TD and its branches are isolated, replace the forceps in the clip portal with a 10-mm hemostatic clip applier. Use curved dissecting forceps in the instrument portal to retract the aorta ventrally for observation of the structures before applying clips. Apply clips with inward pressure across visible branches of the TD. If you cannot identify individual branches, place the clips on the dissected tissue in an en bloc fashion. If possible, verify complete TD ligation after clip application with lymphangiography of a mesenteric lymphatic vessel (see p. 945) or coloration of a popliteal lymph node with methylene blue. For the latter, incise the skin and subcutis over the popliteal lymph node, allowing access to the afferent, or convex, surface of the lymph node. Use a butterfly catheter and syringe with dilute methylene blue (1:60 with saline) to slowly infuse the lymphatics. Place the needle in the medulla of the lymph node and gradually inject dilute methylene blue until the TD is dilated and colored blue. Clip or ligate with suture branches noted to bypass the ligation site. Perform a partial pericardiectomy. Close the incisions routinely.

NOTE Wear magnification and use a headlight to help visualize the thoracic duct and its small branches!

Active Pleuroperitoneal or Pleurovenous Shunting

Commercially made shunt catheters (see Suture Materials and Special Instruments) are available and can be used to pump pleural fluid into the abdomen (Fig. 30.32) or into a vein (i.e., jugular, azygous, or caudal vena cava). Two types of shunts are available: a pleuroperitoneal shunt (Box 30.9) and an ascites (peritoneovenous) shunt (Box 30.10). The latter is meant to pump fluid from the abdomen into a vein and does not require manual pumping (i.e., it functions in an active fashion). This shunt can be placed from the pleural space into a vein (pleurovenous); when used in this manner, manual pumping is required (the shunt will not function in an active fashion). Close observation of these patients for several weeks after pleurovenous shunt placement is necessary, and preoperative heparinization and maintenance on heparin, aspirin, or other anticoagulants may be warranted. Other complications are discussed later. Both types of catheters are placed under general anesthesia.

BOX 30.9 Pleuroperitoneal Shunt Specifications

27-cm fenestrated pleural end
Two one-way valves
49-cm fenestrated peritoneal catheter
Each complete pump of the reservoir dome transferring 1.5 mL of fluid

Data from Denver Biomaterials, Inc.

BOX 30.10 Pleurovenous (Peritoneovenous) Shunt Specifications[a]

- 27-cm fenestrated pleural end
- Single or double one-way valves
- 66-cm fenestrated venous catheter
- Double valve: comes in a standard flow rate (26–40 mL/min at 10-cm head of water) and a low flow rate (<26 mL/min at 10-cm head of water)

[a]This shunt comes in a single- and double-valve form; the double-valve catheter is indicated when the shunt is to be placed in a pleurovenous fashion.
Data from Denver Biomaterials, Inc.

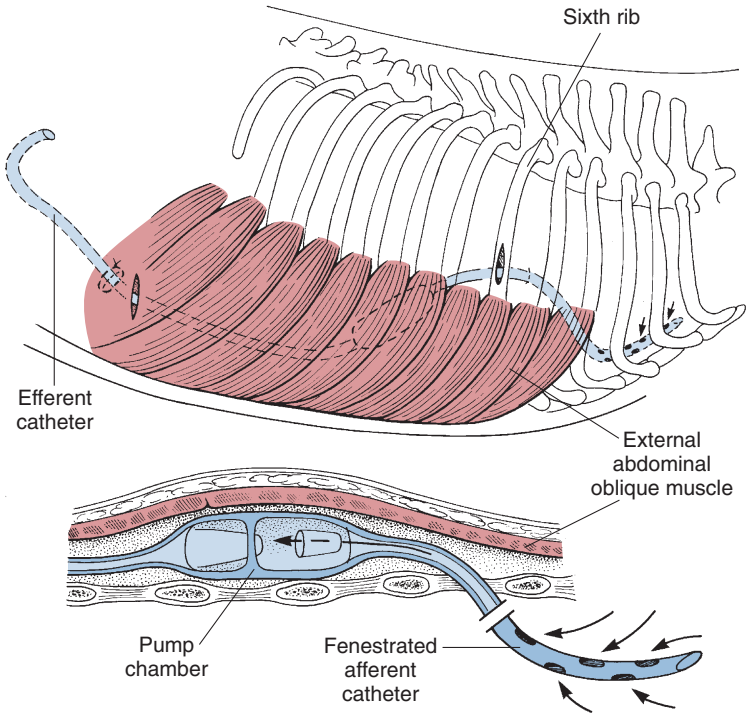


FIG. 30.32 To place a pleuroperitoneal shunt, put the afferent end of the catheter into the thoracic cavity and the efferent end into the abdominal cavity. Make sure the pump chamber overlies a rib so that the chamber can be compressed effectively. Arrows show the direction of the flow of fluid.

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Place the pump chamber and tubing in a bowl of sterilized, heparinized saline. Prime the pump by compressing the valve repeatedly until the system is filled with fluid and flow is established. Expel any remaining air bubbles from the tubing or valve. Make a vertical incision over the middle of the sixth, seventh, or eighth rib. Bluntly insert the pleural end of the shunt catheter into the thoracic cavity. For a pleuroperitoneal shunt, use blunt dissection to create a tunnel under the external abdominal oblique muscle and pull the pump chamber through the tunnel. Place the efferent (peritoneal) end of the catheter into the abdominal cavity just caudal to the costal arch through a small skin incision and a preplaced purse-string suture in the abdominal musculature. For a pleurovenous shunt, tunnel the efferent (venous) end of the catheter over the shoulder to the ventral cervical region. Make a small incision over the jugular vein and insert the venous end of the catheter into the vein. Using fluoroscopy, place the distal end of the catheter at the caudal aspect of the cranial vena cava, just proximal to the right atrium (the venous end of the catheter may be shortened if necessary). As an alternative, the venous end of the catheter may be placed in the azygous or caudal vena cava through an abdominal incision. *Make sure the pump chamber overlies a rib so that the chamber can be effectively compressed.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

The advantage of using hemoclips is that they can be used as a reference point on subsequent radiographs if further ligation is necessary. However, it is best to also ligate the duct with nonabsorbable suture (e.g., silk) if hemoclips are used. Shunts for active drainage include the Denver double-valve pleurovenous and pleuroperitoneal shunts.

POSTOPERATIVE CARE AND ASSESSMENT

If chylothorax resolves spontaneously or after surgery, periodic reevaluation for several years is warranted to detect recurrence. Fibrosing pleuritis is the most common serious complication of chronic chylothorax (see p. 942). Immunosuppression may occur in patients undergoing repeated and frequent thoracentesis because of lymphocyte depletion.

COMPLICATIONS

The most common complication of TD ligation is continued chylous or serosanguinous effusion. With proper technique, the incidence of continued chylous effusion is minimal. Unfortunately, when a serosanguinous effusion occurs after surgery, further surgery involving the TD is of no benefit. Somatostatin (see p. 944), pericardectomy, or a pleuroperitoneal shunt should be considered.

The most common complication of pleurovenous shunt placement is shunt occlusion (Fig. 30.33). Conscientious pumping of the catheter multiple times a day may reduce this complication. It is also less likely to occur if the fluid is serosanguinous rather than chyle. If a clot occludes the pump, injection of streptokinase may help dissolve it. A possible complication of pleurovenous shunt placement is the formation of a right atrial and/or ventricular thrombus. This complication may be life-threatening; therefore pleuroperitoneal shunting is preferred if there is no reason to believe that the animal may not reabsorb the fluid from its abdominal cavity (e.g., the presence of diffuse lymphatic disease or cardiac disease).

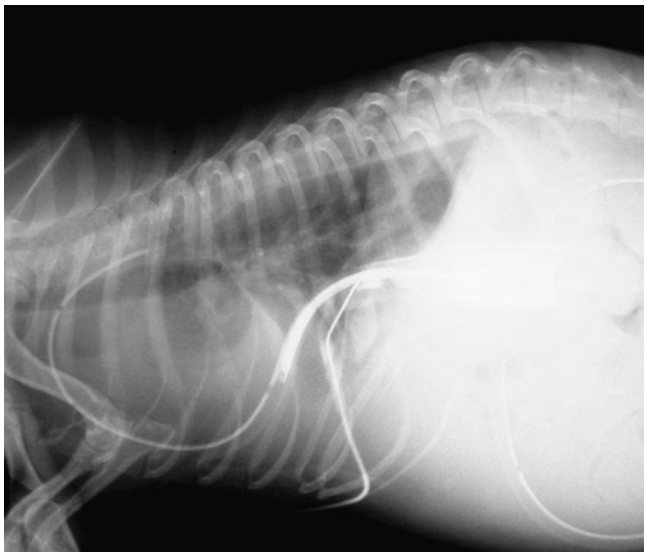


FIG. 30.33 Radiograph of a dog with a pleuroperitoneal shunt in which contrast has been injected into the catheter just proximal to the pump chamber. The inflow catheter has a large, occluding clot in it. (Courtesy Dr. Blaine Andrews.)

PROGNOSIS

This condition may resolve spontaneously or after surgery. Untreated or chronic chylothorax may cause severe fibrosing pleuritis and persistent dyspnea. Euthanasia is frequently performed in animals that do not respond to surgery or medical management. A treatment of TD ligation plus pericardectomy resolves pleural effusion in most animals if properly performed. Continued chylous effusions are typically due to technical failures in completely ligating the duct and its branches. Postoperative serosanguinous fluid occurs in some dogs after surgery; octreotide may be beneficial in these animals. If octreotide is unsuccessful, a pleuroperitoneal shunt may be indicated.

PYOTHORAX

DEFINITION

Pyothorax *(thoracic empyema)* is suppurative inflammation of the thoracic cavity with a resultant accumulation of pus.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The route by which the thoracic cavity becomes infected is not usually evident (e.g., hematogenous spread; migrating foreign objects such as plant awns; penetrating wounds, particularly bite wounds; extension from discospondylitis; extension from pneumonia; pulmonary neoplasia or abscessation; pulmonary or thoracic wall trauma; esophageal perforation; and postoperative infection). Immunosuppressive diseases (e.g., feline leukemia virus and feline immunodeficiency virus) should be excluded in animals with pyothorax, but there is no evidence that development of pyothorax requires debilitation or an increased susceptibility to infection. Aspirations of oral flora may be a common mechanism of pleural space infection in cats, whereas thoracic puncture (e.g., bite wounds) may be a less common cause of

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TABLE 30.9 Morphologic Characteristics and Typical Antibiotic Susceptibilities of Bacteria Commonly Associated With Pyothorax in Small Animals

Organism	OXYGEN Requirements	Gram Stain	Antibiotic Sensitivity[a]
Actinomyces spp.	Facultative anaerobe to strict anaerobe	Gram positive	**Ampicillin, amoxicillin +/– clavulanic acid, penicillin G,** clindamycin, chloramphenicol, erythromycin, minocycline
Bacteroides spp.	Obligate anaerobe	Gram negative	Most *Bacteroides* spp.: **ampicillin, amoxicillin plus clavulanic acid,** clindamycin, chloramphenicol, metronidazole *Bacteroides fragilis*: **amoxicillin plus clavulanic acid,** clindamycin, metronidazole, chloramphenicol
Clostridium spp.	Obligate anaerobe	Gram positive	Most *Clostridium* spp.: ampicillin, **amoxicillin plus clavulanic acid,** chloramphenicol, **metronidazole** *Clostridium perfringens*: **ampicillin, amoxicillin plus clavulanic acid,** cefoxitin, clindamycin, chloramphenicol, metronidazole
Escherichia coli	Aerobe	Gram negative	**Amikacin, enrofloxacin,** ceftizoxime, cefotaxime, potentiated sulfa drugs
Fusobacterium spp.	Obligate anaerobe	Gram negative	**Ampicillin, amoxicillin plus clavulanic acid,** clindamycin, chloramphenicol, metronidazole
Klebsiella spp.	Facultative anaerobe	Gram negative	**Amikacin, ceftizoxime, cefotaxime, ceftriaxone,** enrofloxacin
Nocardia spp.	Aerobe	Gram positive (partly acid fast)	**Amikacin,** cefotaxime, doxycycline, imipenem,[b] minocycline, **trimethoprim-sulfa**
Pasteurella spp.	Facultative anaerobe	Gram negative	**Ampicillin, amoxicillin plus clavulanic acid, cephalosporins,** aminoglycosides
Pseudomonas spp.	Aerobe	Gram negative	**Amikacin, ceftazidime, enrofloxacin**

[a]Drug names in boldface type are the typical drugs of choice.
[b]Considered a drug of last resort; see p. 82.

pyothorax than previously believed. Direct extension from pulmonary disease may be a cause in many animals. In a 2013 report, 6.5% of dogs undergoing thoracic surgery developed postoperative pyothorax, and this complication resulted in death in 66.7% of the affected dogs.[26]

A number of organisms are often cultured from animals with pyothorax; however, there is a high incidence of obligate anaerobes as sole pathogens (Table 30.9). Obligate anaerobic infections or gram-positive filamentous organisms (e.g., *Nocardia* and *Actinomyces* spp.) are frequently cultured from dogs with pyothorax (Fig. 30.34); obligate anaerobes and/or *Pasteurella* spp. are the most common feline isolates.[27]

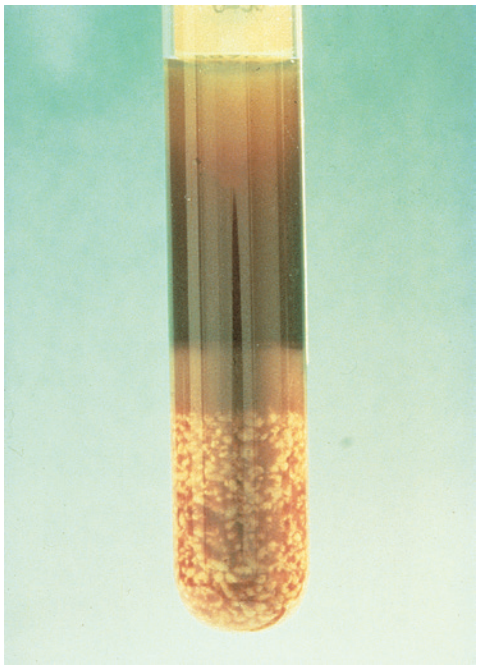


FIG. 30.34 Nocardial pleural effusion.

DIAGNOSIS

Clinical Presentation

Signalment

There is no breed predisposition, and pyothorax may occur in animals of any age. It has been widely held that young, male cats that fight and receive thoracic wounds are at increased risk; however, more recently the parapneumonic spread of infection after colonization and invasion of lung tissue by oropharyngeal flora has been suggested to be the most frequent cause of feline pyothorax. Cats from multicat households appear to be at increased risk. Adult, large-breed dogs (particularly hunting dogs) may be more commonly affected because they often inhale plant foreign material and suffer penetrating thoracic wounds.

History

A delay of several weeks between the trauma that induced the pyothorax and the onset of clinical signs is not uncommon. Most animals are presented for evaluation of respiratory distress or anorexia or both. Coughing is a common presenting complaint in cats with pyothorax.

Physical Examination Findings

Affected animals usually have a restrictive respiratory pattern (i.e., rapid, shallow respirations), and many are febrile. Additional

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findings in patients with pyothorax may include depression, anorexia, weight loss, dehydration, muffled heart and lung sounds, and pale mucous membranes. The thoracic wall may seem incompressible in cats with thoracic effusion.

Diagnostic Imaging

Thoracic radiographs usually reveal pleural effusion (see p. 938). The cause of the pyothorax is seldom apparent radiographically; however, increased opacity in the thoracic cavity after thoracentesis may indicate an abscess or foreign body. Consolidated lung lobes that do not reexpand after fluid removal may indicate fibrosing pleuritis (see p. 942) or lung lobe torsion (see p. 905).

Laboratory Findings

Neutrophilia (with or without a degenerative left shift) may be present on a complete blood cell count. Fluid analysis is necessary to differentiate pyothorax from other exudative effusions. The fluid can range from amber to red or white (in some cases it is initially mistaken for blood). The protein content is usually greater than 3.5 g/dL, and the fluid appears turbid or opaque because of the high nucleated cell count. Nucleated cells consist primarily of degenerative neutrophils (Fig. 30.35), but nondegenerative neutrophils can predominate, depending on the causative agent and prior antibiotic therapy. Effusions associated with fungi and higher bacterial agents such as *Actinomyces* and *Nocardia* spp. are often characterized cytologically by nondegenerative neutrophils and macrophages or may appear hemorrhagic. Macrophages and reactive mesothelial cells are present in purulent effusions in variable numbers, depending on the cause and the chronicity of the fluid. Bacteria are often found, and filamentous rods are suggestive of *Nocardia* spp. or *Actinomyces* spp., as well as *Filifactor villosus*. Performing cytology of any "sulfur granules" in the fluid (yellow masses that resemble sulfur) increases the chance of finding *Nocardia* and *Actinomyces*. Occasionally, fungal elements may be noted in the pleural effusion of animals with fungal disease involving the pulmonary parenchyma. Positive culture results may not be obtained in all animals with pyothorax, particularly if anaerobic organisms are present. Hyponatremia, hypochloremia, hypoalbuminemia, and increased serum total bilirubin concentrations and aminotransferase activity may occur in cats with pyothorax.

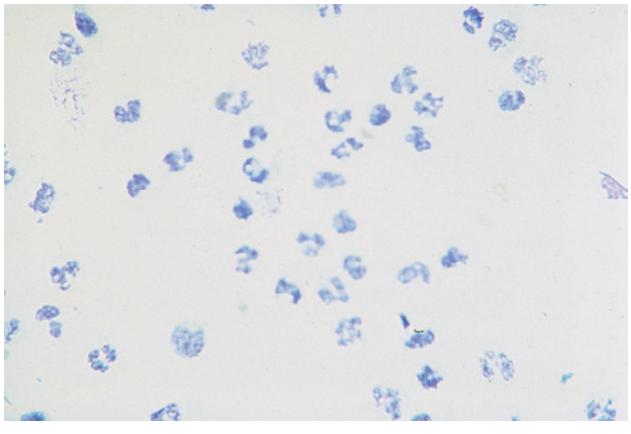


FIG. 30.35 Cytologic study of pleural fluid from a cat with pyothorax. Note the predominance of degenerative neutrophils.

DIFFERENTIAL DIAGNOSIS

Any cause of dyspnea is a differential diagnosis (i.e., cardiac disease, pulmonary disease, mediastinal neoplasia, diaphragmatic hernia). Once pleural effusion has been diagnosed, other causes of exudative effusion (e.g., chylothorax, feline infectious peritonitis) or transudative effusion (e.g., hypoalbuminemia, congestive heart failure) must also be considered. Foul-smelling fluid suggests anaerobic bacteria.

MEDICAL MANAGEMENT

The approach to treatment for pyothorax remains controversial. Reported treatment of pyothorax has included single thoracentesis plus long-term antibiotics or intermittent or continuous thoracic drainage plus thoracic lavage and long-term antibiotic therapy. Despite all of the available options, the optimal treatment to ensure successful short- and long-term outcome, including the avoidance of recurrence, remains unknown.

Although the cause of the effusion is often not discernible, attempts should be made to find and, if possible, correct underlying diseases. Management of these animals needs to be aggressive (Box 30.11). After diagnosis, placement of a thoracostomy drain should be considered. If available, continuous suction devices can be used; however, most animals can be managed with intermittent aspiration. Lavage should be performed two or three times daily. Isotonic fluid, such as saline or lactated Ringer's solution (warmed to room temperature), should be used at a dosage of 20 mL/kg of body weight. The fluid remains in the thoracic cavity for 1 hour and is then removed. Addition of antibiotics to the lavage fluid offers no advantage over the use of appropriate systemic antibiotics. If antibiotics are used in the lavage fluid, the systemic dose should be reduced to minimize toxicity. The use of proteolytic enzymes is controversial and is no longer recommended by most authors. However, the addition of heparin (1500 U/100 mL of lavage solution, up to 10 mL/kg, or administering 100 U/kg subcutaneously every 8 hours) appears beneficial. Lavage may be required for 5 to 7 days. Systemic antibiotic therapy should be based on the results of microbial culture and sensitivity testing (see Table 30.9).

The prevalence of anaerobic infections is high (discussed previously), and a combination of drugs that are active against obligate anaerobic bacteria (e.g., ampicillin, clindamycin, amoxicillin-clavulanic acid, or metronidazole) and facultative bacteria, especially *Escherichia coli* (e.g., amikacin, enrofloxacin, ceftizoxime, or potentiated sulfa drugs), should be used in dogs. In cats, *Pasteurella* spp. and anaerobes are commonly isolated and penicillin and its derivatives (e.g., ampicillin,

BOX 30.11 Management of Animals With Pyothorax

1. Perform Gram stain and culture and sensitivity of fluid and initiate broad-spectrum antibiotic therapy (see text and Table 30.9).
2. Place one or more thoracostomy tubes and lavage thoracic cavity with 20 mL/kg of body weight of warmed, isotonic fluid with heparin added (1500 U heparin/100 mL lavage solution; up to 10 mL/kg).
3. Alter antibiotic therapy based on culture and sensitivity results (but treat for anaerobes even if none are grown in culture), and continue antibiotics for 4 to 6 wk, and 2 wk beyond apparent clinical resolution.

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BOX 30.12 Selected Antibiotics for Use Against *Nocardia* spp. and *Actinomyces* spp.[a] in Dogs

Actinomyces

Ampicillin
22 mg/kg IV q6–8h

Clindamycin
11–33 mg/kg q12h PO, or 11 mg/kg IV q12h (dilute and give slowly when administering IV)

Doxycycline
5 mg/kg PO q12h

Chloramphenicol
40–50 mg/kg PO q8h

Amoxicillin[b]
20–40 mg/kg SC, PO q6h

Nocardia

TMS (Tribrissen; First Choice)[c]
45–60 mg/kg PO q12h

Amikacin[d]
20–25 mg/kg IV, SC q24h[e]

[a]Treat for a minimum of 6 wk, often longer.
[b]May not be effective against L-phase variants.
[c]Observe for side effects (e.g., anemia, thrombocytopenia, hepatic disease, arthritis, vasculitis, keratoconjunctivitis sicca).
[d]May be used if sensitivity shows resistance to TMS; can be used in conjunction with TMS.
[e]Monitor periodically for renal dysfunction. Do not use in combination with nonsteroidal antiinflammatory drugs or other potentially nephrotoxic drugs. Do not give to dehydrated patients.
IM, Intramuscular; *IV,* intravenous; *PO,* orally; *SC,* subcutaneous; *TMS,* trimethoprim-sulfadiazine.

amoxicillin-clavulanic acid) may be indicated while awaiting results of culture and susceptibility testing. Antibiotics should be continued for a minimum of 4 to 6 weeks. Recommended antibiotics for *Actinomyces* and nocardial infections are provided in Box 30.12.

In animals where medical management fails, surgery to identify and remove necrotic tissue and/or foreign bodies, remove adhesions that cause fluid loculation, and if necessary help ensure proper placement of thoracostomy tubes should be considered.

NOTE *Escherichia coli* isolates appear to be less sensitive to enrofloxacin than in the past; this should be considered when choosing antibiotics before receiving the results of culture and sensitivity testing.

SURGICAL TREATMENT

Surgery is indicated in animals that have underlying disease (e.g., lung abscess, lung lobe torsion, foreign body) and in those that do not respond to medical management in 3 to 4 days. If pyothorax is chronic or localized, or if the patient remains dyspneic in the absence of significant volumes of pleural fluid, then fibrosing pleuritis may be present (see p. 942). Surgical exploration of the thoracic cavity may be warranted in such patients.

Preoperative Management

Thoracentesis should be performed before induction of anesthesia if the animal is dyspneic. The animal should be normally hydrated, and significant electrolyte and acid-base abnormalities should be corrected before surgery.

Anesthesia

See Table 30.5 for anesthetic recommendations for patients with pleural effusion undergoing thoracotomy.

Surgical Anatomy

See p. 918 for a description of the pleural space and fluid movement in normal animals.

Positioning

See the description of thoracic procedures beginning on p. 889.

SURGICAL TECHNIQUE

Approach the thorax by an intercostal thoracotomy (see p. 890) if an abnormality can be localized to one hemithorax or by a median sternotomy (see p. 891) if localization is not possible. Explore the thoracic cavity for abscesses, foreign bodies, or other abnormalities and remove affected tissues. If possible, remove fibrin covering the lung tissues. Submit appropriate samples for microbiologic examination and culture. Place a thoracostomy tube for postoperative lavage. Before closure, lavage the thoracic cavity with warmed, sterile saline solution.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Braided, nonabsorbable, multifilament suture (e.g., silk) should not be used for lobectomy or partial lobectomy in these patients. Absorbable suture (e.g., polydioxanone, polyglyconate) is preferred if infection is present.

POSTOPERATIVE CARE AND ASSESSMENT

Thoracic lavage should be continued postoperatively until the infection resolves (discussed previously). Serum protein and electrolytes (e.g., potassium) should be monitored and fluid therapy continued until the animal is eating and drinking normally. Antibiotic therapy should be continued for at least 4 to 6 weeks and for 2 weeks beyond apparent resolution of the disease (discussed previously). Refer to the postoperative management of animals undergoing thoracotomy procedures (p. 897) for additional recommendations.

PROGNOSIS

The prognosis for most animals with pyothorax is good if they are managed as described previously. A review of the current veterinary literature revealed an overall reported survival rate of 83% in dogs and 62% in cats.[28] Recurrence is common in animals treated with antibiotics only (i.e., without thoracic lavage). Long-standing empyema may resorb, leaving a pleural "peel," which is a thick sheet of fibroblasts and inflammatory cells attached to the visceral pleura. This pleural peel may inhibit normal expansion of lung tissue (fibrosing pleuritis; see p. 942). If multiple lung lobes are fibrosed and cannot expand normally, the prognosis may be poor. Most cats with pyothorax can be

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treated successfully with thoracostomy drainage and lavage; hypersalivation, possibly associated with pain, may be a negative prognosticator. Interestingly, bradycardia may be a negative predictor for survival in cats, similar to what has been reported in cats with sepsis. Most cats that die or are euthanized do so within the first 48 hours after presentation. Management with a thoracostomy tube and thoracic lavage with heparin and systemic antibiotics may result in fewer recurrences than single pleural drainage with or without ultrasound guidance plus antibiotics.

THYMOMAS, THYMIC BRANCHIAL CYSTS, AND MEDIASTINAL CYSTS

DEFINITIONS

Thymomas are tumors that arise from epithelial tissues of the thymus. **Thymic branchial cysts** develop from vestiges of the fetal branchial arch system. **Mediastinal cysts** are fluid-filled structures located in the cranial mediastinum that are typically benign.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Masses in the mediastinum of dogs and cats are often neoplastic, although abscesses, granulomas, and cysts are occasionally found. Cranial mediastinal cysts have been reported in cats. The majority of affected cats had no evidence of respiratory disease. The masses were all benign, and the cats remained asymptomatic for 3 to 45 months after diagnosis. Thus many older cats presenting with cystic mediastinal masses have benign lesions requiring no treatment. Although opacities in the cranial mediastinum on thoracic radiographs should be sonographically investigated, asymptomatic cats with cystic lesions may only need serial evaluation with radiography or ultrasound.

Lymphoma is the most common cranial mediastinal tumor in dogs and cats. Other tumors occasionally found here include thymomas, chemodectomas (aortic and carotid body tumors), and ectopic thyroid and parathyroid tumors. Thymomas are the most common surgically treatable neoplasm of the cranial mediastinum in dogs; most are benign. However, because the histologic appearance of the tumor correlates poorly with clinical behavior, the terms *invasive* or *noninvasive* are preferred. Stage I (i.e., noninvasive) thymomas are well circumscribed and do not extend beyond the thymic capsule (Table 30.10). Others may extend beyond the capsule locally or may invade surrounding organs or metastasize to other thoracic or extrathoracic structures. Thymic epithelial tumors in dogs were recently evaluated histologically and subclassification of these tumors was found to be similar to that described in humans.[29]

The clinical signs associated with thymomas may be due to occupation of space and/or paraneoplastic syndrome. Thymomas may cause respiratory distress by compressing the lungs or trachea and/or inducing pleural effusion as they enlarge. Effusions associated with thymomas may be serosanguinous or chylous. Paraneoplastic syndromes are distant effects of a tumor. In a 2013 study, 20 of 116 (17%) dogs with thymoma had signs of myasthenia gravis (MG).[30] MG is an autoimmune neuromuscular disorder characterized by muscular weakness (see Chapter 44). The weakness is due to a deficiency of functional acetylcholine receptors in the neuromuscular postsynaptic membrane caused by autoantibodies that bind to and block the receptors. Acquired MG in cats is most commonly associated with a mediastinal mass (thymoma) but has been reported in cats after methimazole treatment for hyperthyroidism (because of the immunomodulatory properties of the medication).[31] In the aforementioned study, at the time of thymoma diagnosis 40 (34%) dogs had hypercalcemia, 8 (7%) dogs had a concurrent immune-mediated disease, and 31 (27%) dogs had another tumor; 16 (14%) dogs developed a second nonthymic tumor at a later date.[30] As thymomas enlarge, they may compress the cranial vena cava and other cranial thoracic vessels, causing edema of the head, neck, and/or forelimbs (cranial vena cava syndrome).

Thymic branchial cysts develop from vestiges of the branchial arch system of the fetus. They may be found in the subcutaneous tissues of the neck or in the thymus. Rupture of these cysts may result in a chronic inflammatory reaction and abscessation.

TABLE 30.10 Staging Scheme for Thymomas

Stage	Description
I	Growth completely within intact thymic capsule
II	Pericapsular growth into mediastinal fat tissue, adjacent pleura, and/or pericardium
III	Invasion into surrounding organs and/or intrathoracic metastasis
IV	Extrathoracic metastasis
Paraneoplastic Syndromes	
P_0	No evidence of paraneoplastic syndrome
P_1	Myasthenia gravis
P_2	Nonthymic malignant tumor

Modified from Aronson M. Canine thymoma. *Vet Clin North Am Small Anim Pract.* 1985;15(4):755–767.

DIAGNOSIS

Clinical Presentation

Signalment

The average age of dogs with thymomas is 8 to 9 years; however, thymomas have been reported in dogs as young as 3 years. In a 2013 study of 116 dogs with thymoma, 44 (38%) were Labrador retrievers or golden retrievers.[30] Large-breed dogs, particularly German shepherds, golden retrievers, and Labrador retrievers, are more commonly affected than small dogs. A gender predisposition has not been identified. Most cats with thymomas have been older than 8 years. There appears to be an increased risk for developing acquired MG in the Akita, Scottish terriers, German shorthaired pointers, and Chihuahuas.[31]

Abyssinian and Somali cats had an increased incidence of MG compared with mixed breed cats or cats of other breeds.[31] There is no sex predilection in cats. Thymic branchial cysts also occur most commonly in middle-aged and older animals; however, dogs as young as 18 months of age have been affected. Mediastinal cysts are found in older cats.

NOTE Even though the thymus involutes with age, both thymomas and thymic branchial cysts occur in middle-aged and older dogs.

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History

Dogs with thymomas may be presented because of dyspnea, coughing, weight loss, lethargy, dysphagia, muscle weakness, vomiting and/or regurgitation, excessive salivation, and/or neck edema. The onset of clinical signs may be acute, despite relatively slow tumor enlargement. Lethargy, anorexia, dyspnea, and pleural effusion are common in cats with thymoma. Occasionally thymomas are fortuitous findings on thoracic radiographs of asymptomatic animals. Clinical findings in dogs and cats with thymic branchial cysts are similar to those in animals with thymomas. Most are presented for evaluation of progressive dyspnea. Lameness and swelling of the head, neck, and forelimbs are also common. Dogs with MG usually have generalized weakness or just esophageal weakness (see p. 1460). The most common clinical sign of MG in cats is generalized weakness without megaesophagus.[31] Dogs often have spontaneous remission of MG; this is not a characteristic of MG in cats.[31]

Physical Examination Findings

Clinical findings in dogs with thymomas vary among patients. Respiratory abnormalities caused by pleural effusion or aspiration pneumonia may be the predominant finding. Other animals may be presented for evaluation of generalized exertional weakness without evidence of respiratory problems. Occasionally, localized forms of myasthenia are found in which the weakness is limited to the esophagus, larynx or pharynx, and/or facial musculature. The most common clinical findings in dogs and cats with thymic branchial cysts are dyspnea and pleural effusion.

NOTE Be sure to evaluate all dogs with thymomas for myasthenia gravis and esophageal weakness.

Diagnostic Imaging

Animals with mediastinal masses may show dorsal elevation of the trachea and caudal displacement of the heart on lateral thoracic radiographs (Fig. 30.36). The mediastinum may appear widened on the ventrodorsal view, and the heart may be deviated laterally. Pleural effusion is commonly associated with invasive tumors, and pneumothorax is rare. Megaesophagus or secondary aspiration pneumonia (or both) may be noted on thoracic radiographs. Ultrasonography is often helpful for assessing mediastinal masses and ruling out extrathoracic metastasis. Ultrasound-guided aspirates or biopsy may also be performed. Ultrasonographic evaluation of a mediastinal cyst usually reveals a thin-walled structure with anechoic fluid in the center. Routine CT may not detect vascular invasion of cranial mediastinal masses; contrast-enhanced CT angiograms should be performed. One study showed that many thymomas (57.1%) were sonographically cystic in appearance and nearly all were heterogeneous (94%) in echogenicity compared with lymphoma ($P = .0028$). Lymphomatous lymph nodes were more likely to be solid (80%) and were equally divided between hypoechoic (47%) and heterogeneous (53%) echogenicities. Sonographic findings of either internal cysts or a heterogeneous echogenicity in mediastinal masses may be suggestive of a thymoma.[32]

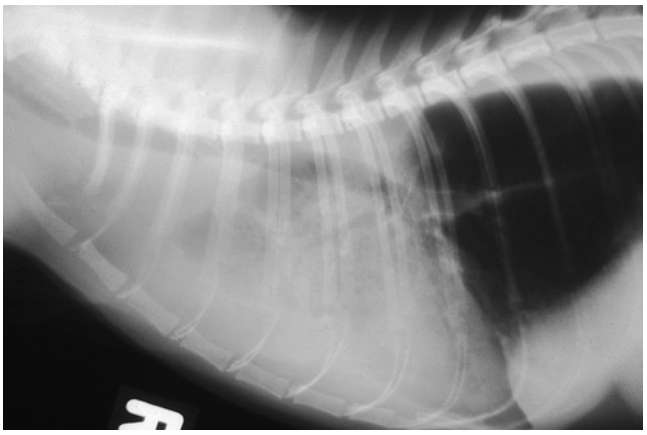


FIG. 30.36 Radiograph of a cat with a well-delineated thymoma. Note the dorsal elevation of the trachea, indicative of a mediastinal mass.

Laboratory Findings

Specific laboratory abnormalities are not found with thymoma or branchial cysts. Leukocytosis may be present if the animal has aspiration pneumonia. Pleural effusion associated with thymoma or thymic branchial cysts may contain mature lymphocytes; however, immature lymphocytes indicate lymphoma. Both lymphoma and thymoma may cause chylous effusions (see p. 941). Cytologic examination of canine and feline mediastinal masses appears to correlate well with final histologic diagnoses.[33]

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for cranial mediastinal masses include both neoplastic and nonneoplastic disease (Box 30.13). The most important differential diagnosis is mediastinal lymphoma because treatment for lymphoma does not involve surgery. Therefore a definitive diagnosis should be made before surgery whenever possible. The presence of hypercalcemia suggests lymphoma, whereas MG or megaesophagus suggests thymoma. However, hypercalcemia has rarely been reported in conjunction with thymoma in dogs. A definitive diagnosis of lymphoma may be made by fine needle aspiration or transthoracic needle biopsy of the mass and/or pleural fluid evaluation. Using ultrasound to pick the site for biopsies reduces the risk of perforating the cranial vena cava or other vascular structures. If a definitive diagnosis cannot be made preoperatively, surgical biopsy with intraoperative cytology or analysis of frozen sections is indicated. The histologic appearance of thymomas does not correlate well

BOX 30.13 Differential Diagnoses for Cranial Mediastinal Masses in Dogs and Cats

Lymphoma
Thymoma
Chemodectoma
Ectopic thyroid or parathyroid tumor
Abscess
Thymic branchial cyst
Schwannoma
Teratoma
Thymic hyperplasia
Granuloma

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with behavior; therefore exploratory thoracotomy is often required to determine if the tumor is invasive.

Acquired MG (see Chapter 44) can be diagnosed by demonstrating circulating antibodies to acetylcholine receptors or by electromyography (i.e., repetitive stimulation). Clinical signs and the response to a cholinesterase inhibitor may help diagnose the disease but are not definitive. When edrophonium chloride is given intravenously, a dramatic but transient improvement in voluntary muscle function occurs in most dogs with MG; however, the test gives false-negative and false-positive results. Furthermore, some dogs can have serious adverse side effects from excessive parasympathomimetic stimulation (e.g., bradycardia, gastrointestinal signs). Pretreatment with glycopyrrolate or atropine is typically recommended, plus access to oxygen. The test is not useful for diagnosing esophageal weakness due to localized MG.

NOTE Be aware of possible complications associated with the use of edrophonium (e.g., possible paralysis), and be prepared to deal with them.

MEDICAL MANAGEMENT

If aspiration pneumonia is present, the dog should be treated with appropriate antibiotics before surgery. Dogs with esophageal weakness often benefit from being fed in an upright, nearly vertical position. Dyspneic animals should have an oxygen-enriched environment plus pleural effusion removed by thoracocentesis. Anticholinesterase therapy (e.g., pyridostigmine bromide) may benefit dogs with clinical problems (e.g., megaesophagus, weakness) caused by MG (Box 30.14). Sometimes immunosuppressive therapy (e.g., mycophenolate, azathioprine) is a useful adjunct in treating myasthenia, but avoid glucocorticoids. Fluid therapy and correction of electrolyte abnormalities may be necessary in animals with severe or frequent regurgitation. Radiation therapy may reduce clinical signs in some animals with thymomas, making surgery unnecessary. Pretreatment with irradiation or chlorambucil may also be done to decrease tumor size prior to surgery.

BOX 30.14 Drugs Used to Treat Acquired Myasthenia Gravis in Dogs

Pyridostigmine Bromide (Mestinon, Regonol; First Choice)
0.5–1.0 mg/kg and adjust dose up as needed, up to 3 mg/kg PO q8–12h to control signs[a]

Azathioprine (Imuran)[a]
2.2 mg/kg PO q24–48h[b]

Mycophenolate (CellCept)
10 mg/kg PO q8–12h[c]

[a]Do not use in patients with gastrointestinal or urinary tract obstructions, cardiac arrhythmias, bronchoconstriction, or pneumonia. Use cautiously in animals receiving potassium bromide. Use only if pyridostigmine does not provide an adequate response, or if its side effects are unacceptable.
[b]Use only if pyridostigmine is ineffective. Watch for myelotoxicity, hepatitis, or pancreatitis. The onset of action is slower if treatment is initiated every other day, but the incidence of side effects is less.
[c]Bloody diarrhea is a major side effect. Has not been shown to be beneficial in outcome.
PO, Orally.

SURGICAL TREATMENT

Long-term survival without thymectomy has been reported in dogs with thymomas. However, surgical removal of stage I or stage II thymomas may be indicated. Concurrent MG makes therapy of thymoma more difficult. Although thymectomy helps resolve MG in many people despite persistent serum antiacetylcholine receptor autoantibody titers, too few dogs with both thymomas and MG have undergone surgery to predict the outcome in these patients. Surgical removal of thymic branchial cysts is indicated.

Preoperative Management

Aspiration pneumonia should be resolved before surgery, and severe muscle weakness should be treated (discussed previously). Before induction of anesthesia, excess pleural fluid should be removed, and fluid and electrolyte abnormalities should be corrected.

Anesthesia

See p. 884 for recommendations for the anesthetic management of patients undergoing thoracotomy procedures. Neuromuscular blocking agents (e.g., atracurium, pancuronium) should be avoided in patients with MG. Patients with megaesophagus should be intubated while positioned sternally rather than laterally, and the endotracheal cuff should be inflated immediately to protect the airway against aspiration. At extubation, the patient should be placed sternally with head down and the endotracheal tube removed when the protective gag reflexes have returned and the patient is fully awake. To prevent aspiration, have suction available prior to intubation and at extubation in the event that removal of esophageal fluid is necessary.

Surgical Anatomy

The thymus is derived from the third and fourth pharyngeal pouches adjacent to the primordial cells of the thyroid gland and migrates caudally into the cranial mediastinum. It reaches maximum size in the dog at approximately 4 or 5 months. The thymus is bordered dorsally by the cranial vena cava and trachea. It is closely associated with many smaller blood vessels (e.g., branches of the brachycephalic trunk and internal thoracic arteries), which often require ligation during thymectomy. The phrenic nerve is closely associated with the dorsal border of the thymus.

Positioning

Depending on the surgical approach chosen (discussed later), either the left thorax or the ventral thorax should be prepared for aseptic surgery.

SURGICAL TECHNIQUE

Thymectomy may be performed through a left third or fourth space intercostal thoracotomy if the tumor is small or through a cranial median sternotomy (see p. 891). If the mass is large, a median sternotomy approach allows better visualization of surrounding structures such as the cranial vena cava. Small, encapsulated thymomas can usually be removed without difficulty, but cytoreduction is often all that is possible with large, invasive tumors. Thymomas are often friable and occasionally cystic, and they should be handled with care to avoid seeding the thoracic cavity with tumor cells. Thymic branchial cysts appear as multilobulated masses containing numerous cysts on transverse

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section. Video-assisted thoracoscopic resection of noninvasive thymomas is possible.

NOTE If the tumor adheres to or closely surrounds the cranial vena cava, temporary occlusion of this vessel may facilitate surgery. Permanent ligation of the cranial vena cava may cause chylothorax.

Explore the thoracic cavity for evidence of metastasis. Identify the cranial vena cava and other associated vessels. Locate the phrenic nerve and preserve it if possible. Ligate small vessels and bluntly dissect the mass and its capsule from surrounding tissues. Try to maintain the integrity of the thymic capsule. If complete removal of the mass is not possible, remove as much as can safely be excised. Submit tissues for histologic examination. Insert a thoracostomy tube before thoracic closure.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Electrocautery is useful when removing thymomas and other vascular neoplasms. See also the recommendations for thoracotomy on p. 896.

POSTOPERATIVE CARE AND ASSESSMENT

Animals with thymomas are at great risk of aspiration during the postoperative period; positioning them with the head elevated may reduce the risk. In addition, suctioning the pharynx before extubation and extubating with the cuff slightly inflated reduces the risk of aspiration if passive regurgitation has occurred during surgery. The animal should be observed for hemorrhage and pneumothorax postoperatively. Adjuvant radiation therapy may benefit animals with invasive tumors that cannot be completely excised. The animal should be closely observed for paraneoplastic disease after therapy. Analgesics should be provided postoperatively in these patients (see Chapter 13; for dosages of opioids, see Table 13.2). With thymomas, the thoracostomy tube can generally be removed within 24 hours if hemorrhage or pneumothorax does not occur. Longer-term tube thoracostomy may be necessary in animals with thymic branchial cysts if rupture of a cyst has caused pleuritis.

PROGNOSIS

The prognosis depends on the invasiveness of the tumor, its size at the time of diagnosis, and the presence or absence of paraneoplastic disease. Most cats will survive 1 year (approximately 90%) and many (approximately 75%) will survive at least 3 years after surgery. A second surgery is warranted in animals with tumor regrowth, and long-term survival (3–5 years) is possible. In a 2013 study of 116 dogs with thymoma, tumor excision was performed for 84 dogs, 14 (17%) of which had tumor recurrence. Median survival time with or without surgical treatment was 635 and 76 days, respectively. Presence of another tumor at the time of thymoma diagnosis, lack of surgical excision, and higher pathologic stage were significantly associated with shorter survival time. Hypercalcemia and presence of MG or megaesophagus at the time of thymoma diagnosis, histopathologic subtype of thymoma, or tumor development at a later date was not associated with survival time.[30] The prognosis for thymic branchial cysts is good.

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31 Principles of Orthopedic Surgery and Regenerative Medicine

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Circumduction is an external swinging of the limb. **Hypermetria** is characterized by a dysfunction of the power to control the range of muscular action which results in movements that are exaggerated or that overreach the intended goal. **Proprioceptive deficits** are abnormal body positions or movements due to a lack of normal perception. Loss of **proprioception** causes abnormal placement reactions in the limbs (legs), abnormal limb position at rest (e.g., paws turned under), and abnormal wearing of the toes. Other definitions are provided in Box 31.1.

PROBLEM IDENTIFICATION

Patients with orthopedic problems represent a significant percentage of the general practice population. Common categories of orthopedic diseases in small animals include (1) bone fractures and malformations (see Chapters 32 and 33); (2) traumatic, developmental, and degenerative joint diseases (see Chapter 34); (3) infectious and immune-mediated joint diseases (see Chapter 34); (4) musculoskeletal neoplasia (see Chapter 36); and (5) tendon, ligament, and muscle injuries (see Chapter 35).

Before selecting the appropriate method for treating an orthopedic problem, it must first be properly diagnosed. Most animals present with obvious lameness and pain, but identifying the cause of the lameness may be difficult. Accurate historical information, thorough general examination, and a complete orthopedic examination are essential for an accurate diagnosis. The importance of a complete, careful, and accurate orthopedic examination cannot be overemphasized because the findings of the orthopedic examination are the most important components for diagnosing orthopedic disease.

DIFFERENTIAL DIAGNOSIS

A differential diagnosis is developed based on results of signalment, history, and physical examination; other tests or procedures may be necessary to define the disease once the aforementioned findings have narrowed the diagnostic possibilities. Additional diagnostics include imaging, joint fluid analysis, infectious disease panels, cytology, and biopsy.

PATIENT SIGNALMENT AND HISTORY

Many orthopedic diseases are more common in certain age groups and breeds, and these are outlined in Table 31.1. Whereas developmental diseases and specific neoplasms may be identified in any breed and at any age, the patient signalment typically aids in prioritizing a list of differentials. Patient history is critical in the proper diagnosis of orthopedic diseases (see Chapter 4). In cases of trauma, clients should be asked about preexisting general and orthopedic diseases.

Physical Examination

The animal's general health is assessed by performing a complete physical examination. A general health evaluation is important before anesthetizing any animal that has orthopedic disease. Baseline examination includes obtaining the animal's temperature, pulse, and respiration; noting the animal's overall appearance (e.g., obesity); and performing thoracic auscultation and abdominal and rectal palpation. Neurologic evaluation (see Chapter 38) is also indicated in many cases, particularly in animals who have sustained recent trauma because they may have simultaneous neurologic and orthopedic injuries; the former injury may be masked by the latter. In animals presenting with gait abnormalities, it is important to differentiate orthopedic and neurologic causes.

Animals presenting for orthopedic evaluation following blunt trauma (automobile trauma, falling injuries) should be evaluated for common comorbidities including (1) cardiovascular shock, hypovolemic shock, and arrhythmias (see Chapter 27); (2) pneumothorax, diaphragmatic hernia, and pulmonary contusions (see Chapter 30); (3) spinal trauma (see Chapters 40 and 41), brain trauma (see Chapter 39), peripheral nerve injuries (see Chapter 44); and (4) urinary bladder rupture (see Chapter 25). Less common blunt trauma injuries include body wall hernias, splenic and hepatic ruptures, and gall bladder rupture (see Chapters 19–21 and 23). Thoracic radiographs and serial electrocardiograms (ECGs) should be strongly considered in animals sustaining significant blunt trauma. Evaluation of the abdominal cavity is done initially with palpation and evaluation of serum chemistry tests. Abdominocentesis (see p. 534) and radiographic or ultrasonographic evaluation of the urinary tract (see p. 698) should be performed if clinical signs (abdominal distention) or chemistry findings (azotemia) suggest injury to the renal system.

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ABSTRACT

Principles of small animal orthopedics include development of excellent orthopedic examination skills and the ability to develop an appropriate differentials list and diagnostic plan.

An understanding of the principles of regenerative medicine will enable the veterinarian to make appropriate recommendations of the use of regenerative medicine and differentiate realistic goals of these techniques from commercial misrepresentations.

KEYWORDS

Orthopedic examination
Orthopedic differentials
Palpation
Stem cells
Platelet-rich plasma

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BOX 31.1 Orthopedic Terms

Allograft—bone transplanted from one animal to another of the same species
Apophyseal osteotomies—procedures performed to enhance surgical exposure of a joint
Autograft—bone transplanted from one site to another in the same animal
Avulsion fracture—occurs when the insertion point of a tendon or ligament is fractured and distracted from the rest of the bone
Bridging plates—span a comminuted fracture
Cerclage wire—orthopedic wire placed around the circumference of the bone and compressing an oblique fracture
Closed reduction—fractured bone alignment performed without a surgical exposure
Compression plates—plates that act to compress the fracture
Corrective osteotomies—elective procedures in which the diaphysis or metaphysis of the bone is cut, realigned, and stabilized until union occurs
Cranial drawer—the abnormal movement elicited during a physical examination that is caused by the tibia sliding cranially in relationship to the femur in the absence of the cranial cruciate ligament
Crepitation—the grating feel or sound associated with manipulating a fractured bone or an arthritic joint
Delayed unions—fractures that heal more slowly than anticipated
Direct bone union—bone formed without evidence of callus
Endochondral bone formation—bone formed on a cartilaginous precursor
External coaptation—fracture fixation using casts or splints
External fixation—fracture fixation in which pins penetrate the bone and skin and are connected externally
Greenstick fracture—an incomplete fracture in which a portion of the cortex is intact
Hemicerclage wire or interfragmentary wire—denotes wire that is placed through predrilled holes in the bone
Indirect reduction—the process of restoring fragment and limb alignment by distracting the major bone segments
Internal fixation—fracture fixation using internal implants to secure the bone
Intramedullary pins—implants that are positioned in the medullary canal of long bones
Intramembranous bone formation—direct differentiation of mesenchymal stem cells into osteoblasts so bone forms without a cartilaginous precursor
Luxation—complete dislocation of a joint
Malunion—healed fracture in which anatomic bone alignment was not achieved or maintained during healing
Neutralization plates—support a reconstructed fracture
Nonunion—a fracture that has an arrested repair process that requires surgical intervention to create an environment conducive to bone healing
Normograde placement—the pin is started at one end of the bone, driven to the fracture area, and then seated at the other end of the bone
Open fracture—one in which the fracture is exposed to the external atmosphere
Open reduction—fracture repair performed after a surgical approach to the bone
Ortolani maneuver—the manipulation used to subluxate a dysplastic hip
Ostectomy—removal of a segment of bone
Osteomyelitis—an inflammatory condition of bone and the medullary canal
Osteotomy—procedure in which the bone is cut into two segments
Procurvatum—cranial bowing of a bone
Recurvatum—caudal bowing of a bone
Reduction—process of reconstructing or realigning a fractured bone
Retrograde placement—the pin is inserted at the fracture area, driven proximally to exit the bone, the fracture is reduced, and the pin is driven distally to seat at the end of the bone
Staged disassembly—process of modifying a fixation frame at approximately 6 weeks after surgery to increase the loading on the healing fracture
Subluxation—partial dislocation of a joint
Valgus deformity—angulation of the distal portion of the limb laterally
Varus deformity—angulation of the distal portion of the limb medially

It is crucial to diagnose and treat these injuries before the animal is anesthetized for fracture repair or joint trauma.

Orthopedic Examination

An orthopedic examination begins by observing the animal standing, sitting, and moving when possible. The patient is then examined by palpation in both standing and recumbent positions when possible.

Standing and Sitting Visual Examination

The patient's conformation (muscle quality, body weight, limb and joint structure) is usually evaluated with the patient standing. Other findings including decreased weight bearing, joint swelling, focal muscle atrophy, limb or joint displacement (internal or external rotation, hyperextension or hyperflexion) and mass effects may also be seen with the patient standing. Commonly observed conformational abnormalities in juvenile dogs include hyperextension of the stifle joints and tibiotarsal joints. These abnormalities may be associated with hip dysplasia or stifle and hock osteochondritis dissecans (OCD) (see Chapter 34).

Observe positioning of the hindlimbs with the dog sitting. Sitting with one leg squarely under the body and the other abducted is often an indication of stifle pain and pathology.

Gait Analysis

Gait abnormalities may be associated with mechanical defects, be due to pain causing lameness, have a neurologic origin, or the gait abnormality may be due to any combination of the three. With experience, observation of a patient while moving aids significantly in differentiating the category of the gait abnormality. *Encourage cats to walk around the examination room to observe for lameness or other gait abnormalities. Walk dogs on a leash at varying speeds (walk, trot) to evaluate for gait abnormalities attributable to orthopedic or neurologic disease.*

Forelimb

Gait abnormalities that may be appreciated in the forelimbs include head nod, circumduction, internal or external rotation, joint collapse, joint hyperflexion or extension, varus or valgus deformity (see Box 31.1), proprioceptive deficits, and hypermetria. Animals with forelimb lameness (painful) will lift their heads as the painful limb strikes the ground in an attempt to remove weight from the affected limb (a head nod), making it appear that they are landing heavily on the opposite, or "good," limb. (A good idiom is "down on the sound.") Animals with bilateral lameness may not limp, but they often show more subtle signs, such as shifting their weight from limb to limb while standing, shortened stride, shaking, and bilateral muscle atrophy. A short stride occurs when the animal has a decreased range of motion of a joint due to pain or mechanical restriction. Circumduction of the affected limb(s) occurs when the animal tries to advance a limb that cannot be adequately flexed. This is often observed in dogs with severe degenerative joint disease of the elbows. Internal and external rotation of the limb may be an effort by the patient to alter loading of the limb to decrease pain or be secondary to mechanical abnormalities (e.g., angular limb

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TABLE 31.1 Differential Diagnoses for Lameness

Signalment	History	Differential Diagnosis	Signalment	History	Differential Diagnosis
Immature, large dogs; front limb	Acute	Fractured physis[a]	Adult, large dogs; rear limb	Acute	Fractured bone[a]
		Fractured bone[a]			Luxated hip[a]
	Chronic	OCD shoulder			Luxated stifle[a]
		OCD elbow			Cruciate and/or meniscus syndrome[a]
		UAP elbow			Ruptured Achilles tendon[a]
		FCP elbow			Luxated tarsus[a]
		Premature closure of physes			Degenerative joint disease caused by cruciate rupture[a]
		Elbow incongruity		Chronic	Panosteitis
		Retained cartilage cores			Patellar luxation[a]
		Panosteitis			Cruciate and/or meniscus syndrome[a]
		Hypertrophic osteodystrophy			Bone and/or soft tissue neoplasia[a]
					Lumbosacral syndrome
					Thoracolumbar disk disease
					Inflammatory joint disease[a]
Immature, large dogs; rear limb	Acute	Fractured physis[a]	Adult, small dogs; front limb	Acute	Fractured bone
		Fractured bone[a]			Luxated shoulder
	Chronic	Hip dysplasia			Luxated elbow
		OCD stifle		Chronic	Degenerative joint disease[a]
		Patellar luxation			Luxating shoulder
		Avulsion of long digital extensor tendon			Bone and/or soft tissue neoplasia
					Inflammatory joint disease
		OCD hock			Radius curvus and/or elbow incongruity
		Panosteitis			Cervical disk disease
		Hypertrophic osteodystrophy			
Immature, small dogs; front limb	Acute	Fractured physis	Adult, small dogs; rear limb	Acute	Fractured bone
		Fractured bone			Luxated hip
	Chronic	Congenital luxation, shoulder			Luxated stifle
		Congenital luxation, elbow			Cruciate and/or meniscus syndrome
					Luxated tarsus
					Degenerative joint disease[a]
				Chronic	Patellar luxation
					Cruciate and/or meniscus syndrome
					Bone and/or soft tissue neoplasia
					Lumbosacral syndrome
					Thoracolumbar disk disease
					Inflammatory joint disease
Immature, small dogs; rear limb	Acute	Premature closure of physes	Adult, large dogs; front limb	Acute	Fractured bone[a]
		Fractured physis			Luxated shoulder[a]
		Fractured bone			Luxated elbow[a]
		Avascular necrosis of femoral head		Chronic	Degenerative joint disease caused by elbow dysplasia
					Panosteitis
	Chronic	Patellar luxation[a]			Bicipital tendinopathy
					Contracture of infraspinatus tendon
					Radius curvus and/or elbow incongruity
					Carpal hyperextension
					Bone and/or soft tissue neoplasia[a]
					Brachial plexus injury[a]
					Cervical disk disease
					Inflammatory joint disease[a]

[a]Denotes potential differential diagnoses in cats.
FCP, Fragmented coronoid process; *OCD,* osteochondritis dissecans; *UAP,* ununited anconeal process.

deformity). Joint collapse in the forelimb is most commonly seen in the carpal joint secondary to traumatic hyperextension injuries or degenerative collapse. Varus or valgus deformities of the forelimb may be due to collateral ligament injuries or angular limb deformities. Proprioceptive deficits and hypermetria are usually indicators of neurologic disease.

Hind Limb

Gait abnormalities that may be appreciated in the hind limbs include hip hike, hip sway, symmetrical simultaneous limb use (bunny hopping), circumduction, internal or external rotation, joint collapse, joint hyperflexion or hyperextension, varus or valgus deformity, proprioceptive deficits, and hypermetria.

A hip or pelvis hike is similar to a head nod in the forelimb and is an attempt by the patient to decrease load and therefore pain in the affected limb. The hemi-pelvis elevates on the affected side when the limb strikes the ground. A hip sway is an attempt by the patient to advance the limb with less motion in the joints of the affected hind limb. This may be due to pain in a joint or a loss of range of motion. Bunny hopping is symmetrical, simultaneous hind limb use and is a compensatory maneuver to limit pain when both hind limbs are affected. This is most

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commonly seen with hip arthrosis secondary to hip dysplasia, but may also be noted with bilateral stifle or hock disease. External swinging (circumduction) of the affected limb(s) occurs when the animal tries to advance a limb that cannot be adequately flexed. Internal and external rotation of the limb may be an effort by the patient to alter loading of the limb to decrease pain (stifle OCD) or be secondary to mechanical abnormalities (patellar luxation). Joint collapse in the hindlimb is most commonly seen in the tibiotarsal joint secondary to Achilles tendon injuries or degeneration (see p. 1284) or intertarsal joint injuries. Varus or valgus deformities of the hind limb may be due to collateral ligament injuries or angular limb deformities. Proprioceptive deficits and hypermetria are usually indicators of neurologic disease.

Neurologic Evaluation

The neurologic examination is described in Chapter 38.

Orthopedic Palpation

Ideally, palpate orthopedic patients in both standing and recumbent positions (particularly if you have little experience); however, experienced surgeons may be able to perform a thorough orthopedic examination with the patient standing. The standing examination is critical for palpation for swellings, masses, atrophy, and other abnormalities, particularly when these changes are asymmetrical (e.g., muscle atrophy, elbow joint effusion, carpal effusion, medial buttressing of the stifle joint, musculoskeletal masses, Achilles tendon abnormalities). *Palpate deeply for pain on muscles and bone and compare from side to side while the patient is standing.* The list of palpated structures below is long, but a thorough standing examination can be quickly performed with experience because of the advantage of symmetrical palpation.

The recumbent examination may permit more thorough and easier palpation of joint range of motion, crepitus, and instability. When the patient is in lateral recumbence, manipulation of a limb does not require it to balance, which may place increased load on a diseased limb, leading to more pain, as occurs in a standing examination when nonaffected limbs are being examined. However, in some situations only a standing examination can be performed. Many patients will not tolerate recumbent examination because of stress, aggression, or most commonly, hyperactivity in puppies. *If the patient requires excessive restraint, perform the entire examination with the patient standing.*

Assess the patient's temperament as this may require you to alter the examination. It is important that the patient be as relaxed as possible when performing an orthopedic examination so that the examiner can have an accurate assessment of range of motion, joint stability, and pain. Stress or tension in a patient can result in nonpathologic changes in range of motion, failure to identify loss of joint stability, and false interpretation of anxiety as pain. In most situations it is best to examine the affected limb(s) last, but in some cases (particularly with active young puppies or kittens) there may only be an opportunity to palpate the diseased limb(s).

Standing Palpation (Suggested Technique)

Head and forelimb. *Starting facing the head of the dog or cat, palpate the skull for symmetry from cranial to caudal including the maxilla, zygomatic arches, cranium, masseter muscles, and mandible. Palpate deeply over the cranium for potential muscle pain.* Abnormal findings may include muscle atrophy, masses, pain response, or crepitus from fractures. *Open and close the jaw to evaluate range of motion and pain. In cases of possible trauma, manipulate the hemimandibles independently in opposite directions to check for symphyseal separation. Next, position yourself directly behind the patient and palpate the neck deeply for pain. If there is no pain, place the neck through range of motion in flexion, extension, and laterally to assess range of motion and pain.* ***Do not manipulate the neck if pain was identified on deep palpation.***

Begin palpation of the forelimbs proximally. Be certain to have the patient standing symmetrically if possible. Asymmetric placement of the feet on the ground alters the symmetry of the limbs and may alter or confuse the results of the examination. *Palpate over the scapula using the spine of the scapula as a reference. Compare muscle mass on both sides.* The infraspinatus and supraspinatus muscles are the easiest location to identify muscle atrophy on the forelimb. Atrophy of these muscles may be due to any cause in the forelimb and is not specific to diseases of the scapula or shoulder joint. *Palpate the shoulder joint using the acromion process and greater tubercle as landmarks.* Increased joint fluid in the shoulder is difficult to palpate due to the surrounding musculature; however, the joint is contiguous with biceps bursa, and increased fluid in this location may indicate either bicipital or shoulder joint pathology. *Identify the proximal biceps tendon and bursa. Palpate deeply over the biceps tendon, monitoring pain response, and feel for increased fluid or fibrosis in the region.*

Palpate the humeral diaphysis medially and laterally keeping in mind that increased pressure on the neurovascular plexus on the medial aspect may give the false impression of pathology in that region. Palpate the distal biceps and tendon medially, monitoring for pain.

Identify the medial and lateral epicondyles of the elbow joint. Slide your fingers caudally feeling for joint effusion, fibrosis, or osteophytosis. Elbow joint effusion and thickening are most easily palpated at the caudolateral portion of the joint although a majority of pathology of the joint is in the medial compartment.

Palpate the diaphysis of the radius from proximolateral to craniodistal, paying particular attention to the distal radius. Palpate deeply for pain and compare the symmetry for potential mass effects secondary to osteosarcoma. Palpate the ulna proximomedial to distolateral. Palpate carefully over the carpal joints for joint effusion. Carpal joint effusion is most easily felt on the craniodorsal surface. *Palpate over the metacarpal regions and dorsally over the toes.*

Thoracolumbar spine and hind limbs. *Identify the dorsal spinous processes (this may be difficult in obese dogs) while supporting the animal with your opposite hand under the thorax and abdomen, and apply gentle pressure on the spine. Progressively increase the pressure to rule out spinal pain.*

Symmetrically position the hind feet. Identify the wings of the ilium, greater trochanters, and ischial tuberosities. Compare the spatial relationships of these three structures. These three structures should form a triangle. If they form a line, then a craniodorsal luxation of the coxofemoral joint should be suspected. *Palpate for muscle mass over the greater trochanter.* The biceps femoris muscle is one of the easiest locations to identify muscle atrophy on the hind limb. Atrophy of these muscles may be due to any cause in the hind limb and is not specific to diseases of the pelvis or coxofemoral joint. *Palpate and compare the muscle mass of the quadriceps and the hamstrings (semimembranosus, semitendinosus, biceps femoris). Palpate the diaphysis of the femur medially, keeping in mind that increased pressure on the neurovascular plexus on the medial aspect may give the false impression of pathology in that region. Cup your hands over the cranial aspects of the stifle*

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joints. Rub your fingertips on the medial aspect of the region feeling for medial buttressing (fibrous proliferation). Palpate the patellas, noting their location. Apply medial and lateral pressure to the sides of the patella, evaluating for possible luxation or subluxation. Some patients will be more relaxed when standing and have less tension in the quadriceps muscles. This may make it easier to diagnose a luxating patella when the patient is standing in comparison to when the patient is in lateral recumbence. *Palpate the patellar tendon from the distal pole of the patella to the tibial crest.* The patella tendon should have sharp medial and lateral borders. The inability to palpate these borders may indicate stifle joint effusion. *Palpate the cranial tibial muscle on the cranial lateral aspect of the tibia. Compare both limbs for symmetry or muscle atrophy. Palpate the tibial diaphysis deeply on the medial aspect of the limb. Palpate the common calcanean tendon (Achilles tendon) from the proximal musculotendinous junction to its insertion on the calcaneus. Palpate the superficial digital flexor tendon as it passes over the calcaneal tubercle. Palpate for effusion or fibrosis. Apply medial and lateral pressure to the sides of the tendon, evaluating for possible displacement. Identify the medial and lateral malleoli and the calcaneal tubercle. Palpate between these structures medially and laterally for joint effusion. Palpate on the cranialdorsal surface of the tibiotarsal joint for effusion. Palpate over the metatarsal regions and dorsally over the toes.*

Recumbent Palpation (Suggested Technique)

A recumbent examination can be performed if the patient is reasonably relaxed in this position. As previously noted, if possible, it is best to examine the affected limb last because pain will often be elicited, stressing the patient. If the patient is not particularly relaxed, then beginning with the affected limb first may be optimal. If the patient will not relax sufficiently to allow a thorough examination to be done, it is best to not use excessive restraint. If necessary, the animal may be sedated; however, this is seldom required (Tables 31.2 and 31.3).

Forelimb. *Begin the examination at the toes. Assess the nails for wearing, which is indicative of toe dragging. Also look for nail injuries that may cause pain and lameness. Observe the nail beds for tumors or infection or foreign bodies. Deeply palpate the phalangeal bones and joints of each digit.* Thickening of the phalangeal bones may be an indication of trauma, neoplasia, infection, or foreign bodies. Thickening of the phalangeal joints

TABLE 31.2 Sedation for Palpation and Radiographs of Dogs[a]

Drug/Combination	Dose	Route	Comments
Protocol 1			Drugs can be combined in a single syringe and administered IM.
Acepromazine[b], *plus*	0.05 mg/kg, maximum of 2 mg in large dogs	IV, SC, IM	• Not reversible. • Animals may require restraint if used alone. • Contraindicated in patients with recent hypotension; blood loss; or hepatic, cardiovascular or renal disease. • A rare idiosyncratic reaction of profound aggression has been reported in dogs following oral or parental acepromazine administration (see p. 126). • Use cautiously, if at all, in boxer dogs due to possible cardiovascular collapse.
Hydromorphone, *or*	0.05–0.2 mg/kg	IV, SC, IM	• Can be reversed with naloxone (0.01–0.02 mg/kg IV, IM) (see Table 13.2).
Oxymorphone, *or*	0.1–0.2 mg/kg	IV, SC, IM	• Butorphanol may cause auditory hypersensitivity or panting.
Butorphanol, *plus* PRN	0.2–0.4 mg/kg	IV, SC, IM	
Atropine, *or*	0.02 mg/kg	IV	Use if bradycardia occurs, especially if lowered heart rate causes subsequent hypotension.
	0.04 mg/kg	SC, IM	
Glycopyrrolate	0.005–0.011 mg/kg	IV, SC, IM	
Protocol 2			Drugs can be combined in a single syringe and administered IM.
Dexmedetomidine, *plus*	2.5–10 µg/kg	IM	• Can be reversed with atipamezole (0.2 mg/kg IM only). • Use only in patients that are young to middle-aged without significant systemic disease.
Hydromorphone, *or*	0.05–0.2 mg/kg	IV, SC, IM	• Can be reversed with naloxone (0.01–0.02 mg/kg IV, IM) (see Table 13.2).
Oxymorphone, *or*	0.1–0.2 mg/kg	IV, SC, IM	• Butorphanol may cause auditory hypersensitivity or panting.
Butorphanol, *plus* PRN	0.2–0.4 mg/kg	IV, SC, IM	
Atropine, *or*	0.02 mg/kg	IV	Dexmedetomidine should not be used in dogs with a resting heart rate of <80 beats/min; an anticholinergic could be given 20 min IM before dexmedetomidine to increase heart rate to >80 beats/min
	0.04 mg/kg	SC, IM	
Glycopyrrolate	0.005–0.011 mg/kg	IV, SC, IM	
Protocol 3			Drugs can be combined in a single syringe and administered IM (this protocol is preferred for patients with cardiovascular compromise or systemic disease).
Midazolam, *or*	0.2–0.4 mg/kg	IV, SC, IM	• Can be reversed with flumazenil (0.01–0.025 mg/kg IV, IM).
Diazepam, *plus*	0.2–0.4 mg/kg	IV only	• Diazepam should be administered IV only.
Hydromorphone, *or*	0.05–0.2 mg/kg	IV, SC, IM	• Can be reversed with naloxone (0.01–0.02 mg/kg IV, IM) (see Table 13.2).
Oxymorphone, *or*	0.1–0.2 mg/kg	IV, SC, IM	• Butorphanol may cause auditory hypersensitivity or panting.
Butorphanol, *plus* PRN	0.2–0.4 mg/kg	IV, SC, IM	
Atropine, *or*	0.02 mg/kg	IV	• Use if bradycardia occurs, especially if lowered heart rate causes subsequent hypotension.
	0.04 mg/kg	SC, IM	
Glycopyrrolate	0.005–0.011 mg/kg	IV, SC, IM	• Not recommended in animals with cardiovascular disease and tachycardia unless patient becomes bradycardic and hypotensive.

[a]Animals receiving sedatives should be carefully monitored. Parameters including (but not limited to) respiratory rate, heart rate, and pulse oximetry should be monitored regularly. Blood pressure should be checked when blood pressure changes are anticipated. Temperature should be monitored if the animal is sedated for longer than 30 minutes.
[b]Acepromazine (0.05 mg/kg IM or SC; maximum of 2 mg) may be used alone for radiographs and palpation; however, it provides minimal restraint.
IM, Intramuscular; *IV,* intravenous; *PRN,* as needed; *SC,* subcutaneous.

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TABLE 31.3 Sedation for Palpation and Radiographs of Cats[a]

Drug/Combination	Dose	Route	Comments
Protocol 1			Drugs can be combined in a single syringe and administered IM.
Acepromazine[b], *plus*	0.05 mg/kg, maximum of 1 mg	IV, SC, IM	• Not reversible. • Animals may require restraint if used alone. • Contraindicated in patients with history of recent hypotension; blood loss; or hepatic, cardiovascular, or renal disease.
Hydromorphone *or*	0.05–0.1 mg/kg	IV, SC, IM	• Hydromorphone may cause elevated temperatures in cats.
Oxymorphone, *or*	0.05–0.1 mg/kg	IV, SC, IM	• Can be reversed with naloxone (0.01–0.02 mg/kg IV, IM) (see Table 13.2).
Butorphanol, *plus* PRN	0.2–0.4 mg/kg	IV, SC, IM	
Atropine, *or*	0.02 mg/kg	IV	Use if bradycardia occurs, especially if lowered heart rate causes subsequent hypotension.
	0.04 mg/kg	SC, IM	
Glycopyrrolate	0.005–0.011 mg/kg	IV, SC, IM	
Protocol 2			Drugs can be combined in a single syringe and administered IM.
Dexmedetomidine, *plus*	5–20 µg/kg	IM	• Can be reversed with atipamezole (0.2 mg/kg IM only). • Use only in patients that are young to middle-aged without significant systemic disease.
Hydromorphone, *or*	0.05–0.1 mg/kg	IV, SC, IM	• Hydromorphone may cause elevated temperatures in cats.
Oxymorphone, *or*	0.05–0.1 mg/kg	IV, SC, IM	• Can be reversed with naloxone (0.01–0.02 mg/kg IV, IM) (see Table 13.2).
Butorphanol, *plus* PRN	0.2–0.4 mg/kg	IV, SC, IM	
Atropine, *or*	0.02 mg/kg	IV	Use if bradycardia occurs, especially if lowered heart rate causes subsequent hypotension.
	0.04 mg/kg	SC, IM	
Glycopyrrolate	0.005–0.011 mg/kg	IV, SC, IM	
Protocol 3			Drugs can be combined in a single syringe and administered IM (this protocol is preferred for cats with cardiovascular compromise or systemic disease).
Midazolam, *or*	0.2–0.4 mg/kg	IV, SC, IM	• May cause paradoxical aggression in healthy cats when used alone.
Diazepam, *plus*	0.2–0.4 mg/kg	IV only	• Can be reversed with flumazenil (0.01–0.025 mg/kg IV, IM). • Diazepam should be administered by IV route only.
Hydromorphone, *or*	0.1–0.2 mg/kg	IV, SC, IM	• Hydromorphone may cause elevated temperatures in cats.
Oxymorphone, *or*	0.1–0.2 mg/kg	IV, SC, IM	• Can be reversed with naloxone (0.01–0.02 mg/kg IV, IM) (see Table 13.2).
Butorphanol, *or*	0.2–0.4 mg/kg	IV, SC, IM	
Alfaxalone, *plus* PRN	2–5 mg/kg	IM, IV	• The addition of alfaxalone works well in the compromised cat that is fractious and in which ketamine or an alpha-2 agonist is not recommended. • Can be mixed in the same syringe with the opioid or benzodiazepine.
Atropine, *or*	0.02 mg/kg	IV	• Use if bradycardia occurs, especially if lowered heart rate causes subsequent hypotension.
	0.04 mg/kg	SC, IM	
Glycopyrrolate	0.005–0.011 mg/kg	IV, SC, IM	• Not recommended in animals with cardiovascular disease and tachycardia, unless patient becomes bradycardic and hypotensive.
Protocol 4			Drugs can be combined in a single syringe and administered IM (this protocol is preferred, but not limited, for use in fractious cats).
Ketamine, *plus*	5 mg/kg	IM	• Provides no muscle relaxation when used alone. • Contraindicated in cats with seizures.
Midazolam, *or*	0.2–0.4 mg/kg	IM	
Dexmedetomidine	5–20 µg/kg	IM	• Can be reversed with atipamezole (0.2 mg/kg IM only). • Use only in patients that are young to middle aged without significant systemic disease.

[a]Animals receiving sedatives should be carefully monitored. Parameters including (but not limited to) respiratory rate, heart rate, and pulse oximetry should be monitored regularly. Blood pressure should be checked when blood pressure changes are anticipated. Temperature should be monitored if the animal is sedated for longer than 30 minutes.
[b]Acepromazine (0.05 mg/kg IM or SC; maximum of 1 mg) may be used alone for radiographs and palpation; however, it provides minimal restraint.
IM, Intramuscular; *IV,* intravenous; *PRN,* as needed; *SC,* subcutaneous.

may be an indication of infection, immune-mediated joint disease, or trauma. *Manipulate the interphalangeal and metacarpal-phalangeal joints in flexion, extension, and laterally, evaluating for crepitus or instability due to collateral ligament damage. Deeply palpate the metacarpal bones for pain, swelling, or mass effects.*

Palpate the carpal joint in flexion-extension and medial-lateral. The carpus can normally flex so that the digital pads can touch the distal antebrachium. A decrease range of motion in flexion is common in active dogs and not necessarily an indication of pathology. *Compare the range of motion of the carpus in extension to the contralateral limb.* An increase in range of motion in extension may indicate trauma to the carpal fibrocartilage. With jumping and falling injuries, hyperextension may be identified in both carpi. An increase in mediolateral motion of the carpus may indicate damage to a collateral ligament. The carpus is also a common location for identification of immune-mediated joint disease.

Palpate the radius and ulna deeply from distal to proximal for pain and swelling. In young patients, pain may be a sign of hypertrophic osteodystrophy or panosteitis. In older patients, pain and firm swelling may be an indication of osteosarcoma.

Flex and extend the elbow joint noting the range of motion, crepitus, and pain. The elbow joint should normally extend so that the humeroradial angle appears to be close to 180 degrees. The joint should normally flex to a very acute angle (Fig. 31.1). Dogs with moderate to advanced elbow arthrosis due to elbow

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FIG. 31.1 Flex and extend the elbow. Notice the angle of greatest flexion of a normal elbow measured with a goniometer. The carpus should almost touch the shoulder. Compare this with the angle of flexion in an elbow with degenerative joint disease (see Fig. 31.2).



FIG. 31.2 Angle of greatest flexion of elbow in dog with degenerative joint disease. Compare this with the angle of flexion in a normal elbow (see Fig. 31.1).

dysplasia frequently have a range of motion in flexion of only 90 degrees (Fig. 31.2). *Manipulate the joint mediolaterally to assess the integrity of the collateral ligaments.* Collateral ligament damage of the elbow joint is uncommon except in cases of luxation. *Identify the medial and lateral epicondyles and the olecranon and palpate for joint effusion, fibrosis, and osteophytosis particularly on the lateral aspect as in the standing examination.*

Palpate the humerus medially and laterally, being careful to minimize pressure on the neurovascular structures on the medial aspect of the limb. Palpate the proximal aspect of the humerus on the lateral aspect paying close attention in older dogs when neoplasia is a differential.



FIG. 31.3 Hyperextend the shoulder while stabilizing the scapula. Pain on hyperextension or hyperflexion may indicate osteochondritis dissecans of the humeral head.

Flex and extend the shoulder joint by grasping the radius/ulna with one hand and stabilizing the scapula or thorax with the opposite hand. When flexing the shoulder joint, keep the elbow joint in moderate flexion to enable differentiation between shoulder and elbow pain. When the shoulder joint is flexed, the humerus should be parallel to the spine in a normal dog. When the shoulder joint is extended, the humerus should reach close to but not completely parallel to the spine (Fig. 31.3). In young dogs, observe for pain during flexion and extension, which may be an indication of osteochondrosis. *Flex the shoulder joint and extend the elbow joint and then apply digital pressure on the proximal or distal aspects of the biceps tendon as an indication of biceps disease. Abduct the humerus while stabilizing the scapula to test the integrity of the medial glenohumeral ligaments. Palpate the scapula from distal to proximal beginning at the acromion process feeling for masses and crepitus.*

Hindlimb. *With the patient in a lateral recumbent position, position yourself behind the hindlimb instead of at the toes to thoroughly palpate the limb.*

Palpate the toes and metatarsal region as described earlier for the forelimb foot.

Flex and extend the tibiotarsal joint with the stifle in moderate flexion. If the stifle is kept in extension, then the tarsus cannot be fully flexed. *Note any crepitus or pain. With the stifle and hock in extension, abduct and adduct the tibiotarsal joint to evaluate the long branches of the medial and lateral collateral ligaments. Flex the stifle and hock and abduct and adduct the tibiotarsal joint with a rolling motion to test the short branches of the medial and lateral collateral ligaments. Identify the region of the superficial digital flexor tendon. Palpate for swelling and push medially and laterally on the tendon, evaluating for instability. Palpate the insertion of the Achilles tendon for swelling or pain. With the stifle joint in extension, flex the hock joint evaluating for hyperflexion associated with Achilles tendon injury (Fig. 31.4).*

Palpate the hock joint caudally and cranially for effusion.

Palpate the tibial deeply on the medial aspect of the limb from distal to proximal for masses or pain.

Identify the following landmarks of the stifle joint: tibial crest, fibular head, lateral fabella, patella, patellar tendon. With the stifle joint in mild flexion, apply pressure to the patella in a medial and lateral direction evaluating for luxation or subluxation. Internal and external rotation of the tibia may aid in detecting patellar

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luxation (Fig. 31.5). *With the stifle joint in full extension, abduct and adduct the tibia to evaluate the medial and lateral collaterals (Fig. 31.6).*

Flex the stifle joint. The hock should be able to touch the ischial tuberosity with normal range of motion. *Extend the stifle joint.* The hind limb should appear to be at 180 degrees with the stifle joint in extension.

Palpation for Cruciate Injury

Testing for cruciate injury is performed by the drawer test or the tibial compression test. The drawer test is a passive motion test, whereas the tibial compression test simulates weight bearing and is an active motion test. It is important when testing for drawer to have a gentle grasp of the limb. A tight grasp will cause tension in the patient and the examiner and result in a false negative test.

Tibial compression test. *Grasp the stifle joint with the hand that is closer to the spine. Place the index finger so that the base of the finger is over the patella and the tip of the finger is over the tibial crest. Position the thumb gently over the lateral aspect of the stifle joint and place your remaining fingers on the medial aspect of the limb.* This hand maintains the stifle in nearly full extension and feels for thrust during the maneuver. *Gently grasp the foot in the lower hand and with the stifle held in moderate extension, flex the tibiotarsal joint while palpating the tibial crest for forward thrust (Fig. 31.7).* Normally the tibial crest will advance only a



FIG. 31.4 To test integrity of the Achilles tendon complex, attempt to flex the hock with the stifle in extension.



FIG. 31.6 To examine for collateral ligament injury, fully extend the stifle, stabilize the distal femur with one hand, and apply medial and lateral pressure to the tibia with the other hand.

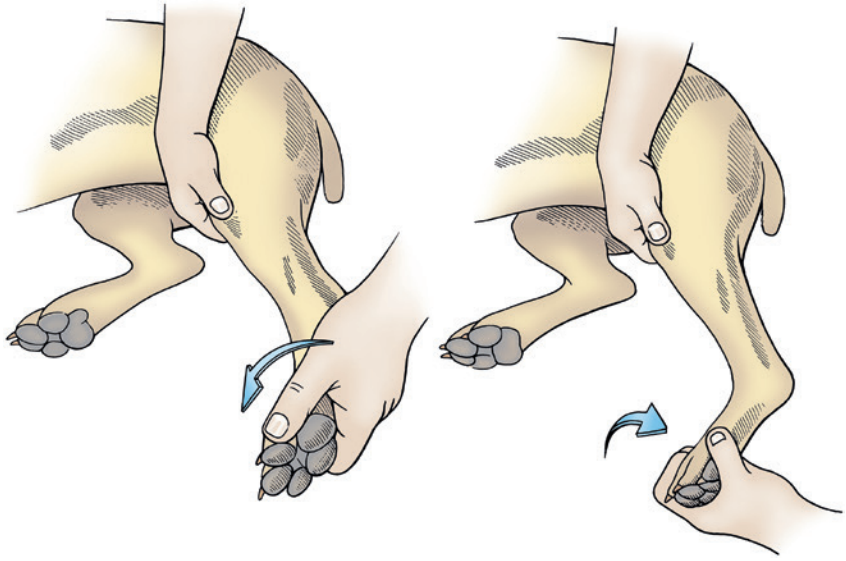


FIG. 31.5 To examine the stifle for medial patellar luxation, extend the stifle, internally rotate the foot, and apply medial pressure to the patella with the thumb. To examine the stifle for lateral patellar luxation, slightly flex the stifle, externally rotate the foot, and apply lateral pressure to the patella with the fingers.

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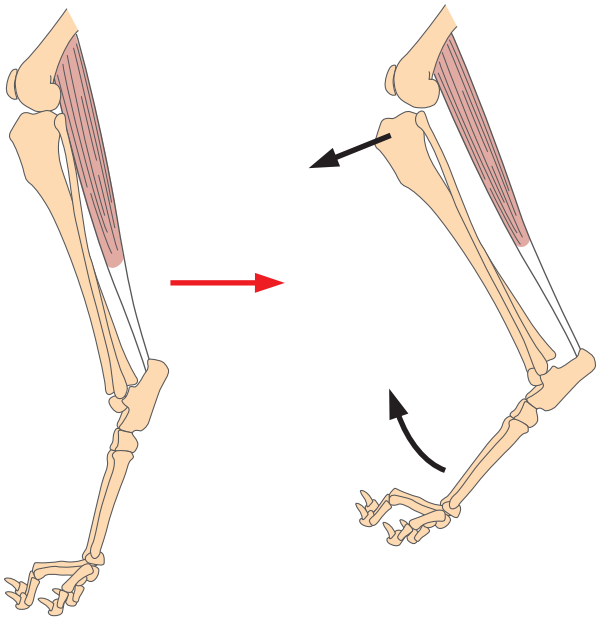


FIG. 31.7 Flexing the hock while the limb is in a standing position will tense the gastrocnemius muscle and force the proximal tibia forward if the cranial cruciate ligament is ruptured.

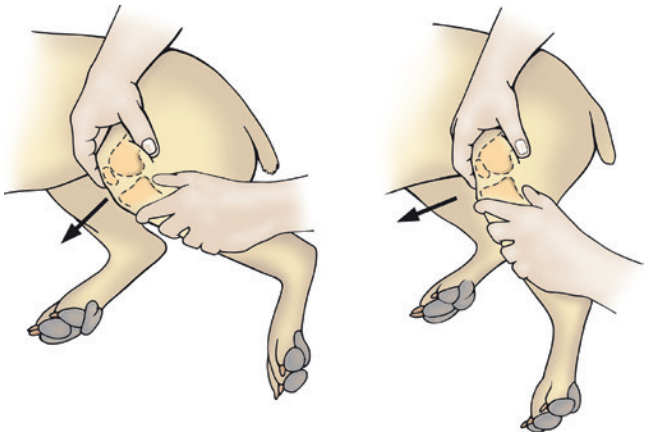


FIG. 31.8 To examine for cruciate ligament injury, place the thumb of one hand over the lateral fabella and the index finger over the patella. Stabilize the femur with this hand. Place the thumb of the opposite hand caudal to the fibular head with the index finger on the tibial tuberosity. With the stifle first flexed and then extended, attempt to move the tibia cranially and distally to the femur.

few millimeters with this maneuver. When the cruciate ligament is completely ruptured, the tibial crest may thrust forward a centimeter or more.

Drawer test. *With the lower hand, gently grasp the proximal tibia, placing the thumb behind the fibular head and the index finger on the tibial crest (Fig. 31.8).* Grasping this area too tightly may cause pain from the peroneal nerve, which runs caudal to the fibular head. *Place the upper hand on the distal femur with the thumb behind the fabella and the index finger over the patella.* In many dogs, only the thumb and index finger of each hand are necessary to grasp the stifle joint. A light grip increases the relaxation of the examiner and patient and increases the chance of an accurate test. *Keep the stifle joint in mild flexion and use the distal hand to push the tibial cranially and slightly distally.* The normal stifle joint should have little to no drawer motion. When the cruciate is completely ruptured, there may be a centimeter or more of drawer motion. Partial cruciate tears may or may not result in increased drawer, and cruciate disease cannot be ruled out based on a normal drawer test.

Palpate the femur deeply on the medial aspect being careful not to apply excessive pressure to the neurovascular structures. Identify the landmarks of the proximal femur and pelvis including: greater trochanter, wing of the ilium, and the medial and lateral aspects of the ischium. Lines between the greater trochanter, dorsal aspect of the ilial wing, and the ischial tuberosity should form an acute triangle. If these structures fall in a straight line, it may indicate a craniodorsal luxation of the hip joint (Fig. 31.9). *Place the thumb and index finger of the upper hand lightly on the greater trochanter. Grasp the flexed stifle joint and flex and extend the hip joint.* When the hip is flexed and extended, the femur should be nearly parallel to the long axis of the spine. *Abduct and adduct the femur feeling for crepitus or subluxation with the upper hand. Internally and externally rotate the femur.* The femur should be able to internally and externally rotate to equal degrees.

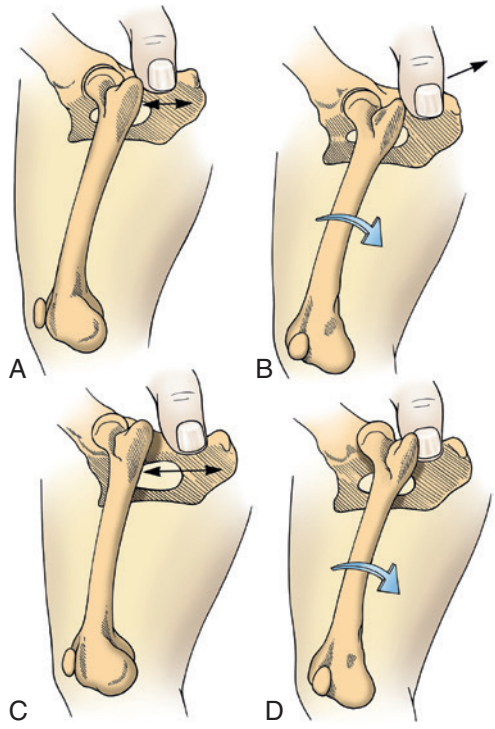


FIG. 31.9 (A–B) To examine for hip luxation, place the thumb in the space caudal to the greater trochanter and externally rotate the femur. If the coxofemoral joint is intact, the greater trochanter will displace the thumb. (C–D) If the coxofemoral joint is luxated, the greater trochanter will roll over the thumb.

Palpation for Juvenile Hip Dysplasia

The Barden and Ortolani tests are the primary palpations for juvenile hip dysplasia and coxofemoral subluxation.

Barden test. *With the patient in lateral recumbence, place the thumb and index finger of the upper hand on the greater trochanter.*

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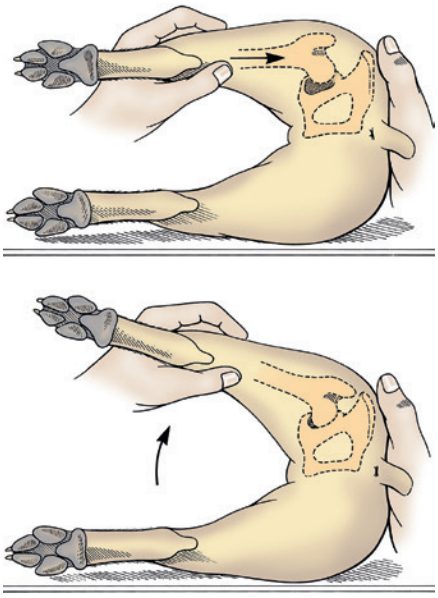


FIG. 31.10 To examine the hip for laxity with the animal positioned in lateral recumbency, place one hand over the back and grasp the stifle with the opposite hand. Hold the femur parallel to the table or in adduction and subluxate the femoral head by pushing the stifle toward the pelvis. While maintaining pressure, abduct the limb. As the femoral head returns to the acetabulum, you will feel a click.

Gently grasp the flexed stifle joint with the lower hand with the thumb on the lateral aspect and the remaining fingers extended on the distal aspect of the femur. Press laterally with the fingers of the lower hand to lever the femoral head out of the acetabulum. Alternately, press down on the greater trochanter to reduce the femoral head back into the acetabulum. A normal hip joint should have little to no subluxation. Juvenile hip dysplasia may result in up to two centimeters of lateral subluxation.

Ortolani maneuver. *With the patient in lateral recumbence, place the upper hand dorsal to the pelvic region to stabilize the patient. Start with the Barden procedure and try to sublux the femoral head (Fig. 31.10). Next, apply proximal pressure on the stifle joint to force the subluxed femoral head against the dorsal acetabular rim. Finally, abduct the femur and feel and watch for the femoral head to reduce into the acetabulum.*

The Ortolani procedure may also be performed with the patient in dorsal recumbence (Fig. 31.11). *Place the patient in dorsal recumbence. Position the hind limbs so the femurs are pointing directly vertically. Flex the stifle joints to 90 degrees and place one hand over each stifle joint. Adduct the stifle joints to sublux the femoral heads and place downward (dorsal) pressure on the stifle joints to lock the femoral heads against the dorsal acetabular rims. Abduct one leg at a time feeling and looking for subluxation.*

Additional Diagnostic Techniques

Although a differential diagnosis is typically developed based on results of history, signalment, and physical examination, definitive diagnoses may require additional diagnostic tools including imaging, hematology, serum biochemistry, cytology, and/or electrodiagnostics. Radiography is often the initial imaging modality used; however, computed tomography, magnetic resonance imaging, ultrasonography, or scintigraphy may be indicated for diagnosis and/or improved visualization of some problems. Joint taps are useful for differentiating suppurative and mononuclear inflammation. Fine needle aspirates or biopsies are essential for diagnosing neoplastic disease.

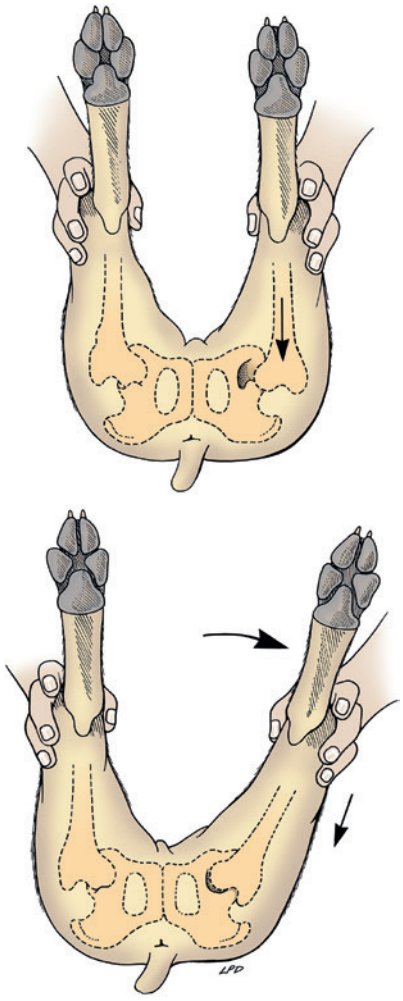


FIG. 31.11 To examine the hip for laxity with the animal positioned in dorsal recumbency, place the hands over the stifles. Hold the femurs parallel to each other and perpendicular to the table. Subluxate the femoral head by pushing the stifle toward the pelvis. While maintaining pressure, abduct the limb. As the femoral head returns to the acetabulum, you will feel a click.

PERIOPERATIVE MANAGEMENT

Perioperative management of surgical patients is discussed in detail in Chapter 4. Orthopedic patients may be admitted for elective surgery (e.g., cranial cruciate ligament injury, hip dysplasia, osteochondritis dissecans) or nonelective surgery (e.g., bone fractures, joint luxations). When the animal is being examined for an elective surgery there is ample time to perform an appropriate preoperative diagnostic evaluation. Younger patients (<5–7 years old) should have selected screening laboratory tests, including packed cell volume, and serum total solids. The

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need for further laboratory evaluation should be based on signalment, physical examination, geographical location, and results of initial screening tests.

Patients older than 5 to 7 years with orthopedic disease must be assessed more carefully than younger patients. Just as the physiologic properties of the musculoskeletal system decline with age, other organ systems also deteriorate. Thorough physical examination remains the foundation of preoperative evaluation and should be supplemented with a complete blood count, chemistry profile, and urinalysis. Special diagnostic tests (e.g., coagulation profile) may be indicated, depending on the history, signalment, and physical findings.

Trauma patients should have a thorough and complete physical evaluation. Serial examinations are important because serious or potentially lethal problems may not become evident for several hours or days after the injury. Although organ dysfunction may be evident (or suspected) on initial physical examination, repeated examinations may be necessary to define the severity of injury. Animals that have received trauma severe enough to disrupt musculoskeletal integrity (e.g., fracture and luxation) often have concurrent external or internal organ system injury. Cardiovascular, pulmonary, urinary, and neurologic systems are most frequently injured. If abnormalities are found, a differential diagnosis and diagnostic plan for each problem should be developed and additional diagnostic tests completed. For example, cardiac arrhythmias and femoral pulse abnormalities may be found in patients with traumatic myocarditis, necessitating thoracic radiographs and an ECG to assess treatment options and anesthetic risk. Auscultation may detect pulmonary injury (e.g., lung contusion and pneumothorax), but physical changes may be subtle and missed on physical examination. Because one-third or more of trauma-associated fracture patients have some degree of pulmonary injury, preoperative evaluation should include thoracic radiographs. A minimum database should include a complete blood count, serum biochemistry profile, and urinalysis. Additional laboratory tests (e.g., coagulation profile and acid-base balance) may be required to assess differential diagnoses developed on physical examination. Abnormal values should be assessed in light of physical findings, and the need for additional or serial tests should be determined. Serial tests are useful to validate abnormal findings and monitor patient progress. Delaying surgical intervention until abnormal organ function returns to normal is optimal; however, it is often not feasible. Some abnormalities may necessitate that surgical repair of the orthopedic disease be delayed (e.g., uroabdomen), whereas others may alter the prognosis such that repairing the orthopedic condition is not justified (e.g., vertebral fracture with loss of deep pain sensation).

PAIN MANAGEMENT AND ANESTHESIA

Orthopedic patients benefit from perioperative analgesics (see Boxes 32.1 and 32.2, respectively). The projected level of postoperative discomfort and the duration of discomfort should be assessed to determine the choice of preoperative analgesic. Most orthopedic surgeries are considered moderately to severely painful. Although there is much debate concerning preemptive analgesia, especially in the human medical literature, there are several principles that must be followed in order for preemptive analgesia to be successful. First, the depth of analgesia must be sufficient to block pain receptors during surgery. Second, nociceptive receptors from the entire field must be blocked. And last, analgesia must be continued in the postoperative period (see Chapter 13).

Anesthetic protocols should be chosen based on signalment, physical examination findings, and laboratory analysis. Patients treated for elective orthopedic problems (e.g., cruciate reconstruction) and who have no preoperative findings suggestive of major organ dysfunction can be managed using a variety of anesthetic techniques (see Table 32.1). Patients with cardiovascular compromise or trauma should be anesthetized with care (see Table 32.2). When appropriate, balanced anesthetic protocols that include analgesic agents supplemented with epidural analgesia or nerve blocks are recommended to decrease intraoperative pain response and reduce the amount of anesthetic needed (see Table 32.3). Epidural anesthesia (using lidocaine, bupivacaine, or ropivacaine) in combination with general anesthesia provides profound relaxation by temporarily paralyzing rear limb muscles, easing fracture reduction of the pelvis, femur, and tibia (see Chapter 13). The duration of action depends on the drug used, ranging from 1 to 6 hours. Morphine or buprenorphine can be added to the epidural injection, providing postoperative pain relief for up to 24 hours. Additionally, opioids may be used alone in the epidural space when relaxation of muscles is not warranted but postoperative analgesia is desired. A brachial plexus block using local anesthetics will provide additional analgesia and muscle relaxation in patients undergoing surgery of the forelimb. A femoral-sciatic nerve block using local anesthetics will provide additional analgesia and relaxation for surgery done the rear limb (see Chapter 13).

Opioid analgesics should be administered for at least 12 to 24 hours after surgery, depending on the procedure and results of serial patient evaluations. Butorphanol or buprenorphine are often sufficient for patients undergoing procedures requiring minimal tissue manipulation. In patients undergoing more painful procedures that require significant tissue manipulation (e.g., triple pelvic osteotomy) or in those with traumatic injuries, hydromorphone, oxymorphone, or morphine is recommended. When there is minimal risk of continued blood loss, nonsteroidal antiinflammatory drugs (NSAIDs) may be administered for pain control.

NSAIDs are advocated for pain control in orthopedic patients (see Chapter 13 and Table 32.4). They can be used alone or in combination with opioids depending on the severity of the animal's pain. Some opioids (e.g., fentanyl) provide immediate relief, whereas the onset of NSAIDs is 30 minutes to 1 hour. An advantage of NSAIDs is that they provide sustained relief; their duration of action is 12 to 24 hours. Intravenous hydromorphone, oxymorphone, and morphine have delayed onsets of action of 15 to 20 minutes and provide analgesia for only 3 to 4 hours. Although when given before surgery carprofen and deracoxib are effective for postoperative pain relief in dogs, the perioperative effect of these drugs is unpredictable. Similarly, meloxicam injection is approved for use in cats preoperatively (one dose only), but its perioperative course may be unpredictable. It is prudent to reserve NSAIDs for postoperative administration, especially if blood loss is anticipated. Depending upon the NSAID chosen, there may be some inhibition of platelet function. Although carprofen, meloxicam, and etodolac only weakly inhibit cyclooxygenase (COX)-1 and thus platelet function, it is reasonable to wait until the postoperative period to add one of these to the pain protocol. Nonselective COX inhibitors (e.g., ketoprofen,

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phenylbutazone) are contraindicated for surgical procedures in which hemorrhage is anticipated. Additionally, NSAIDs should not be used in patients with marked hypotension or those with severe pulmonary disease (especially asthmatic patients). NSAIDs can be continued orally for several days after surgery to provide pain relief after the animal has been discharged from the hospital. See Chapters 13 and 34 for additional information on NSAIDs used in association with orthopedic surgery. Given the aforementioned complications of NSAIDS, opioids are often the preferred medication for use as a preoperative analgesic because they do not alter coagulation. Additionally, opioids are also less apt to potentiate hypotension than are inhalants.

ANTIBIOTICS

The use of perioperative and postoperative prophylactic antibiotics is discussed in Chapter 9. In general, perioperative antibiotics are indicated in orthopedic surgery, and the use of postoperative prophylactic antibiotics may significantly decrease the incidence of surgical site infections when orthopedic implants are used. Postoperative antibiotics should be chosen based on culture results when performing surgery on patients with contaminated and infected wounds.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative instructions for each orthopedic technique are presented in the discussions on various orthopedic conditions. Common to the success of all orthopedic procedures is adequate client communication concerning postoperative care, regular reevaluations, and the use of appropriate analgesics and physical rehabilitation methods.

Adequate follow-up care for bandages and splints is essential for preventing complications such as ischemic injury and tissue sloughing. Owners should receive explicit instructions for aftercare. The bandage and the middle toenails should be observed twice a day for evidence of odor or swelling. Separation of the toenails indicates swelling of the digits. Excessive licking and chewing of the bandage indicates irritation; the bandage or splint should be removed as soon as possible to inspect the limb if these are noted. The bandage or splint must be kept clean and dry. If it is wet outside, a plastic bag should be temporarily placed over the limb to cover the bandage. Velpeau (see p. 1167) and Ehmer (see p. 1221) slings should not be used for more than 2 weeks. Long-term limb immobilization in a flexed position makes rehabilitation difficult.

General postoperative protocols for fracture patients include postoperative radiographs made to document fracture reduction or alignment and implant position. If possible, the limb is supported for a few days with a soft padded bandage to reduce swelling. Patient activity is generally restricted to leash walking and physical rehabilitation until the fracture has healed. Physical rehabilitation encourages controlled limb use and optimal limb function after fracture healing. Care must be taken to develop customized protocols for each patient depending on location of the fracture, stability and type of fracture fixation, potential for healing, abilities and attitudes of the patient, and willingness of the client (see Chapters 11 and 33). Fracture fixation systems have specific follow-up care that is detailed in the discussions of each system and specific fractures. Establishing excellent client communications and a reevaluation schedule for each patient are paramount for successful fracture management.

Regenerative Medicine and Stem Cell Therapy

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Regenerative medicine is the process of creating living, functional tissues to repair or replace tissue or organ function lost because of disease, damage, or congenital defects. It incorporates numerous biomedical approaches including tissue engineering, stem cell therapy, and medical devices. **Stem cells** are undifferentiated cells that are capable of giving rise to more cells of the same type indefinitely, and can be induced under specific physiologic or experimental conditions to become tissue or organ specific cells (Box 31.2). **Embryonic stem cells** (ESCs) are primitive or undifferentiated cells derived from a 5-day embryo that are capable of dividing without differentiating for a prolonged period in culture and are known to develop into cells and tissues of the three primary germ layers (Fig. 31.12A). Based on their ability to differentiate into cells of numerous organs, embryonic stem cells are considered **pluripotent stem cells**. **Embryonic stem cell lines** are embryonic stem cells that have been cultured under in vitro conditions that allow proliferation without differentiation for months to years. **Tissue specific stem cells** are **"adult"** or **somatic stem cells** that reside within a particular tissue or organ and can develop into the mature cell types of that tissue or organ (see Fig. 31.12B). Tissue specific stem cells usually exist in small populations but have been identified in numerous tissues including skin, blood, intestine, and brain. Based on their ability to differentiate into multiple cell types within a specific tissue or organ these cells are considered *multipotent*.

Bone marrow derived stem cells are a type of multipotent stem cell that are able to give rise to bone, cartilage, and fat cells and able to support formation of blood cells (see Fig. 31.12B). **Mesenchymal stem cells** (MSCs) is a term that is currently used to define nonblood adult stem cells from a variety of tissues, although it is not clear that MSCs from different tissues are the same. Stromal cells are connective tissue cells found in virtually every organ. In bone marrow, stromal cells support blood formation. **Induced pluripotent stem cells** are the product of reprogramming a somatic cell into an ESC-like state through viral gene delivery. This technique, first reported in 2007, provided a new approach to the generation of ESC-like cells.

Stromal vascular fraction (SVF) is obtained by use of proteolytic enzymes and centrifugation of adipose tissue (see Fig. 31.12B). This produces a mixture of hematopoietic stem cells,

BOX 31.2 Categorization of Stem Cell Activity

Totipotent—capable of forming all the cell types in a body, plus the extra-embryonic or placental cells
Pluripotent—capable of forming all the cell types in a body
Induced pluripotent—a pluripotent stem cell artificially derived from a nonpluripotent cell such as a stromal cell
Multipotent—capable of forming several unrelated cell types
Oligopotent—capable of forming only a few related cell types
Unipotent—capable of forming only one cell type

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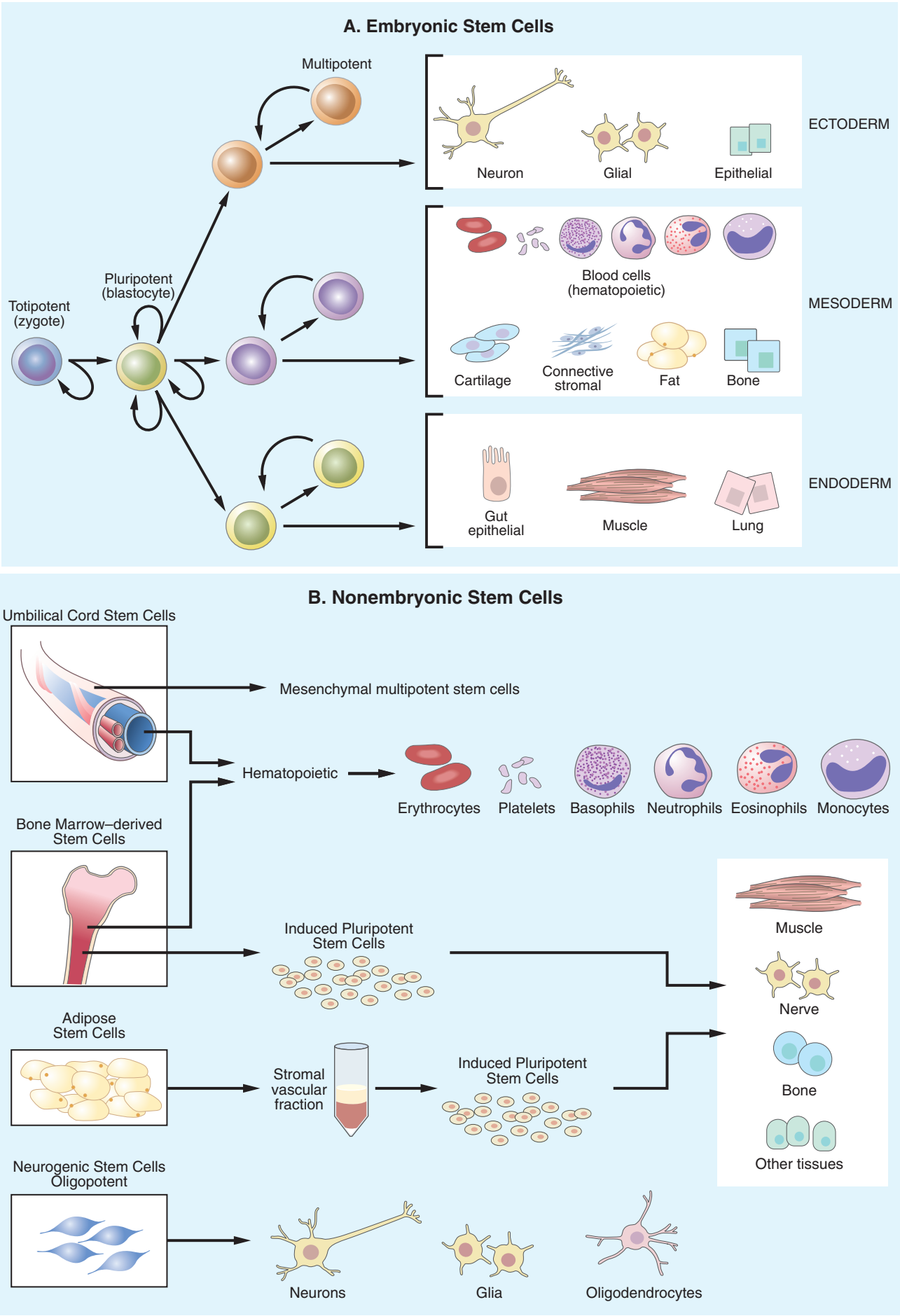


FIG. 31.12 Categorizations of stem cells. (A) Embryonic stem cells. (B) Nonembryonic (adult) stem cells.

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endothelial cell, and adipose derived stem cells. Disagreement remains regarding the strict definition of what constitutes a stem cell and the relationship between stem cells and stromal cells. **Platelet-rich plasma** (PRP) is an autogenous fluid concentrate composed primarily of platelets and white blood cells used to accelerate healing of tendon, ligament, bone, and other tissues. There are no commonly accepted definitions of PRP. The most lenient definition of PRP is any plasma that has more platelets than the baseline blood from which the PRP is made, whereas other definitions are more stringent and define PRP as containing a minimum of 5.5×10^{10} platelets/50 mL. **Protein-rich gel** (PRG) or **platelet-rich fibrin** is formed by combining PRP with thrombin and calcium chloride to form a semisolid material. **Conditioned media** is the collection of cytokines, chemokines, and growth factors from the media in which stem cells are cultured.

STEM CELLS

Research and application of stem cell therapies are still in the early stages. They constitute a major portion of the regenerative medicine treatment market, which is expected to reach 8 billion dollars by the year 2020.[1] Minimal regulation, particularly in veterinary medicine, has led to numerous unsubstantiated claims regarding the efficacy of many stem cell treatment protocols. The International Society for Stem Cell Research (ISSCR) provides guidelines for responsible use of stem cell therapies and information regarding efficacy and risks.

Stem cells are distinguished from other cell types by their ability to renew themselves through cell division (even after long periods of dormancy) and their ability to be induced to become tissue- or organ-specific cells with special functions under certain physiologic or experimental conditions. In some organs (e.g., gut, bone marrow) stem cells regularly divide to replace damaged tissues. In other organs, including the heart and pancreas, stem cells only divide under special conditions. Thus in many tissues stem cells serve as an internal repair system, dividing to replenish other cells; each new cell has the potential either to remain a stem cell or to become a more specialized cell (e.g., muscle, red blood, cartilage, brain cell). Until recently, scientists primarily worked with two kinds of stem cells from animals and humans: embryonic stem cells and nonembryonic "somatic" or "adult" stem cells.

Adult Stem Cells

Adult stem cell is the term used to describe postnatal stem cells that persist throughout life and function to repair or replace cells within certain tissues in response to traumatic events or natural cell turnover. Adult stem cells have been identified in many organs and tissues, including brain, bone marrow, peripheral blood, blood vessels, skeletal muscle, skin, teeth, heart, gut, liver, and the reproductive organs of males and females. They are thought to reside in a specific area of each tissue (called a *stem cell niche*). Among tissues identified to harbor stem cells throughout postnatal life, one of the most studied is bone marrow, which serves as a source of both hematopoietic stem cells (HSCs) and MSCs.

Stem cells may remain quiescent (nondividing) for long periods of time until they are activated by disease or tissue injury or by a normal need for additional cells to maintain tissues. When needed, these stem cells may differentiate into (1) HSCs, which give rise to all the types of blood cells (e.g., red blood cells, white blood cells) including cells that regulate immune function; (2) MSCs, which give rise to a variety of cell types including osteocytes, chondrocytes, adipocytes, and other kinds of connective tissue cells (e.g., tendon); (3) neural stem cells, which give rise to its three major cell types: neurons, astrocytes, and oligodendrocytes; (4) epithelial stem cells in the lining of the digestive tract, which occur in deep crypts and give rise to absorptive cells, goblet cells, Paneth cells, and enteroendocrine cells; (5) skin stem cells, which occur in the basal layer of the epidermis and at the base of hair follicles; (6) epidermal stem cells, which give rise to keratinocytes; and (7) follicular stem cells, which give rise to both the hair follicle and epidermis.

Strategies that utilize these cells for therapy are known as *cell-based therapies*. The best-known clinical application of cell-based therapies is bone marrow transplantation to repopulate the marrow and restore blood cells after high-dose chemotherapy or radiotherapy. However, because the need for donated organs and tissues often used to replace ailing or destroyed tissue far outweighs the available supply, the use of stem cells directed to differentiate into specific cell types is becoming increasingly important. Stem cells offer the possibility of a renewable source of replacement cells and tissues to treat many diseases including spinal cord injury, burns, heart disease, diabetes, osteoarthritis, and rheumatoid arthritis.

Adipose Derived Stem Cells

Adipose tissue is the richest source of stem cells in the body. There are four basic methods for obtaining and preparing stem cells for administration. (1) Adipose grafts have been injected into joints in combination with bone marrow concentrate. (2) The SVF is obtained by use of proteolytic enzymes and centrifugation. This produces a mixture of hematopoietic stem cells, endothelial cells, and adipose derived stem cells. Kits are available to produce SVF at point of care. Flow cytometry, based on MSC reactivity to a number of antibodies, may be used to select for expressed antigens to remove nonmesenchymal cells from the sample. For instance, antibodies against CD34, a surface marker found on hematopoietic cells, are frequently used to identify and remove nonmesenchymal cells from a marrow culture. $CD34^+$ cells represent a major category of human stem cell populations, and vascularization is critical in early healing of regenerating tissues. ($CD34^+$ is presumably a common feature of human MSCs, but canine MSCs are considered $CD34^-$). (3) A third technique is to isolate the cells and treat them with growth factors/cytokines to stimulate their differentiation into the cell type of interest (e.g., chondrocyte, cardiac myocyte). Osteogenic differentiation of MSCs can be induced in vitro by treating a monolayer culture with a pro-osteogenic cocktail such as dexamethasone, ascorbic acid-2-phosphate, and β-glycerophosphate. For chondrogenic differentiation, bovine insulin, transferrin, selenious acid, linoleic acid, bovine serum albumin, sodium pyruvate, proline, L-glutamine, or transforming growth factor-β1 (TGF-β1) may be added. MSCs have been shown to have the ability to form muscle cells and cardiomyocytes when treated with the demethylating agent 5-azacytidine. This technique may still result in a relatively low yield of stem cells (albeit predifferentiated cells). (4) The most complex technique (which is considered to be the gold standard) is to obtain the cells, expand the cell population in culture, and then treat them with growth factors. This latter technique, in which an expanded cell population is administered, results in significantly greater cell numbers being produced.

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Bone Marrow Derived Stem Cells

Bone marrow is commonly used as a bone graft in healing fractures in veterinary medicine. Postoperative techniques involve aspirating bone marrow and injecting percutaneously with or without morselization of the aspirate. These point of care techniques are referred to as *bone marrow concentrate* or *bone marrow aspirate concentrate*. Stem cells (mesenchymal) account for only 0.01% to 0.001% of bone marrow cells in comparison to adipose tissue, which contains 1% to 10% stem cells (MSCs, hematopoietic, and others). Abrasion arthroplasty and microfracture are arthroscopic means to direct bone marrow and its stem cells to healing cartilage lesions.

MSC concentrations from bone marrow are increased by centrifugation and culture. This enables removal of $CD34^+$ cells. The addition of growth factors can enable the production of billions of MSCs from a small bone marrow aspirate.

Numerous factors may affect the number and function of stem cells, including the age and physical status of the donor, tissue source (e.g., fat, bone marrow), method of harvest, and manipulation, handling, and storage of the sample.

Autologous Versus Allogeneic Stem Cells

Mesenchymal stem cells are multipotent adult stem cells that can differentiate into different tissues originating from mesoderm, ranging from bone and cartilage to cardiac muscle. MSCs are an excellent candidate for cell therapy because they are easily accessible (e.g., fat, bone marrow, skin), their isolation is straightforward, they can be greatly expanded (thus many cells can be derived from a single donor), they can be biopreserved with minimal loss of potency, and there appears to be little or no significant adverse reactions to allogeneic versus autologous MSC transplants. There is minimal information on repeated use of allogenic cells and possible subsequent immune responses. Stem cells appear to have potent immunosuppressive properties, allowing them to modulate the function of all major immune cell populations, thus impeding immune responses; however, the underlying mechanisms of their differentiation and function are not thoroughly understood.

MSCs may be *autologous* (from self), *allogeneic* (from a donor of the same species), or *xenogeneic* (from another species). Although human MSCs have been shown to maintain their multipotential capacity after transplantation into sheep, the immune response to xenografts (adult) is much more vigorous than to allografts because of the greater antigenic differences that exist among different species than within a species. Thus most current research is being done on allogeneic and autologous MSCs. MSCs represent an advantageous cell type for allogeneic transplantation because evidence suggests that MSCs are immune-privileged with low major histocompatibility complex I and no major histocompatibility complex II expression, therefore reducing risks of rejection and complications for transplantation. Although MSCs may be protected from the immune system, many clinicians believe that autologous MSCs are preferred over allogeneic stem cells. There are advantages and disadvantages of each approach. Minimal federal regulation makes commercial production of autogenous stem products substantially cheaper and easier than allogeneic products, which are regulated. However, controversy exists as to the number of stem cells that are being returned to the patient and their activity when autologous processes are used. Several studies have provided evidence that the ability of stem cells to respond to environmental demands may be diminished during aging; thus patients that are old or sick and perhaps most in need of stem cells may have the fewest or least functional stem cells with which to repair their own tissues.

Use of allogeneic MSCs is currently being studied by numerous groups and has been widely tested in clinical trials of cardiovascular, neurologic, and immunologic disease in humans, with encouraging results. There are examples of MSC utilization in the repair of ischemic heart disease, chronic kidney disease, and inflammatory lung disease (e.g., acute lung injury, chronic obstructive pulmonary disease, asthma, and pulmonary hypertension) and in the treatment of chronic wounds; however, much research is still needed before using these cells is considered a routine part of clinical practice. Of concern is whether differentiation of MSCs will lead to induced immunogenicity and thus limit their long-term benefit in diseases such as myocardial infarction.

PLATELET-RICH PLASMA

PRP has the potential to aid in the management of numerous surgical diseases by encouraging healing through the delivery of growth factors. Other advantages include the autogenous source and point of care convenience. Numerous studies have investigated the use of PRP in combination with stem cells to provide both growth factors and pluripotential regenerative cells. Extrapolation of laboratory studies and human clinical studies must be done with caution, and currently there is inadequate information regarding ideal ratios and concentrations of platelets and leukocytes. This is particularly true in small animals where there are limited published data. However, results to date of laboratory studies and clinical equine and human studies support the investigation of the use of PRP in small animal surgery.

Platelets are among the first cells to migrate to sites of tissue trauma. In addition to their role in hemostasis, they contain growth factors that stimulate tissue healing (see Fig. 31.12B). Alpha granules in platelets contain numerous growth factors including platelet-derived growth factor (PDGF), TGF-β1, TGF-β2, vascular endothelial growth factor (VEGF), basic fibroblastic growth factor, and epidermal growth factor (EGF). Other factors contained in platelets that may affect tissue inflammation and healing include antibacterial and fungicidal proteins, metalloproteases adenosine diphosphate, adenosine triphosphate, calcium ions, histamine, serotonin, and dopamine.

PDGF and TGF-β are critical modulators of tissue healing. PDGF is active in early wound healing, including stimulation of mitogenesis to increase the number of regenerative cells, angiogenesis, and activation of macrophages, which are responsible for wound cleaning and the generation of additional growth factors. TGF-β accelerates the production of collagen from fibroblasts in the later stages of healing. TGF-β1 and -β2 are active in chemotaxis and mitogenesis of preosteoblasts and stimulate collagen deposition during connective tissue healing and bone formation. Insulin-like growth factor-I (IGF-1) has been demonstrated to induce proliferation, differentiation, and hypertrophy of multiple cell lines. IGF-1 and IGF-2 increase the number of osteoblasts and appear to accelerate bone formation. PRP may also decrease excessive early inflammation and subsequent scar formation through modulation of interleukin-1 production.

Formation of PRP

PRP is created by centrifugation of autologous blood followed by extraction of the plasma and buffy coat portion, which contain

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high concentrations of platelets (Figs. 31.13 and 31.14). Several manufacturers have developed devices for the production of PRP, although variations in centrifuge design result in clinically significant differences in the resulting product (Table 31.4). Differences among the systems include the volume of whole blood, time and speed of centrifugation, use of anticoagulant, and resulting volume and quantity of platelets in the concentrate. Platelet gels may be subsequently formed from the concentrate by addition of calcium chloride and thrombin. The gel product may aid in limiting the disbursement of the platelets and growth factors.

The American Red Cross defines protein-rich plasma as 5.5 $\times 10^{10}$ platelets/50 mL. The concentrations of platelets in PRP from the differing preparation methods vary from 2.5 to 8 times that of whole blood (see Table 31.4). It has been recommended that a four times increase in concentration be used, although there is little clinical evidence to support this recommendation.

Of additional concern in the formation and efficacy of PRP may be the concentration of leukocytes within the preparation. Increased leukocyte concentration has been associated with increased scar tissue formation and increased collagen degradation ex vivo. Higher leukocyte concentrations may correlate negatively with markers of matrix production in tendons. Leukocyte concentrations in different manufacturers' products vary tremendously, with platelet to white blood cell ratios ranging from 800:1 to 51:1 (see Table 31.4). The characteristics of PRP from commercially available systems have been reported in two recent studies.[2,3]

Conditioned Media

Conditioned media or conditioned culture media (CM) is the collection of cytokines chemokines and growth factors from the media in which cells are cultured. Cells in culture are exposed to specific environmental conditions that results in a cellular secretome that may aid in the treatment of specific conditions. The primary mechanisms of action of CM are trophic and

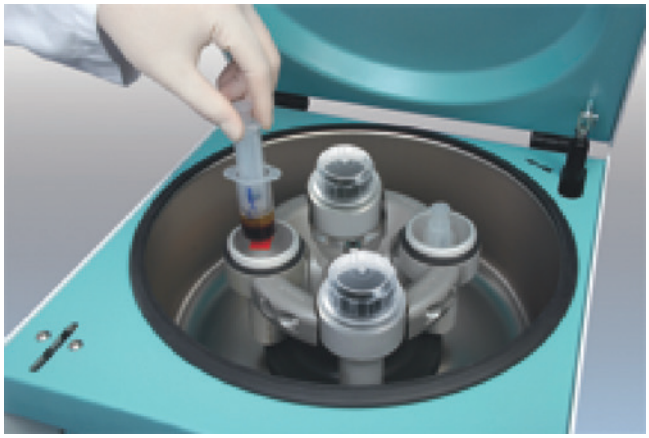


FIG. 31.13 Centrifuge for formation of platelet-rich plasma. (Courtesy Arthrex Corporation.)



FIG. 31.14 Arthrex Angel platelet-rich plasma (PRP) centrifuge and resulting PRP. (Courtesy Arthrex Corporation.)

TABLE 31.4 Characteristics of Canine Platelet-Rich Plasma Prepared With Five Different Systems

	PLATELET-RICH PLASMA CONCENTRATING SYSTEM				
Variable	**1**	**2**	**3**	**4**	**5**
Whole Blood					
Platelet count (cells/μL)	207,133 ± 37,444	226,533 ± 40,555	249,933 ± 37,051	260,867 ± 47,885	252,867 ± 39,279
Leukocyte count cells/μL)	8,467 ± 2,605	7,733 ± 2,198	8,200 ± 2,303	8,047 ± 2,474	7,787 ± 2,451
Hct (%)	42.2 ± 3.7	39.9 ± 2.9	43.3 ± 3.5	42.5 ± 0.7	41.3 ± 4.4
PRP					
Platelet count (cells/μL)	169,933 ± 3,695	743,000 ± 301,719	452,800 ± 185,747	1,340,677 ± 285,520	135,667 ± 514,614
Leukocyte count (cells/μL)	1,100 ± 600	1,393 ± 1,128	19,967 ± 4,936	25,807 ± 6,993	10,927 ± 4,894
Hct (%)	0	0	22,2 ± 1.6	13.4 ± 4.0	0.06 ± 0.2

Mean ± SD values for characteristics of whole blood samples obtained from 15 dogs between 18 months and 9 years of age with no concurrent disease, except for osteoarthritis in some dogs, and PRP products prepared by use of a variety of commercially available systems.
1, Protec PRP, PulseVet, Alpharetta, GA; *2,* MediVet PRP, MediVet America, Nicholasville, KY; *3,* C-PET, Pall Corp, Port Washington, NY; *4,* SmartPReP 2, Harvest Technologies, Plymouth, MA; *5,* Angel, Arthrex Vet Systems, Naples, FL.
Hct, Hematocrit.
From Franklin SP, Garner BC, Cook JL. Characteristics of canine platelet-rich plasma prepared with five commercially available systems. *Am J Vet Res.* 2015;76(9):822–827.

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paracrine effects on target cells and tissues. Conditioned media are not yet available commercially in veterinary medicine but hold the promise of being an off the shelf product.

PROPOSED MECHANISMS OF ACTION OF STEM CELLS, PRP, AND CONDITIONED MEDIA

Stem Cells

Numerous mechanisms of action have been proposed regarding the beneficial response to stem cell treatment in certain diseases (Fig. 31.15). (1) Stem cells may engraft in the existing tissue if the environmental conditions (biochemical, vascular, and mechanical) are favorable. Engrafting may be limited or nonexistent in diseases such as osteoarthritis due to poor nutrient supply, high mechanical forces, and high levels of proteases and other enzymes. (2) Stem cells may have trophic effects through the secretion of growth factors, cytokines, and chemokines in addition to antiapoptotic factors. This potential effect is also the proposed mechanism of action of PRP and conditioned medium from stem cell cultures. The MSC and CM secretome and PRP may also act on the regional stem cell niche to promote proliferation. (3) Stem cells may have significant antiinflammatory actions in the target tissue. Mesenchymal stem cells may be induced by local inflammatory chemicals to produce iNOS, interleukin-1Ra, and TSG-6 among other agents to decrease inflammation.

Clinical Applications and Evidence

Tendon and Ligament

Tendons and ligaments have limited blood supply, limited cellularity, and poor healing characteristics; however, the frequency of these injuries in humans and animals makes this an area of high interest for the use of regenerative medicine. Studies in laboratory animals have demonstrated increased tendon callus production and increased force to failure of tendon healing when the tendon repair sites were treated with PRP. Growth factors found in PRP including TGF-β1, PDGF, VEGF, and EGF increase type I collagen production and tenocyte proliferation.

Human. Critical analysis of currently available clinical data on the use of autologous stem cells and PRP as treatment for various tendon and ligament abnormalities was reported recently.[4] Evidence of efficacy is strongest for PRP as a treatment for lateral epicondylitis and patellar tendinopathy; however, current evidence does not suggest that PRP provides a substantial benefit when used for the treatment of disorders of the rotator cuff or anterior cruciate ligament. There are currently not enough data to strongly recommend either for or against autologous stem cell therapies. A lack of standardization among studies severely limits the ability to draw conclusions from the currently available data on biological treatments for tendon and ligament abnormalities.

Canine. Experimental studies have provided moderate to strong evidence of the benefit of PRP application in tendon healing,[5] and intraarticular PRP injection in the canine model of ligament injury (Video 31.1).[6] The effect of postsurgical intraarticular injections of autologous platelet concentrates after cranial cruciate ligament reconstruction by fascia lata autograft was studied in 10 dogs, reporting marginal benefit in limb function 90 days postoperatively.[7] A case series of 10 dogs with supraspinatus tendinopathy reported subjective improvement of lameness and function after regional PRP injection; however, objective kinetic data failed to demonstrate any benefit of PRP injection.[8]

Cartilage and Osteoarthritis

Osteoarthritis is one of the most common diseases in humans and veterinary patients and the poor healing of articular cartilage makes treatment exceedingly challenging. Articular cartilage has a limited capacity for self-repair because it is both avascular and aneural; untreated injuries of cartilage often result in osteoarthritis (OA) (see Chapter 34). Numerous forms of stem cells have been

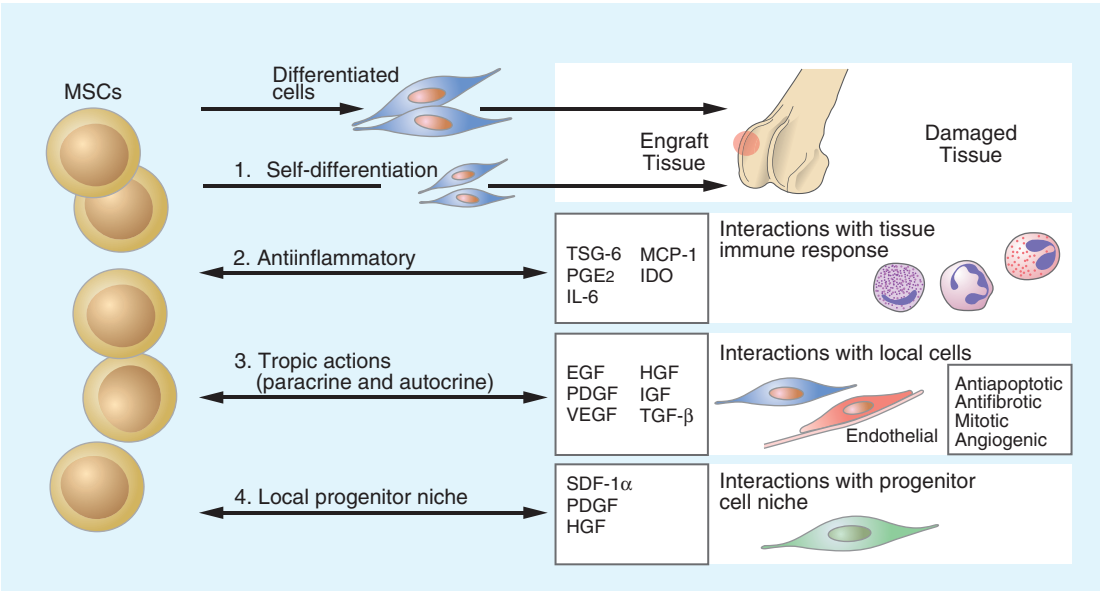


FIG. 31.15 Proposed mechanisms of action of stem cells. *EGF,* Epidermal growth factor; *HGF,* hepatocyte growth factor; *IDO,* indoleamine 2,3-dioxygenase; *IGF,* insulin-like growth factor; *IL,* interleukin; *MCP,* monocyte chemotactic protein; *PDGF,* platelet-derived growth factor; *PGE2,* prostaglandin E2; *SDF,* stromal cell-derived factor; *TGF,* transforming growth factor; *TSG,* TNF-stimulating gene; *VEGF,* vascular endothelial growth factor.

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studied clinically in the treatment of joint disease. Adverse events are rare. The two major methods of delivery are syringe injection and arthroscopic/surgical application.

Human. Intraarticular stem cell injections have been reported to result in minimal meniscal regrowth, cartilage protection, and decreased joint damage in patients undergoing partial meniscectomy. The number of patients with meniscal regrowth was very small, and the improvement was only transient.[1] Numerous studies have examined the effects of needle injection of stem cells for the management of cartilage lesions and osteoarthritis. The sample size in these studies was small but generally showed minimal complications and fair to good improvement.[1] A combination of stem cells and PRP has been used in studies of OA with most studies demonstrating an improvement in visual analog scale.[1] Complications included joint pain and swelling, as well as tendonitis. Surgical/arthroscopic implantation of stem cells with and without PRP has been performed for variations of cartilage lesions in humans. Clinical studies have generally demonstrated only modest success in healing of cartilage lesions.[1]

The efficacy of intraarticular cellular therapy has not yet been established. A 2016 systematic review of the current literature regarding the safety and efficacy of cellular therapy delivered by intraarticular injection in the knee suggested positive results with respect to clinical improvement and safety.[9] However, the improvement was modest, and a placebo effect cannot be disregarded. The value and effective use of cell therapy in OA remain unclear largely because of the absence of (1) rigorous blinded clinical trials, (2) standardized use of nomenclature to define cell populations, and (3) quantitative metrics to define cell populations and clinical and structural outcomes. Although many of the studies reported were randomized, patients had not been blinded. Because cellular therapy carries a high level of expectation for possible benefits, it can constitute a strong source of bias in enrollment and in perception of patient-reported outcome.

The efficacy of intraarticular PRP therapy has not yet been established.[10] In a 2015 randomized, blinded (patients and outcome assessors), controlled trial with 12 months of follow-up in patients with OA, injections of PRP did not provide more benefit than injections of hyaluronic acid.[11]

Canine. Experimental studies have provided moderate to strong evidence of benefit of intraarticular PRP injection in canine models of OA.[12] A randomized, controlled clinical trial in 20 client-owned dogs with OA involving a single joint (predominantly stifle) reported significant improvement in subjective assessment and objective kinetic data at 12 weeks after autologous platelet intraarticular injection.[13]

A prospective, randomized, controlled clinical trial in 30 client-owned dogs evaluated the efficacy of multiple intraarticular injections of adipose stromal cell therapy for treatment of elbow OA and fragmented medial coronoid process when used as an adjuvant to standard of care treatment.[14] Objective kinetic data showed the benefit of autologous SVF 6 months after injection, and failed to show the benefit of allogeneic cultured stem cell injection. A randomized, blinded clinical trial in 93 client-owned dogs with predominantly hip and elbow OA reported significant benefits of allogenic adipose-derived stem cells compared with the control group based with a validated subjective outcome measure.[15] This study did not report objective kinetic data, and the placebo effect was extremely high.

A clinical trial to evaluate effects of simultaneous intraarticular and IV injection of autologous adipose-derived SVF and PRP to 10 client-owned dogs with hip OA reported significant improvement in objective kinetic data in dogs treated with SVF and PRP compared with the control group.[16] The benefits of the reported treatment remain controversial based on the limitations of this study.

Bone

PRP may aid in fracture repair and healing by providing additional growth factors that are critical to bone formation. TGFs initiate bone healing and support long-term bone regeneration and remodeling. TGF-β1 and -β2 support chemotaxis and mitogenesis of preosteoblasts and promote collagen deposition. TGF stimulates increased numbers of fibroblasts and preosteoblasts and supports cell differentiation into mature osteoblasts, resulting in increased bone matrix. Additionally, TGF appears to inhibit osteoclast formation and bone resorption. IGF-1 and IGF-2 also appear to increase the number of osteoblasts and accelerate bone deposition during fracture healing. PRG has been used for bone formation most extensively in human dental and maxillofacial applications. PRG has frequently been combined with an osteoinductive material such as demineralized bone matrix.

Results of clinical and laboratory studies on the efficacy of PRP in accentuation of bone healing are conflicting. Canine laboratory studies have shown a negative effect of PRG and allogenic bone in comparison to allogenic bone alone in a model intended to simulate in-growth of a cementless prosthesis. However, other laboratory studies have demonstrated a significant increase in bone formation attributable to PRP when PRP was combined with other osteoinductive treatments.[21]

Human. The efficacy of biologic product in bone healing has not been established in any clinical trial.[17] A systematic review and meta-analysis was conducted to determine the efficacy of autologous blood concentrates in decreasing pain and improving healing and function in patients with orthopedic conditions, including bone injuries.[18] Fifteen randomized controlled trials and five prospective cohort studies showed no clinical benefit with PRP. This study suggested considerable uncertainty about the evidence regarding a clinical benefit of the use of autologous blood concentrates for a variety of orthopedic disorders.

Canine. Experimental studies have provided moderate evidence of benefits of autologous stem cell application in augmentation of bone healing in canine ostectomy models.[19] Experimental studies have provided weak evidence of benefits of regional PRP injection in canine models of fracture healing.[20] There is currently no sufficient clinical evidence to support efficacy of stem cell or PRP applications in bone healing.

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32

Principles of Fracture Diagnoses and Management



DEFINITIONS

Pathologic fracture is a fracture secondary to disease of the bone (e.g., neoplasia, infection). **Coaptation**, which literally means "joining together," refers to the joining of two parts of a fractured bone. A **composite** is a combination of two or more structures. In fracture repair it refers to the combination of orthopedic implants or external stabilization with the reconstructed bone, which provides biomechanical stability for healing and weight bearing. **Comorbidities** refer to the presence of more than one disease or injury. **Comminution** denotes multiple fragmentation of a fractured bone.

PERIOPERATIVE MANAGEMENT

Fractures are the result of either trauma or underlying bone pathology, or both. Differentiation between these causes is based on patient signalment, history, radiographic findings, and additional diagnostics, including cytology and biopsy. In cases of trauma, it is critical to first triage the patient for comorbidities.

Patient Triage, Wound Management, and Preoperative Analgesia

The purpose of triage is to determine if there are problems that require immediate attention prior to complete assessment of the patient. If a complete physical examination cannot be performed at the time of presentation, a strategy should be developed to complete the examination as soon as possible to avoid missing concurrent medical conditions.

Animals presenting for orthopedic evaluation following blunt trauma (e.g., automobile trauma, falling injuries) should be evaluated for comorbidities common in these patients, including those of the following systems: cardiovascular (e.g., shock and arrhythmias), respiratory (e.g., pneumothorax, diaphragmatic hernia, pulmonary contusions), neurologic (e.g., spinal trauma, brain trauma, peripheral nerve injuries), and urinary (e.g., urinary bladder rupture, urethral tear). Less common blunt trauma injuries include body wall hernias, splenic and hepatic ruptures, and gall bladder rupture. Thoracic radiographs and serial electrocardiograms should be performed in animals with significant blunt trauma. Evaluation of the abdominal cavity is done initially with palpation and evaluation of serum chemistry tests. Abdominocentesis (see p. 534) and radiographic or ultrasonographic evaluation of the urinary tract (see p. 698) should be performed if clinical signs (abdominal distention) or chemistry findings (azotemia) suggest injury. It is crucial to diagnose and treat these injuries before the animal is anesthetized for fracture repair or joint trauma.

Once the patient has been triaged and is stable, a thorough examination must be completed. The patient should be thoroughly examined for preexisting diseases that may affect surgical repair or recovery, or may affect the owner's willingness to have surgery performed when the prognosis and treatment of these comorbidities are taken into consideration. Fracture repair should never proceed unless a complete physical examination has been conducted.

Proper wound management is critical to successful treatment of trauma patients. Large or severe wounds may require extensive management and multiple sedations or anesthesia, and may also significantly increase cost, affect function due to muscular or neurologic injury, lead to increased length of hospitalization and risk of infection, and/or delay bone healing due to damage to blood supply. Wound management is discussed in Chapter 15.

Pain Management and Anesthesia

Orthopedic patients benefit from perioperative analgesics (Boxes 32.1 and 32.2). The projected level of postoperative discomfort and duration of discomfort should be assessed to determine the choice of preoperative analgesic. Most orthopedic surgeries are considered moderately to severely painful. Although there is much debate concerning preemptive analgesia, especially in the human medical literature, there are several principles that must be followed in order for preemptive analgesia to be successful. First, the depth of analgesia must be sufficient to block pain receptors during surgery. Second, nociceptive receptors from the entire field must be blocked. And last, analgesia must be continued in the postoperative period (see Chapter 13).

Anesthetic protocols should be chosen based on signalment, physical examination findings, and laboratory analysis. Patients treated for elective orthopedic problems (e.g., cruciate reconstruction) and who have no preoperative findings suggestive of major organ dysfunction can be managed using a variety of anesthetic techniques (Table 32.1). Patients with cardiovascular compromise or trauma should be anesthetized with care (Table 32.2). When appropriate, balanced anesthetic protocols that include analgesic agents supplemented with epidural analgesia or nerve blocks are recommended to decrease intraoperative pain response and reduce the amount of anesthetic needed (Table 32.3). Epidural anesthesia (with lidocaine, bupivacaine, or ropivacaine) in combination with general anesthesia provides profound relaxation by temporarily paralyzing rear limb muscles, easing fracture reduction of the pelvis, femur, and tibia (see Chapter 13). The duration of action depends on the drug used, ranging from 1 to 6 hours. Morphine or buprenorphine can be added to the epidural injection, providing postoperative pain relief for up to 24 hours. Additionally, opioids

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ABSTRACT

An understanding of the principles of fracture repair is critical to the successful treatment of fractures in dogs and cats. Failure to provide adequate stability can lead to nonunion, malunion, and serious secondary medical complications.

KEYWORDS

Fracture
comminution
reduction
nonunion
malunion
bone graft

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BOX 32.1 Perioperative Analgesia for Dogs[a]

- Fentanyl CRI (2 µg/kg IV loading dose, then 2–20 µg/kg/h IV), *or*
- Morphine[b] (0.5–2 mg/kg IM, SC q4h *or* 0.18 mg/kg IV q1h CRI), *or*
- Oxymorphone (0.03–0.1 mg/kg IV, IM q2–4h), *or*
- Hydromorphone (0.05 mg/kg IM, SC q2–4h *or* 0.05–0.1 mg/kg IV q2–4h) *or*
- Hydromorphone CRI (0.025–0.1 mg/kg per hour IV), *or*
- Buprenorphine (0.005–0.02 mg/kg IV, IM q4–8h), *plus*
- +/– Ketamine CRI (2 µg/kg/min IV; if no previous loading dose, give 0.5 mg/kg IV before CRI)

[a]Avoid nonsteroidal antiinflammatory drugs in hypotensive and hemorrhaging patients.
[b]Give slowly to prevent histamine release.
CRI, Constant-rate infusion; *IM,* intramuscular; *IV,* intravenous.

BOX 32.2 Perioperative Analgesia for Cats

- Fentanyl CRI (1 µg/kg IV loading dose, then 4 µg/kg/h IV), *or*
- Morphine[a] (0.05–0.4 mg/kg IM, SC q3–4h), *or*
- Oxymorphone (0.01–0.05 mg/kg IV, IM q2–4h), *or*
- Hydromorphone (0.03-0.1 mg/kg IM, SC q2–4h *or* 0.05–0.1 mg/kg IV q2–4h), *or*
- Buprenorphine (0.01–0.02 mg/kg OTM q6–12h), *plus*
- +/– Ketamine CRI (2 µg/kg/min IV. If no previous loading dose, give 0.5 mg/kg IV prior to CRI)

[a]Give slowly to prevent histamine release.
CRI, Constant-rate infusion; *IM,* intramuscular; *IV,* intravenous; *OTM,* oral transmucosal.

TABLE 32.1 Anesthetic Considerations in the Stable Patient Undergoing Fracture Repair

Preoperative Considerations

Associated conditions	Minimal; usually healthy
Bloodwork	HCT TP In patients >5–7 y, consider electrolytes, liver enzymes, BUN, and Cr
Physical exam	May be painful at presentation
Premedications	If patient is anxious give: • Diazepam (0.2 mg/kg, IV), *or* • Midazolam (0.2 mg/kg IV, IM), *plus* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM in dogs; 0.05–0.1 mg/kg IV, IM in cats), *or* • Oxymorphone (0.05–0.2 mg/kg IM, IV), *or* • Morphine[b] (0.1–0.2 mg/kg IV or 0.2–0.4 IM), *or* • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM)

Intraoperative Considerations

Induction	If premedicated, give: • Propofol (2–4 mg/kg IV), *or* • Alfaxalone (2–3 mg/kg IV) If not premedicated given, then: • Propofol (4–8 mg/kg IV), *or* • Alfaxalone (2–5 mg/kg IV), *or* • Ketamine (5.5 mg/kg IV) with diazepam (0.28 mg/kg IV)
Maintenance	• Isoflurane or sevoflurane, *plus* • Fentanyl (2–10 µg/kg IV PRN in dogs; 1–4 µg/kg IV PRN in cats) for short-term pain relief, *plus* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM in dogs; 0.05–0.1 mg/kg IV, IM in cats), *or* • Oxymorphone (0.05–0.2 mg/kg IV), *or* • Morphine[b] (0.1–1 mg/kg IV PRN in dogs; 0.1–0.2 mg/kg IV PRN in cats), *or* • Buprenorphine[c] (0.005–0.02 mg/kg IV), *plus* • Ketamine (low dose) (0.5–1 mg/kg IV), *or* • Ketamine CRI (0.5 mg/kg IV loading dose, then 10 µg/kg/min IV) • For muscle relaxation, give: *(need to provide intermittent positive ventilation with these agents. It is recommended to monitor the level of blockade with a train of four.)* • Vecuronium (0.01–0.1 mg/kg IV), *or* • Atracurium (0.1–0.25 mg/kg IV), *or* • Cisatracurium (0.02–0.1 mg/kg IV)
Fluid needs	• 5–10 mL/kg/h plus 3 × EBL • Colloids 2–5 mL/kg/h
Monitoring	Blood pressure HR ECG Respiratory rate SpO_2 Temperature $EtCO_2$
Blocks Epidural:	If relaxation/paralysis of rear limb required, then: • Bupivacaine (0.5% 0.22 mL/kg) duration 2–5 h, *or* • Ropivacaine (0.5% 0.22 mL/kg) duration 2–5 h, *or* • Lidocaine (2% 1 mL per 4.5 kg of body weight), duration of 1–2 h If relaxation/paralysis is not desired, then: • Morphine (0.1 mg/kg preservative free), *or* • Buprenorphine (0.003–0.005 mg/kg diluted in saline)

Continued

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TABLE 32.1 Anesthetic Considerations in the Stable Patient Undergoing Fracture Repair—cont'd

Postoperative Considerations

Analgesia	• Fentanyl CRI (1–3 µg/kg IV loading dose, then 2–20 µg/kg/h IV), *or*
	• Hydromorphone CRI (0.025–0.1 mg/kg/h IV, 0.1–0.22 mg/kg IM, SC q4–6h in dogs), *or*
	• Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q2–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats), *or*
	• Morphine[b] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.1–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h in cats), *or*
	• Morphine CRI (0.2 mg/kg loading dose followed by 0.1 mg/kg/h), *or*
	• Oxymorphone (0.05–0.2 IV, IM q2–4h), *or*
	• Buprenorphine[c] (0.005–0.02 mg/kg IV, IM q4–6h in dogs and cats; 0.01–0.04 mg/kg OTM q8h in cats), *plus*
	• +/– Ketamine CRI (if no previous loading dose, give 0.0.3–0.5 mg/kg IV prior to CRI of 0.3–0.6 mg/kg/h during surgery, give 0.12 mg/kg/h after surgery), *plus*
	• Consider NSAIDs
	  • In dogs: carprofen, deracoxib, meloxicam (see Table 32.4)
	  • In cats: meloxicam,[d] robenacoxib (see Table 32.4)

Monitoring	SpO_2 Blood pressure HR Respiratory rate Temperature
Bloodwork	HCT and TP if large blood loss
Estimated pain score	Can be severe depending on etiology or procedure; some of these patients have been in chronic pain

[a]Monitor for hyperthermia in cats.
[b]Give slowly to prevent histamine release.
[c]Buprenorphine is a better analgesic than morphine in cats.
[d]Black box warning added by the Food and Drug Administration in October 2010 identified cases of renal failure and death in cats with repeated uses of meloxicam. Meloxicam is approved for single use only in cats in the United States.
BUN, Blood urea nitrogen; *Cr,* creatinine; *CRI,* constant-rate infusion; *EBL,* estimated blood loss; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *NSAID,* nonsteroidal antiinflammatory drug; *OTM,* oral transmucosal; *PO,* orally; *PRN,* as needed; *SC,* subcutaneous; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein.

TABLE 32.2 Anesthetic Considerations for Fracture Stabilization in the Acute Trauma Patient

Preoperative Considerations

Associated conditions	Anemia Hypotension Dehydration Electrolyte abnormalities Dysrhythmias; often ventricular Presence of other trauma (e.g., pelvic, abdominal, thoracic)
Bloodwork	HCT Electrolytes BUN Cr TP Albumin +/– Blood gas
Physical exam	• May be dehydrated or in shock, hypotensive, tachycardic or bradycardic, and hypothermic • There will be an absence of pulmonary sounds in the dorsal lateral chest wall with auscultation if there is a pneumothorax; absence of pulmonary sounds in the ventral chest wall and muffled heart sounds if there is hemothorax; the abdomen may be painful if the animal has a uroabdomen or hemoabdomen
Other diagnostics	• Blood pressure • ECG • SpO_2 • Radiographs (thoracic and abdominal) • +/– Abdominal ultrasound • Thoracocentesis if dyspneic

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TABLE 32.2 Anesthetic Considerations for Fracture Stabilization in the Acute Trauma Patient—cont'd

Premedications	• Oxygen with flow-by or face mask
	• Give shock dose of fluids if necessary to stabilize. Reconsider need for surgery if patient is unstable.
	• Give blood transfusion if HCT <20% in dogs; <18% in cats (see pp. 27 and 32)
	• Avoid sedatives in depressed or dyspneic patients
	• In patients that have minimal respiratory compromise give:
	  • Midazolam (0.1–0.2 mg/kg IV, IM), *or*
	  • Diazepam (0.1–0.2 mg/kg IV)
	• In patients that are not depressed or dyspneic, give:
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM in dogs; 0.05–0.1 mg/kg IV, IM in cats), *or*
	  • Oxymorphone (0.05–0.2 mg/kg IV, IM), *or*
	  • Morphine[b] (0.1–0.2 mg/kg IV or 0.2–0.4 IM), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg, IV, IM)
	• If pulmonary involvement, avoid all respiratory depressants before induction, including but not limited to opioids, xylazine, medetomidine, and dexmedetomidine.
	• If hypotensive, avoid acepromazine, xylazine, medetomidine, and dexmedetomidine.
	• If tachycardic, avoid induction dose ketamine, atropine, and glycopyrrolate.

Intraoperative Considerations	

Induction	• Preoxygenate for 3–5 min with face mask or flow-by oxygen
	• Consider balanced induction technique:
	  • Fentanyl (5 µg/kg IV) plus midazolam (0.25 mg/kg IV) plus propofol (2–4 mg/kg IV)
	  • If normal to low heart rate, consider using ketamine (2–4 mg/kg IV) instead of propofol
	• If hypotensive, give:
	  • Etomidate (0.5–1.5 mg/kg IV); administer a benzodiazepine IV before etomidate to avoid myoclonus, use cautiously in patients that are in shock due to adrenocortical suppression.
	• If normotensive, give:
	  • Propofol (2–6 mg/kg IV), *or*
	  • Alfaxalone (2–5 mg/kg IV), *or*
	  • Ketamine (5 mg/kg with benzodiazepine 0.25 mg/kg IV)

Maintenance	• Isoflurane or sevoflurane, *plus*
	  • Fentanyl (2–10 µg/kg IV PRN in dogs; 1–4 µg/kg IV PRN in cats) for short-term pain relief, *plus* PRN
	  • Fentanyl CRI (1–5 µg/kg IV loading dose, then 2–30 µg/kg/h IV), *or*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV PRN), *plus*
	  • Ketamine (low dose 0.5–1 mg/kg IV), *or*
	  • Ketamine (0.5 mg/kg IV then CRI at 10 µg/kg/min IV), *or*
	  • Lidocaine (1–2 mg/kg IV loading dose then CRI at 1–3 mg/kg/h IV in dogs)
	• If hypotensive (to keep MAP 60–80 mm Hg), give phenylephrine, ephedrine, dopamine, norepinephrine, vasopressin, or fluids as needed
	• If pulmonary involvement, ventilation parameters:
	  • SpO_2 >95%
	  • Small tidal volumes: 5–7 mL/kg
	  • Respiratory rates: 10–30
	  • Peak airway pressures < 12–15 mm Hg if able to achieve adequate tidal volumes

Fluid needs	• 10–20 mL/kg/h to replace evaporative losses plus 3 × EBL • Consider colloids or blood for EBL replacement in 1:1 ratio
Monitoring	BP ECG Respiratory rate SpO_2 $EtCO_2$ Temperature +/– Arterial line U/O
Blocks	Epidural: • Morphine (0.1 mg/kg preservative free) *or* • Buprenorphine (0.003–0.005 mg/kg diluted in saline) • Avoid local anesthetics for spinals and epidurals in hypotensive patients Incisional: • Lidocaine (<5 mg/kg in dogs and 2–4 mg/kg in cats), *or* • Bupivicaine (<2 mg/kg)

Continued

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TABLE 32.2 Anesthetic Considerations for Fracture Stabilization in the Acute Trauma Patient—cont'd

Postoperative Considerations

Analgesia	• Fentanyl CRI (1–10 µg/kg IV loading dose, then 2–20 µg/kg/h IV), *or* • Morphine[b] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.05–0.2 mg/kg IV or 0.1–0.5 IM q1–4h in cats) if no hypotension, *or* • Oxymorphone (0.05–0.2 mg/kg IV, IM), *or* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV, IM q3–4h in cats), *or* • Hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs), *or* • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM q4–8h or 0.01–0.02 mg/kg OTM q6–12h in cats), *plus* • +/– Ketamine CRI (2 µg/kg/min IV); if no previous loading dose, give 0.5 mg/kg IV before CRI • Avoid NSAIDs in hypotensive and hemorrhaging patients.
Monitoring	SpO_2 Blood pressure ECG HR Respiratory rate Temperature U/O
Bloodwork	HCT TP Electrolytes +/– Blood gas +/– Albumin
Estimated pain score	Severe depending on amount of trauma

[a]Monitor for hyperthermia in cats.
[b]Give slowly to prevent histamine release.
[c]Buprenorphine is a better analgesic than morphine in cats.
BP, Blood pressure; *BUN,* blood urea nitrogen; *Cr,* creatinine; *CRI,* constant-rate infusion; *EBL,* estimated blood loss; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *HCT,* hematocrit; *HR,* heart rate; *IM,* intramuscular; *IV,* intravenous; *MAP,* mean arterial pressure; *NSAID,* nonsteroidal antiinflammatory drug; *OTM,* oral transmucosal; *PO,* orally; *PRN,* as needed; *SC,* subcutaneous; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein; *U/O,* urine output.

TABLE 32.3 Epidural Anesthesia in the Dog[a]

Epidural Volumes

Low Epidural

0.2 mL/kg results in analgesia/anesthesia of pelvis and caudal abdomen

High Epidural

- 0.3 mL/kg results in analgesia/anesthesia of pelvis, abdomen, and thorax
- Usually performed with only an opioid (saline can be used to increase the volume)
- Local anesthetics should be avoided in high epidurals to prevent sympathetic trunk blockade and intercostal nerve paralysis

Drug	Dose	Onset of Action (min)	Duration of Action (h)
Lidocaine 2%[b]	1 mL/4.5 kg	10	1–1.5
Bupivacaine (0.25% or 0.5%)[b] (preservative free)	1 mL/4.5 kg	20–30	4.5–6
Ropivacaine (0.5%)[b]	1 mL/4.5 mg per kg	20–30	4.5–6
Fentanyl	0.001 mg/kg	15–20	3–5
Morphine (preservative free)[c]	0.1 mg/kg[a]	30–60	10–24
Buprenorphine	0.004 mg/kg	10–45	12–18

[a]Dosage, onset, and duration are based on clinical experience; each patient should be evaluated individually. Reduce the dose by at least half for spinal administration of local anesthetics; reduce the epidural dose by 25% in geriatric, obese, and pregnant animals and in those with space-occupying lesions of the spinal cord or with conditions in which venous engorgement is expected.
[b]A block to T1 leads to intercostal nerve paralysis and sympathetic blockade resulting in bradycardia; a block to C5–C7 leads to phrenic nerve paralysis.
[c]Most common complication with opioid epidural is dose-related respiratory depression.

may be used alone in the epidural space when relaxation of muscles is not warranted but postoperative analgesia is desired. A brachial plexus block using local anesthetics will provide additional analgesia and muscle relaxation in patients undergoing surgery of the forelimb (see p. 152). A femoral-sciatic nerve block using local anesthetics will provide additional analgesia and relaxation for surgery on the rear limb (see p. 153).

Opioid analgesics should be administered for at least 12 to 24 hours after surgery, depending on the procedure and results of serial patient evaluations. Butorphanol or buprenorphine are often sufficient for patients undergoing procedures requiring minimal tissue manipulation. In patients undergoing more painful procedures that require significant tissue manipulation (e.g., triple pelvic osteotomy) or in those with traumatic injuries, hydromorphone, oxymorphone, or morphine is recommended. When there is minimal risk of continued blood loss, nonsteroidal antiinflammatory drugs (NSAIDs) may be administered for pain control.

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TABLE 32.4 Nonsteroidal Antiinflammatory Drugs Approved for Treating Dogs and Cats With Orthopedic Disease

Carprofen	*Dogs:* 2.2 mg/kg PO, SC q12h or 4.4 mg/kg PO, SC q24h *Cats:* 4 mg/kg SC, IV once
Deracoxib	*Dogs:* 1–2 mg/kg PO q24h[a]
Etodolac	5–15 mg/kg PO q24h
Meloxicam	*Dogs:* 0.2 mg/kg q24h IV, SC, PO once for 1 day, followed by 0.1 mg/kg q24h (in food) *Cats:* 0.05 mg/kg PO q24h with reduction in dose if long-term treatment is pursued. Long-term treatment may be reduced to 0.05 mg/kg PO q48h and as low as q72h. Single dose of 0.15 mg/kg SC but approved for doses up to 0.3 mg/kg SC.
Firocoxib	*Dogs:* 5 mg/kg PO q24h
Robenacoxib	*Dogs:* 1.0-2.0 mg/kg q24h *Cats:* 2 mg/kg SC between the shoulder blades or in cats weighing 2.5–6 kg give 6 mg/cat PO q24h for up to 3 days; in cats weighing 6.1–12 kg give 12 mg/cat PO q24h for up to 3 days. For days 3–11 give 1 mg/kg PO q24h.

[a]A higher dose (3–4 mg/kg/d) may be used short term for postoperative pain (do not confuse the two dosages).
PO, Orally; *SC,* subcutaneous.

NSAIDs are advocated for pain control in orthopedic patients (see Chapter 13 and Table 32.4). They can be used alone or in combination with opioids, depending on the severity of the animal's pain. Some opioids (e.g., fentanyl) provide immediate relief, whereas the onset of NSAIDs is 30 minutes to 1 hour. An advantage of NSAIDs is that they provide sustained relief; their duration of action is 12 to 24 hours. Intravenous hydromorphone, oxymorphone, and morphine have delayed onsets of action of 15 to 20 minutes but provide analgesia for only 3 to 4 hours. Although when given before surgery carprofen and deracoxib are effective for postoperative pain relief in dogs, the perioperative course can be unpredictable. Similarly, meloxicam injection is approved for use in cats preoperatively (one dose only), but its perioperative course may be unpredictable. It is prudent to reserve NSAIDs for postoperative administration, especially if blood loss is anticipated. Depending upon the NSAID chosen, there may be some inhibition of platelet function. Although carprofen, meloxicam, and etodolac only weakly inhibit cyclooxygenase-1 and thus platelet function, it is reasonable to wait until the postoperative period to add one of these to the pain protocol. Nonselective cyclooxygenase inhibitors (e.g., ketoprofen, phenylbutazone) are contraindicated for surgical procedures in which hemorrhage is anticipated. Additionally, NSAIDs should not be used in patients with marked hypotension, renal or hepatic disease, or those with severe pulmonary disease (especially asthmatic patients). NSAIDs can be continued orally for several days after surgery to provide pain relief after the animal has been discharged from the hospital. See p. 150 for additional information on NSAIDs. Given the aforementioned complications of NSAIDs, opioids are often the preferred medication for use as a preoperative analgesic because they do not alter coagulation. Additionally, opioids are also less apt to potentiate hypotension than are inhalant anesthetics.

Antibiotics

Prophylactic antibiotics are effective in preventing surgical site infections in orthopedic patients. Despite careful adherence to aseptic technique, bacteria are present during surgical procedures. Infection occurs when host defenses cannot contain the microorganisms. Factors influencing the number of bacteria required to cause infection include the virulence of the organism, condition of the wound, presence of implants, and host defenses. Prophylactic antibiotics help contain the bacterial population to that which can be managed by the host defenses.

Prophylactic antibiotics must be used properly to be effective. Antibiotic selection, timing of administration, dose, and duration are all important components. Antibiotic selection is based on two criteria: (1) which microorganisms are most likely to cause orthopedic wound infections and (2) which antibiotics are most likely to be effective against potential offending microorganisms. Coagulase-positive *Staphylococcus* species and *Escherichia coli* are the preponderant aerobic bacteria isolated from surgical wounds in small animal patients. Methicillin-resistant *Staphylococcus aureus* is frequently recognized as a pathogen in veterinary facilities. Anaerobic bacteria (e.g., *Bacteroides, Fusobacterium,* and *Clostridium* spp.) are infrequently recognized as important pathogens in orthopedic patients. Antibiotics are most effective when present in the tissues before bacterial contamination. Thus antibiotics must be administered intravenously 30 to 60 minutes before making the incision. Concentrations of the drug equal to or greater than the minimum inhibitory concentration needed to prevent proliferation of known pathogens are desirable. The duration of antibiotic administration should serve to maintain adequate serum and tissue concentrations while the wound is open. Antibiotic administration should be repeated every 2 to 4 hours and discontinued either at completion of surgery or no longer than 24 hours after surgery. Cefazolin, 22 mg/kg IV, administered after anesthetic induction and repeated every 2 to 4 hours during the procedure, is the protocol of choice for most orthopedic patients. Use of perioperative and postoperative antibiotics are discussed in detail in Chapter 9.

NOTE Antibiotics initiated after the procedure are not effective in preventing infection.

TEMPORARY FRACTURE STABILIZATION

Temporary fracture support may be considered prior to definitive fracture stabilization, although it is not appropriate in all cases. Fractures or luxations below the elbow and stifle joint are best managed with a soft padded bandage, with or without additional support using fiberglass casting or metal splints. Fractures above the elbow or stifle joint are very difficult to coapt; thus temporary support, which has limited efficacy and the potential for significant complications, is rarely indicated for such fractures. Advantages and disadvantages of temporary fracture support are included in Box 32.3. The most common temporary methods of fracture support are Robert Jones bandages and soft padded bandages supported with spoon splints or other coaptation materials.

Robert Jones Bandages

Robert Jones bandages and their modifications are often used in veterinary patients. These bulky, cotton-gauze wrappings are typically used before or after surgery for temporary limb splintage. The original Robert Jones bandage used commercially available, 12-inch (30.5 cm) rolled cotton that was liberally applied to the limb to a thickness of 4 to 6 inches (10–15 cm). Modified Robert Jones bandages use less cotton, but still provide compression. The thick cotton layer provides mild compression of soft tissue

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and immobilizes fractures without causing vascular compromise. Soft tissue and bone immobilization enhance patient comfort, prevent further soft tissue damage from sharp bone fragments, and minimize swelling, which enhances visualization and palpation of anatomic landmarks during surgery. In addition, Robert Jones bandages help eliminate dead space after surgery.

NOTE Robert Jones bandages extend from the ends of the toes (with only toenails and pads visible) to the midfemur or midhumerus and are only useful when applied to injuries below the stifle or elbow joint.

BOX 32.3 Advantages and Disadvantages of Temporary Support

Advantages

- Minimizes fracture movement, which decreases the risk of additional comminution
- Prevents wearing of fracture edges
- Reduces damage to adjacent soft tissue structures
- Decreases the potential for fracture contamination through skin puncture
- Decreases pain and swelling
- Aids in maintaining tissue viability

Disadvantages

- Difficult to stabilize fractures above the elbow or knee
- Additional cost
- Potential self-destruction of the bandage by the patient
- Possible vascular compromise
- Dermatitis
- Increased pain

Prepare the limb by clipping long hair from the midhumerus (midfemur) to the toes and treat any open wounds. Apply adhesive tape stirrups to cranial and caudal surfaces of the foot from carpus (tarsus) to 6 inches (15 cm) beyond the toes (Fig. 32.1A). Wrap 3 to 6 inches (7.5–15 cm) of cotton padding (from a 12-inch (30.5 cm) roll or cast padding) around the limb from the end of the toes to the midhumerus (midfemur) (Fig. 32.1B). Ensure that the nails of the third and fourth digits are visible so that limb swelling can be detected. Then wrap elastic gauze firmly over the cotton to compress it (Fig. 32.1C). Apply at least two to three layers of gauze to achieve smooth, even tension; sufficient compression will cause the bandage to sound like a ripe watermelon when tapped with a finger. Invert the tape stirrups and stick them to the outer layer of gauze. Then apply elastic tape (e.g., Elasticon, Vetrap) to the outer surface of the bandage (Fig. 32.1D).

Spoon Splints

Spoon splints (metasplints) are used to provide support to injuries of the distal radius and ulna, carpus or tarsus, metacarpus or metatarsus, and phalanges. Metasplints are used for ancillary support of internal fixation devices or as a primary means of fixation when the patient fracture assessment score (see p. 985) indicates minimal stress and rapid healing. Spoon splints are commercially available in aluminum or plastic in a variety of lengths and sizes. *Clip long hair and treat open wounds (see p. 183) before covering them with a sterile dressing. Apply adhesive tape stirrups from the carpus (tarsus) to toes, leaving the ends extending 6 inches (15 cm) beyond the toes. Firmly apply cast padding around the limb in a spiral fashion with a 50% overlap (Fig. 32.2A). Begin the padding at the ends of the toes and extend it proximally 1 inch (2.5 cm) beyond the proximal aspect of the*

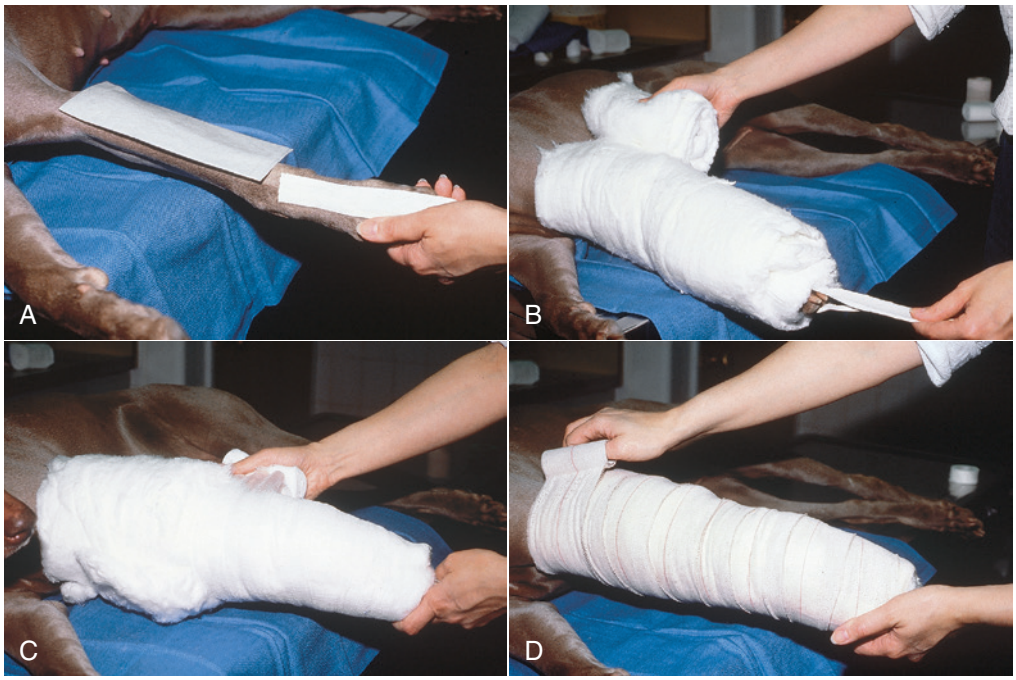


FIG. 32.1 (A) When applying a Robert Jones bandage, place adhesive tape stirrups on the cranial and caudal surfaces of the foot from the carpus (tarsus) to 6 inches (15 cm) beyond the toes. Cover any wounds with a nonadhesive pad. (B) Then wrap 3 to 6 inches (7.5–15 cm) of cotton padding around the limb. (C) After applying tape stirrups and cotton padding on the limb, wrap elastic gauze firmly over the cotton to compress it. (D) Then apply elastic tape to the outer surface of the bandage.

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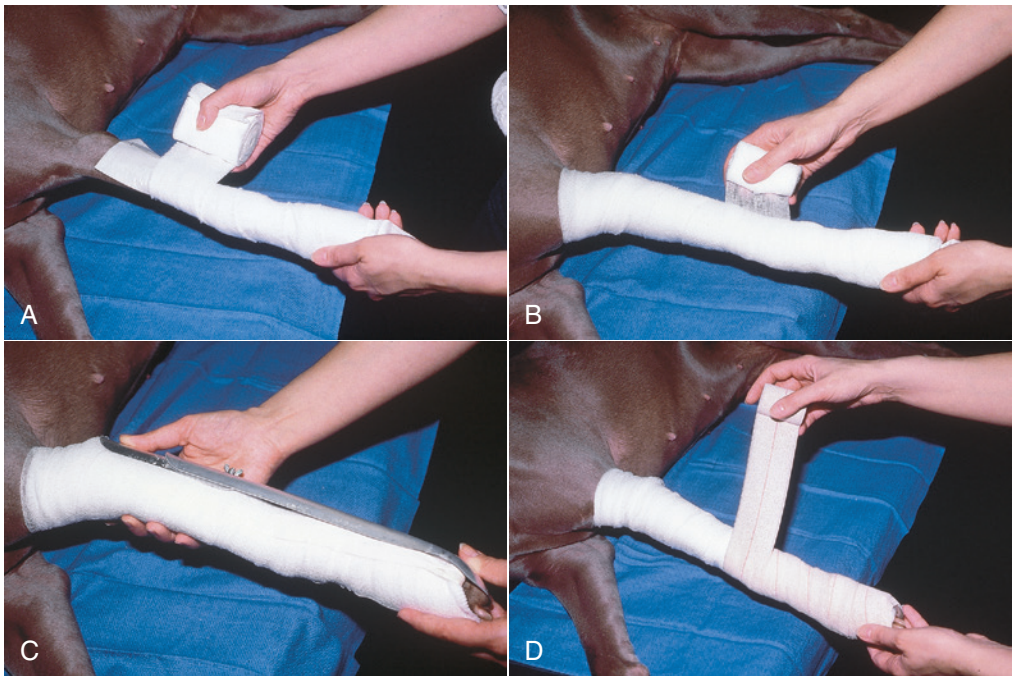


FIG. 32.2 (A) When placing a spoon splint on a limb, firmly apply cast padding around the limb in a spiral fashion with a 50% overlap. (B) Wrap elastic gauze firmly over the cast padding. (C) Place the padded limb in an appropriate-sized splint. (D) Secure the splint to the limb with Vetrap or elastic adhesive tape.

splint. Apply just enough cotton to prevent skin abrasions and pressure sores, but do not make the bandage so bulky that it will be awkward for the patient. Cover bony prominences with excess padding. Wrap elastic gauze over the cotton to mildly compress it (Fig. 32.2B). Place the padded limb in an appropriate-sized splint and secure it to the limb with Vetrap or elastic adhesive tape (Fig. 32.2C–D). Invert and stick the stirrups to the final wrapping.

Soft Padded Bandages

Soft padded bandages are used when excessive compression of the tissue is not desired. *Apply the bandage initially as described earlier for metasplints. Instead of using a preformed metasplint, apply the bandage without a splint, or insert a lateral splint made of fiberglass casting material or heat-molded plastic as reinforcement.* Reinforcing a padded bandage applied to the rear limb with a lateral splint also prevents the bandage from folding as the limb is used.

OPERATIVE PLANNING

The goal of fracture repair is to achieve complete bone healing with optimum function in as little time as possible. Modern fracture stabilization techniques and implants provide outstanding stability enabling a more rapid return to activity. The value of a rapid return to activity includes decreased muscle atrophy, decreased weight gain, maintenance of joint range of motion, minimization of decubital injuries, improved patient attitudes, and higher client satisfaction.

The process of selecting an appropriate method of fracture repair includes the following steps: (1) patient assessment, (2) fracture assessment, (3) fracture assessment scoring, and (4) selection of the fracture repair method. Other factors that may influence the selection of the method of fracture repair include financial and client compliance issues; however, repair selection should never be compromised solely based on cost to the point that there is an increased risk of failure or significant complications.

PATIENT ASSESSMENT

Assessment of the patient includes the initial triage, assessment of all medical diseases, and evaluation of specific biological factors that may affect bone healing and the fracture assessment score as described below.

FRACTURE ASSESSMENT

Assessment of the fracture is best achieved by a systematic, repeatable description of the fracture. Fractures are described to allow accurate communication with owners and colleagues and to aid in planning the appropriate treatment. Both the animal's limb and radiographs are evaluated. An accurate description of the fracture includes (1) bone affected, (2) location of the fracture in the bone, (3) whether the fracture is open or closed to the environment, (4) degree of comminution, and (5) displacement of the fracture (Box 32.4). Determination of open versus closed requires examination of the limb in addition to evaluation of the radiographs. If there are bandages on the limb, they should be removed when possible to complete the assessment. Moderate or severe soft tissue damage and bleeding as evidenced by the degree of limb swelling and bruising should also be noted as they might affect bone and soft tissue healing.

The degree of comminution is described using the terms *simple* (two-piece fracture), *minimally*, *moderately,* or *severely comminuted*. Comminuted fractures have multiple fracture lines. Comminuted fractures range from three-piece fractures with a

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BOX 32.4 Description of Fractures

1. Bone affected
   - List specific bone (list forelimb or rear limb, if necessary)
   - Note side (left or right)
2. Location of fracture in the bone
   - Articular: fracture extends into the joint
   - Metaphyseal: bone between the epiphysis and diaphysis. Contains the growth plate (physis). Further classify as:
     - *Salter-Harris type I:* fracture is through the physis
     - *Salter-Harris type II:* fracture is through the physis and a portion of the metaphysis
     - *Salter-Harris type III:* fracture is through the physis and epiphysis; generally these are articular fractures
     - *Salter-Harris type IV:* fracture is through the epiphysis, across the physis, and through the metaphysis; these are also articular fractures
     - *Salter-Harris type V:* crushing injuries of the physis that are not visible radiographically but become evident several weeks after the injury when physeal function ceases
     - *Salter-Harris type VI:* used to describe the partial physeal closures resulting from damage to a portion of the physis and causing asymmetric physeal closure
   - Epiphyseal: the end of the long bone
   - Diaphyseal: the narrow shaft of the bone
     - Further classify fractures as proximal, central, or distal third
3. Open or closed

   Further classify open fractures as:
   - *Grade I:* there is a small puncture hole located in the skin in the proximity of the fracture that was caused by the bone penetrating to the outside. The bone may or may not be visible in the wound.
   - *Grade II:* has a variably sized skin wound associated with the fracture that resulted from external trauma. More damage to the soft tissue is generally associated with grade II than with grade I open fractures. Although the extent of the soft tissue damage may vary, the fracture is minimally comminuted.
   - *Grade III:* there is severe bone fragmentation associated with extensive soft tissue injury, with or without skin loss. These fractures are usually high-velocity comminuted fractures, such as gunshot injuries or shearing type of injuries of the distal extremities.
4. Degree of comminution
   - Describe as simple (two-piece fracture), minimal, moderate, or severe
   - Describe further as having or being:
     - *Butterfly fragments*
     - *Greenstick fracture:* incomplete fracture in which a portion of the cortex is intact, thus stabilizing the bone to some extent
     - *Avulsion fracture:* the insertion point of a tendon or ligament is fractured and distracted from the rest of the bone
     - *Transverse fracture:* fracture line is perpendicular to the long axis of the bone
     - *Oblique fracture:* lines run at an angle to the perpendicular line to the long axis of the bone
       - *Short oblique* fractures: ≤45 degrees
       - *Long oblique* fractures: >45 degrees
       - *Spiral* fractures are similar to long oblique fractures but wrap around the long axis of the bone.
5. Nondisplaced or displaced

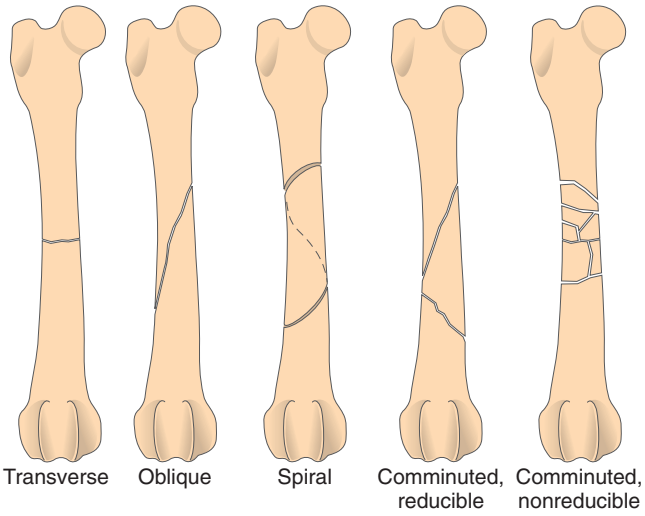


FIG. 32.3 Classification of fractures based on direction and number of fracture lines.

butterfly fragment (fragment with two oblique fracture lines resembling a butterfly's silhouette) to highly comminuted fractures with five or more pieces (Fig. 32.3). The velocity of the forces causing the fracture dictates the number of fragments and damage to surrounding soft tissues. *Low-velocity forces* result in single fractures with little energy dissipated into the soft tissue. Conversely, *high-velocity forces* result in comminuted fractures with the high energy dissipated through fracture propagation and surrounding soft tissue injury. Fractures may be *complete* or *incomplete* and may be further classified as *greenstick*, which occurs in an immature animal, *avulsion*, or *transverse* (see Box 32.4). *Oblique* fracture lines run at an angle to the perpendicular line of the long axis of the bone; they are described as *short oblique* fractures if they are 45 degrees or less or *long oblique* fractures if they are greater than 45 degrees to the perpendicular of the long axis of the bone (see Box 32.4). *Spiral* fractures are similar to long oblique fractures but wrap around the long axis of the bone (see Box 32.4). Lastly, fractures may be *nondisplaced* or *displaced*. The direction of displacement is described as the location of the distal segment relative to the proximal segment. Most fractures are proximally displaced due to muscle contracture and swelling. **Open fractures** are classified according to (1) the mechanism of puncture and (2) the severity of soft tissue injury, and are noted as a *grade I*, *grade II*, or *grade III* open fracture (see Box 32.4).

Physeal Fractures

Physeal fractures are identified according to the Salter-Harris classification scheme, which identifies the location of the fracture line (Fig. 32.4). *Salter-Harris type I* fractures run through the physis. *Salter-Harris type II* fractures run through the physis and a portion of the metaphysis. *Salter-Harris type III* fractures run through the physis and the epiphysis and are generally articular fractures. *Salter-Harris type IV* fractures are also articular fractures, running through the epiphysis, across the physis, and through the metaphysis. *Salter-Harris type V* fractures are crushing injuries of the physis that are not visible radiographically but that become evident several weeks later when physeal function ceases. An additional *Salter-Harris type VI* classification has been used to describe the partial physeal closures resulting from damage to a portion of the physis and causing asymmetric physeal closure.

NOTE An example of an appropriate fracture classification would be an "open, complete, displaced, severely comminuted, nonreducible fracture of the diaphysis of the left femur."

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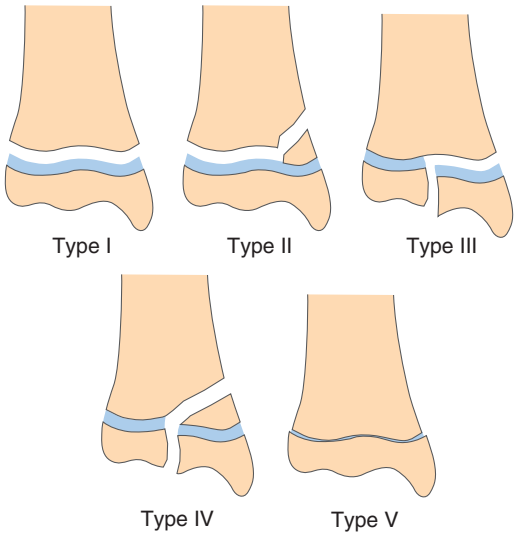


FIG. 32.4 Salter-Harris classification of physeal fractures based on the radiographic location of the fracture line.

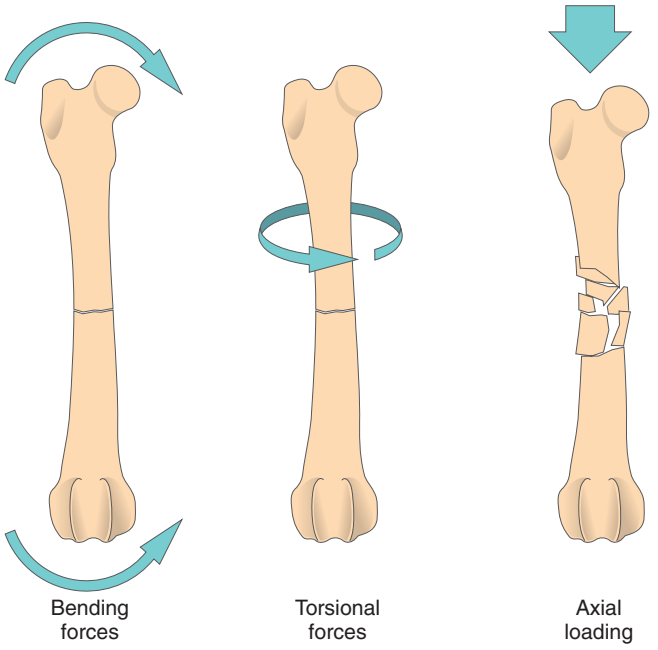


FIG. 32.5 Forces acting on fractured bones.

FRACTURE ASSESSMENT SCORING

The objectives in treating fractures, nonunions, or bone deformities are bone union and the patient's return to normal function. Using appropriate decision-making processes to choose implants and plan the procedure should produce consistent and predictable results. The surgeon must evaluate the fracture, patient, and client to identify which implants will achieve the necessary stability for the appropriate time to satisfy the objectives (i.e., develop a fracture assessment score). Additionally, the surgeon must ensure the selected fixation is able to counteract the compressive, bending, and torsional forces applied to the stabilized bone by weight bearing and adjacent muscular contraction (Fig. 32.5). It is important to make a detailed plan for the entire surgical operation, including method of fracture reduction, sequence of implant application, and possibilities for bone grafting (see p. 991). Failure to plan the procedure results in prolonged operating times, excessive soft tissue trauma, and technical errors. The ultimate outcomes of improper planning are implant failure, delayed healing, infection, and nonunion.

SELECTION OF THE FRACTURE REPAIR METHOD

After the patient has been thoroughly examined and life-threatening problems corrected, preoperative data must be analyzed to start the decision-making process. Preoperative data include patient information such as age, weight, general health, activity level, and presence of other orthopedic pathology; radiographs of the fractured and corresponding contralateral intact bones, which include the proximal and distal joints; and client information, such as their expectations and ability to perform postoperative care. These data can be summarized as a fracture assessment score that reflects the (1) mechanical factors, (2) biological factors, and (3) clinical environment in which the implants must function, and guides the types of implants chosen.

Mechanical Factors

An accurate mechanical evaluation indicates how strong the fixation must be for the patient. Mechanical factors include the number of limbs injured, patient size and activity, and ability to achieve load-sharing fixation between the bony column and the implant (Fig. 32.6). The combination of the reconstructed bone and the implants is referred to as the *composite*. Reducibility of the fracture must be determined. In general, two-piece fractures and fractures with large butterfly fragments that can be secured with cerclage wire or lag screws are considered *reducible,* allowing the reconstructed cortex to share the load of weight bearing with the implants. Fractures with multiple large fragments arranged like barrel staves or multiple small fragments that cannot be secured with implants are considered *nonreducible,* with the implants carrying the load of weight bearing until callus is formed.

Patients with multiple limb injuries are referred to as having *polytrauma* and will likely bear more weight on the repaired limb or limbs than a patient with a single limb injury. Although a moderate degree of weight bearing on the operated limb is desirable postoperatively to maintain muscle mass and range of motion, excessive loading due to lack of restraint or polytrauma will increase the risk of complication. Anticipation of excessive loads should be recognized during fracture assessment scoring.

The degree of load sharing between implants and the bony column also influences complication rates. Ideal load sharing occurs when a transverse fracture is repaired because much of the force is transmitted axially through the limb. Loading of the implant is minimized, so loosening and fatigue failure are less likely. Conversely, when loads are transmitted from bone segment to bone segment through implants rather than through the bony column (e.g., highly comminuted fractures, such as gunshot wounds that cannot be anatomically reconstructed, segmental bone resections, and limb-lengthening procedures), implant loosening and fatigue failure are more common.

The use of modern implants, particularly locking screws, dramatically increases the stiffness of the construct and decreases the incidence of implant loosening; but these improvements are not a substitution for thorough evaluation and proper implant selection and application.

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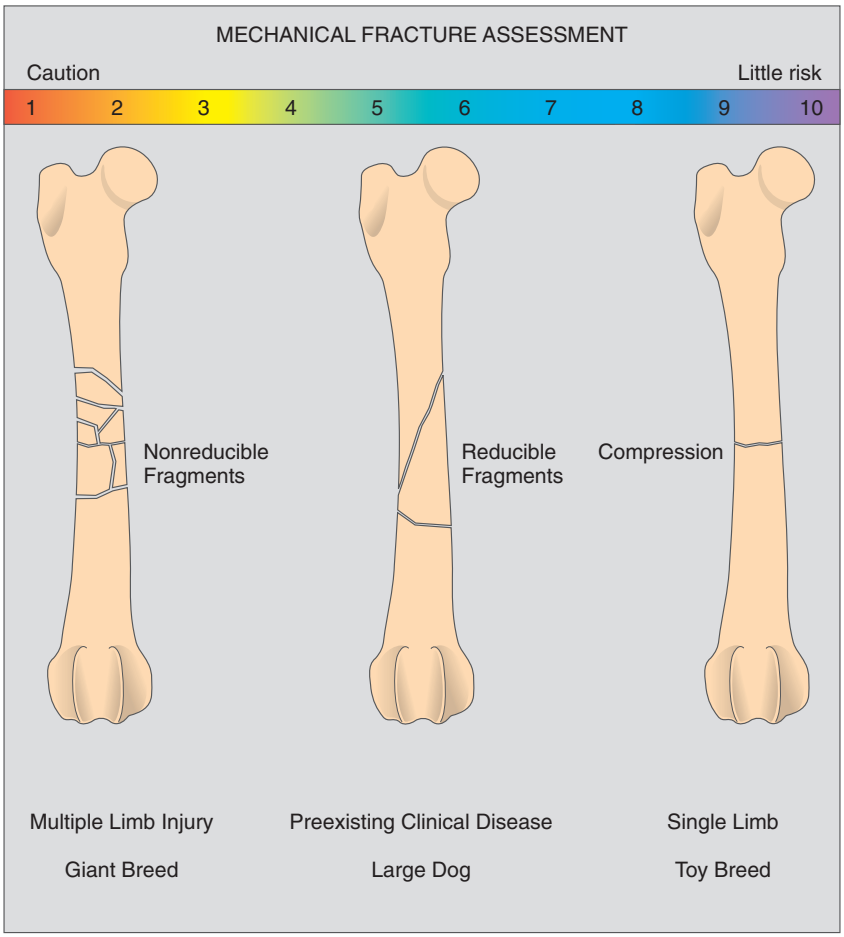


FIG. 32.6 Mechanical factors to be considered when determining patient fracture assessment. Conditions occurring on the left (e.g., buttress function and multiple limb injury) place maximum stress on an implant system and require thoughtful implant choice and application. In contrast, if conditions on the right are present, less stress is applied to the implant system, reducing the risk of complications.

Biological Factors

An accurate biological evaluation gives an indication of how fast callus may be formed, thus indirectly determining how long the implants need to function to support the bone. Many biological factors influence the rate of bone healing (Fig. 32.7). The age and general health of the patient are important. A young (<6 months of age) healthy patient is a "healing machine" and requires functional fixation devices for only a limited time. Conversely, the same fracture in an older animal will require stable fixation for a significantly longer time. Other biologic factors to consider are whether the fracture is open or closed and if it resulted from a low-energy or high-energy injury. A significant degree of soft tissue injury and bony comminution accompanies open, high-energy fractures (e.g., gunshot wounds). More time is required for bone union because the adjacent source of vascular supply is damaged. The implant-bone construct must have high initial rigidity for neovascularization and healing of fragile tissue. It must also be rigid to function as a support until biological callus is formed. With closed or low-energy fractures, less soft tissue damage is present, and bone union proceeds more rapidly.

Another factor influencing biological assessment is whether open reduction is required. If the fracture must be opened, iatrogenic vascular damage occurs. A powerful biological influence is the surgeon's skill in minimizing soft tissue envelope damage during open reduction. Obtaining desired reduction and stability with minimal soft tissue manipulation and operative time allows greater success than with longer surgeries in which reduction and stability are obtained at the expense of significant soft tissue manipulation. Preservation of the soft tissue envelope with open reduction is exceedingly important. This concept has led to a fracture management technique termed *bridging osteosynthesis*, in which there is minimal or no manipulation of the soft tissue envelope. For example, a comminuted fracture may be repaired through closed application of an external fixator or through open reduction and bridging of the fracture site with a bridging plate without manipulation of fracture fragments. Minimally invasive methods of plate application also preserve the surrounding biology.

The bone injured and injury location influence biological assessment because the soft tissue envelope surrounding various long bones differs. Distal radial or distal tibial diaphyseal fractures (i.e., locations with a sparse soft tissue envelope) have delayed

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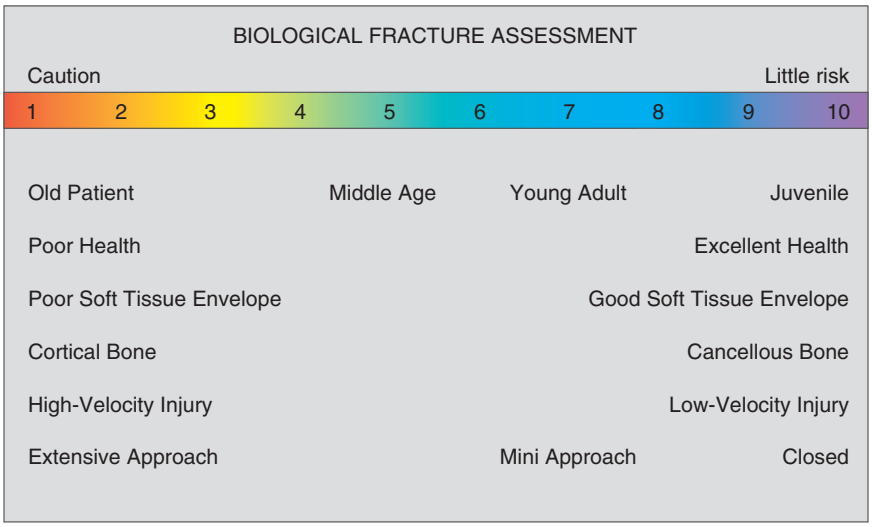


FIG. 32.7 Biological factors to be considered when determining patient fracture assessment. Patient factors listed on the left do not favor rapid healing; thus the implant system must remain in place for prolonged periods. In contrast, patient factors on the right dictate rapid healing and necessitate that the implant function for a short time.

unions or other complications more often than similar femoral or humeral fractures. Fractures occurring in cancellous metaphyseal or epiphyseal regions heal more rapidly than diaphyseal fractures because cancellous bone has a greater surface area for contact between fracture ends. Cancellous bone also has abundant osteoblasts and other biological factors that favor bone union, which is fortunate because articular fractures require precise reduction for optimal outcome. The surgical manipulation required for reduction is balanced by the inherent healing potential of the region.

Clinical Factors

Clinical factors are patient and client factors that affect healing during the postoperative period and thus influence the fracture assessment score (Fig. 32.8). Factors include the willingness and ability of clients to attend to their pet's postoperative needs, anticipated patient cooperation after surgery, and anticipated postoperative limb function. Unwilling clients or those unable to commit the time needed to care for stabilization systems requiring moderate or intensive postoperative maintenance (e.g., external coaptation, external skeletal fixations) should not be given this task. This is particularly valid if the biological assessment dictates an extended time to bone union. Bone plates and screws would be more appropriate in these cases.

Patient cooperation is an important clinical factor after surgery. Very active, uncontrollable patients are not good candidates for external stabilization systems because high activity levels increase the likelihood of complications with these systems. Hyperactive patients are not good candidates for external coaptation because casts or splints are difficult to maintain without shifting or sliding in them. Likewise, external skeletal fixators may be poor choices because these patients may continuously bump the external bar against objects, causing premature transfixation pin loosening.

Anticipated postoperative limb function must also be considered. Rapid return to normal limb function is a goal of fracture management. Therefore patient comfort during healing must be considered when selecting implants, including the patient's ability to cope with discomfort (i.e., a stoic animal vs. one that is less pain tolerant) and estimated time to bone union. When early bone union is not anticipated (>6 weeks), patient comfort that facilitates limb use and allows physical rehabilitation to be performed after surgery is essential. Implant systems vary in degree of comfort, depending on the involved bone and individual patient tolerance. As a general rule, bone plates provide the greatest level of postoperative comfort.

INTERPRETATION OF THE FRACTURE ASSESSMENT SCORE

Fracture assessment scores are assigned on a scale of 1 to 10 but are generally grouped as high (8–10), moderate (4–7), and low (1–3) (see Figs. 32.6 to 32.8). Fractures with high scores generally heal successfully with few complications, whereas fractures at the lower end of the scale potentially do not heal as well and have more complications. *Mechanical assessment* estimates the strength of implant necessary. *Biological assessment* estimates the length of time implants must be functional (i.e., time to bone union). If the fracture assessment score falls at the lower end of the scale, the implant will likely carry most, if not all, of the physiologic loads immediately after surgery. The implant must assume this function until bony callus forms. This period will be prolonged for patients with low fracture assessment scores because poor biological factors prolong bony callus formation. Higher fracture assessment scores mean less stress on the fixation system and less time required for bone healing. When the fracture assessment score falls at the upper end of the scale, implants share the physiologic loads with the bone immediately after surgery, and bone union occurs relatively rapidly.

Low Scores (1 to 3)

Generally, these are nonreducible fractures in older animals in which healing will be affected by other extenuating circumstances. Implants must bridge these fractures and therefore must have sufficient strength to prevent permanent bending or breakage for more than 6 weeks. Suggested implants (or combinations of implants) with sufficient strength and stiffness to function at

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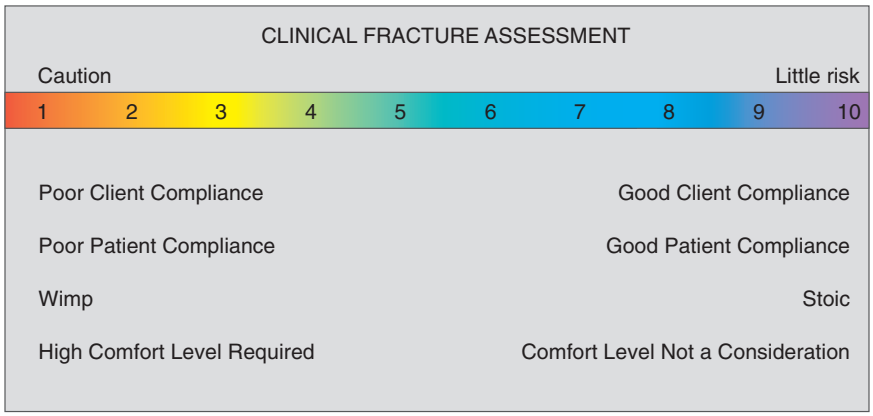


FIG. 32.8 Clinical factors to be considered when determining patient fracture assessment. Clinical factors on the left necessitate a comfortable implant system that requires little postoperative maintenance, whereas any implant system (regardless of postoperative maintenance) is appropriate with clinical factors on the right.

the lower end of the fracture assessment scale are lengthening bone plates (see p. 1022), locking bone plates, bone plate–intramedullary (IM) pin combinations ("plate-rod") (see p. 1022), interlocking nails (see p. 1011), or type III external fixators (see p. 998). These patients are not candidates for casts, IM pin, and cerclage wire fixation. Bone grafting should be considered in patients with low fracture assessment scores.

Moderate Scores (4 to 7)

Overlapping biological and mechanical factors affect healing and implant selection when the fracture assessment score moves toward midscale. For example, in an older dog with a transverse fracture, the implant and bone share the load after surgery, and the implant will be subjected to less stress, but healing may be delayed. Alternatively, in an immature dog with a nonreducible fracture, the biological assessment may indicate early callus formation, despite the implant being subjected to high initial loads as it functions to bridge the fracture. In both situations, less implant strength and endurance are required than in patients with low assessment scores because of either immediate load sharing or early callus formation. Suggested implants include bone plates, type II external skeletal fixators, IM pin–external skeletal fixator combinations, and interlocking nails. Bone grafting may be considered in patients with moderate fracture assessment scores.

High Scores (8 to 10)

When the fracture assessment score is high, mechanical assessment indicates minimal implant stress because of load sharing, and biological assessment indicates enhanced healing potential. Immediate load sharing between the bone-implant construct and rapid bone union are expected. Suggested implants include type I external skeletal fixators, IM pin–cerclage wires, and external coaptation. Bone grafting is rarely indicated in patients with high fracture assessment scores.

FRACTURE REDUCTION

Reduction is defined as the process of either reconstructing fractured bone fragments to their normal anatomic configuration or restoring normal limb alignment by reestablishing normal limb length and joint alignment while maintaining spatial orientation of the limb (Fig. 32.9). Historically surgeons have attempted to completely reconstruct the fractured bone; however, meticulous reconstruction is not always necessary or desirable. Accurate reconstruction is critical in articular fractures to limit the progression of arthritis. In nonarticular fractures, reconstruction of the fractured bone is only necessary as a means of providing load sharing with the implants in the construction of the construct. Accurate reconstruction does not necessarily accelerate bone healing, as manipulation of the fragments to achieve accurate

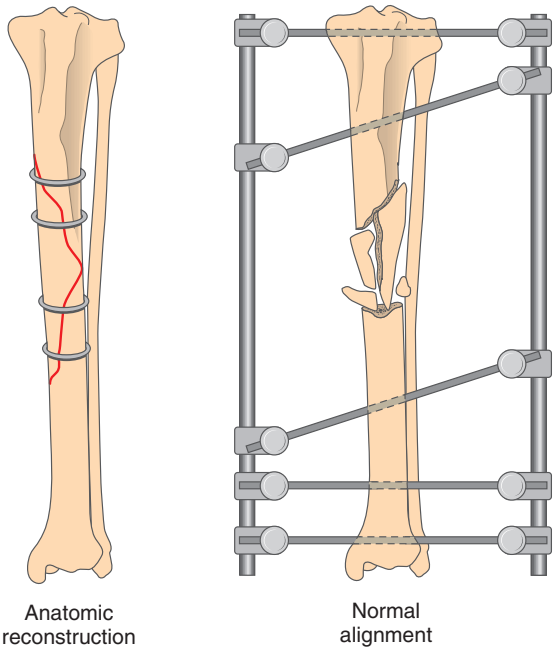


FIG. 32.9 Anatomic reconstruction of cortex is achieved by replacing all fracture fragments and holding them securely. Normal alignment of the limb is achieved by aligning adjacent joints, restoring normal bone length, and ensuring no rotation of the distal bone segment.

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BOX 32.5 Indications for Open or Closed Reduction

Open Reduction

- Articular fractures
- Simple fractures that can be anatomically reconstructed
- Comminuted nonreducible diaphyseal fractures of long bones—"open, but do not touch"

Closed Reduction

- Greenstick and/or nondisplaced fractures of long bones below the elbow and stifle
- Comminuted nonreducible diaphyseal fractures of long bones treated with external fixators

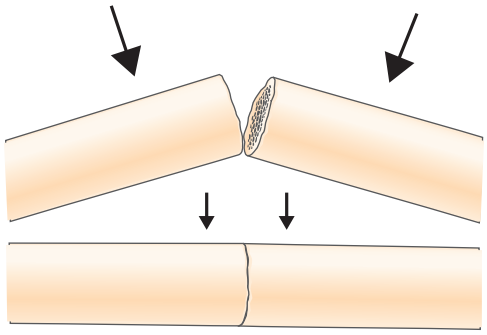


FIG. 32.10 To reduce a transverse fracture, lift the bone segments from surrounding soft tissue until the fracture surfaces are apposed. While maintaining contact, slowly replace the segments into a normal position.

reconstruction may devitalize bone segments and disrupt the healing process.

Techniques used to reduce fractures or align limbs must overcome the physiologic processes of muscle contraction and fracture fragment overriding. The initial decision when planning fracture treatment is to determine whether to use closed or open reduction (Box 32.5). *Closed reduction* refers to reducing fractures or aligning limbs without surgically exposing fractured bones. Closed reduction enhances the biological environment by (1) preserving soft tissue and blood supply, which speeds healing; (2) decreasing risk of infection; and (3) potentially reducing operating time. The main disadvantage of closed reduction is the difficulty of gaining accurate reconstruction of reducible fractures. *Open reduction* refers to using a surgical approach to expose fractured bone segments and fragments so that they can be anatomically reconstructed and held in position with implants. Open reduction is further classified as *limited open reduction* in which a small exposure is made to lever a transverse fracture into position or secure an oblique fracture with lag screws or cerclage before placing an external fixator or interlocking nail, or "*open but do not touch*" *reduction* in which a lengthy exposure is made for realigning the bone and placing a plate but the fracture fragments and hematoma are not manipulated. Advantages of open reduction include (1) visualization and direct contact with bone fragments facilitates anatomic fracture reconstruction; (2) direct placement of implants (e.g., cerclage wire, lag screws, and plates) is possible; (3) bone reconstruction allows bone and implants to share loads, which results in stronger fracture fixation (i.e., improving the mechanical environment); and (4) cancellous bone grafts can be used to enhance bone healing (see p. 991). Disadvantages of open reduction include increased surgical trauma to soft tissue and blood supply, diminishing the biological environment, and greater opportunity to introduce bacterial contamination. The advantages and disadvantages of each method must be considered before selecting the reduction method.

CLOSED REDUCTION

Fractures that are appropriately treated with closed reduction include incomplete or nondisplaced fractures of bones distal to the elbow and stifle stabilized with casts or external fixators (see pp. 995 and 998) or severely comminuted radial and tibial fractures treated with external fixators (see Box 32.5). Goals for closed reduction of both nondisplaced and nonreducible fractures are to achieve normal limb alignment. Attention must focus on eliminating rotational and angular deformity of distal segments. True lateral and craniocaudal radiographic projections of the joint proximal to the fractured bone allow the surgeon to determine if joint surfaces above and below the fractured bone are parallel to each other and in correct rotational alignment.

OPEN REDUCTION

Fractures that can be anatomically reconstructed (e.g., most single displaced fractures, those with large fragments and long oblique fracture lines classified as reducible fractures) or those that are displaced and involve joint surfaces are appropriately managed with open reduction. Bone columns and articular surfaces are restored and stabilized. Surgical approaches for exposing bones for fracture repair are found in subsequent chapters pertaining to individual bones. General principles of surgical approaches are to (1) follow normal separations between muscles, (2) obtain adequate exposure of fractured bones, (3) handle soft tissue gently and preserve soft tissue attachments to bone fragments, and (4) prevent trauma to major nerves and vessels.

DIRECT REDUCTION

The major difficulty in achieving anatomic reduction is counteracting the muscle contraction that has caused bone segments to override. Slow, manual distraction of segments using bone-holding forceps and levers will eventually fatigue muscles and allow reduction. Transverse fractures can be reduced by applying traction, countertraction, and bending forces. The bone ends should be lifted from the incision and brought into contact (Video 32.1). Force is slowly applied to replace the bones in a normal position (Fig. 32.10). A lever can also be used to reduce transverse fractures (Fig. 32.11). In some cases, fracture reduction is aided by applying a precontoured plate to one bone segment then reducing the fracture and maintaining reduction by securing the plate to the second bone segment. This technique is particularly useful for ilial body fractures and distal radial fractures. Long oblique fractures can be hard to reduce because the fracture line configuration makes bending or levering techniques difficult, and overriding may occur even after reduction. Two self-retaining reduction forceps can be used to force distraction of segments slowly until reduction is achieved (Fig. 32.12). Rough handling of bone with

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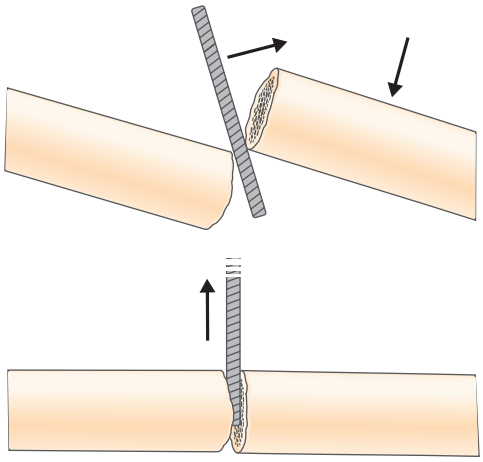


FIG. 32.11 To reduce overriding bone segments of a transverse fracture, carefully place a lever (small periosteal elevator or scalpel blade handle) between the overriding bone segments. Use the lever to apply pressure gently and help distract and reduce the bone segments.

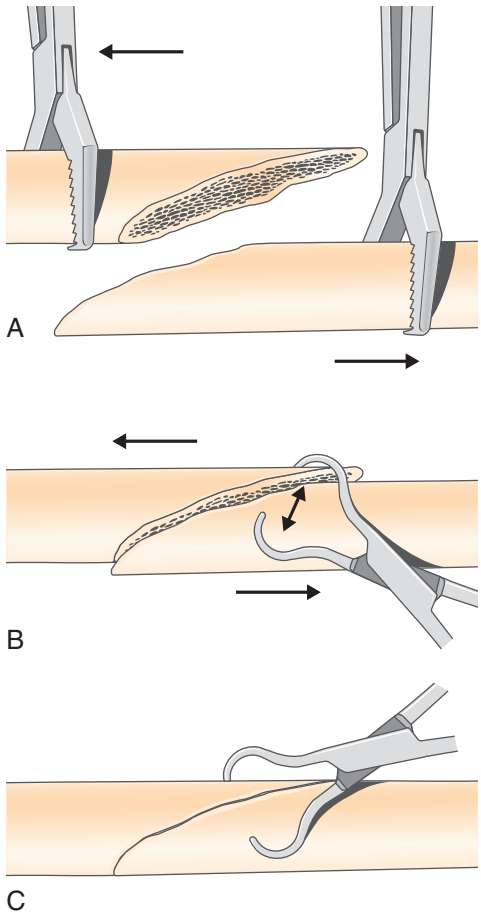


FIG. 32.12 (A) Manually return the bone segments of a long oblique fracture to close approximation with bone-holding forceps. (B) Place pointed reduction forceps at an angle to the fracture line. (C) As the pointed reduction forceps are gently closed, the bone ends are distracted. The pointed reduction forceps should be manipulated to aid reduction and secured perpendicular to the fracture line. Multiple attempts may be necessary before reduction is achieved and held with pointed reduction forceps.

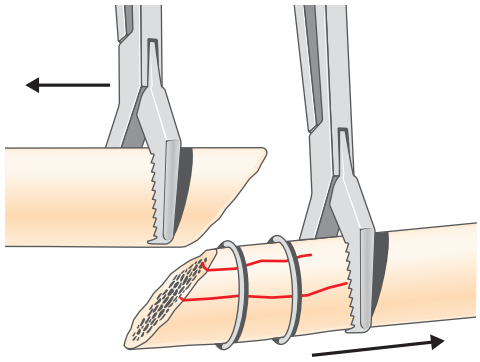


FIG. 32.13 Bone segment ends are secured with cerclage wire if fissure fractures are present.

any of these techniques can cause additional fragmentation. The bone should be inspected for fissure lines. Weak bone segments should be supported by cerclage wires before attempting reduction (Fig. 32.13). Reducible fractures with more than two pieces are first treated by securing loose fragments to one segment with lag screws or cerclage wire. The two-piece fracture is then carefully distracted and aligned for definitive fixation (Fig. 32.14).

Reduced fractures should be securely apposed for effective implant application. Transverse long bone fractures are generally held by the forces exerted by surrounding musculature. Oblique fractures must be held with reduction forceps for accurate and effective lag screw or cerclage application. However, some fractures, such as proximal femoral physeal fractures, can only be hand-held in reduction during implant application.

INDIRECT REDUCTION

Nonreducible fractures are best managed with indirect reduction to preserve the biology, and bridging fixations to provide the mechanical support. *Indirect reduction* is the process of restoring fragment and limb alignment by distracting the major bone segments. One of the most effective means of reduction in the femur, humerus, or tibia is to place a *temporary* IM pin in the medullary canal. Once the pin is in place, additional reduction in rotation or translation can be achieved. The pin is driven normograde or retrograde through the proximal bone segment. It is then centered into the distal segment and driven distally until it engages metaphyseal bone. The proximal segment is steadied with bone-holding forceps while the pin is advanced distally until appropriate distraction is achieved (Fig. 32.15). With the IM pin in place, cerclage wires, interfragmentary screws, and bone plates and screws can be placed. The IM pin may then be removed prior to surgical closure if desired.

The animal's own weight can be used to advantage to achieve indirect reduction of fractures by suspending the fractured limb from a secure ceiling bolt. The animal is draped and the surgical procedure performed with the limb suspended. Temporarily lowering the operating table causes the animal's weight to distract the fracture. This method can be used during both closed and open reduction of fractures stabilized with external fixators or open reduction of fractures stabilized with plates. When the fracture is secured in the appropriate position, the operating table is raised to remove the traction force from the limb (Fig. 32.16).

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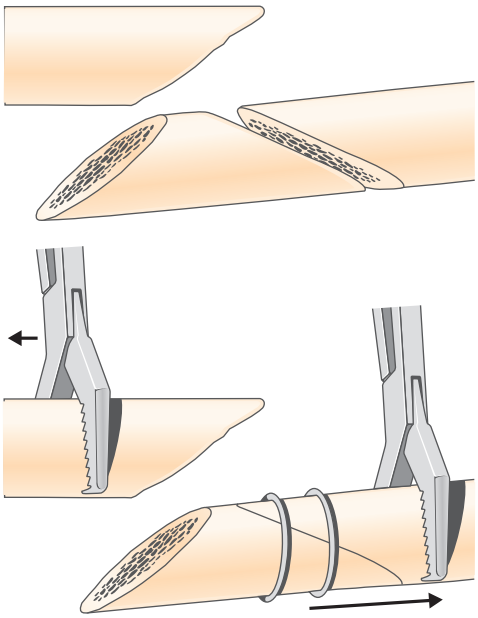


FIG. 32.14 Anatomic reconstruction of a fracture with a large butterfly fragment is achieved by first reducing the fragment and securing it to one segment of bone. This creates a two-piece fracture to be reduced and stabilized.

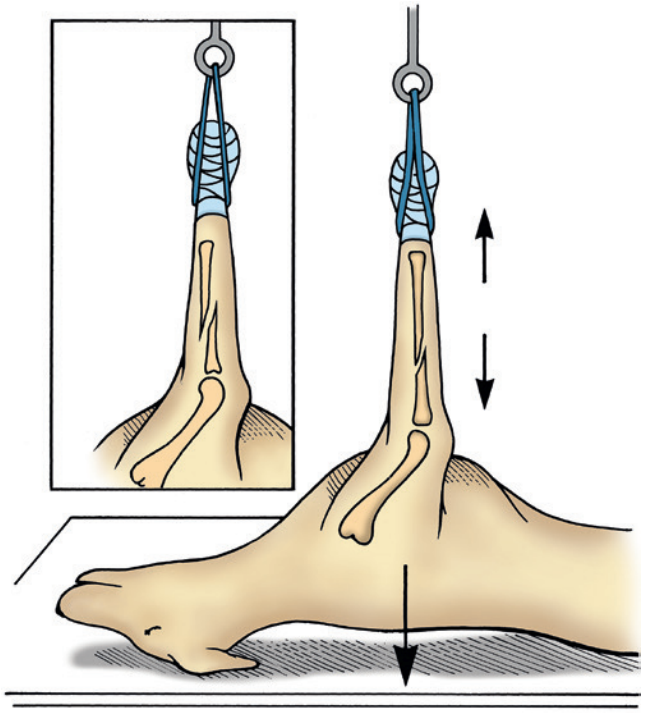


FIG. 32.16 Suspending the fractured limb from the ceiling allows the animal's weight to aid in fracture distraction.

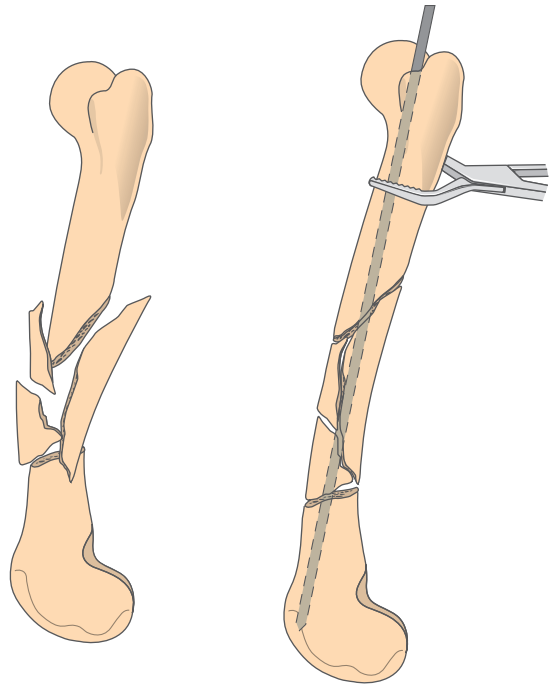


FIG. 32.15 When using an intramedullary (IM) pin to distract fractures, stabilize the proximal segment with bone-holding forceps and use an IM pin to push the distal segment of bone away from the proximal segment.

Bone Grafting

Bone grafting to enhance bone healing is standard practice in fracture management and joint arthrodesis. Options for grafting include autograft (bone transplanted from one site to another in the same animal), allograft (bone transplanted from one animal to another of the same species), biomaterials (demineralized bone matrix [DBM], collagen), synthetic bone substitutes (tricalcium phosphate ceramics, bioglass, and polymers) and composites of osteogenic cells, osteoinductive growth factors, and synthetic osteoconductive matrix.

Bone grafts and bone substitutes have specific characteristics applicable to fracture repair and bone healing and should be selected based on those properties. *Osteogenesis* is the ability of cells to survive transplantation and serve as a source of osteoblasts. *Osteoinduction* is the ability of the material to induce the migration and differentiation of mesenchymal stem cells (MSCs) into osteoblasts via the presence of the transforming growth factor-β (TGF-β) superfamily. *Osteoconduction* is the ability of the material to provide a scaffold for host bone invasion, which determines the speed of *osteointegration,* the surface bonding between graft and host bone. The structural properties (ability of the material to provide mechanical support to the fracture) must be considered. Advantages and disadvantages of each material should also be considered during the selection process (Table 32.5).

CANCELLOUS BONE AUTOGRAFTS

Cancellous bone autografts are considered the gold standard against which all graft materials are compared, as they provide optimal osteogenic, osteoinductive, and osteoconductive properties and are not immunogenic. Autogenous cancellous bone

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TABLE 32.5 Properties of Materials Used for Bone Grafting

Material	Osteogenesis	Osteoinduction	Osteoconduction	Strength
Cancellous autograft	+++	+++	+++	–
Cortical autograft	++	++	++	+++
Cancellous allograft	–	+	++	–
Cortical allograft	–	+	+	+++
Demineralized bone matrix	–	+	+	–
Collagen	–	–	++	–
Bone morphogenic protein	–	+	–	–
Bone marrow aspirate	++	+	–	–
Mesenchymal stem cells	+	–	–	–
Platelet-rich plasma	–	+	–	–
Ceramics	–	–	+	+
Bioglass	–	–	+	–
Polymers	–	–	+	–

grafts are recommended when rapid bone formation is desired, to assist healing when optimal healing is not anticipated (e.g., cortical defects after fracture repair, adult and elderly animals with fractures, delayed unions, nonunions, osteotomies, joint arthrodesis, and cystic defects), or to promote bone formation in infected fractures. Disadvantages include the additional surgical time required to harvest the graft, the potential for morbidity associated with the donor site, and the limited availability of cancellous bone in small or elderly patients.

Cancellous bone may be harvested from any long bone metaphysis; however, the proximal humerus, proximal tibia, distal femur, and ilial wing are most often used because they are accessible and contain large amounts of cancellous bone. The graft is usually harvested after fracture stabilization; however, it may be harvested before the primary orthopedic procedure if the donor site could be contaminated with either tumor cells or bacteria from the recipient site. Alternately, a separate surgical team and instrumentation may be used to harvest the graft. The graft site is selected because of accessibility when the animal is positioned for fracture repair.

Proximal Humerus

Prepare the graft site for aseptic surgery. Perform a craniolateral approach to the proximal humerus by incising through skin and subcutaneous tissue. Retract the acromial head of the deltoid muscle caudally and expose the flat aspect of the craniolateral metaphysis, just distal to the greater tubercle. Make a round hole in the bone cortex, using an IM pin or drill bit (Fig. 32.17A). Ensure that the hole's location is distal to the physis in the immature animal. A round hole is made through the cortex to minimize formation of a stress riser, which could contribute to fracture through the cortical defect. *After penetrating bone cortex, insert a bone curette and harvest cancellous bone (Fig. 32.17B). Place the cancellous bone directly into the recipient bed or store it in a blood-soaked sponge or stainless steel cup (Fig. 32.17C–D). Add blood to the graft if it is placed in a cup to keep it moist.* The blood will clot and form a moldable composite with the graft, which facilitates handling. *Secure stored grafts on the instrument table to prevent inadvertent disposal. Flush the fracture site, and loosely pack all defects and fracture lines with graft material. Try to fill the defect as much as possible for optimal osteogenesis. Close adjacent tissue around the grafts to hold them in position. Close subcutaneous tissue and skin of the graft site routinely.*

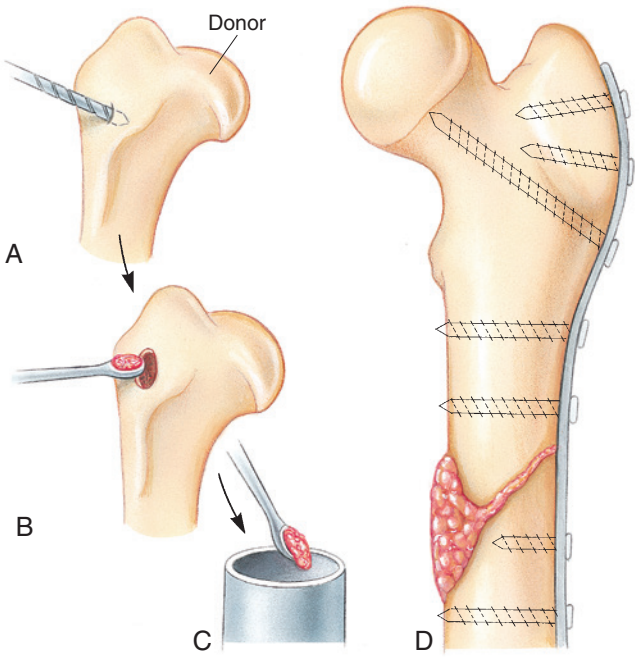


FIG. 32.17 (A) To obtain cancellous bone from the humerus, make a round hole through the near cortex. (B) Use a bone curette to harvest cancellous bone. (C) Place the bone into a stainless steel cup with whole blood for short-term storage. (D) Loosely pack the cancellous bone in the fracture gap or along fracture lines.

Proximal Tibia

Make a craniomedial skin incision over the medial surface of the proximal tibia. After incising subcutaneous tissue, harvest cancellous bone as described previously.

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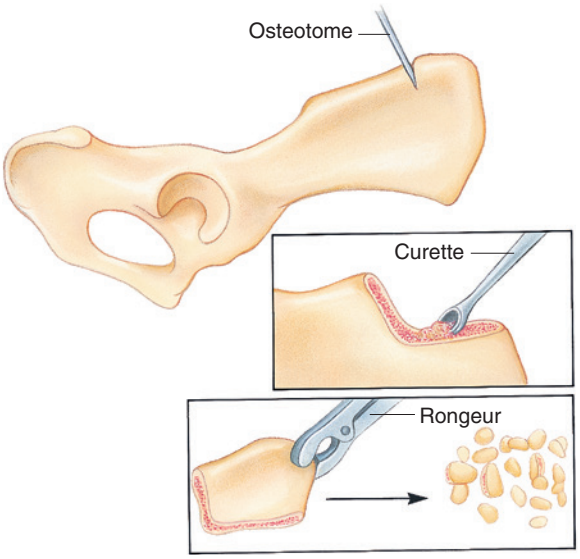


FIG. 32.18 To obtain cancellous bone from the wing of the ilium, use an osteotome to remove a wedge from the dorsocranial ilial wing. Use a bone curette to harvest cancellous bone. Use rongeurs to fragment the osteotomized wedge of corticocancellous bone.

Distal Femur

Make a lateral skin incision over the lateral aspect of the femoral condyle. Bluntly dissect the soft tissues over the lateral femoral condyle and maintain reduction with a Gelpi retractor. Harvest cancellous bone as described previously.

Ilial Wing

Make a skin incision over the craniodorsal iliac spine. Incise subcutaneous tissue and expose the dorsal surface of the ilial wing. Elevate gluteal musculature from the lateral surface and harvest cancellous bone as described previously. Alternately, obtain a corticocancellous graft by using an osteotome to remove a cortical wedge from the ilial wing. Morselize the wedge with rongeurs (Fig. 32.18) and place it into the recipient site. Revascularization of cancellous bone autografts begins as early as 2 days after grafting and is usually completed within 2 weeks (Fig. 32.19A–B). Transplanted osteogenic cells or undifferentiated mesenchymal cells become active osteoblasts, secreting osteoid on transplanted trabecular bone (Fig. 32.19C). This osteoid is mineralized and forms new host bone in fracture sites (Fig. 32.19D). This new bone also incorporates the graft into host bone. Eventually the necrotic cores of trabecular bone are resorbed by osteoclasts, and grafts are totally replaced by host bone. Trabecular new bone is remodeled into cortical bone in response to the mechanical environment (Fig. 32.19E). This healing response can be monitored radiographically by observing filling of the defect with cancellous bone, followed by cortical reconstruction. The donor site is initially filled with hematoma, which is later replaced with fibrous connective tissue. Osteoblasts migrate to the area and deposit osteoid. Mineralization occurs, and new trabecular bone is formed within defects. This process takes approximately 12 weeks; additional cancellous bone should not be harvested from the same area before this time.

Few complications are associated with autogenous cancellous bone grafting techniques in dogs and cats. Donor site pain is seldom clinically evident. Seroma formation or wound dehiscence may occur at donor sites. Infection or seeding of tumor to donor sites occurs rarely and can be prevented by proper sequencing of bone graft harvesting. Fractures through the donor site have been reported infrequently. Complications at the recipient site (e.g., failure of grafts to stimulate bone formation) are difficult to recognize.

CORTICAL BONE AUTOGRAFTS

Cortical autografts are harvested from areas where cortical bone can be removed without adversely affecting function (i.e., ribs, ilial wing, distal ulna, fibula). The most common use of a cortical autograft is transplantation of a rib to form a segmental strut for mandibular fractures. Cortical autograft harvest is done during fracture repair, and the graft is incorporated into the fracture site as a *segmental graft* (placed between fracture segments) or as a *sliding onlay graft* (placed over the fracture site). Cortical autografts are usually held in place with the same implant used to stabilize the fracture.

CANCELLOUS BONE ALLOGRAFTS

Cancellous bone allografts are commercially available as frozen chips or as chips mixed with demineralized bone powder. Advantages to using prepared allografts are reduced operating time, availability of graft, and elimination of morbidity at a donor site. Disadvantages are the cost of the grafts and the lack of osteogenic properties in the cancellous bone chips. Allogeneic bone graft can be mixed to extend cancellous autograft to fill large defects or can be mixed with autologous marrow to increase the osteogenic properties.

The same stages of incorporation occur in cancellous allografts as in cancellous autografts. Autogenous graft is superior to allogeneic graft in promoting rapid new bone formation because of osteogenic properties, but no difference exists in long-term outcome.

CORTICAL BONE ALLOGRAFTS

Cortical bone is harvested and banked to provide a ready source of cortical alloimplants. Harvesting must be done under aseptic conditions unless the bone is sterilized after collection. Frozen cortical allografts are available commercially. With the advent of biological fracture repair techniques (see p. 1023) that incorporate fragments in comminuted fractures, cortical bone allografts are now used more often for limb-sparing procedures than for fracture repair. Plate and screw fixation is necessary to ensure stability of host-graft interfaces for prolonged periods while fractures heal and grafts remodel.

Complications associated with cortical allografts include infection, graft rejection, failure of fracture repair, and graft fracture. Graft infection usually results from graft or fracture site contamination, coupled with instability. This results in a large, sequestered piece of foreign material that must be debrided when fracture stabilization is performed. Cancellous bone autografts may be used to fill resultant fracture gaps. Signs of graft rejection (e.g., failure of graft and host bone to unite, graft resorption without replacement) are rarely noted clinically. Plate fracture may be observed when reduction and fixation of host-graft interfaces provide inadequate reconstruction of the bone column. Grafts may also fracture after plate removal.

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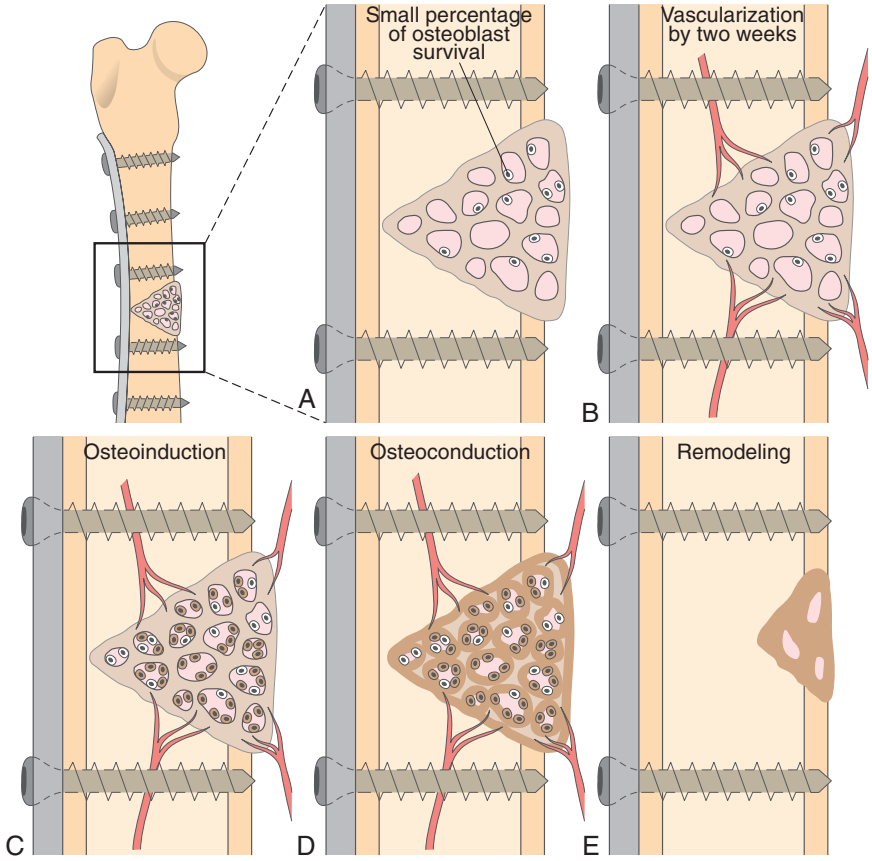


FIG. 32.19 Stages of cancellous bone incorporation into a healing fracture. (A) Graft placement. (B) Vascularization of the graft. (C) Osteoinduction. (D) Osteoconduction. (E) Remodeling.

DEMINERALIZED BONE MATRIX

DBM is obtained from processed allograft bone. Calcium in canine DBM is reduced to less than 3% from 22% to 25% in intact bone. The remaining collagen network provides osteoconductive properties, whereas the inherent bone morphogenic proteins (BMPs) liberated during demineralization provide osteoinduction. The processing also reduces the immunogenicity of the product. The efficacy of DBM may vary depending on the method of processing, origin of the material, and storage conditions. DBM is available for dogs and cats as a 50:50 combination of DBM with varying sizes of allogeneic cancellous chips. The material is either frozen or freeze dried.

Indications for DBM use are the same as those for cancellous bone autograft. The material is often used to extend the available cancellous bone autograft but may be used by itself. DBM may also be combined with carriers such as putty and with growth factors or stem cells. The advantage of DBM is its availability. The disadvantages are the lack of osteogenic properties and the possibility of immune reaction. The combination of DBM and cancellous bone allograft are effective in the management of arthrodeses, osteotomies, and fractures.

COLLAGEN

Collagen is present in extracellular bone matrix and tendon sources. Collagen is potentially immunogenic but provides favorable environment for bone regeneration. Collagen's primary use is as a delivery system or scaffold for other osteoconductive agents and for osteogenic and osteoinductive agents.

BONE MORPHOGENIC PROTEIN

The BMPs are part of the TGF-β superfamily of growth and differentiation factors. The pure form of BMP is nonimmunogenic and non–species specific. The BMPs are osteoinductive, causing undifferentiated perivascular mesenchymal cells (stem cells) to change directly into osteoprogenitor cells, and osteoprogenitor cells to differentiate into osteoblastic cells. Recombinant human (rh) gene technology has resulted in the production of BMP 2 and BMP 7 for clinical use; however, the product is costly. The BMPs are generally combined with a matrix carrier for optimal bone formation. Currently, BMPs are not marketed to veterinarians and are costly, although several studies report their use in individual cases or clinical trials.

BONE MARROW

Bone marrow is a source of MSCs. Autologous bone marrow can be aspirated and injected into a delayed union or nonunion site to stimulate bone production. However, the ability of marrow osteoprogenitor cells to stimulate bone formation varies from individual to individual and between species. The lack of an osteoconductive scaffold hinders the effectiveness of this material.

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MESENCHYMAL STEM CELLS

MSCs are cells found in periosteum, bone marrow, muscle, fat, and synovium and are capable of differentiating into osteoblasts, chondrocytes, tenocytes, adipocytes, and myoblasts. Autologous or allogenic cells can be isolated and culture expanded to provide an osteogenic material that can be delivered in a matrix carrier. See Chapter 31 for further details.

PLATELET-RICH PLASMA

Platelet-rich plasma is an autologous material produced by centrifugation of the patient's blood. Platelets release proliferative and morphogenic proteins, which provide an excellent environment for healing in tissues such as bone, tendon, and cartilage. Platelets support chemotaxis of MSCs, proliferation and differentiation to osteoblasts, and stimulate angiogenesis. Platelet-rich plasma has been combined with other natural and synthetic biomaterials for treatment of bone defects in humans with inconsistent results. See Chapter 31 for further details.

CERAMICS

Ceramics are synthetic combinations of calcium phosphates, generally hydroxyapatite, and/or tricalcium phosphate, which provide a biocompatible scaffold for bone formation. Synthetic ceramics do not possess osteogenic or osteoinductive properties and must be combined with other materials to achieve those functions. Hydroxyapatite is slower to resorb than tricalcium phosphate. Porosity, pore size, and particle size can also be manipulated to improve function and resorption rate. Dense materials offer improved mechanical strength but are slower to degrade. Injectable calcium phosphate may be used to fill voids in fractures or improve screw-holding power. The material hardens and is subsequently replaced with bone. Calcium phosphate ceramics are available in paste and block forms.

BIOGLASS

Bioglass is a synthetic graft substitute composed of calcium salts, phosphates, and silicon dioxide that bonds directly with living tissues when implanted into a surgical site. Bioactive glass has osteoconductive properties similar to ceramics but is significantly stronger mechanically. However, blocks of bioglass are too brittle to use in fracture reconstruction. Bioglass is generally used in particulate form to repair periodontal defects. It can also be used as an autologous bone graft expander.

POLYMER

Polymers are compounds formed by joining smaller, usually repeating, units linked by covalent bonds. Polymers designed as bone substitutes can be divided into natural (organic) polymers and synthetic polymers, and further categorized into degradable and nondegradable types. Polymers are used alone as osteoconductive scaffolds, in combination with osteogenic and osteoinductive materials, or as extenders for cancellous autografts and allografts.

Fracture Fixation Systems

Five major types of loads are applied to bones that need to be considered in the selection of a fracture repair method (Table 32.6). Categorization of the fracture determines if these loads will be borne by the bone itself or if the implants need to counteract these forces. For example, a tibial crest fracture results in tension forces that must be counteracted, whereas most diaphyseal fractures are under compression. Fracture repair implants have differing abilities to counteract loads. As an example, IM pins do not provide rotational stability, whereas cerclage wires placed on a long oblique fracture will contribute to rotational stability. A significant part of the fracture assessment process is determining the types of forces that will be applied to the implants and selecting those that can withstand these forces.

EXTERNAL COAPTATION

External coaptation may be used to provide patient comfort before surgery and decrease soft tissue damage. It also may be used as the primary repair in some conditions. Bandages and splints for perioperative coaptation are described on pp. 981 to 983.

CASTS

Indications and Biomechanical Principles

Full leg casts encircle the limb to stabilize a fracture. They can be used as a primary means of stabilization or as a supplement

TABLE 32.6 Mechanical Factors of Fixation Methods

Protects Against:	Bending	Rotation	Compression	Tension	Shear
Cast	Yes	Yes	No	No	Yes
Intramedullary pin	Yes	No	No	No	Yes
Cross pin	Yes	Yes	No	Yes	Yes
Cerclage wire	Yes	Yes	No	No	No
Tension band	No	No	No	Yes	No
External fixator	Yes	Yes	Yes	Yes	Yes
Interfragmentary screw	No	Yes	No	No	Yes
Interlocking nail	Yes	Yes	Yes	Yes	Yes
Bone plate	Yes	Yes	Yes	Yes	Yes

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to internal fixation devices of fractures below the elbow or stifle joint. **Casts should never be applied in any manner for fractures above the elbow or stifle joint as they provide no stabilization and actually increase instability of the fracture.** As a primary stabilizer, full leg casts are most useful in stable fractures in which the fracture assessment (see p. 985) indicates rapid bone union. The functional period for a cast is short, usually no longer than 6 weeks before cast complications dictate removal. The classic casting material is plaster of Paris, but with the development of synthetic casting materials, its use has declined. Synthetic casts made of fiberglass or polypropylene substrate impregnated with water-activated polyurethane resin have considerable advantages over plaster casts. Fiberglass bandages impregnated with polyurethane resin make the stiffest casts without being brittle when compared with other casting material. They are easy to apply, with maximal adhesiveness between layers, minimal wastage, and considerable durability. However, fiberglass bandages interfere with radiographic observation of the healing fracture because the weave pattern of the casting material is visible. Polypropylene substrates impregnated with polyurethane resin are more radiolucent, with a less distracting pattern.

Mechanically, a properly applied cast immobilizes the joints above and below the fractured bone, neutralizing bending and rotational forces on the fracture. A cast does not counteract axial forces applied to the fractured bone. The cast does not rigidly stabilize the fracture, resulting in indirect bone healing.

NOTE Full leg casts cannot be applied above the midhumerus or midfemur and therefore should only be used for fractures of the distal limb (radial and/or ulnar, tibial, and metacarpal and/or metatarsal fractures).

Application

General anesthesia is usually indicated for cast placement to allow closed fracture reduction (see p. 976). If the fractured bone ends cannot be reasonably reduced (i.e., varus-valgus and rotational alignment are restored with at least 50% contact of major fragment ends), then casting should not be performed (Video 32.2). Most radial and tibial fractures are presented in a valgus deformity because of the strong pull of the lateral musculature on the distal fragment. This deformity may be neutralized by holding the limb in a neutral or slight varus position during cast application (Box 32.6). *Clip long hair and apply tape stirrups to the distal limb. Avoid circumferential tape. Apply a single layer of cast stockinette onto the limb over the stirrups. Extend the stockinette from 2 inches (5 cm) distal to the toes to 2 inches above the estimated proximal extent of the cast. Fit the stockinette snugly against the limb. Have an assistant stretch the stockinette by pulling the ends during cast application to prevent folds or wrinkles in the material. Apply cast padding in a spiral fashion to the limb from the toes (i.e., exposing only the toenails of the middle digits) to the estimated proximal extent of the cast, overlapping the material 50%. Use sufficient cast padding to protect the limb from developing cast sores, but do not use excessive padding that would prevent the cast from resting snugly against and conforming to the limb (Fig. 32.20A).* Generally, cast padding should be only two layers thick. *Immerse the casting tape into cold water, squeeze excess water gently from the roll, and apply it to the limb, beginning at the toes. Wrap the cast material in a similar fashion as the cast padding, with 50% overlap (Fig. 32.20B). Encompass the toes, but leave the nails of the third and fourth phalanges exposed to allow detection of limb swelling. As the cast tape is applied above the elbow or stifle, use firm pressure to compress the larger muscles and conform the cast to the limb. Use two layers of cast tape with 50% overlap (i.e., four layers on cross section) in small and medium-sized dogs and three layers (six on cross section) in larger dogs (>30 kg).* Apply the cast tape quickly because it will set in 4 to 6 minutes. *Before the cast tape sets, roll the edges of the cast outward by pulling the proximal aspect of the stockinette over the end of the cast. Apply elastic adhesive or Vetrap around the cast and stick the stirrups to this layer.* An oscillating saw is required for cast removal. Sedation facilitates cast removal because the vibration and noise generated by the saw often frightens the animal (see Tables 31.2 and 31.3). *Cut the medial and lateral sides of the cast, separate the two halves, and remove it.* Frequent removal of a cast used for fracture stabilization is not desirable because fracture reduction may be lost. If skin abrasion or loosening of the cast occurs, replacement may be necessary. *If frequent removal is anticipated, plan to use a bivalve cast. Slightly increase the amount of cast padding applied, place a layer of gauze over the cast padding, wrapping from the toes proximally, and bivalve the full cast on initial application. Use the oscillating saw to cut the medial and lateral walls. Separate the two halves slightly, then tape them securely with elastic bandage. When the underlying skin needs to be treated or the cast needs modification, separate the halves by removing the elastic bandage.*

BOX 32.6 Key Concepts for Applying Casts

- Casts must span the joints above and below the fractured bone to be effective.
- Apply the cast with the leg held in slight varus and flexion.
- Excessive padding results in a loose cast after the padding compresses.
- Extend the cast distally to encompass the toes, leaving only the toenails of the middle digits exposed.

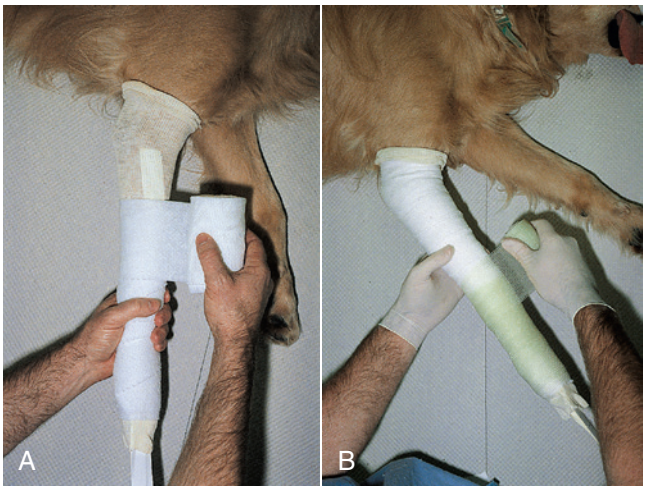


FIG. 32.20 (A) When applying a fiberglass cast, place adhesive stirrups on the limb and cover them with a stockinette. Apply cast padding firmly over the stockinette using an overlapping pattern. (B) Then place four to six layers of casting material on the limb, overlapping each layer 50%.

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Apply new cast padding, replace the halves, and secure them with elastic bandage.

NOTE A bivalve cast is used to supplement internal fixation devices, such as bone plate and screws in fractures of the carpus, tarsus, metacarpal and metatarsal bones and digits, and carpal or tarsal arthrodesis. Bivalve casts may also be applied in very young animals where minimal stability is required and rapid growth in a solid cast might lead to skin complications.

Postoperative Care

Specific written instructions outlining proper at-home cast care should be provided for the client. Casts should be evaluated 24 hours after application and weekly thereafter. Clients should be instructed to observe the toes daily for evidence of swelling (spreading the exposed toenails), excessive chewing or licking at the cast, or a foul odor. Any of these signs requires that the animal be evaluated immediately. The cast should be kept clean and free of moisture. Instruct the client to place a plastic covering over the cast when the animal goes outside. In growing animals, the cast may need to be changed every 2 weeks; in adults, the cast may last 4 to 6 weeks. Physical rehabilitation may be necessary after cast removal to restore range of motion and encourage rapid return to normal function (see Table 32.7 and Chapter 11).

NOTE Be sure to have the owners check the toes daily and call immediately if they note any swelling or other abnormalities.

TABLE 32.7 Sample Rehabilitation Protocol for Patient With a Diaphyseal Fracture (Internal Fixation or External Fixator)

Treatments/ Modalities	Day 1–7	Day 7–21	3–4 Weeks	5–7 Weeks	8–12 Weeks
Pain medications	As directed	As directed	PRN	PRN	PRN
Cryotherapy	10–15 min 3 times daily before walks or exercises; first session immediately after surgery	Use after exercise for 15 minutes	PRN	PRN	PRN
Heat therapy		Apply heat to the muscles before exercise or if dog is stiff	PRN	PRN	PRN
Massage	5 min 3 times daily before starting exercises; massage from toes toward heart	Continue twice daily	Twice daily	Twice daily	Only if desired
Passive range of motion	10 repetitions 3 times daily, all joints and affected limb—start above and below knee	Continue twice daily; if dog is using leg well, decrease	Discontinue if dog is walking well and range of motion is normal		
Laser therapy	Daily or every other day for first week	Every other day for first week then twice weekly	Twice weekly	Twice weekly	Discontinue
Walks	5 min supported leash walk 2–3 times daily	Increase each walk by 2–3 min each week	Increase by 5 min each week	Increase by 5 min each week	15–20 min walks 2–3 times daily
Neuromuscular electrical stimulation	10 min twice daily	10 min twice daily	Discontinue if dog is doing well		
Balancing		5 min twice daily	5 min twice daily	Unileg standing up to 5 min	Replace with hill walks
Cavalettis/obstacles/ sit to stand		5 min twice daily	5 min twice daily	5 min twice daily	5 min twice daily
Weaving/circles		2–3 min once daily	5 min twice daily	5 min twice daily	5 min twice daily
Hills				Zigzag low slow hills 5 minutes up and down[a]	Increase to 10 minutes twice daily
Underwater treadmill		10 min 3 times weekly after sutures are removed[b]	10 min 3 times weekly[b]	15–20 min twice weekly[b]	15–30 minutes twice weekly until released from rehab[b]
Swimming					8 wk post–internal fixation for 5–10 min if all healed[c]

[a]Wait until after 8 wk for external fixators.
[b]For external fixators, wait until device is removed at 14+ wk to start water therapy.
[c]For external fixators, wait 4 wk after device is removed.

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EXTERNAL FIXATORS

LINEAR EXTERNAL FIXATOR

Indications and Biomechanical Principles

External skeletal fixators are a versatile and affordable treatment for long bone fractures, corrective osteotomies, joint arthrodesis, and temporary joint immobilization. They are not indicated for articular fractures and are rarely used for pelvic and spinal fractures. External fixators are well suited for stabilization after closed reduction of comminuted fractures. External fixators can be adjusted during and after surgery to improve fracture alignment. The functional period for external fixators varies depending on the frame constructed, but it is related to the onset of pin loosening.

Fixation frames are classified by the number of planes occupied by the frame and the number of sides of the limb from which the fixator protrudes. Using this system, common frames are unilateral-uniplanar (type Ia), unilateral-biplanar (type Ib), bilateral-uniplanar (type II, further divided into maximal type II frames filled with full pins and minimal type II frames constructed with a minimum of two full pins), and bilateral-biplanar (type III) (Fig. 32.21).

Fixation frames can be created to meet initial mechanical stabilization needs of the fracture and can subsequently be modified or destabilized to provide optimal stabilization throughout the healing period. Factors influencing the strength and stiffness of the fixator and its ability to resist the axial loading, bending, and rotation associated with weight bearing include pins (type, size, number, location, and length), connecting bars (number and material), frame configuration (unilateral, bilateral, and biplanar), and fracture configuration (Box 32.7). Threaded pins have an interlocking hold with the bone and resist pull-out and loosening. Increasing pin diameter increases pin stiffness exponentially, but pin diameter should not exceed 25% to 30% of the bone diameter. Increasing the number of fixation pins in the proximal and distal major fragments increases the fixator's stiffness and enhances the distribution of the physiologic loads among pins. This is true for up to four pins per major proximal and distal fragment; beyond this number the increase in mechanical advantage is negligible. Locating pins both close to the fracture and at the ends of the bone increases the stiffness of the fixation and decreases the motion at the fracture site. Decreasing the distance between the bone and the fixation clamp (decreasing the working distance of the pin) by orienting the clamp so the pin-clamping bolt is closest to the skin increases the fixator's stiffness.

Increasing the number of bars and pin planes increases the strength and stiffness of the fixator. Types Ia, Ib, II, and III external fixators are successively stronger and stiffer. The

BOX 32.7 Key Concepts for Increasing the Strength and Stiffness of an External Fixator

- Predrill before inserting positive profile threaded pins.
- Increase the pin numbers (up to four pins per bone segment).
- Increase the pin size (up to 25% of bone diameter).
- Locate pins near the joints and near the fracture.
- Decrease the distance between the bone and the pin-clamp interface.
- Increase the connecting bar size or use augmentation plates.
- Increase the number and planes of connecting bars.
- Tie the intramedullary pin into the fixator frame.

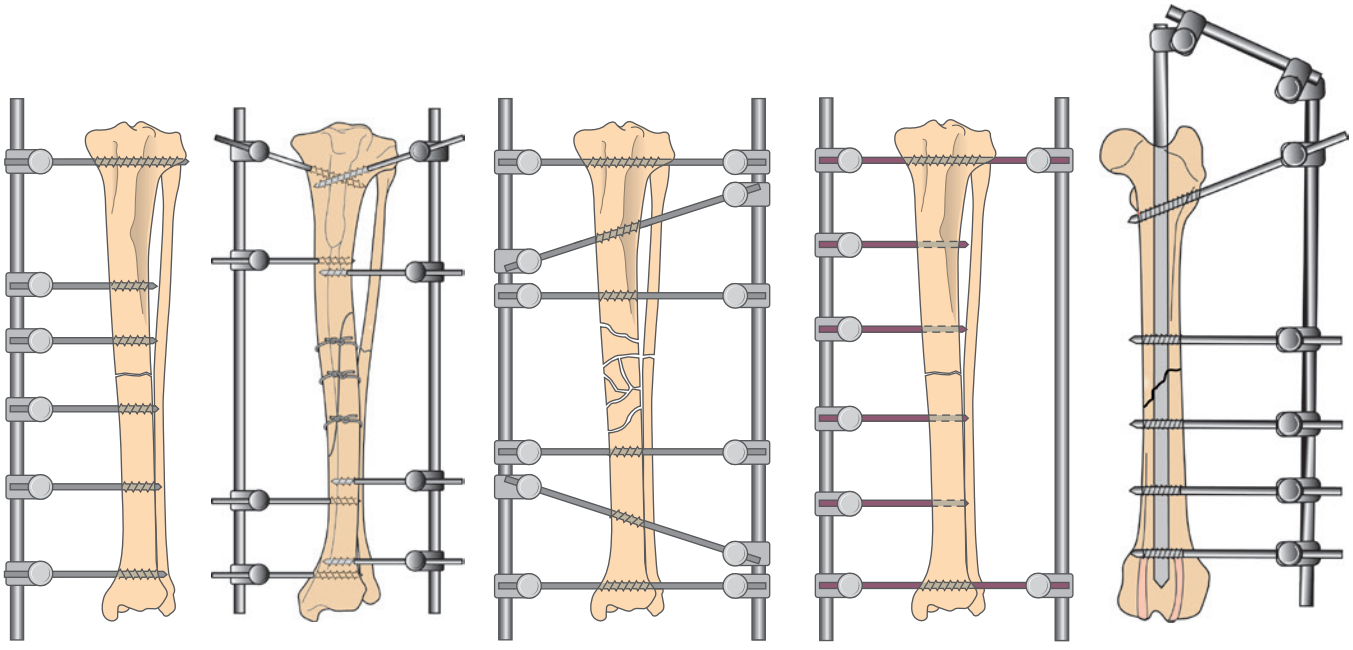


FIG. 32.21 Position of external bars and nomenclature for common external fixator frames. (Second and fourth images modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat*. St. Louis: Elsevier; 2005.)

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strength of a type Ia fixator may be increased by adding a second bar, using a larger diameter bar, or supplementing with an augmentation plate. Connecting bar material (stainless steel, aluminum, carbon fiber, acrylic, putty) affects the stiffness of the bar and subsequently the fixator. Decreasing the distance between the two central pin-gripping clamps on the bar (the working length of bar over the fracture) also increases the stiffness of a fixator. Type Ia frames are subject to bending when axial loads are applied unless the load is shared with a reduced transverse fracture. Type Ib and type III frames resist axial compression, bending, and rotation. Type II frames resist axial compression, bending in the same plane as the fixator, and rotation. Combining external skeletal fixators with IM pins is an effective way to increase the rigidity of the fracture fixation in the humerus and femur. These systems are referred to as *tie-in configurations* (see Fig. 32.21).

Equipment and Supplies

External fixation devices comprise three basic units: (1) fixation pins inserted into bone to hold major fragments, (2) external connectors to support fractured bone, and (3) linkage devices that attach the fixation pins and external connector.

Fixation Pins

Fixation pins may be categorized by implantation method (e.g., half pin or full pin) or structural design (e.g., positive profile threaded or negative profile threaded). Half pins are end-threaded and are inserted so that they penetrate both cortices, but only one skin surface (Fig. 32.22A), whereas full pins are centrally threaded and penetrate both cortices and skin surfaces (see Fig. 32.22B). Centrally threaded pins are often used as full pins with type II or type III external fixator frames (see later discussion) and are sometimes used in the distal humerus or femur in multiple frame modified type Ib constructs. The central threads engage bone, and the smooth pin ends extend beyond the skin surface. End-threaded pins are often described according to the number of cortices to be engaged by the threads (i.e., one-cortex or two-cortex end-threaded pins). One-cortex end-threaded pins have threads near the pin tip; therefore, although the pin itself penetrates both cortices, the threads engage only the far cortex. Two-cortex end-threaded pins have sufficient thread length to engage both cortices. Most end-threaded pins available are two-cortex pins. Threaded pins can be further described according to thread profile (negative or positive). Centrally threaded and end-threaded pins in which the core diameter of the threaded section is smaller than the diameter of the smooth section have a negative thread profile. If the core diameter is consistent between smooth and threaded regions, the thread profile is positive. The thread height and thread pitch are specifically designed to engage dense cortical bone (threaded cortical pins) or spongy cancellous bone (threaded cancellous pins) (Fig. 32.23). Negative threaded pins have an increased risk of failure at the juncture between the threaded portion and the solid portion unless the junction is tapered. Tapered negative threaded pins have the advantages of ease of insertion of negative threaded pins and the strength of positive threaded pins.

Acrylic pins are small negatively or positively threaded stainless steel pins that have the end of the pin opposite the threads etched to improve the grip of acrylic fixation bars. These pins are often used on mandibular fractures, small bone fractures, and avian fracture repairs.

External Connectors

External connectors are made of stainless steel, titanium alloy, carbon fiber, aluminum, acrylic, or putty (Fig. 32.24). The carbon fiber rods are radiolucent, allowing radiographic visualization of the healing fracture. The different sizes of metal rods have correspondingly sized pin grippers (linkage devices, discussed below). The external fixator and linkage devices may be fashioned from acrylic using commercial kits for acrylic-pin external fixation or from supplies designed to allow "homemade" acrylic splints to be applied (Fig. 32.25). Acrylic-pin external fixation systems contain threaded fixation pins, prepackaged acrylic, and sterilized acrylic column-molding tubes. The application kit also contains reusable temporary alignment clamps. Homemade splints use individually purchased materials for construction of frames. The acrylic in homemade splints is polymethylmethacrylate or epoxy.

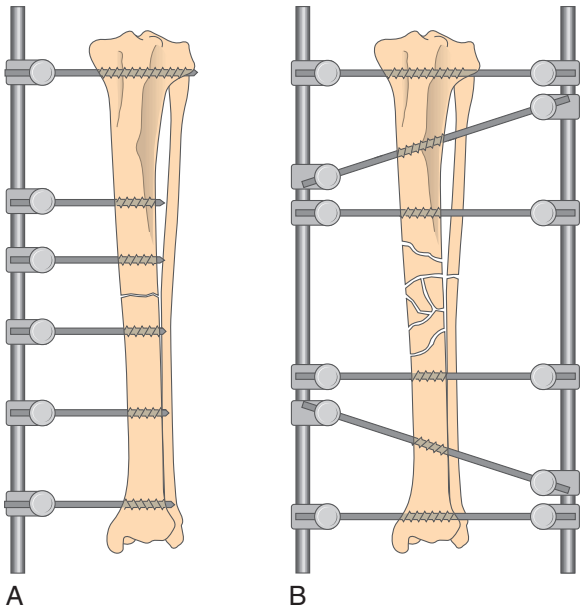


FIG. 32.22 Placement of (A) half pins and (B) full pins. Note that half pins are inserted so that they penetrate both cortices but only one skin surface, whereas full pins penetrate both cortices and skin surfaces.

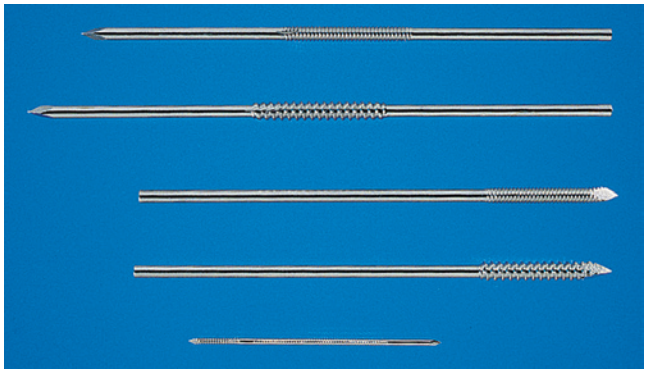


FIG. 32.23 Positive-profile fixation pins used with external skeletal fixation. *Top to bottom:* Centrally threaded cortical pin, centrally threaded cancellous pin, end-threaded cortical pin, end-threaded cancellous pin, and mandibular fixation pin.

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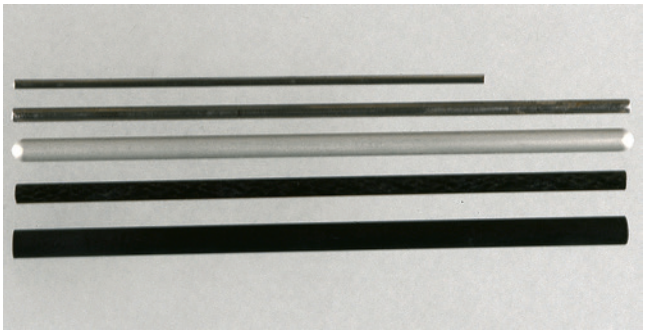


FIG. 32.24 Small, medium, and large external connectors (bars) used with external skeletal fixation are made of stainless steel, titanium alloy, and carbon fiber.

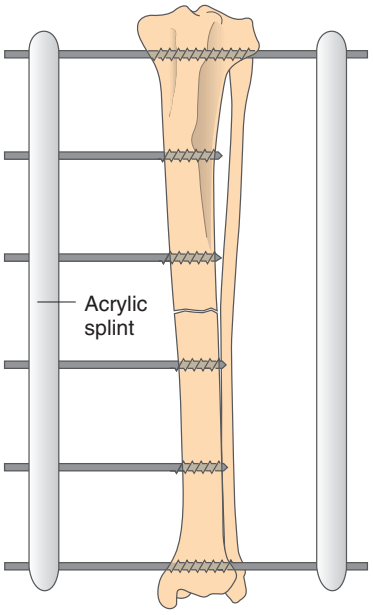


FIG. 32.25 Use of acrylic materials as external connectors rather than standard metal bars.

The fixation pins are purchased individually, and pediatric anesthesia tubing is used for the column-molding tubes, or the material may be molded by hand.

Linkage Devices

There are two commercially available clamps that enhance frame application and fracture fixation. The bar-gripping portion of the SK clamp (IMEX Veterinary) is separable to allow easy positioning on the connecting bar and is secured by two bolts. The hole in the bolt accepting the fixation pin is large enough to accept positive-profile threaded pins. Fixation pins are secured with a specially designed washer positioned on the pin fixation bolt. Double clamps (clamping connecting rod to connecting rod) are also available in this system. The Secur-U clamp (Securos Surgical) has an altered U-shaped component that can be easily placed on the connecting bar. The head component of the clamp has two holes, one to accept a larger pin shank and the other to accept a smaller pin shank. Securing the head with a bolt secures the fixation pin (Fig. 32.26).

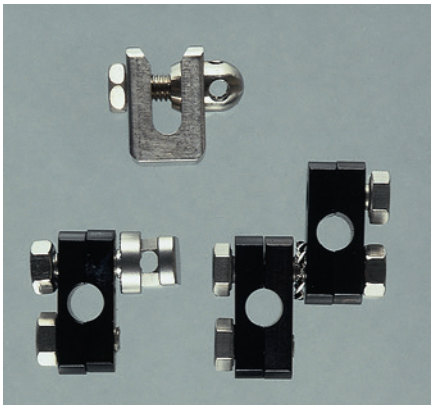


FIG. 32.26 Linkage devices for joining fixation pins to external connecting bars and external connecting bars to each other. Note the larger holes for the external connecting bars and smaller holes in the bolts for fixation pins. Secur-U clamp (Securos Surgical), SK single and double clamps (IMEX Veterinary).

Application

Preoperative planning for choosing the external fixator frame and the number and location of pins is essential for optimal outcome. The patient fracture assessment score (see p. 985) must be considered when choosing an appropriate frame configuration. The lower the fracture assessment score, the longer the fixator must remain in place and the stronger it must be (and vice versa). It is always better to err on the side of increased strength and stiffness rather than insufficient strength and stiffness. An insufficiently strong or durable frame may lead to complications, whereas an overly rigid frame can be destabilized to a less rigid frame as healing progresses. Therefore it is important to understand the factors that significantly influence the strength and stiffness of the external fixator-bone combination, including frame configuration, pin number, size, placement, position, design, and bar placement (see Box 32.7).

External fixators are most commonly used on the radius and tibia. Application to the femur or humerus is limited by the lack of access to the medial aspect of the bone. In addition, lateral pins must be inserted through large muscles, particularly on the femur. Insertion of pins through the muscles increases pain and morbidity.

A variety of frame configurations and pin designs are available, but certain principles of application are common for all external fixation devices. One of the most important principles is adherence to the practice of aseptic surgery. Proper aseptic technique, including patient preparation, gloving, gowning, draping, and instrument preparation, is just as important, if not more important, in the application of external fixators as for any other fixation method. *Suspend the injured limb from hooks in the ceiling. Scrub the liberally clipped area with an antiseptic soap.* The limb may be left suspended during application of the external fixator or released from the suspension after it has been draped (see p. 40). Once the number and size of pins have been selected, the method of placement and position of the pins relative to the fracture need to be considered. The most effective way to place a fixation pin is by using a mini-approach to expose the pin site and predrilling the hole before fixation pin insertion (Box 32.8). *Make a small (1-cm) longitudinal skin incision over the proposed pin site. Use a hemostat to dissect bluntly through the soft tissue*

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BOX 32.8 Key Concepts for Inserting Fixation Pins

- Expose the pin insertion site.
- Center the pin in the bone.
- Predrill the pin hole.
- Insert the pin with low revolutions per minute power.
- Release the incision around the pin to prevent skin tension.

from the skin surface to the bone to create a soft tissue tunnel that allows free gliding motion of surrounding muscles around the fixation pin. The tunnel also prevents the pain and discomfort that can result from impingement of soft tissue against fixation pins.

Create the soft tissue tunnel between large muscle bellies rather than through them, and avoid neurovascular structures. Protect the soft tissue in the walls of the tunnel from trauma using a drill sleeve, or retract and stabilize the tissue with a hemostat.

Predrill the bone using a high revolutions per minute speed drill and a twist drill bit 0.1 mm smaller than the core diameter of the fixation pin. Place the fixation pin through the drilled hole with a power drill, using low-revolutions per minute speed. Be sure the pin tip extends beyond the opposite cortex. The site at which each fixation pin will be placed relative to the fracture is a factor that can positively influence the mechanical performance of external fixators. Generally, the most proximal and distal pins are placed in the respective metaphyses, and remaining pins are spaced evenly in the proximal and distal fragments. One pin should be placed 1 cm proximal and one pin 1 cm distal to the fracture. An alternative strategy is to place these pins one bone diameter away from the fracture region (a guideline that is self-adjusting with regard to patient size). The closer these pins are placed on either side of the fracture, the shorter the distance between connecting clamps on the external bar. Therefore the length of the bar that must sustain the load is shortened, resulting in a stiffer external bar.

NOTE Each fixation pin should be drilled into the bone at the point of greatest cross-sectional diameter (usually the center of the bone). When using type 1 (half) pins, the trocar point should exit the far cortical surface for a distance of 2 to 3 mm to make sure that thread fully engages the far cortex of the bone.

The distance between the external bar and the body affects the length of the fixation pin from its attachment to the bolt on the clamp to its point of entrance into the bone. The shorter this distance, the less flexible is the fixation pin and the less micromovement at the pin-bone interface. *Once the fixation pins and external bar(s) are in place, adjust the position of the bar relative to the skin surface. Place the external fixation bar as close to the body as possible without allowing the clamps (pin grippers) or bar to impinge on the skin surface. Turn the pin-gripping clamps so the bolt is closest to the skin. Place the external bar so that one forefinger can be inserted between the clamps and the skin surface (approximately 1 cm). Tighten the clamps to secure the bar.*

Unilateral-Uniplanar (Type Ia) Fixators

Type Ia fixators (see Fig. 32.21) are usually applied to the cranial medial surface of the radius, the medial surface of the tibia, and the lateral surface of the femur or humerus. The fixation pins are referred to as half or type 1 pins because they only penetrate the near skin surface and the bone. *Begin by placing a half pin in the metaphyses of the proximal and distal bone fragments. Place the pins in the center of the bone cylinder, perpendicular to its long axis and through both cortices. For efficiency with SK clamps, place an appropriate number of pin grippers on the bar to accommodate placement of subsequent pins. For example, if three pins are to be inserted in the proximal fragment and three pins in the distal fragment, then slide six empty pin grippers onto the external bar. Reduce the fracture (open or closed) and connect the two pins to the two pin grippers on the ends of the external bar. Place the additional half pins directly through the four open central pin grippers. Once all the intermediate pins have been placed, tighten the pin grippers, and make a radiographic examination of the limb to assess fracture reduction and pin placement. Trim the fixation pins to prevent injury to the owners and immediate environment. Application technique varies slightly with Secur-U clamps. When this type of clamp is used, pins are placed in the bone using a special aiming tool applied to the connecting rod. Clamp components are then applied to the pin to secure it to the rod.*

NOTE Either SK or Secur-U clamps can be easily added to an external connecting bar if an additional fixation pin is required.

Unilateral-Biplanar (Type Ib) Fixators

Type Ib frame configurations (see Fig. 32.21) are applied most often to the radius and tibia. With the radius, one external bar is placed over the craniomedial surface of the bone and a second bar over the craniolateral surface. With the tibia, one external bar is placed over the medial surface of the bone and a second bar over the cranial surface. *Apply a type Ia frame to the cranial medial surface of the radius or tibia as described previously. Then apply another type Ia frame approximately 90 degrees to the first frame. In general, plan to place a total of four fixation pins per major bone segment. Strengthen the frame for animals with low fracture assessment scores by adding diagonal connecting bars, which span from the proximal cranial lateral connecting bar to the distal cranial medial connecting bar and vice versa. Connect the diagonal bars to the existing connecting bars with double clamps, or connect the diagonal bars to the existing pins with single clamps. Once all pins have been placed, tighten the pin grippers, and make a radiographic examination of the limb to assess fracture reduction and pin placement. Trim the fixation pins to prevent injury to the owners and immediate environment.* A modified type Ib fixator can be developed for the humerus by placing a full fixation pin through the condyle and constructing a unilateral frame on the lateral aspect of the humerus, coupled with a unilateral frame connecting the medial aspect of the full fixation pin to half pins secured to the cranial aspect of the proximal humerus.

Bilateral-Uniplanar (Type II) Fixators

Because of the adjacent body trunk, type II frames (see Fig. 32.21) cannot be placed on the femur or humerus. They are applied only to the radius or tibia and are usually placed in the frontal plane (a vertical plane that divides the bone into cranial and caudal sections). Minimal type II frames are generally applied when using the SK equipment, but they may be upgraded to a maximal type II frame for animals with low fracture assessment scores. Maximal type II frames are generally applied when using Securos equipment because of the versatility of the aiming device. *First, place full pins in the proximal and distal metaphyses so that*

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they lie in the frontal plane of each segment. Place the pins perpendicular to the bone surface and parallel to the adjacent joint line to facilitate restoring limb alignment. If necessary, use these pins to apply traction to the limb to aid in fracture reduction. Reduce the fracture, place the appropriate number of empty pin grippers on each external bar to accommodate placement of the preplaced pins and subsequent intermediate pins, and connect the proximal and distal full pins to the medial and lateral connecting bars. Insert intermediate pins as half pins or full pins. Determine which pin type to use based on the fracture assessment score. Commercial aiming devices help direct the fixation pins used in maximal type II frames to the contralateral clamp. A guide device can be constructed with an additional connecting bar and clamp. *Once all the intermediate pins are in place, tighten the pin grippers and take a radiograph of the limb to assess fracture reduction and pin placement. Trim the fixation pins to prevent injury to the owners and immediate environment.*

Bilateral-Biplanar (Type III) Fixator

Type III configurations cannot be applied to the femur or humerus because of the position of the body wall. Type III frames are only indicated in very large dogs with low fracture assessment scores. *To apply type III fixators to the radius or tibia, place a type II in a frontal plane, then place a type Ia frame in the midsagittal plane (a vertical plane that divides the limb into lateral and medial sections).*

External Skeletal Fixators With Intramedullary Pins

Humeral and femoral fractures are not usually stabilized with external skeletal fixators alone because the most stable frames (type II and type III) cannot be applied to these bones. To provide the strength and stiffness desired with complicated humeral or femoral fractures, a combination IM pin and type Ia or type Ib external fixator is often used (see Fig. 32.21). The fixation pin design and number of fixation pins are based on the fracture assessment score, but normally the number of fixation pins is limited to two or three pins placed above and below the fracture. More fixation pins are not used because the greater the number of pins, the more intense the discomfort associated with pins placed through the large muscle groups. *Begin by reducing the fracture and inserting an IM pin that fills 30% to 40% of the medullary canal.* Use of an IM pin the same diameter as the pin shank of the fixation pins used in the fixator facilitates attachment of the IM pin to the fixation frame in tie-in configurations. *Use cerclage wire to support long oblique fractures or spiral fractures. If multiple fragments are present, bridge the comminuted section of bone with the IM pin and external fixator without disturbing soft tissue attachments to bone fragments. Once the fracture is reduced and the IM pin has been placed, add the external fixator. Use the largest size fixation pin that does not exceed 25% of the diameter of the bone and will pass adjacent to the IM pin. If unsure about the track of the IM pin inside the bone relative to the chosen site for insertion of a fixation pin, drill the proposed fixation pin site with a small Kirschner wire. If the IM pin is encountered, select an alternative location; otherwise insert the fixation pin at this site. Insert the intermediate pins through preplaced pin grippers as described earlier. Connect the fixation pins to an external bar placed 1 cm from the skin surface.* Increasing the number of fixation pins strengthens the external fixator but also increases postoperative discomfort. Two methods are used to strengthen external fixators without increasing the number of pins. One method is to add more external bars (the addition of a single external bar doubles the strength of the system), or an augmentation plate can be added with the Securos fixator. Additionally, the IM pin may be left protruding above the skin surface at the exit point proximal to the greater trochanter. The IM pin is then "tied" into the external fixator by connecting the two with an additional short segment of external bar (see Fig. 32.21).

Acrylic Splints

Application of acrylic splints involves a one-stage or two-stage technique. The advantage of two-stage techniques is that pin placement and fracture reduction can be assessed before hardening of the acrylic. If the single-stage technique is used and if postoperative radiographs show fracture reduction or fixation pin placement to be unsatisfactory, a small section of the acrylic column must be removed before corrective measures can be taken. *With either technique, insert the fixation pins in the bone fragments following the same principles and guidelines described for construction of standard external fixator frames. Place acrylic column-molding tubes over the ends of the fixation pins 2 cm from the skin surface. If using a single-stage technique, reduce the fracture and pour the acrylic into the columns. Place saline-moistened sponges or use a saline drip on the fixation pins to dissipate heat generated by the acrylic. Allow the acrylic to cure for 5 to 10 minutes. If using a two-stage technique, place the tubes over the ends of the fixation pins, but do not add the acrylic. Instead, reduce the fracture and apply a temporary alignment frame. Take radiographs to assess pin placement and fracture reduction; if satisfactory, pour acrylic into the columns and allow it to cure. If alignment needs to be changed after the acrylic has hardened, cut the acrylic column(s) at the fracture line with a saw. Once the appropriate adjustments are made, patch the acrylic column by adding new acrylic to fill the gap. Peel the plastic molding back from each end at the gap, and drill several holes in the remaining acrylic to provide a site of attachment for the old and new acrylic. Mold a small amount of new acrylic by hand and place it into the gap, then allow the acrylic to cure.*

Postoperative Care

Postoperative analgesia should be provided (see Boxes 32.1 and 32.2 and Table 32.4). Immediately after surgery, the pin-skin interface should be cleaned with antiseptic solution using cotton swabs. Incisions around the pins should be released or extended if there is skin tension around a pin during limb flexion and extension. Sterile gauze sponges are opened, crumpled, and placed around and between fixation pins and the limb wrapped with an elastic bandage material. After surgery, the pin-skin interface should be cleaned and the bandage changed daily. After approximately 1 week, gauze packing may be discontinued. A bandage may be applied to the fixator bars and pins to protect the frame and the animal's environment. The pin-skin interface should be cleaned daily until little or no serosanguineous transudate is noted at the surface. Once the interface seals, the daily observations should continue. Activity should be restricted to leash walking and physical rehabilitation until the fracture has healed. Physical rehabilitation encourages controlled limb use and optimal limb function after fracture healing (see Table 32.7 and Chapter 11). After the animal has been released from the hospital, postoperative examinations should be done at 2 and 6 weeks after surgery, then every 6 weeks. The pin-skin interface should be carefully examined and the clamp and skin observed for separation. If irritation or drainage is present, the involved skin surface should be cleaned and the area around the pins packed with gauze.

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Staged disassembly is the process of modifying a fixation frame at approximately 6 weeks after surgery to increase the loading on the healing fracture. Increased loading on the fracture further stimulates bone healing and remodeling. For example, type Ia SK frames may be destabilized by trading a large connecting bar for a smaller one. Type Ib frames are destabilized to type Ia frames by removing one frame. Type II frames are destabilized to type Ia frames by removing a connecting bar. Type Ib or II acrylic frames may be destabilized by removing the middle section of acrylic (over the fracture) from one bar. Frames may also be destabilized by removing fixation pins; however, a minimum of two pins on either side of the fracture must be maintained. In addition, the increased load on the remaining pins will result in pin loosening. The fixator should be removed completely once the bone has healed. The patient should be sedated, the pin-gripper loosened, and the fixation pin removed with a hand chuck.

CIRCULAR EXTERNAL FIXATORS

Indications and Biomechanical Principles

AP Circular external fixators are used to stabilize fractures, compress or distract fractures or nonunions, transport bone segments, and dynamically correct bone angular and length deformities. The circular external fixator is uniquely suited for controlled distraction of bone segments, resulting in new bone formation in the trailing pathways called *distraction osteogenesis* (see p. 1005). Small-diameter tensioned wires provide adequate stability to bone segments but allow controlled axial micromotion at the fracture site without compromising the fixator's stability.

For optimal mechanical stability, the frame comprises four rings securing four pairs of wires that are placed as close to perpendicular as the soft tissue anatomy allows. Ring diameter affects wire length and mechanical properties of the circular fixator. Increasing ring diameter decreases axial stiffness and to a lesser extent, torsional and bending stiffness. The smallest ring diameter, allowing a minimum of 2 cm between skin and ring, provides the optimal mechanical performance. The most proximal and most distal rings are placed at their respective metaphyseal locations, and the inner two rings are placed close to the fracture. This "far, near, near, far" construction provides optimal control and stabilization of the major bone segments.

The angle of intersection of the wires and the positioning of the bone relative to the center of the ring affect the fixator's mechanical properties. Wires that intersect at 90 degrees maximize stability and minimize shearing forces. Often wires must intersect at less than 90 degrees to avoid tendons and vital neurovascular structures. Decreasing the angle of intersection reduces axial and bending rigidity. In general, wire angles less than 45 degrees should be avoided. Similarly, concentric placement of the bone within the ring is optimal. Often the soft tissue dictates that the bone is eccentrically placed, which decreases torsional stiffness and increases axial stiffness. Addition of olive wires increases the bending, axial, and torsional stiffness of the fixator. Increasing wire tension also increases the bending and axial stiffness but decreases the torsional stiffness of the fixator.

Equipment and Supplies

Circular external fixators are composed of transosseous wires attached to rings or partial rings with wire fixation bolts or other assembly elements. The rings are linked with threaded or telescopic rods, connecting plates, hinges and posts, and nuts and bolts to form a frame. A wire tensioner and various wrenches complete the set (Fig. 32.27). Although many specialized components can be used with the circular fixators, the main components of the system are described next.

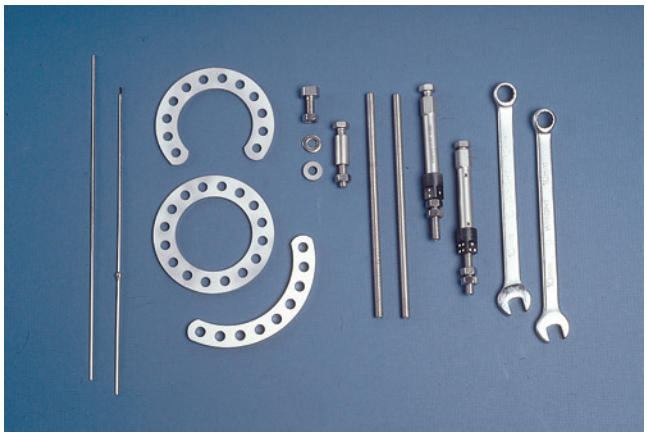


FIG. 32.27 Equipment used for circular external fixation includes wires with and without olives, rings, wire fixation bolts, threaded rods, nuts, and wrenches.

Wires

Wires used for dogs and cats are generally 1, 1.2, or 1.5 mm in diameter. Wire strength and stiffness increase proportionately to the diameter of the wire. Cats and small dogs weighing up to 10 kg require 1-mm wires, dogs 10 to 20 kg require 1.2-mm wires, and dogs more than 20 kg require 1.5-mm wires. Wires with a bayonet point are preferred for drilling through cortical bone. Wires with a trocar point are reserved for drilling through cancellous bone. Wires with a bead positioned midway along the wire are called *olive wires* and are placed with the bead adjacent to the cortex to minimize bone translation along the wire. Appropriately placed olive wires can provide interfragmentary compression and increase the stability of the frame construct.

Wires are placed so that one wire is adjacent to each surface of the ring for a total of two wires per ring. Additional wires may be added to each ring with the addition of two posts to the ring to allow for a drop wire positioned at a distance from the ring surface.

Rings

Partial rings and full rings are selected based on size of the limb and location of the ring along the limb. Five-eighths rings or stretch rings are used when full rings would limit joint motion in areas adjacent to the elbow and stifle. Judicious wire tensioning (≤50 kg) is used with partial rings to prevent ring deformation. Partial rings are versatile and can be used separately or bolted together to form full rings. Full rings have more available holes for rods and wires but are less versatile. Ring size is dictated by the animal's size. The smallest ring that allows at least 2 cm in distance between skin and inner circumference of the ring at all points should be selected for frame construction.

Wire Fixation Bolts

Cannulated wire fixation bolts allow wire passage through a concentrically placed hole at the base of the bolt head. The fixation bolt is then tightened to the ring surface with a nut, securely

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clamping the wire. Slotted wire fixation bolts have an eccentric slot located under the bolt head and parallel to its long axis. Wires are fixed between the slot and the ring surface when the nut is tightened. Some slotted wire fixation bolts are also cannulated to accept wires in both positions. The wire must be fixed without deformation. Both slotted and simple washers are used to raise a wire fixation bolt to a wire not lying directly on the ring.

Frame Construction

Threaded rods are used to connect the rings and form a frame. The rings are stabilized on each rod using one nut above and one nut below each ring. The nuts are tightened to clamp the ring securely. Compression or distraction of the fracture can be achieved by turning the nuts that fix the rods to the rings in the appropriate direction. Telescopic rods are hollow rods used as supports and connecting elements of the rings. These rods provide additional stability when relatively long distances must be spanned.

In general, frames are constructed so that one ring and its wires are placed at the proximal end of the long bone, and another ring and wires are placed at the distal end of the long bone. Two additional rings are placed so that their wires penetrate the proximal and distal bone segments close to the fracture. This "far, near, near, far" construction provides optimal control and stabilization of the major bone segments.

Hinges are specialized posts that accept threaded rods and that can interface at an angle with each other when connected by a bolt and nut. Used in pairs, hinges connect proximal and distal ring frames and allow gradual reduction of an angular deformity.

Wire Tensioner

The tensioner is an instrument used to tension the wires to an exact force (Fig. 32.28). Wire tension affects the overall rigidity of the fixator construct. The exact amount of tensioning needed depends on the animal's weight, the local bone quality, the treatment plan, ring diameter, and the frame construction. Cats and small dogs do not require tensioning of the wires. Dogs weighing 5 to 10 kg require 20 to 30 kg of tension in the wires. Dogs weighing 10 to 20 kg require 30 to 60 kg of tension in the wires. Dogs more than 20 kg require 60 to 90 kg of tension in the wires. Balanced tension, using two tensioners simultaneously to tension the wires for a single ring, is preferable. When using a tensioner that is not calibrated, tensioning the wire until the tensioner remains parallel with the ring without support is sufficient.

Wrenches

At least two appropriately sized crescent wrenches are necessary for tightening bolts and nuts simultaneously.

Application

Fracture Treatment

AP Circular fixators are most applicable for fractures of the extremities below the elbow and stifle (Fig. 32.29). Modified circular fixators have been used for femoral and humeral fractures, but this application requires extensive use of arches and olive wires. Principles of frame construction and circular fixator application must be followed for optimal outcome in patients with all ranges of fracture assessment score.

Radiographs are evaluated to determine fracture location and soft tissue diameter. The rings are selected to fit the soft tissue diameter while allowing 2 cm of distance between soft tissue and the rings. To expedite operating time, frames are generally preconstructed with four rings and three threaded rods. The rods are placed to form an equilateral triangle if possible. The rings are positioned on the rods in a "far, near, near, far" manner, with the proximal ring at the proximal metaphysis and the distal ring at the distal metaphysis. In most cases the proximal ring is a partial ring designed to limit interference with the stifle or elbow motion. The two inner rings are positioned approximately 1 to 2 cm proximal and distal to the fracture line. The rings are stabilized in position on the threaded rods by tightening nuts placed adjacent to the top and bottom surfaces of the rings. Care is taken to secure the rings parallel to each other. Measuring the distance between rings on all three rods helps to ensure accurate placement. A short distal or proximal segment may limit the frame to three rings. The single ring will secure three wires placed in the short segment, two wires will be secured to the ring, and the third wire will be secured on posts arising from the ring. *Prepare the patient for aseptic surgery. Place the preconstructed frame around the limb.*

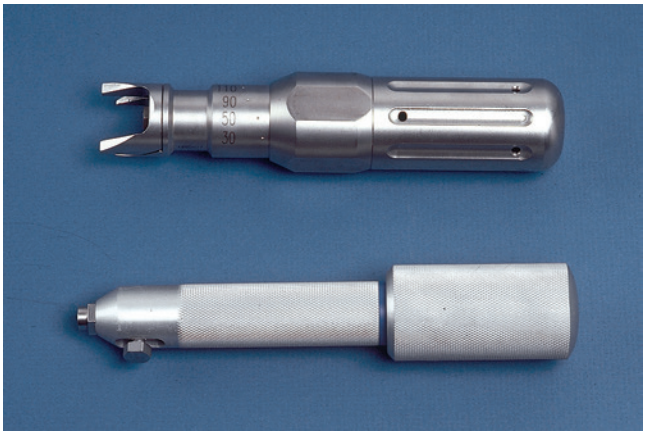


FIG. 32.28 Wire tensioners (calibrated and uncalibrated) used with circular fixators.

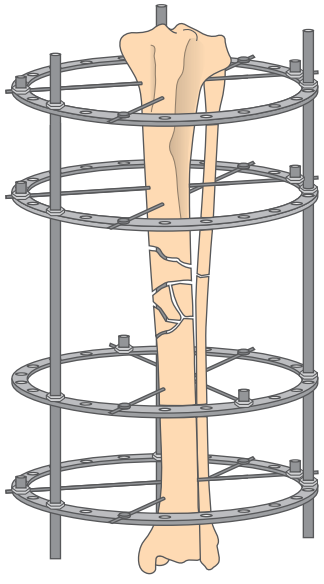


FIG. 32.29 Standard circular fixator frame for fracture management.

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If the limb is suspended for reduction purposes (see p. 990), it must detached, the frame positioned, and the limb reattached in suspension. Wire size is selected according to patient size. *Hold the frame in an approximate position, and select an appropriately sized wire to drive through the distal metaphysis of the bone, parallel to the distal joint surface, avoiding vital soft tissue structures. If necessary, as when an adjacent surgical approach disrupts normal skin position where the wire is to be located, hold the skin in approximately normal position while the wire is driven.* This precaution ensures minimal tension on the skin adjacent to the wire. *If the wire is penetrating muscle, position the limb to stretch the muscle maximally as the wire is driven. Manually push the wire through the skin and adjacent soft tissue until it centers on the bone, penetrating the periosteum. Then drive the wire through both cortices with a high-speed power drill. For optimal wire placement, hammer the wire out through the remaining soft tissue until it is centered in the bone. Secure the wire to the distal surface of the distal ring with slotted or cannulated bolts and nuts. Reposition the frame to ensure that the limb is centered in the frame and that the rings do not compromise the soft tissue. Position a second wire adjacent to the proximal surface of the distal ring and drive it through the bone. Position the wire as close to 90 degrees (generally, 60–70 degrees is achieved) to the first wire as soft tissue constraints will allow.*

Examine the position of the wire in relationship to the ring. If the wire lies immediately adjacent to the ring, secure it with a cannulated bolt if it passes over a hole or with a slotted bolt if it passes adjacent to a hole. If the wire is located above or below the ring surface, use washers to raise the surface to the wire, which is then secured as described earlier. Pulling a wire to meet the ring causes undue forces on the wire that may cause pain, malalignment due to translation of the bone segment, and pin tract infection. *Reexamine the position of the frame and place a second pair of wires through the proximal metaphysis, adjacent to the proximal ring, in a similar manner (Box 32.9).* Circular fixators are unique in that fracture reduction can be controlled with the frame and wire application during a closed procedure. Fracture reduction can be achieved by distracting the proximal and distal rings to overcome fracture fragment overriding or using fixation with olive wires to bring fragments into contact. Alternately, using an arch wire technique involves placing a wire through the diaphysis of a bone segment and arching the wire before securing it to the ring. When the wire is tensioned, the arch is straightened and the bone segment drawn into alignment. Closed fracture reduction using the circular fixator is monitored with radiographic control. *Fill out the remainder of the frame with additional wires, using appropriate techniques to reduce the fracture and align bones as needed. Tension each pair of wires as they are placed, to the appropriate tension for the patient's size. Optimally, tension each pair of wires simultaneously to prevent ring deformation. To tension the wire, tighten the nut securing one end of each wire. Apply the tensioner to the opposite end of the wire and tension. Secure the remaining end of the wire before removing the tensioner. After tensioning, cut the wires flush with the securing bolt or cut 2 to 3 cm from the bolt and curl the ends toward the ring. Examine the limb and the frame to ensure that (1) all rings clear the soft tissue, (2) all wire sites allow comfortable skin position, (3) all joints move freely, (4) all wires are tensioned, and (5) all nuts are tight. Make a radiographic assessment of the fracture alignment and fixation placement.*

BOX 32.9 Key Concepts for Inserting Fixation Wires

- Position soft tissues before driving wire.
- Drive the wire with a high-speed drill.
- Place wire pairs as close to 90 degrees with each other as possible.
- Do not pull the wire to the ring.
- Tension the wire appropriately.
- Use olive wires to increase stability.

Arthrodesis

The circular external fixator can be used for carpal and tarsal arthrodesis. After articular cartilage removal and cancellous bone graft application, a preconstructed frame is placed and secured with two rings located proximally to the joint and two rings distally to the joint. Appropriate angulation of the frame to accommodate the joint angle can be achieved using hinges in place of threaded rods.

Bone Lengthening

The ultimate advantages of the circular fixator over other forms of fracture fixation are its versatility and effectiveness in bone distraction or intercalary bone transport and the resultant distraction osteogenesis, or production of new bone in the trailing pathway (see p. 1006). *Bone lengthening* is used to correct a shortened bone after premature physeal closure. *Bone transport* is used to fill a bone defect produced by traumatic bone loss or resection of a bone tumor. Successful application of this technique requires close attention to the details of appropriate osteotomy technique; preservation of marrow, periosteum, and extraosseous blood supply; application of a stable circular construct; and correct rate and rhythm of distraction.

Bone lengthening can be achieved unifocally or bifocally. *Unifocal* lengthening occurs when a single osteotomy is created in the bone. *Bifocal* bone lengthening occurs when two osteotomy sites are created in the bone, effectively doubling the rate of distraction. A preconstructed frame similar to the four-ring frame developed for fracture fixation is used for bone lengthening. Bifocal bone lengthening requires an additional ring to stabilize the center bone segment. The frame can be constructed using the threaded rods. However, telescoping rods or linear motors can be inserted for increased strength and ease of distraction.

Optimal osteotomy techniques for distraction osteogenesis have been the subject of extensive investigation. Ilizarov determined that a subperiosteal partial cortical osteotomy (called a *corticotomy* because the osteotome does not penetrate across the medullary canal) around three-quarters of the bone's circumference, followed by osteoclasis or manually fracturing the remaining quarter of the bone, maximized preservation of endosteal and periosteal tissues and adjacent circulation. Other investigators have shown that osteotomy performed with an osteotome may be as effective as corticotomy and that preservation of periosteal tissue enhances the regenerative and reparative potential. An oscillating saw cooled with saline can be used to perform the osteotomy but may cause delayed consolidation. This concern may not be clinically relevant for young animals with greater osteogenic potential.

After frame application and osteotomy, a delay or latency period before initiation of distraction is recommended to allow local cellular and vascular responses to occur. Recommendations vary from 1 to 3 days for immature animals to 5 to 7 days for

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BOX 32.10 Factors Used to Determine Latency Period

Decrease Latency Period
Immature animal
Excellent health
Subperiosteal corticotomy
Metaphyseal osteotomy
Minimal soft tissue trauma

Increase Latency Period
Adult animal
Compromised health
Osteotomy with oscillating saw
Diaphyseal osteotomy
Massive soft tissue trauma

adult animals. Early distraction may cause diminished callus production. Delaying distraction may cause premature consolidation. In general, the animal's age and health, type and location of the osteotomy, and associated soft tissue trauma are considered when establishing a latency period (Box 32.10).

An optimum rate of 1 mm per day divided into a rhythm of four distractions per day (i.e., 0.25 mm per distraction) has been shown to favor regenerate bone formation without causing soft tissue discomfort and to be clinically achievable. Rate and rhythm of distraction can be varied slightly depending on the patient and on radiographic evidence of regenerate formation. Distraction is accomplished either by turning the nuts securing the rings to the threaded rods or by turning the cubes on the linear motors.

NOTE Take care to turn the nuts in the appropriate direction to cause distraction. Measure between the rings to ensure that distraction is occurring. Be sure the nuts are secured after distraction to restore frame stability.

Radiographs should be taken every 2 weeks to observe the appearance of the regenerate bone and adjust the rate of distraction accordingly. The regenerate bone should appear as definite columns of longitudinally oriented new bone extending from each osteotomy surface toward a central transverse radiolucent area. Bone should also retain a constant diameter radiographically. An hour-glass configuration of the regenerate bone indicates an overly rapid distraction rate. Inconsistent radiopacity, irregular bone columns, and focal failures of bone formation indicate instability or poor vascularity; the rate of distraction should be decreased until a normal regenerate is observed. A cross-sectional diameter of the regenerate bone exceeding the cortical diameter indicates a rapidly forming regenerate and the possibility of premature consolidation; the rate of distraction should be increased.

Bone Transport (Advanced Technique)

The principles and techniques described for bone lengthening are used when moving a segment of bone into a bone defect while stimulating new bone formation in the trailing pathway. The technique of bone transport is used to fill large bone column defects occurring after trauma, nonunion, osteomyelitis with sequestered bone, or bone tumor resection (Fig. 32.30) *Preconstruct a frame with four stabilizing rings and a center transport ring. Apply the frame to the affected limb as described earlier. Approach and identify the osteotomy site. Secure the segment of bone to be transported to the center transport ring with two tensioned wires. Perform the osteotomy. Ensure that the osteotomy is complete and the bone segment movable. After the appropriate latent period, move the transport ring toward the defect at an appropriate rate and rhythm for the animal.* When the transported bone segment contacts the bone on the far side of the defect, called *docking*, compression is applied with the ring fixator to stimulate bone union. Occasionally, cancellous graft is necessary to achieve bone union.



FIG. 32.30 Regenerated bone appears in the trailing pathway of a distracted bone segment in this dog treated with bone resection and distraction osteogenesis. (Courtesy Dr. Nicole Ehrhart.)

Angular Limb Deformity Correction (Advanced Technique)

Circular fixators are indicated for dogs that have severe limb length discrepancy, when additional extensive growth is anticipated, and in dogs that have significant craniocaudal deformity. Circular fixators can be used to treat dogs with varus-valgus deformities, although techniques using the standard linear external fixator are also effective for treating this deformity. An advantage of circular fixators in treating angular deformities is that the flexibility of the system allows angular deformities and length discrepancies to be corrected simultaneously. Disadvantages of circular fixators for correcting deformities include an intensive and often lengthy learning curve for surgeons, extensive preoperative planning, and intensive postoperative monitoring of the deformity correction process.

Initially, mediolateral and craniocaudal radiographs of the affected limb and the opposite control limb (if normal) are used to define the varus-valgus and craniocaudal components of the deformity. Length discrepancy is also determined by comparing lateral radiographs of the affected bone(s). Rotational deformity may be estimated from radiographs, although often it is best determined from physical examination. Rotational deformity is

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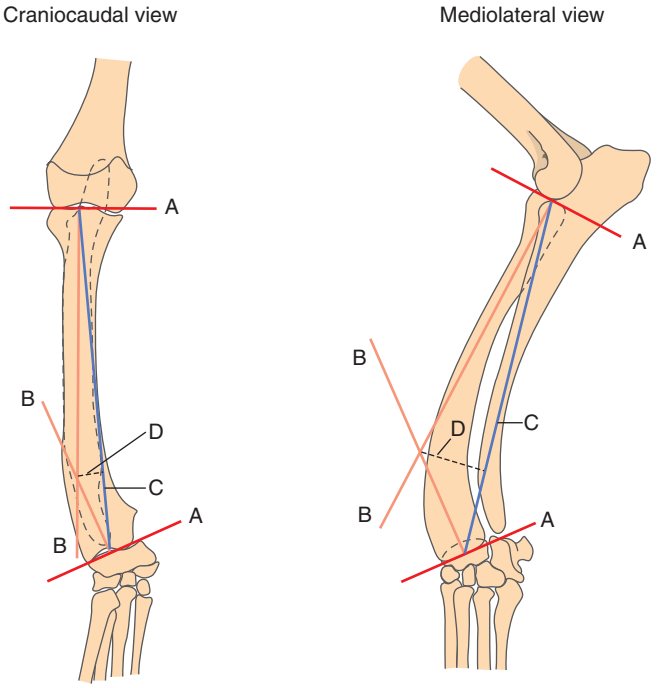


FIG. 32.31 Schematic for drawing the lines on tracings of the craniocaudal and lateral radiographs. Lines *A* parallel the joint surfaces. Lines *B* bisect the metaphyses and form the apex of the deformity. Lines *C* connect the intersections of the metaphyseal lines and the lines paralleling the articular surfaces. Lines *D,* connecting the apex of the deformity to line *C* and perpendicular to line *C,* are the vector components.

measured directly on the dog by comparing the planes of flexion and extension of the adjacent joints. *To determine the degree and plane of angular deformity and the optimal hinge and motor placement, trace the craniocaudal and mediolateral radiographic images of the affected bone. Draw two lines that bisect their respective metaphyses and are perpendicular to lines paralleling the adjacent articular surface on each tracing (Fig. 32.31).* The point of intersection of the two lines is the apex of the deformity. *To calculate the varus-valgus and craniocaudal components of the deformity, draw a line connecting the intersections of the metaphyseal lines and the lines paralleling the articular surfaces on both radiographic views. Then draw a line from the point of intersection (i.e., apex of the deformity) to the line connecting the articular surface lines on each tracing (see Fig. 32.31).* These lines are perpendicular to the lines connecting the articular surface lines and represent the vector components of the deformity. *Draw the appropriately sized fixator ring with all holes represented. Identify and draw two lines within the circle connecting the craniocaudal and mediolateral aspects of the ring.* The circle lines should intersect perpendicularly at the center of the circle. *Plot a line the distance of the medial or lateral deformity (i.e., vector component), starting at the intersection of the circle lines and extending the appropriate distance and direction. Plot a second line the distance and direction of the cranial or caudal deformity, starting at the end of the mediolateral distance line and paralleling the craniocaudal circle lines. Drop a tangential line from the intersection of the circle lines, and terminate it at the end of the craniocaudal distance line; this line defines the plane of deformity. Place the motor on the ring in the plane of deformity and on the concave side of the deformity (Fig. 32.32).*

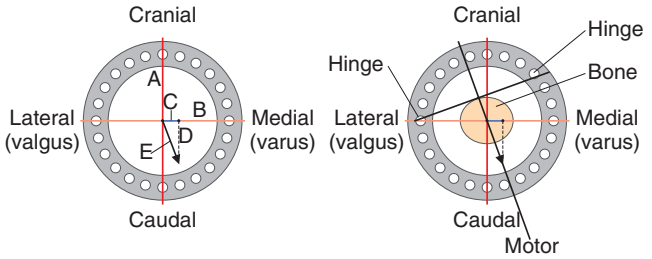


FIG. 32.32 Schematic of the ring drawing. Lines *A* and *B* connect the cranial and caudal aspects and the medial and lateral aspects of the ring and intersect perpendicularly in the center of the ring. Line *C* is the medial to lateral vector component (determined from the craniocaudal radiograph). Line *D* is the cranial to caudal vector component (determined from the lateral radiograph). Line *E* is the tangential line defining the plane of deformity. The motor is placed in the plane of deformity and on the concave side of the limb. The hinges are placed at the apex of the deformity.

NOTE The hinges are placed at the apex of the deformity. The positioning of the hinges is critical to the outcome. Hinges placed at the apex of the deformity and level with the convex surface of the bone will correct the angular deformity, with an increase in length on the concave side of the bone equal to an opening wedge osteotomy. Hinges placed at the apex of the deformity and at a distance from the convex surface of the bone result in simultaneous opening of the wedge and an increase in the length of the convex surface of the bone.

The frame is constructed with the hinges and motor in the appropriate positions before the surgical procedure. *Following the protocol outlined earlier for application of a circular fixator in fracture fixation, place the preconstructed frame over the affected limb, and secure the proximal and distal rings with tensioned wires. Ensure that the tensioned wires are parallel to their respective articular surfaces. Place additional tensioned wires to fill out the frame. Approach the bone and perform an osteotomy at the apex of the deformity.* The osteotomy should parallel the closest articular surface. Rotational correction is usually performed acutely at surgery. *Close the incision. Ensure that the hinges and motor are appropriately placed, and secure all nuts in the frame.*

After an appropriate latency period, initiate the distraction with the motor (see Box 32.10). The rhythm of distraction is two to four times per day. The rate of distraction will vary depending on the animal and on radiographic evaluation of the regenerate bone. Because the motor is offset from the deformity, calculations must be performed to ensure appropriate distraction at the motor to achieve 1 mm per day at the osteotomy site.

Postoperative Care

Postoperative analgesia should be provided (see Boxes 32.1 and 32.2 and Table 32.4). Immediately after surgery the wire-skin interface should be cleaned with antiseptic solution using cotton swabs. Sterile gauze sponges should be opened, crumpled, and packed around and between tensioned wires and the limb wrapped with Vetrap or a similar bandage material. Holes are cut in the bandage to allow access to the nuts involved in distraction. After surgery the wire-skin interface should be cleaned and the bandage changed daily or every 2 days. After approximately 1 week, gauze packing can be discontinued. The frame may still be protected

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with an external wrap. The wire-skin interface should be cleaned daily. Activity should be restricted to leash walking and physical rehabilitation until the fracture has healed. Physical rehabilitation encourages controlled limb use and optimal limb function after fracture healing.

If frame distraction is occurring, explicit instructions must be given to the owners to ensure proper rate and direction of distraction. After release from the hospital, the animal should be reevaluated weekly for the first 2 to 3 weeks postoperatively or during the distraction phase. The wire-skin interface should be carefully examined and the ring and skin observed for separation. If irritation or drainage is present, the involved skin surface should be cleaned and packed with gauze. Radiographs are made every 7 to 10 days during distraction and appropriate adjustments made to the rate of distraction. When the distraction is complete, the hinges are replaced with threaded rods, and the frame is stabilized by tightening all nuts. The fixation remains in place until radiographic evidence of bone consolidation. Occasionally, distraction will cause musculotendinous contracture, leading to flexural deformities or joint stiffness. Physical therapy and limb use are keys to preventing this complication. The circular fixator is removed once the bone has healed. The appropriate time to take postoperative radiographs for healing depends on the estimated time to bone union and the patient fracture assessment. The patient should be sedated and the wires cut close to the bolts and removed with a hand chuck or vise grip. Smooth wires can be removed from either direction, whereas olive wires must be removed from the side of the olive.

HYBRID CIRCULAR EXTERNAL FIXATORS

Indications and Biomechanical Principles

AP Hybrid circular external fixators are a combination of a ring fixator and a linear fixator. Hybrid fixators are indicated for fractures with a short juxtaarticular bone segment. Generally, the frame consists of a ring with wires or fixation pins securing the short segment of bone, and fixation pins and a connecting rod securing the long segment of bone. The connecting rod articulates with the ring to stabilize the fracture. Hybrid fixators may be applied to the radius, tibia, femur, and humerus. Generally, types Ia and Ib hybrid fixators are applied to the radius and tibia. Modified frames that employ fixation pins through the ring and may tie into an IM pin are applied to the humerus and femur. Hybrid fixators can also be used to stabilize corrective osteotomies for some angular limb deformities.

The mechanical properties of the fixation frames are similar to those described for the circular and linear fixation systems.

Equipment and Supplies

See the sections on linear external fixators and circular external fixators for information on equipment and supplies. Additional frame components unique to the hybrid fixator frame are connecting bars that are threaded on one end (hybrid rods). These rods are inserted into the ring and secured with paired nuts. Spherical nuts and washers are also available to provide some angular adjustment between the rod and the ring.

Application

Type Ia Hybrid Fixators

Begin by placing the appropriately sized ring, with a connecting bar attached, around the juxtaarticular (short) bone segment. Hold the frame in an approximate position, and select an appropriately sized wire to drive through the metaphysis of the bone, parallel to the adjacent joint surface. Secure the wire (see Box 32.9). Add the appropriate number of pin-gripping clamps to the connecting rod. Reduce the fracture and secure the proximal segment with a fixation pin inserted appropriately (see p. 1000). Check alignment and secure the frame by clamping the fixation pin with the proximal pin-gripping clamp. Check alignment then add the second wire to the ring and, if room permits, add a third wire (drop wire) secured to posts connected to the ring. Tension and secure the wires. Place additional half pins directly through the pin grippers to achieve three to four pins in the long bone segment (Fig. 32.33A). Once all the intermediate pins have been placed, tighten the pin grippers, and make a radiographic examination of the limb to assess fracture reduction and pin and wire placement. Trim the fixation pins and wires to prevent injury to the owners and immediate environment.

Type Ib Hybrid Fixators

Apply a type Ia hybrid frame to the bone. Then secure another connecting bar to the ring at approximately 90 degrees to the first frame. Fill out the second connecting bar with fixation pins securing the long segment of bone. Once all the intermediate pins have been placed, tighten the pin grippers, and make a radiographic examination of the limb to assess fracture reduction and pin and wire placement (Fig. 32.33B). Trim the fixation pins and wires to prevent injury to the owners and immediate environment.

IM Pin Tie-in Hybrid Fixators

Use an IM pin to reduce a comminuted fracture or as part of the reconstruction of a single fracture. Secure an incomplete ring to the juxtaarticular bone segment with either olive wires or fixation pins (see Box 32.9). Secure one hybrid connecting rod in one of the lateral holes of the ring with paired hemispherical washers and nuts. Place the appropriate number of half pins to secure the longer bone segment to the hybrid connecting rod. Secure a second hybrid connecting rod to one of the craniomedial holes of the ring with articulated posts or a hinge assembly, and then secure the other end of the rod to the proximal portion of the first hybrid rod or to a cranially positioned half pin placed in the proximal fracture segment. Secure the protruding end of the IM pin to the hybrid connecting rod with a pin-gripping clamp (Fig. 32.33C). Tighten the pin grippers, and make a radiographic examination of the limb to assess fracture reduction and pin and wire placement. Trim the fixation pins and wires to prevent injury to the owners and immediate environment.

Postoperative Care

Postoperative care, including destabilization, is similar to that provided for linear external fixators (see p. 1003).

INTRAMEDULLARY FIXATION

INTRAMEDULLARY PINS AND KIRSCHNER WIRES

Indications and Biomechanical Principles

IM pins are used for diaphyseal fractures of the humerus, femur, tibia, ulna, and metacarpal and metatarsal bones. IM pins are contraindicated for the radius because the insertion point of the pin generally interferes with the carpus. The biomechanical advantage of IM pins is their resistance to applied bending loads (Fig. 32.34A). In contrast to other implants (e.g., bone plates and external fixators), IM pins are equally resistant to bending

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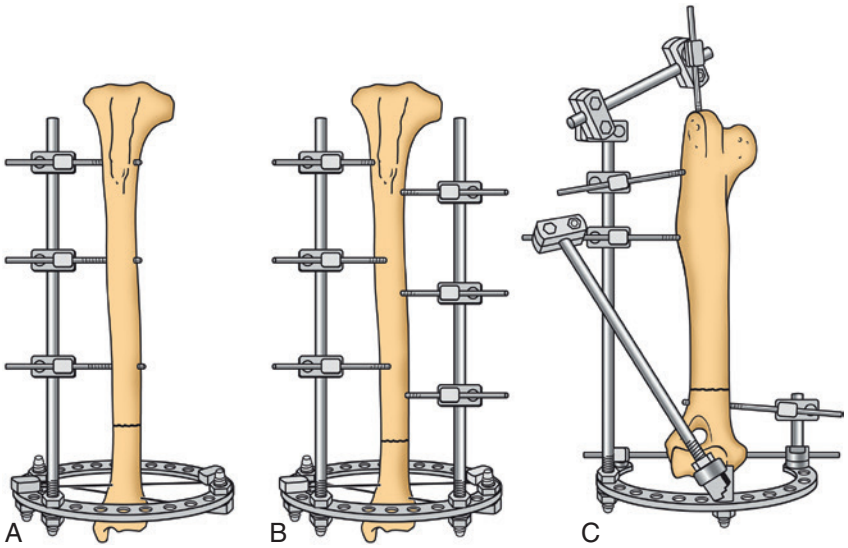


FIG. 32.33 Hybrid circular external fixators are a combination of a ring fixator and a linear fixator. (A) A hybrid Ia frame consists of a ring and a uniplanar connecting bar and fixation pins. (B) A hybrid Ib frame consists of a ring and two uniplanar connecting bars and pins placed at approximately 90 degrees to each other. (C) An intramedullary (IM) pin tie-in hybrid frame consists of a ring and modified Ib frames with an IM pin connected to the frame. The pin may be bent to secure it to the frame. The pin may also be bent before securing it to the frame, eliminating the proximal connecting bar.

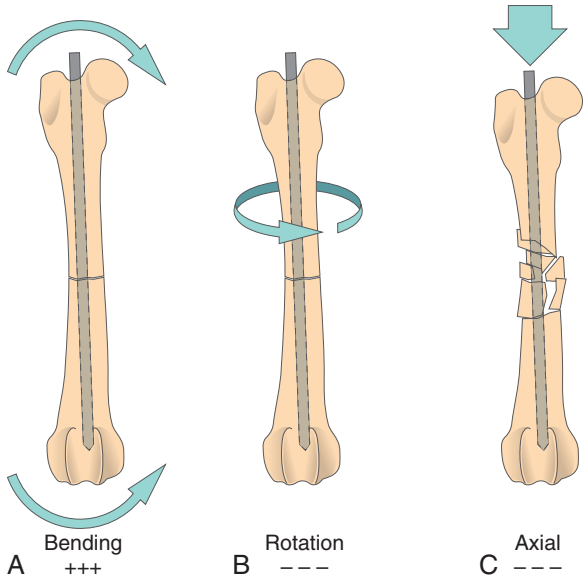


FIG. 32.34 (A) A biomechanical advantage of intramedullary (IM) pins is that they are equally resistant to bending loads applied from any direction because they are round. (B–C) Biomechanical disadvantages of IM pins include poor resistance to rotational or axial (compressive) loads and lack of fixation (interlocking) with bone.

loads applied from any direction because they are round and generally centered in the medullary canal. Biomechanical disadvantages of IM pins include poor resistance to axial (compressive) or rotational loads and lack of fixation (interlocking) with bone (see Fig. 32.34B–C). The only resistance to rotation or axial loads provided by an IM pin is friction generated where pin and bone contact. Contact between pin and cortical bone is variable because the cross-sectional marrow cavity diameter varies. Contact between pin and epiphyseal cancellous bone varies with the amount of cancellous bone and accuracy of pin placement. Because friction created between pin and bone also prevents premature pin migration, stress associated with unstable fractures results in micromotion at the pin-bone interface, bone resorption, and pin migration. Because of these limitations in mechanical support afforded by either single-pin or stacked pin stabilization, IM pins should be supplemented with other implants (e.g., cerclage wire, external fixator, or plate) to provide rotational and axial support.

Steinmann pins or Kirschner wires may be used as crossed pins (wires) or placed in a triangulated pattern to stabilize metaphyseal and physeal fractures in young animals (see Chapter 33). Kirschner wires are also used as IM pins in very small animals.

Equipment and Supplies

IM pins are smooth, round, 316L stainless steel rods. The most common IM pins used in veterinary medicine are *Steinmann pins,* which are available in sizes ranging from 1/16 to 1/4 inch (1.6–6.3 mm) and come with a variety of point designs (Fig. 32.35). They can be *single armed* (one end with a point and one end blunt) or *double armed* (a point at each end). The most popular point designs are trocar points and chisel points. *Chisel points* have a two-sided cutting edge that is slightly more effective in cutting through dense cortical bone because it generates less heat than trocar points. *Trocar points* have a triple cutting edge and cut through cancellous bone easily. Because Steinmann pins are generally used as IM rods and are seated in proximal or distal epiphyseal cancellous bone, trocar points are most often used.

IM pins may be smooth or may have threads near the end of the pin. The end-threaded Steinmann pin was developed to increase the pin's holding power in cancellous bone. However,

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FIG. 32.35 Intramedullary pins with chisel *(left)* and trocar *(right)* points.

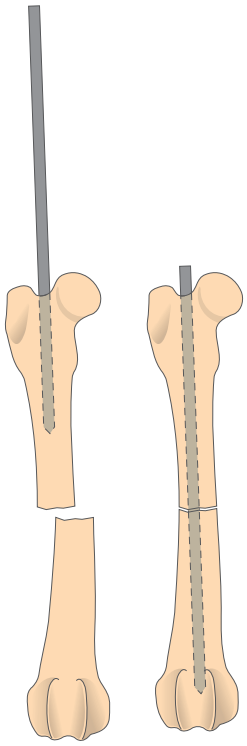


FIG. 32.36 For normograde placement of an intramedullary pin in the femur, insert the pin so that it enters the bone proximally in the craniolateral trochanteric fossa. Direct it caudally to glide along the caudal cortex and seat it in the caudocentral aspect of the condyle.

use of end-threaded pins is controversial. Threaded Steinmann pins have increased potential for premature failure compared with smooth Steinmann pins. In the manufacturing of the end-threaded Steinmann pin, threads are cut into the tip of a smooth Steinmann pin, causing the pin diameter to be greater in the nonthreaded section of the pin than in the threaded section. This difference in cross-sectional area acts as a stress concentrator that predisposes to premature pin bending or breakage. Tapering the junction between the threaded and nonthreaded portion of the pin significantly increases the strength of this juncture.

Kirschner wires are small in diameter, ranging from 0.035-inch (0.9 mm) to 0.062-inch (1.6 mm) smooth pins, which generally have trocar points on each end.

Steinmann pins used as IM pins are generally driven with a Jacobs chuck or power drill. Steinmann pins and Kirschner wires used as crossed pins may be driven with a high-speed wire driver. An appropriately sized pin cutter or wire cutter is also required.

Application

Select an IM pin that equals 60% to 70% of the diameter of the marrow cavity at the isthmus of the bone when pairing the IM pin with cerclage wire. Choose a smaller diameter pin when pairing the pin with an external fixator or bone plate (Box 32.11). Although IM pins function most effectively if they fill the medullary canal, the curvature of most canine bones dictates that a smaller pin must be used if the fracture is to be reconstructed anatomically. Pin size determination is made after evaluating radiographs of the fracture. Appropriate size may be confirmed by comparing the pin with the medullary canal after the fracture is exposed. *Select two pins of the same length before starting the procedure.* The second pin serves as a guide pin when laid over the external surface of the limb. The guide pin is aligned with the bone so the operator can aim the IM pin in a similar direction.

An IM pin may be inserted *normograde* or *retrograde* for placement in the femur or humerus. IM pins must be inserted normograde in the canine tibia to prevent damage to the stifle. *To place a pin in a normograde fashion, start the pin at the appropriate location at one end of the long bone and drive it down the medullary canal to the fracture, reduce the fracture, and continue to drive the pin until it seats in metaphyseal bone (Fig. 32.36).* The advantages of normograde placement are that the pin can be placed more precisely and there is less manipulation of the fracture area. The disadvantage of normograde placement is that it may be difficult to identify the correct entry point into the bone if the insertion of the pin is done blindly. *To place a pin in a retrograde fashion, first expose the fracture and insert the pin into the medullary canal of the appropriate bone segment. Drive the pin to exit the bone segment. Replace the pin chuck on the exited portion of the pin, and withdraw it until the pin point is*

BOX 32.11 Key Concepts for Applying Intramedullary Pins

- Select a pin sized 60%–70% of the medullary canal width to pair with cerclage wire.
- Select a pin sized 50%–60% of the medullary canal width to pair with an external fixator.
- Select a pin sized 40%–50% of the medullary canal width to pair with a plate.
- Span the length of the bone with the intramedullary pin.
- Use retrograde or normograde pin insertion in the humerus and femur.
- Use normograde pin insertion in the tibia.
- Check pin location with reference pin and by manipulating the joint.
- Always use additional fixation to control rotation and axial loading.

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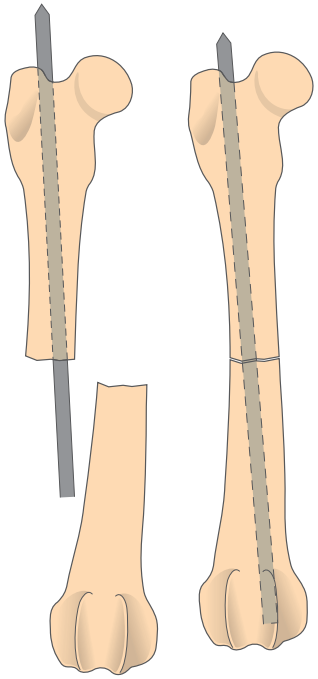


FIG. 32.37 For retrograde placement of an intramedullary pin in the femur, insert the pin into the marrow cavity at the fracture surface. Force the shaft of the pin against the caudomedial cortex and drive the pin proximally. Reduce the fracture and drive the pin distally to seat in the caudocentral aspect of the femoral condyle.

within the medullary canal. Reduce the fracture and drive the pin to seat in metaphyseal bone (Fig. 32.37). Check the pin insertion length by using the second pin as a reference. Approximate the position of the proximal tips of both pins, and lay the reference pin over soft tissue outside the limb to determine the position of the distal tip of the IM pin. Check for pin interference in the distal joint by flexing and extending the joint. Crepitation or decreased range of motion may indicate pin interference. Radiographs will confirm the presence of a pin in the joint. *If necessary, retract the pin so it no longer interferes with the joint. Check the fracture for anatomic reduction or correct bone alignment and apply either cerclage wire (long oblique fractures in animals with high fracture assessment scores), external fixators (frame construction depends on fracture assessment score), or a bridging plate (animals with comminuted fractures and low fracture assessment scores) (see Box 32.11, see also p. 1010). When satisfied with the pin position, cut the excess pin by pushing down on the soft tissue and cutting the pin below the skin level with a pin cutter. Suture the soft tissue over the pin.*

To apply Steinmann pins or Kirschner wires as crossed pins to secure a physeal or metaphyseal fracture, reduce the fracture and insert the pins on the lateral and medial surfaces on the epiphyseal segment. Drive the lateral pin across the fracture to exit on the medial surface of the metaphyseal segment. Drive the medial pin across the fracture to exit on the lateral surface of the metaphyseal segment (Fig. 32.38). The pins should exit sufficiently that the trocar tips are clear of the cortices, but they should not be so long as to interfere with soft tissue. *Take care that the pins cross within the metaphyseal segment and not at the fracture. Check the fracture stability and pin position. With small diameter pins, bend the pin end and cut off the excess. With larger diameter pins, which resist bending, simply cut the excess pin. The cut ends may be seated deeper in the bone with a mallet and pin set or fully closed Jacobs chuck.*

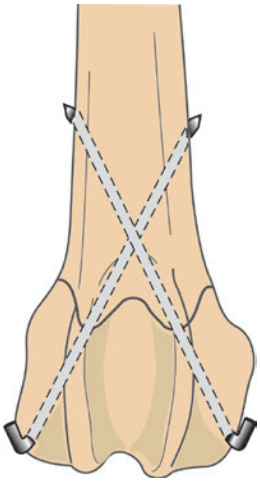


FIG. 32.38 Steinmann pins and Kirschner wires may be applied as crossed pins to secure juxtaarticular segments in animals with high fracture assessment scores. (Modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat*. St. Louis: Elsevier; 2005.)

Postoperative Care

Postoperative analgesia should be provided (see Boxes 32.1 and 32.2 and Table 32.4). Postoperative radiographs are made to evaluate implant location. Activity should be restricted to leash walking and physical rehabilitation until the fracture has healed. Physical rehabilitation encourages controlled limb use and optimal limb function after fracture healing (see Table 32.7 and Chapter 11). Radiographs should generally be repeated 6 to 8 weeks postoperatively. The functional period of a Steinmann pin or Kirschner wire is short because of its reliance on frictional hold. IM pins supported with an external fixator or a plate have a longer functional period. Clients may become concerned about subcutaneous fluid swelling surrounding the pin end: this is a seroma caused by irritation of the pin moving in soft tissue. IM pins may be removed once bone union has occurred; if a seroma is present, it will resolve after pin removal. If the pin is easily palpable, then it may be removed with sedation and local anesthetic. If the pin is buried within soft tissue, then the patient must be anesthetized and taken to the operating room. Current radiographs are necessary for the surgeon to pinpoint the exact location of the pin. Kirschner wires and crossed pins are generally not removed unless they cause a problem. *To remove a pin, sedate or anesthetize the patient and clip and aseptically prepare the skin surface overlying the end of the pin. Instill a local anesthetic (if the patient is sedated) and make a small skin incision over the palpable end of the pin. Bluntly dissect soft tissue from the pin, grasp it with a pin remover, and extract it from the bone. Place a suture(s) in the skin wound.*

INTERLOCKING NAILS

Indications and Biomechanical Principles

Interlocking nails are inserted in the medullary canal and locked in place with screws or cross-locking bolts placed through the

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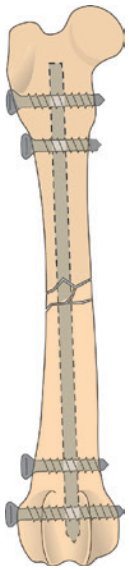


FIG. 32.39 Interlocking nail. The interlocking screws provide additional rotational and axial support.

proximal and distal fracture segments and the nail (Fig. 32.39). Interlocking nails resist all the forces acting on fractures. The nail provides bending support, whereas the locking bone screws or bolts provide axial and rotational support. Interlocking nails may be used effectively in animals with high and medium fracture assessment scores.

The interlocking nail is used primarily for middiaphyseal humeral, femoral, or tibial fractures; it is contraindicated for the radius. The nails come in various sizes and lengths and have holes in each end for screw insertion. The nails are indicated for both single and comminuted middiaphyseal fractures. Ideally, intact cortical bone should be sufficient proximal and distal to the fracture to allow insertion of two screws in each segment, although this is not always possible in the humerus. Screws should be placed at least 2 cm from the fracture line to prevent excessive forces on the nail. Interlocking nails are weakest at the screw hole. Fatigue failure at the screw hole occurs when an inadequate-sized nail is used or when the nail hole is adjacent to the fracture line. Screws placed in the nail hole do not decrease the likelihood of nail failure if the nail is inappropriately placed. As stronger nails were developed with smaller nail holes to protect the integrity of the nail, the subsequently smaller screws occasionally failed. Solid cross-locking bolts may be used to minimize the occurrence of locking screw failure.

Equipment and Supplies

Interlocking nails available for veterinary use are 6, 7, and 8 mm in diameter, with several lengths available in each size. Each nail has three or four screw holes (up to two in each end). The distal end may be a trocar or blunt point, and the proximal end has an internally threaded hole and two alignment tabs, to which an extension piece can be attached. An insertion tool is attached to the extension piece to insert the nail in a normograde direction. A drill-guide jig is then attached to the extension piece and used to align the drill bit with the holes in the nail (Fig. 32.40). The appropriate-sized cortex bone screws or locking bolts are needed for each size nail. The cross-locking bolts are solid bolts with self-tapping positive profile threads below the head of the bolt.

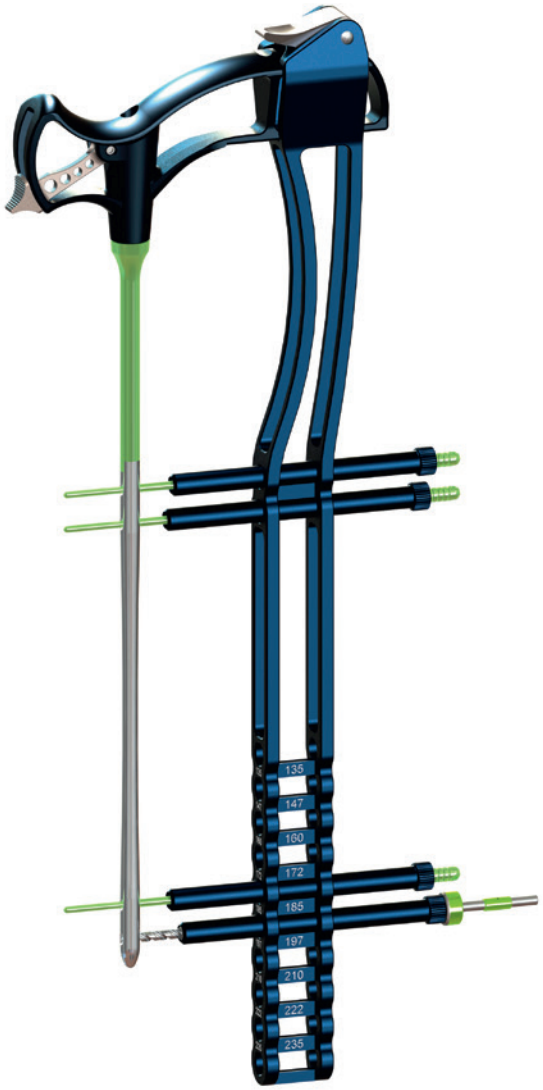


FIG. 32.40 Equipment used for interlocking nail fixation, including jig, nail, drill bit in the distal hole, and alignment posts in the proximal three holes. (Courtesy BioMedtrix.)

The solid intersection provides greater resistance to failure. The bolts are cut to an appropriate length before application.

Application

Select the largest nail that will fit into the bone. Be sure the nail is long enough to span the normal length of the fractured bone. Prepare the marrow cavity for nail placement by inserting a series of progressively larger Steinmann pins or a hand reamer in either a retrograde or normograde fashion, removing mainly medullary contents and metaphyseal cancellous bone. Attach the appropriate extension and then the insertion tool to insert the appropriate length nail to a position just below the bone or joint surface. Remove the insertion tool, be sure the extension is firmly attached to the nail, and then secure the drill-guide jig to the extension. The guide system is mandatory to ensure that the drilled hole intersects the bone at a site where a hole exists in the nail (Fig. 32.41). Typically the distal-most hole is drilled first as it may be the

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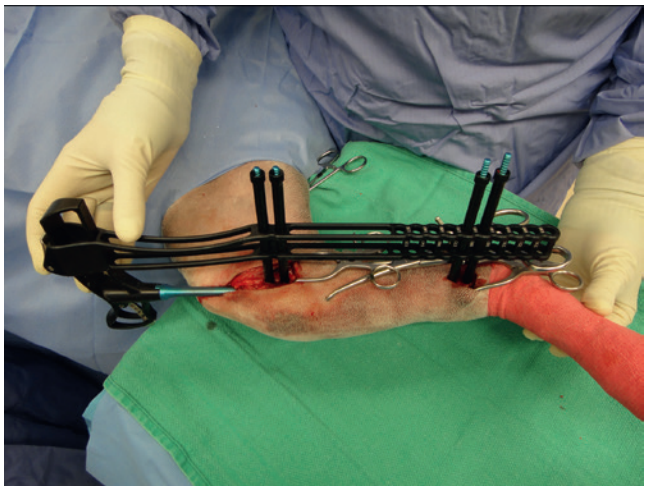


FIG. 32.41 Intraoperative image of an interlocking nail on the tibia of a dog. (Courtesy BioMedtrix.)

BOX 32.12 Key Concepts for Applying Interlocking Nails

- Use the largest nail that fits in the bone.
- Span the length of the bone with the nail.
- Ream the medullary canal with a Steinmann pin or use the reamers.
- Insert the nail in a normograde fashion.
- Position the nail so the holes are 2 cm away from the fracture.
- Secure the nail with four screws or fixation bolts for optimal fixation.

most difficult to locate. *Drill a hole in the distal fragment through the near cortex and the nail hole and exiting the far cortex. Measure and tap the hole and insert the appropriate length bone screw to interlock the nail with the cortices. Rotate the distal segment and check to see if the pin is also rotating. Check rotational alignment and axial length of the bone before placing the proximal screws and the remaining distal screw (Box 32.12).*

When using locking bolts, drill a hole equal to the shaft diameter of the bolt. Measure to determine the bolt length necessary. Cut the bolt approximately 2 mm longer than the measured distance. Gently advance the bolt so the self-tapping threads engage the near cortex. See Chapter 33 for interlocking nail application to specific bones.

Postoperative Care

Postoperative analgesia should be provided (see Boxes 32.1 and 32.2 and Table 32.4). Activity should be restricted to leash walking and physical rehabilitation until the fracture has healed. Physical rehabilitation encourages controlled limb use and optimal limb function after fracture healing (see Table 32.7 and Chapter 11). Generally, postoperative examinations should be done at 2 and 6 weeks after surgery and then every 6 weeks. The functional period of an interlocking nail is longer than that of a Steinmann pin or Kirschner wire because of the interlocking hold of the screws or bolts with the bone. Most interlocking nails are left in place after bone healing. The recessed end of the nail does not generally impinge on soft tissue. If a nail is removed, the patient must be placed under general anesthetic. *Clip and aseptically prepare the skin surface and make small skin incisions over the interlocking screws. Remove the screws and make an incision over the end of the nail. Apply the distraction device and remove the nail.*

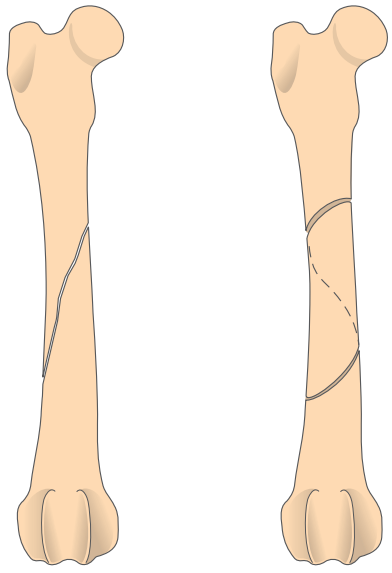


FIG. 32.42 Fracture configurations in which cerclage wire is useful for providing mechanical support through interfragmentary compression.

ORTHOPEDIC WIRE

Cerclage and Hemicerclage Wire

Orthopedic wire is often used as cerclage wire or hemicerclage wire. It is used in combination with other orthopedic implants to supplement axial, rotational, and bending support of fractures. The term *cerclage wire* is used to denote the use of orthopedic wire placed around the circumference of the bone. *Hemicerclage wire* or *interfragmentary wire* is the term used to denote wire that is placed through predrilled holes in the bone. Cerclage wire can be combined with Kirschner wires to prevent wire slipping in areas where the bone diameter changes or to secure cerclage wires at an oblique angle to the long axis of the bone.

Indications and Biomechanical Principles

Cerclage wire is used either to provide stability to anatomically reconstructed long oblique or spiral fractures (Fig. 32.42) or to hold multiple fragments in position. To function as a stabilizer, wire must generate sufficient compression between fracture surfaces to prevent the fragments from moving or collapsing under weight-bearing loads. To accomplish this, two criteria must be met: (1) the length of the fracture line should be a minimum of two times the diameter of the diaphysis at the level of the fracture line, and (2) the fracture must be anatomically reduced. When these criteria are met, cerclage wire can provide additional stability by generating sufficient compression between fragments to hold them in place during healing (Fig. 32.43). Cerclage wire is always supported by additional implants (e.g., IM pins, external fixators, or plates) that control the large weight-bearing (primarily bending) forces (Box 32.13).

If more than two or three bone segments are present or if the fracture lines are not of sufficient length, cerclage wire can

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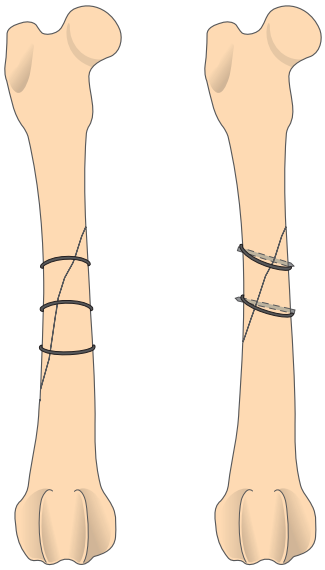


FIG. 32.43 Proper placement of cerclage wire. Wires are placed at least 5 mm from fracture ends and spaced one-half to one times the diameter of the bone. Kirschner wires may be used to prevent cerclage wire slippage. (Modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat.* St. Louis: Elsevier; 2005.)

BOX 32.13 Key Concepts for Applying Cerclage Wire

- Use only on anatomically reconstructed long oblique or spiral fractures.
- Use 18-gauge wire for large dogs and 22- or 20-gauge wire for cats and small dogs.
- Place two to three cerclage wires per fracture line.
- Place wires perpendicular to the long axis of the bone.
- Space wires one-half to one bone diameter apart.
- Support cerclage wires with an intramedullary pin, interlocking nail, external fixator, or plate.

only be used to hold fragments in position; it cannot generate the compression needed to resist weight-bearing loads. The attempt to gain stability with cerclage wire in multifragmented fractures is the most common cause of cerclage wire failure. When multiple fragments are present, movement postoperatively may cause one of the pieces to shift in position, which would allow the entire segment of reconstructed bone to collapse. An analogy to the use of cerclage wire for stabilizing multiple bone fragments is the use of metal rings to hold slats in a wooden barrel. Collapse would occur if one slat (bone fragment) in the barrel were to loosen. When wire is misused, probable outcomes include collapse of the fracture, loss of stability, and wire loosening, which further delay healing.

There is some variation in mechanical properties depending on the method of tightening (loop versus twist) and the wire pattern (single-wrap versus double-wrap cerclage wires). In general, twist tightening generates less tension in the wire than loop tightening, and double-wrap patterns generate greater compression of the fragments with increased resistance to the load of weight bearing. Clinical performance of the two types of wire tightening and the single-wrap and double-wrap patterns appears similar as long as principles of wire application are followed.

Cerclage wire fixation is not recommended for stabilizing short oblique (45 degrees or less) or transverse fracture lines. Use of a Kirschner wire crossing the fracture line, with a cerclage wire looped around the diaphysis of the bone and resting above and below the Kirschner wire, has been described for short oblique fractures, but it is inferior to lag screw fixation.

Equipment and Supplies

Cerclage wire is made from a malleable form of 316L stainless steel. It may be purchased in a spool or as preformed loop wire and is available in sizes ranging from 22 gauge (0.64 mm) to 18 gauge (1 mm). The use of 22- or 20-gauge wire is recommended for cats and small dogs, whereas 18-gauge wire is recommended for larger dogs. Hemicerclage wire should be 18- or 20-gauge 316L stainless steel. A variety of instruments are available to secure the encircling wire to bone. Twist knots may be formed by using pliers or old needle holders. Cerclage wire with a preformed loop at one end is secured with a specially designed wire tightener.

Application

Cerclage Wire

Use a wire passer to place the wire around the bone without extensively reflecting soft tissue. Do not entrap tissue between the wire and periosteum. To prevent slippage and loosening of the wire, place the wires perpendicular to the bone surface. To tighten a wire with a twist knot, pull the wire tight to the bone and form the first three twists by hand. Grasp the twists with the pliers and pull the wire perpendicular to the long axis of the bone. Twist the wire while maintaining the tension. Once the wire is tight, either cut the wire near the third twist and leave it extended, or cut the wire near the fifth or sixth twist and bend it over in the direction of the twist (Fig. 32.44). A fibrous cap will rapidly form over the protruding end of the wire, providing protection from soft tissue irritation. *To tighten a cerclage wire with a preformed loop, pass the free end of the wire around the bone, through the loop, into the nose of the wire tightener, and through the hole in the crank. Turn the crank until the wire is secure. Bend the wire tightener over so the free end of the wire folds on itself. Reverse the crank to expose more of the wire and complete the fold. Cut the excess wire, leaving approximately 0.5 to 1 cm in length, and push the wire end to the bone (Fig. 32.45).* The instrument used for tightening the wire is not critical; however, the wire must be tight after securing the knot. *Ascertain that the wire is perpendicular to the long axis of the bone. Ascertain the degree of tightness by attempting to move the wire with a pair of needle holders. Replace the wire if it is loose. When all the wires have been placed, recheck the tightness of each wire because loosening of the initial wires may occur with subsequent wire placement.* Cerclage wire used to stabilize shorter oblique fractures or in metaphyseal fractures must be perpendicular to the fracture to achieve compression, but the wire placed in this manner will slip. *To prevent slipping, place a Kirschner wire across the fracture and leave the ends of the Kirschner wire protruding 1 mm beyond the bone surface at both the near and far cortex. Place the cerclage wire around the bone such that the loop rests above the Kirschner pin at the far cortex and below the Kirschner pin at the near cortex (see Fig. 32.43).* For a description of placing interfragmentary wires, see application of wire to mandibular fractures on p. 1038.

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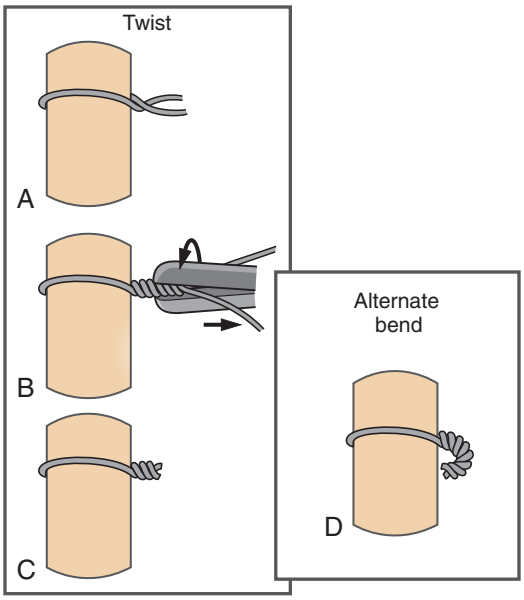


FIG. 32.44 (A) For plain orthopedic wire, begin twisting the wire ends by hand. (B) Place the wire twisting pliers or needle holders onto the twist and tighten the wire by pulling and twisting. (C) When the wire is tight, cut the wire 3 mm from the start of the twist. (D) Alternatively, cut the wire 5 to 7 mm from the twist and bend it over in the direction of the twist.

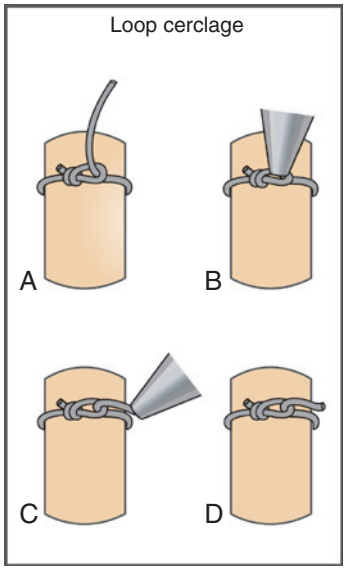


FIG. 32.45 (A) For loop cerclage, tighten the wire by passing the plain end of the wire through the eye of the wire. (B) Insert the wire into the wire tightener and crank the wire tight. (C) Bend the wire over the eye. Retract the wire tightener and finish bending the wire. (D) Cut the wire. (Modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat*. St. Louis: Elsevier; 2005.)

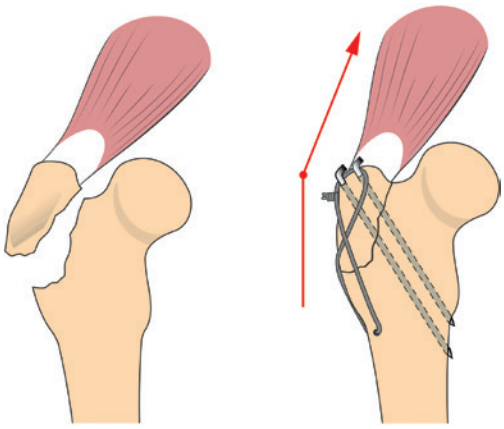


FIG. 32.46 Placement and mechanical principle of a tension band. Tightening the wire exerts a force that counters the force of muscle contraction and compresses the fracture surface.

Postoperative Care

Postoperative analgesia should be provided (see Boxes 32.1 and 32.2 and Table 32.4). Wire used for fractures does not require special considerations postoperatively. Wire is generally not removed once the fracture has healed, unless it causes problems.

TENSION BANDS

Indications and Biomechanical Principles

Tension is the predominant force when avulsion fractures occur at a point where groups of muscles originate or insert in bone (e.g., greater trochanter, olecranon, and supraglenoid tuberosity of scapulae). Contraction of the muscle group at these sites generates tension that pulls the bony insertion or origin from its anatomic location. The most effective way to resist tension is through application of a tension band (Fig. 32.46). The purpose of a tension band is to convert distractive tensile forces into compressive forces.

Equipment and Supplies

Equipment needed for placement of a tension band includes small Steinmann pins or Kirschner wires and orthopedic wire. A high-speed wire driver is preferred for placing the Kirschner wires. The orthopedic wire is secured with a wire tightener (see the previous discussion).

Application

When using pins and wire to apply a tension band, reduce the fracture first, then place two small pins or Kirschner wires across the fracture to maintain reduction. Place the pins perpendicular to the fracture line and parallel to each other. Try to seat the pins in the opposite cortex. Drill a small hole through the bone below the fracture at approximately the same distance present between the fracture and the insertion point of the pins. Locate the hole so the wire will rest on the bone's tension surface when tightened. In the femur, for example, drill the hole from cranial to caudal so that the wire will rest on the lateral, or tension, surface of the femur. Pass the wire through the drill hole and around the two small pins used to stabilize the fracture. Twist the ends to form a figure eight. The twist portion of the wire should be on top of the flat portion of the wire. Wind the twist knot to tighten the arms

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BOX 32.14 Key Concepts for Applying Tension Band Wires

- Use two Kirschner wires or small Steinmann pins.
- Place wires parallel to each other and perpendicular to the fracture.
- Seat the wires in the opposite cortex.
- Place the hole for the orthopedic wire the same distance below the fracture as the pins are inserted above the fracture.
- Tighten the wire so it is in direct contact with the bone.

of the tension band. As the wire is tightened, tension is created that opposes that generated by muscle contraction. Bend the exposed ends of the Kirschner wires 90 degrees, and turn the ends into the tendon (Box 32.14).

Postoperative Care

Postoperative analgesia should be provided (see Boxes 32.1 and 32.2 and Table 32.4). Special postoperative care is not required for tension bands. If the ends of the pins irritate soft tissue, the pins should be removed. Otherwise, tension bands are not removed once the fracture has healed.

PLATE AND SCREW FIXATION

BONE PLATES AND SCREWS

Stabilization of fractures with bone plates and screws is a popular method of fracture fixation. Modern plating technology began in the early 1960s when a group of Swiss surgeons formed an association for the study of fracture treatment in humans. This group, the Swiss Arbeitsgemeinschaft fur Osteosynthesefragen (AO), is referred to as the Association for the Study of Internal Fixation (ASIF) in the United States. The AO and/or ASIF developed and continues to develop recommendations for application of orthopedic devices, which have led to increased success and fewer complications associated with fracture management. In the 1970s, an arm of the AO group, the AO-Vet, was established to document and address problems specifically associated with fracture management in animals. From this group's work, specialized instrumentation and bone plates were designed to treat animal injuries. To achieve consistent and predictable results with bone plating, a thorough understanding of the principles and techniques of application is essential. Although numerous companies are marketing varied designs of equipment, the AO-ASIF principles apply to all of these systems and are used to describe the principles of application in this text.

Indications and Biomechanical Principles

Bone plates and screws offer a versatile method of fracture stabilization and can stabilize any long bone fracture. They are often used for fractures of the axial skeleton and are imperative for fractures involving joint surfaces. Bone plates and screws are particularly useful when postoperative comfort and early limb use are the highest priority (e.g., fractures involving joint surfaces, geriatric patients, revision of nonunions, patients with fracture disease). Bone plates and screws are used to treat animals with high, medium, and low fracture assessment scores, but they are particularly useful for animals with low fracture assessment scores.

Screws used as lag screws cause compression at the fracture, increasing the friction between fragments and resisting the forces acting on the fracture. Two or more screws must be used to counteract bending forces in the diaphysis, but they are not sufficient to withstand the large loads generated by weight bearing without plate support. Screws may be used to reconstruct articular fractures without plate support in some cases. Screw resistance to bending load is determined by core diameter and increases by raising the radius to the fourth power. Screw-holding power (resistance to pull-out) increases in a linear relationship with increasing diameter of the threads.

Nonlocking bone plates achieve fixation of the fracture by friction generated by the application of a well-contoured plate to the bone surface with screws. When applied properly, bone plates effectively resist the axial loading, bending, and torsional forces acting on fractured bones. There is some concern that the force between plate and bone prevents periosteal perfusion, although the clinical significance of this is likely minimal when the bone plate is properly applied. Plates have been developed to minimize interference with blood supply. The surface on which the plate is located influences the degree of stability obtained. In general, all long bones are subject to bending forces because physiologic loads are applied eccentrically to the bone center. When a bone is subject to this type of loading, a bend will occur that causes compression on the concave surface of the bone and tension on the convex surface. This tension must be prevented because it will cause a fracture line to pull apart. This is accomplished by laying the plate on the tension surface and thereby allowing the plate to absorb the tensile stress that would separate the fracture. In addition, bone plates are stronger in tension than compression. Plates are susceptible to repeated bending stresses because of the plate's eccentric location in relationship to the long axis of the bone. Implant fatigue failure occurs when the opposite cortex is not reconstructed and fails to bridge with bone early enough to protect the plate. Plate holes concentrate stress, and failure generally occurs in this area. Increasing the plate size, using a broad lengthening plate, or using a plate-pin combination may strengthen the implant or reduce the strain sufficiently to reduce risk of fatigue failure.

Locking screw plate systems act as internal fixators and have mechanical and application advantages over conventional plate systems. The screw head locks into the plate hole, allowing the plate and screws to act mechanically as a single unit. Additionally, the locking screw is not creating undue stress in the bone because it is in a neutral position. The result is an increase of the construct yield strength with the locking screws. Because the locking mechanism between the plate and the screw provides the fracture fixation, precise contouring of the plate is unnecessary. The disadvantage is that most locked screws must be placed perpendicular to the plate to lock. Some bone plates are specifically designed with locking holes oblique to the length of the plate (tibial plateau leveling osteotomy [TPLO] plates, arthrodesis plates) to decrease the risk of a screw entering the adjacent joint. Locking plates enable the use of either locking or nonlocking screws. In most cases, a combination of both screw types is used. The addition of a single locking screw in each segment of a two piece fracture will roughly double the stiffness of the construct when compared with a standard plate. The addition of a second locking screw further increases stiffness of the construct to a lesser degree, and the addition of more screws does not significantly increase stiffness. Locking screws also have the advantage of a very low risk of screw back-out and may be applied in certain circumstances for this reason alone. Locking plates can be effectively used with indirect reduction and minimally invasive surgical techniques. Minimally invasive plate osteosynthesis,

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where a plate is inserted percutaneously and secured to the bone above and below the fracture, minimizes biological damage.

Equipment and Supplies

Bone plating sets are available that contain the instrumentation necessary to apply the implants. Drill guides and sleeves, taps, depth gauge, and screwdrivers are used to insert screws. Bending and torquing irons are used to contour or shape the plates.

Screws

Cortical and cancellous bone screws are made of 316L stainless steel or titanium and may or may not be self-tapping. A non–self-tapping screw requires that threads be cut into bone with a tap; a self-tapping screw has a cutting tip to cut threads into bone and flutes to accept bone debris. Cortical screws are fully threaded and designed for use in compact cortical bone. The pitch (number of threads per inch) of the screw is greater than that of a cancellous screw (Fig. 32.47). This allows a greater number of threads to engage the matrix of the relatively narrow-diameter cortical bone. Cancellous screws are either completely or partially threaded and are used primarily in the epiphysis or metaphysis. The thread height (difference between the core diameter and outer screw diameter) of cancellous screws is greater than the thread height of cortical screws, which allows deep purchase into the soft, spongy epiphyseal or metaphyseal bone. Cortical and cancellous screws are named for the outside diameter; in 3.5-mm cortical screws, for example, the outer screw diameter is 3.5 mm. Cancellous and cortical screws are available in sizes ranging from 1.5 to 6.5 mm. Locking screws are designed with threaded heads that screw or lock into specially designed plates with threaded plate holes, such as the locking compression plate (Fig. 32.48). Generally, locking screws have a large core diameter and shallow thread profile. Locking screws may be self-drilling and self-tapping and may be used monocortically or bicortically.



FIG. 32.47 Types of bone screws. *Left to right,* cortical, fully threaded cancellous, and partially threaded cancellous.

Bone screws are used either to anchor bone plates to bone or to hold bone fragments in place. When used to anchor a bone plate to bone, these screws are called *plate screws*. The screws used to hold bone fragments in anatomic position and prevent them from collapsing into the marrow cavity are called *positional screws*. Positional screws can be inserted through a plate hole or placed in bone independent of the plate. *Lag screws* (also termed *compression screws*) are used to apply compression between fragments.

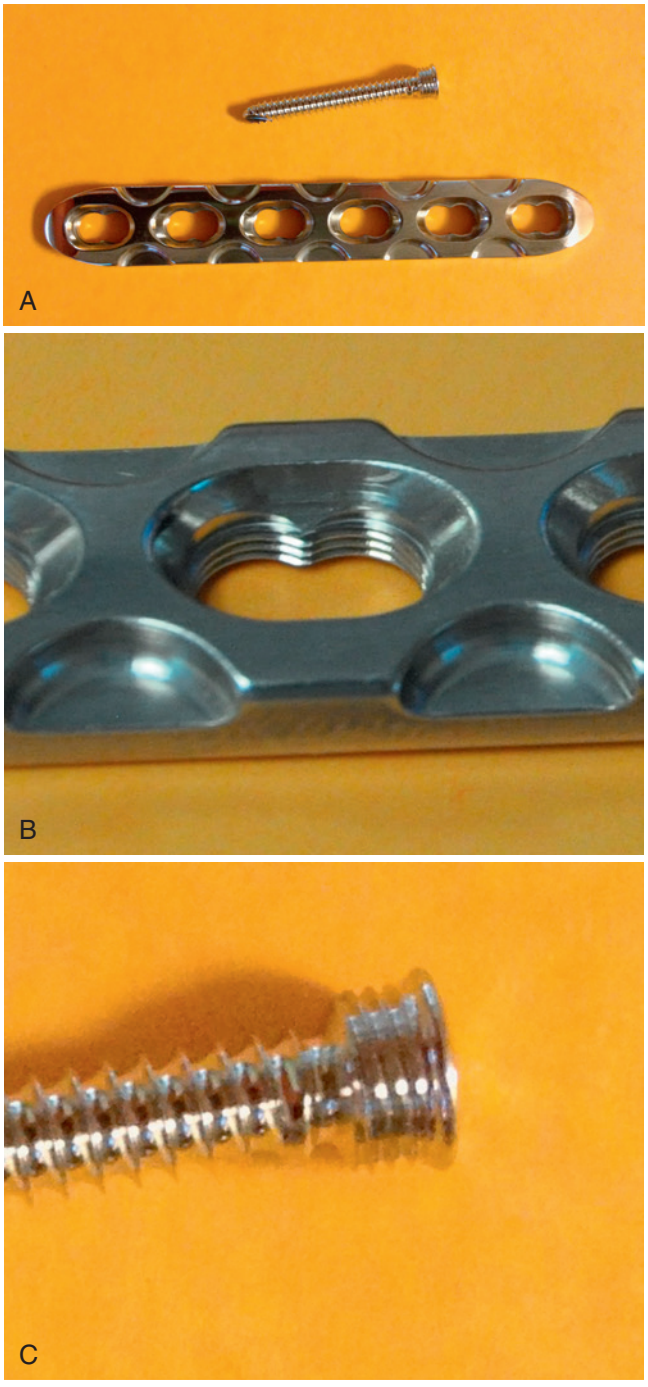


FIG. 32.48 (A) Locking bone plate and screw. (B) Close-up of locking bone plate showing combination threaded and dynamic compression hole. (C) Close-up of locking screw showing dual thread pitch.

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Whether a screw is used as a plate screw, a positional screw, or a lag screw, appropriate instrumentation must be used to implant the screw correctly. Specific drill guides are used for neutral and eccentric placement of plate screws and placement of screws independent of a bone plate. Each different screw size has a drill bit corresponding to the inner core diameter (shaft) of the screw, a drill bit corresponding to the outer diameter of the screw, and a tap corresponding to the threads of the screw. Additional instrumentation includes a depth gauge to measure the length of the screw desired and a countersink to cut a circular groove in the cortex to accept the head of the screw. The countersink is used when a lag screw is inserted into the cortex independent of a bone plate.

Plates

Bone plates are made of 316L stainless steel or titanium; however, because titanium plates and screws are more expensive than stainless steel plates, the latter are used more often. Bone plates are designated in several different ways, including plate length, screw size that the plate hole will accept, plate and screw hole configuration, and function. Plate length is designated by the number of plate holes. Each of the different plate sizes is available in a wide range of lengths. The 3.5 broad plates range in length from 4 to 22 holes, and the 2.7 plates range from 4 to 12 holes. Plate size is determined by the cortical screw that the plate holes will accept; in a 3.5 broad plate, for example, the plate holes will accept 3.5-mm cortical bone screws. Similarly, 2.7 bone plates accept 2.7-mm cortical bone screws, and 4.5 bone plates accept 4.5-mm cortical bone screws (Fig. 32.49).

Plate hole configuration is also used to designate the type of plate. A plate hole can be round (e.g., veterinary cuttable plate [VCP]) or oblong (e.g., dynamic compression plate [DCP]). A bone plate with oblong holes is referred to as a DCP because compression can be applied to the bone through the dynamic action of the screw being tightened. The configuration of the oblong hole is based on a spherical gliding principle modeled after a ball rolling down an inclined plane. The conical shape of the screw head is representative of the ball, and the oblong plate hole is representative of the inclined plane. With the plate hole being inclined, the screw head will slide toward the center of the oblong hole as the screw is tightened. When the screw slides toward the center of the plate hole, horizontal movement of bone beneath the plate occurs. If this is done on each side of the fracture line, the bone is pushed together from both sides, resulting in compression at the fracture line. Proper screw placement is ensured by using drill guides that center the drill hole in either a loading or neutral position. In the loading position, approximately 1 mm of compression is achieved for each screw tightened, whereas in the neutral position, approximately 0.1 mm of compression is achieved. The spherical gliding principle is implemented on both ends of each plate hole in the *limited-contact dynamic compression plate* (LC-DCP). Locking plates have threaded holes that accept and lock with the locking head screw. The *locking compression plate* has a combination plate hole that can accept either standard screws or locking screws (Fig. 32.50).

In addition to plate hole design, *bone plate configuration* is also used to designate the plate type. The 3.5 and 4.5 bone plates are available as standard plates and broad plates. The broad plates are wider, which gives them increased strength and stiffness, an important feature when bone plates are used in large-breed and giant-breed dogs. Both titanium and stainless steel plates are designed as LC-DCPs. They are manufactured so that there is limited contact between the plate and bone to minimize interruption of blood flow. This is accomplished by undercutting the bottom surface of the plate between the screw holes. Undercutting the screw holes also evenly distributes the stress on the plate, eliminating the effect of the plate hole as a stress concentrator. The screw holes are based on the dynamic compression principle but differ in that the oblong screw hole is inclined from both ends of the screw hole toward the center, allowing compression to be applied in either direction. Special drill guides are required. The undercutting also allows greater angulation (up to 40 degrees) of plate screws. Specialized bone plates (e.g., reconstruction plates,

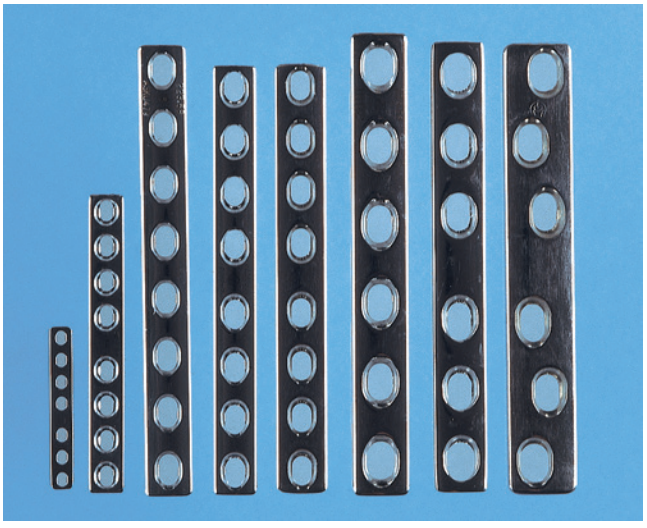


FIG. 32.49 Types of bone plates. *Left to right,* 2 dynamic compression plate (DCP), 2.7 DCP, 3.5 limited-contact (LC)-DCP, 3.5 narrow DCP, 3.5 broad DCP, 4.5 LC-DCP, 4.5 narrow DCP, and 4.5 broad DCP.

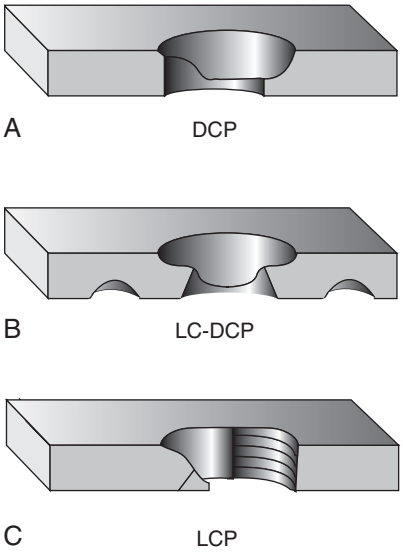


FIG. 32.50 (A) Cross sections of plate hole in a dynamic compression plate (DCP), (B) limited-contact dynamic compression plate (LC-DCP), and (C) locking compression plate (LCP).

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angled plates, and condylar screw plates) are available for selected orthopedic conditions.

Although bone plates are designated as to their intended function (compression, neutralization, and bridging or buttress), depending on how they are applied to the bone, it is important to realize that the plate configuration (e.g., DCP, VCP, broad plate) does not change. A 3.5 broad DCP may serve as a compression plate, neutralization plate, or buttress plate, depending on how it is applied to the bone. A bone plate serves as a *compression plate* when compression is applied to the fracture line through proper application of the plate and screws. A DCP may only function as a compression plate if the fracture line is transverse or short oblique (<45 degrees). If the fracture line is greater than 45 degrees or is comminuted, the plate cannot be used to compress the fracture lines. A *neutralization plate* neutralizes physiologic forces acting on a section of bone that has been anatomically reconstructed and stabilized with screws and wire. Indications for a neutralization plate include reducible comminuted fractures and oblique fractures in which the fracture line exceeds 45 degrees. A *bridging plate* spans a fragmented section of bone, and a *buttress plate* holds a collapsed epiphysis in position. The most common application of a bridging plate is with fragmented diaphyseal fractures in which surgical reduction and stabilization of the bone fragments are not technically feasible (i.e., nonreducible fractures) (Fig. 32.51).

The plate size (2, 2.7, 3.5, or 4.5) required varies depending on patient weight and bone dimensions. ASIF and AO have developed charts that can be used to select a suitable plate relative to body weight. The plate length should be sufficient to prevent premature loosening of plate screws and subsequent loosening of the plate from the bone surface. In most cases of diaphyseal fractures, the plate should span the bone length for optimal performance. The minimum length of plate should allow purchase of six cortices (three screws if both cortices are purchased by each screw) in the main bone fragment above the fracture and six cortices in the main fragment below the fracture. This number of screws will ensure adequate distribution of stress among the plate screws. However, the minimum of six cortices on each side of the fracture is often exceeded to ensure that the plate spans the diaphyseal length.

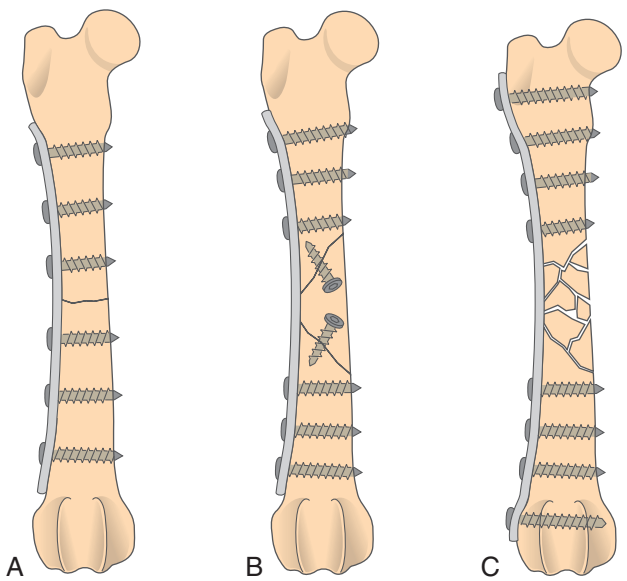


FIG. 32.51 Functions of a bone plate. (A) Compression plate. (B) Neutralization plate. (C) Buttress plate.

Other plates useful in small animals include the reconstruction plate, VCP, canine acetabular plate, T plate, arthrodesis plate, distal femoral osteotomy plate, and TPLO plate. These plates are advantageous for specific injuries. *Reconstruction plates* have deep indentations in the sides of the plate between plate holes. These plates may be contoured in three planes, making them especially useful for treating fractures of bones with complex three-dimensional geometry, such as the pelvis, the distal humerus and femur, or the mandible. VCPs are available in two sizes, designated by the size screw that the plate hole will accept. The 2/2.7 VCP can be used with either a 2- or 2.7-mm cortical screw, whereas the 1.5/2 VCP can be used with either a 2- or a 1.5-mm cortical screw. The VCP is popular because it is available in varying lengths up to 50 screw holes (300 mm). The plate can be cut so that it has the desired number of holes. VCPs are often used in a stacked configuration to bridge comminuted fractures in smaller patients. Stacking two plates onto each other increases the strength and stiffness of the fixation compared with using a single plate. The *canine acetabular plate* is manufactured to conform to the dorsolateral surface of the canine acetabulum and is available in two sizes. This plate is particularly useful in large and giant breeds because it is strong and stiff. The *T plate* is used for distal radial fractures in small breeds. Typically, this fracture has a very short distal segment, which makes it difficult to place an adequate number of plate screws. The plate has a "T" configuration, with the horizontal bar conforming to the epiphysis and/or metaphysis of the distal radius. The shape and size of the plate allow adequate plate screws to be placed in the short metaphyseal segment. Distal femoral osteotomy and TPLO plates are intended for osteotomy procedures, but their shape can be advantageous in the management of certain fractures, including those of the distal femur where there is often limited bone stock distally.

Application

A precise order of maneuvers is followed when inserting a lag screw, position screw, plate screw, or locking screw.

Lag Screw

A lag screw functions to compress a fracture line between two bony fragments. It may be inserted through a plate hole or directly into bone, independent of the bone plate. The optimal position of a lag screw is perpendicular to the fracture line. On shorter oblique fractures, the lag screw is positioned to bisect the angle formed between a line perpendicular to the fracture surface and one perpendicular to the long axis of the bone to prevent slipping of the fragments. The drill hole in the near cortex must be a *glide hole* (a hole equal in diameter to the outside diameter or thread diameter of the screw), whereas the drill hole in the far cortex must be a *thread hole* (a hole equal in diameter to the inner core diameter or shaft of the screw) (Box 32.15). *Use a drill bit that corresponds to the outer diameter of the screw or thread diameter to create a glide hole through which the screw will pass without purchasing bone. When creating the glide hole, drill the bone using a drill guide to maintain alignment and protect soft tissue. Insert a drill sleeve into the glide hole in preparation for creating the thread hole in the far cortex (the drill sleeve insert centers the thread hole in the far cortex relative to the glide hole, which prevents stripping the thread hole on screw insertion). After the glide hole and thread hole are made, use a*

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BOX 32.15 Key Concepts for Applying Lag Screws

- Reduce and secure the fracture before placing the lag screw.
- For optimal compression, place the screw perpendicular to the fracture.
- Drill the near cortex with a bit equal to the screw thread diameter.
- Drill the far cortex with a bit equal to the screw core diameter.
- When using a partially threaded screw, be sure the threads do not cross the fracture.

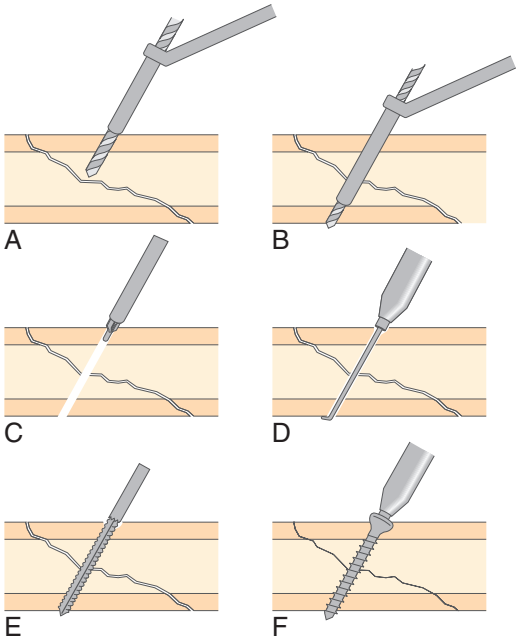


FIG. 32.52 (A) To insert a cortex screw with lag function, drill a glide hole in the near bone segment with a drill bit that has the diameter of the outside screw thread. Use the drill guide to protect soft tissue and align the drill bit. (B) Place an insert sleeve through the glide hole until the far bone segment is engaged. Drill a thread hole with a drill bit the same diameter as the core of the screw. The drill sleeve keeps the thread hole centered relative to the glide hole. (C) Use a countersink to cut a bevel in the cortical bone at the entrance of the glide hole. This increases the contact area between the bone and screw and decreases the amount of the screw head exposed. This step is not necessary if the lag screw is placed through a plate hole. (D) Determine the length of screw to be inserted with a depth gauge. (E) Use a tap to cut threads for the screw in the far bone segment. This step is not necessary if self-tapping screws are used. (F) Insert the screw and tighten it to create interfragmentary compression.

countersink to prepare a site for the screw head in the cortex, and use a depth gauge to determine the appropriate length screw to use. Tap the thread hole through a tap sleeve to maintain alignment and protect soft tissue. Insert the appropriate length screw and tighten it with the fingers only on the screwdriver (Fig. 32.52). The threads of the screw will glide through the hole in the near cortex (glide hole) and purchase the bone in the far cortex (thread hole). As the screw is tightened, the screw head contacts the near

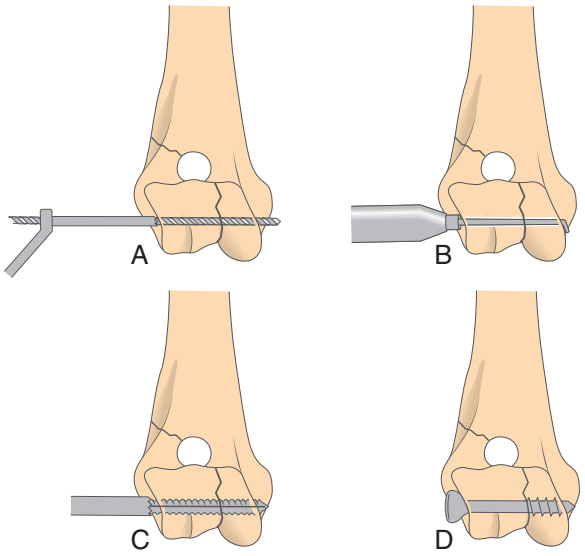


FIG. 32.53 (A) To insert a partially threaded cancellous bone screw, drill the near and far cortices as threaded holes. (B) Measure the depth of the hole. (C) Tap the holes. (D) Insert the screw to compress the fracture. For compression to occur, the threads must not be present at the fracture line. Note that the screw threads have crossed past the fracture line and that the smooth shaft of the screw lies within the fracture plane.

cortex. As the threads purchase the far cortex, the fracture line is compressed. A lag screw can be placed through a plate hole by following the same procedure. Because the screw head rests against the bone plate, however, it is not necessary to countersink the near cortex. Fully threaded cancellous screws can also be inserted as lag screws, either through the plate or independent of the plate, by following the same procedures. The only difference in placement is the instrumentation needed to match the screw size.

Partially threaded bone screws can also be used as lag screws. *With partially threaded screws, drill the near and far cortices as thread holes (Fig. 32.53). Use a depth gauge to determine the appropriate length screw, and tap both cortices before inserting the screw.* Because the screw is only partially threaded, there are no threads to engage the bone on the near side of the fracture, and bone is purchased only in the far cortex. As the screw head contacts bone and the screw tightens, compression is achieved. It is critical that the smooth shaft of the screw crosses the fracture line; if threads are present at the fracture line, compression cannot be achieved.

Warning: the 4-mm partially threaded cancellous screw is weaker than the 3.5-mm cortex screw when placed across the humeral condyle and may fail when used to treat dogs with incomplete ossification of the humeral condyle, which is slow to heal.

Position Screw

Either a cortical screw or a fully threaded cancellous screw can function as a position screw. A position screw is used to hold two bone fragments in anatomic alignment when compression would cause one fragment to collapse into the marrow cavity (Video 32.3). *Hold the fragments in position with bone-holding forceps and drill a thread hole through the cortex of each fragment*

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with a drill bit corresponding to the inner core diameter (shaft) of the screw. Use a depth gauge to determine the appropriate length screw and cut threads in both cortices with the appropriate tap. Insert the screw, using bone-holding forceps to hold the fragments in position and prevent distraction at the fracture line. Gently tighten the screws ("finger tight") until the screw head rests adjacent to the near cortex (or bone plate) (Video 32.4). The screw holds the fragments in position while the bone-holding forceps is removed.

Plate Screw

Standard plate screws secure the plate to the bone by the force generated by the torque applied to the screw. *When inserting a plate screw in the diaphysis, drill a thread hole through the near (cis) and far (trans) cortices. Use the neutral drill guide to place the screw in the center of the plate hole. Use the load or eccentric drill guide with the arrow pointed toward the fracture line to place the screw eccentrically initially and cause compression at the fracture line when the screw is tightened (see Compression Plate below). To insert a 3.5-mm plate screw, use a drill bit that corresponds to the inner core diameter (shaft) of the screw (2.5 mm) and a tap that corresponds to the outer thread diameter of the screw (3.5 mm). Determine the length of screw needed with the depth gauge placed through the plate hole and cut threads into the near and far cortices with a tap. Use a tap sleeve when cutting threads to maintain axial alignment relative to the thread hole and to prevent soft tissue from winding around the tap threads. Remove the tap, and flush the hole with sterile saline to eliminate bone debris and lubricate the hole. Insert a cortical screw and use fingers only on the screwdriver to tighten it (Fig. 32.54). In spongy metaphyseal or epiphyseal bone, use a cancellous bone screw as a plate screw and place in a similar manner.*

Locking Screw

Locking screws must be accurately inserted perpendicular to the plate hole for the threads to match and secure the screw. Additionally, the fracture must be reduced and the plate positioned appropriately before the screw is inserted (Video 32.5). *Screw the threaded drill guide into the locking hole. Be sure the guide is secured. Drill with the appropriately sized drill bit. Remove the drill guide and measure the hole to determine screw length.* Locking screws may be used as monocortical or bicortical screws. If using a monocortical screw, take care that the screw does not contact the far cortex as this may interfere with securing the screw head. *Use the screwdriver to seat the screw. Carefully tighten the screw, avoiding excessive force. Alternatively, use a power driver with a torque-limiting attachment to seat the screw.*

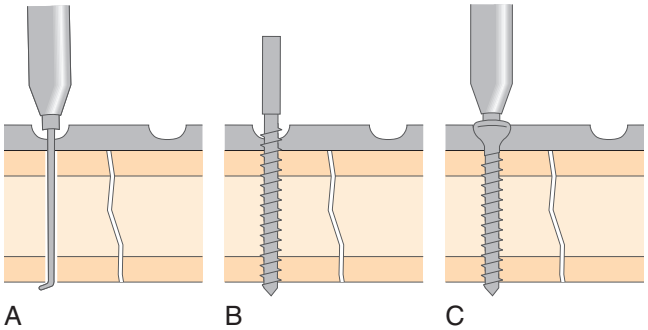


FIG. 32.54 Placement of a plate screw. (A) Drill the thread hole, then measure the screw length. (B) Tap threads in the near (cis) cortex and far (trans) cortex. (C) Insert the screw.

Plates (Box 32.16)

Compression Plate

The compression plate is used to generate axial compression at the fracture. To achieve this function, it is important to contour the plate properly relative to the bone surface. *To apply a plate as a compression plate, contour it so that the plate remains slightly offset (1 to 2 mm) from the surface of the bone at the fracture line (Fig. 32.55). Secure the plate to the bone with plate-holding forceps, ensuring that the ends of the plate lie over the bone.* If the plate is contoured to conform accurately to the bone surface, the fracture line will load asymmetrically. This occurs because the compression generated through the plate is applied to the bone eccentrically (it is greatest on the surface on which the plate rests). The net result is compression of the fracture line beneath the plate and widening of the fracture near the far cortex. Because a gap exists in the far cortex, the plate supports all the applied loads without any significant contribution from the bony column. The gap in the far cortex is prevented through prestressing the bone plate by slightly overcontouring it relative to the bone surface, so the center of the plate is lifted 1 to 2 mm above the bone surface. When the plate screws on each side of the fracture line are tightened, each main bone fragment is pulled up against

BOX 32.16 Key Concepts for Applying Bone Plates

- Select the appropriate plate size.
- Select a plate that spans the bone length for diaphyseal fractures.
- Accurately contour the plate.
- Place a minimum of three screws or secure six cortices above and below the fracture.
- Use a longer and stronger plate as a bridging plate or augment it with an intramedullary pin.

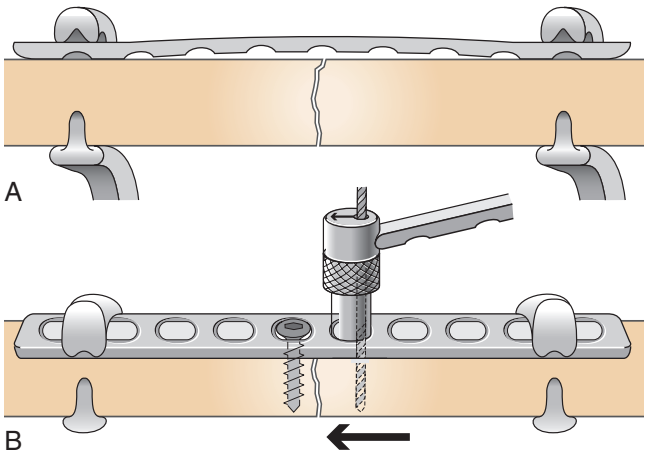


FIG. 32.55 To apply a dynamic compression plate, prestress the plate by slightly overbending it at the fracture site during contouring. (A) This creates a 1- to 2-mm gap between the plate and bone over the fracture. Secure the plate to the bone. (B) Use the eccentric drill guide to locate a screw eccentrically through a plate hole over the unattached segment of bone. (B, Modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat.* St. Louis: Elsevier; 2005.)

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the plate, compressing the far cortex. *Insert the two plate screws nearest the fracture first. Place both screws in a loaded position and tighten them to achieve compression of the fracture line.* Similar compression can be achieved by placing the first two screws in any of the plate holes as long as there is one screw on either side of the fracture. *Insert subsequent plate screws in holes in an alternating manner on either side of the fracture, working toward the plate ends. Adequate compression of the fracture is generally achieved with loading of the first two screws.*

NOTE If you desire greater compression, place an additional (secondary) screw on each side of the fracture in the loaded position. After drilling and inserting the additional screws approximately 90%, loosen the initial screws to permit the bone to translate towards the fracture line while the secondary screws are tightened. Then tighten the primary screws and insert the remaining screws in a neutral position.

Neutralization Plate

First, reduce and stabilize the fracture with a series of lag screws, multiple cerclage wires, or a combination of both. Because the screws and wires are not sufficiently strong to resist physiologic forces generated by weight bearing, use a bone plate to bridge the area and to neutralize forces that would act to collapse the fracture. As with a compression plate, apply a neutralization plate to the tension surface of the bone, but contour it to the anatomic surface of the bone. Separation of the fracture line beneath the plate will not occur because the fracture lines have already been compressed with lag screws or cerclage wire (see Fig. 32.51). The neutralization plate protects the reconstructed bone from all torsional, bending, and shearing forces. The recommended minimum number of cortices (six) engaged on each side of the fracture is the same as for a compression plate. *With a neutralization plate, insert all screws in the neutral position, beginning from the ends of the plate and working toward the center. If a plate screw cannot be inserted because it lies over a fracture line, leave the hole empty. If the plate hole lies over a lag screw placed through the bone, leave the hole empty, or insert a screw that purchases only the near cortex.*

Bridging Plate

Precontour the plate to match the normal anatomic shape of the bone. Use a radiograph of the intact bone of the opposite leg as a template to help contour the plate if the affected bone is severely comminuted. Align the bone to restore length and correct rotational orientation before securing the plate (see Fig. 32.51). The bridging plate serves as a splint to maintain the spatial alignment of the bone during healing. The plate and screws will carry all the applied loads during the early postoperative period. This results in greater stress on the bone screws than with compression or neutralization plates, in which the applied loads are shared with the bone. *Therefore purchase a minimum of eight cortices rather than six. Similarly, use a stronger and stiffer plate because it also will be subject to substantial loads until bone is deposited within the fracture gap to form a bony column. For optimal strength and stiffness, use a broad plate, lengthening plate, or stacked VCP rather than a standard plate. Alternately, support the plate with ancillary implants (IM pins or external skeletal fixators) that share the applied loads during the early healing period. With a plate-IM pin combination, insert an IM pin approximately 30% of the diameter of the marrow cavity, being careful to maintain the rotational alignment and axial length of the bone. Contour a plate of appropriate length and apply it to the appropriate surface of the bone. Insert the most proximal and distal plate screws so that they avoid the IM pin and engage both near and far cortices. Insert the plate screws near the center of the plate so that they engage either both cortices or only the near cortex (monocortical screws) (Fig. 32.56).*

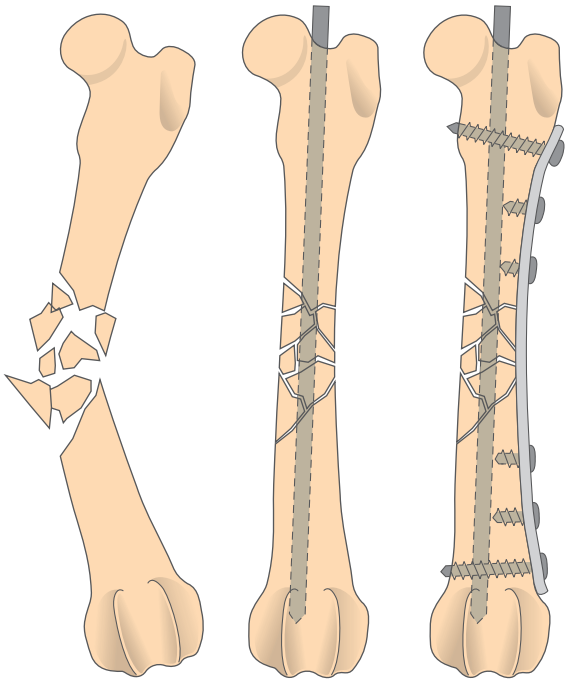


FIG. 32.56 Stabilization of a comminuted fracture with a plate-rod combination.

Buttress Plate

Apply a buttress plate to shore up a metaphyseal fracture or protect a screw repair of an intraarticular slab fracture. The buttress plate functions to prevent collapse of the adjacent articular surface. *To prevent any slipping of the plate and collapse of the articular surface, insert the screws in the juxtaarticular portion of the plate by first placing the drill guide adjacent to the part of the plate hole nearest the fracture. Butting the screw head to that portion of the plate during the initial screw insertion prevents the plate from sliding.*

Locking Plate

To use the locking plate as a bridging plate, select a plate long enough to span the bone. Precontour the plate to match the normal anatomic shape of the bone. Use a radiograph of the intact bone of the opposite leg as a template to help contour the plate. If all locking screws are used, the plate does not have to conform completely to the bone (Box 32.17). *Align the bone to restore length and correct rotational orientation, and hold the plate in position with plate-holding forceps.* The alignment must be correct before securing the plate, as the locking screws will hold the bone in that position. *Secure the plate to the bone with locking screws or a combination of standard and locking screws.* If using a combination of standard screws and locking screws, the plate should conform to the bone and the standard screws should be applied first to pull the bone to the plate.

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BOX 32.17 Key Concepts for Applying Locking Plates

- Plate contour is not critical.
- Locking screws must be perpendicular to the plate.
- Reduce the fracture before locking screws are tightened.
- Use a longer plate with fewer screws.

Minimally invasive percutaneous osteosynthesis. *To use the locking plate as a percutaneously placed bridging plate, select a plate long enough to span the bone. Precontour the plate to match the normal anatomic shape of the bone. Use a radiograph of the intact bone of the opposite leg as a template. Reduce the fracture indirectly using percutaneously placed forceps, a temporary external fixator, or an IM pin. Make two small incisions at either end of the bone and create a tunnel in the soft tissues adjacent to the periosteal surface of the bone. Insert the plate through the tunnel and secure it to the bone at both ends with plate-holding forceps. Check the bone alignment and plate placement with fluoroscopy. Secure the plate to the bone with locking screws placed at either end. Insert additional screws if needed through stab incisions over the plate holes.*

Postoperative Care

Bone plates and screws require minimal postoperative maintenance. Postoperative analgesia should be provided (see Boxes 32.1 and 32.2 and Table 32.4). Activity should be restricted to leash walking and physical rehabilitation until the fracture has healed. Physical rehabilitation encourages controlled limb use and optimal limb function after fracture healing (see Table 32.7 and Chapter 11). Generally, postoperative examinations should be done at 2 and 8 weeks after surgery and then as needed. The functional period of plates and screws is relatively long because of the interlocking hold of the screws with the bone. Most interfragmentary screws are left in place after bone healing. Plate removal may be indicated for soft tissue complications, cold conduction, or infection. Clients may also rarely elect plate removal to reduce the low risk of implant-associated sarcoma. Plate removal should be performed aseptically with the patient under general anesthetic. *Incise the skin overlying the plate screws and bluntly dissect through soft tissue to the head of the screw. Once all plate screws are removed, lift the plate from the bone surface at one end and extract it.* Locking plate screws may become cold-welded to the plate, making removal difficult and requiring the use of carbide drills or high-speed metal cutting tools.

POSTOPERATIVE RADIOGRAPHIC ASSESSMENT FOR FRACTURE REPAIRS

All fracture repairs should be radiographed immediately postoperatively to document and enable assessment of the repair. The three principle aspects of postoperative radiographic assessment are alignment, apparatus, apposition. Recheck radiographs include a fourth principle, which is activity. Assessment of alignment is to ensure that the joints above and below the fracture are properly aligned. Proper function of the limb requires avoidance of varus, valgus, internal, and external rotation, and minimization of cranial and caudal deformity and translation. Assessment of the apparatus is to ensure that the implants are adequate to support bone healing and without evidence of complications such as implants in the joint. Assessment of apposition is to ensure that the bone ends are close enough to support bone healing. In some fractures the bone ends may be compressed to encourage primary bone healing. In comminuted fractures, apposition of the bone ends is not possible. Recheck radiographs are performed, usually several weeks following the repair, depending on the age of the patient and characteristics of the fracture. Recheck radiographs should also be evaluated for activity, meaning evidence of appropriate bone healing and absence of evidence of infection or stabilization failure.

FRACTURE HEALING

NORMAL FRACTURE HEALING

Fracture healing is the biological process after cartilage and bone disruption that restores tissue continuity necessary for function. The goals of fracture treatment are to (1) support healing and (2) restore function to the affected bone, surrounding soft tissue, and limb. Each goal should be kept in mind when selecting treatment regimens and fixation devices (see sections on decision making and fixation systems). Fracture healing varies depending on biological factors (e.g., patient age, fracture location in cortical bone, cancellous bone, or physeal cartilage; cellular responses; circulation; and concurrent soft tissue injury) and mechanical factors (e.g., patient body weight, stability of bone segments and fragments after fixation device placement) that influence the sequence of cellular events in fracture healing.

VASCULAR SUPPLY

All physiologic processes occurring within bone, including repair processes during fracture healing, are dependent on an adequate blood supply. Normal circulation to long bones consists of an afferent supply from the principal nutrient artery, proximal and distal metaphyseal arteries, and periosteal arteries that enter bone at areas of heavy fascial attachment. The direction of blood flow through the diaphysis is centrifugal, from medullary canal to periosteum. Under normal conditions, medullary pressure probably restricts periosteal blood flow to the outer third of the cortex (Fig. 32.57A). In contrast to mature animals, immature animals have numerous arteries that perforate newly formed appositional bone running longitudinally over the periosteal surface. The metaphysis and epiphysis have separate blood supplies and generally do not communicate across the cartilaginous physis (see Fig. 32.57B). The epiphyseal blood supply nourishes the cartilaginous reserve cell zone and growing physeal cells. Interruption of this portion of the circulation results in death of growing cells and cessation of physeal function. Metaphyseal arteries supply cells involved with endochondral ossification; disruption of metaphyseal blood flow delays endochondral ossification, resulting in widening of the cartilaginous physis. When circulation is reestablished, endochondral ossification resumes. Flat bones with extensive muscle attachment (e.g., pelvis and scapula) have tremendous extraosseous blood supply in addition to that provided by nutrient arteries. Irregular bones (e.g., carpal and tarsal bones) generally have multiple nutrient arteries.

Medullary circulation is disrupted in most long bone fractures. Initially, existing components of the normal vasculature (i.e., metaphyseal vessels) are enhanced to supply the injured area. Additionally, a transient extraosseous vascular supply develops in soft tissue and surrounding fractures to nourish the early

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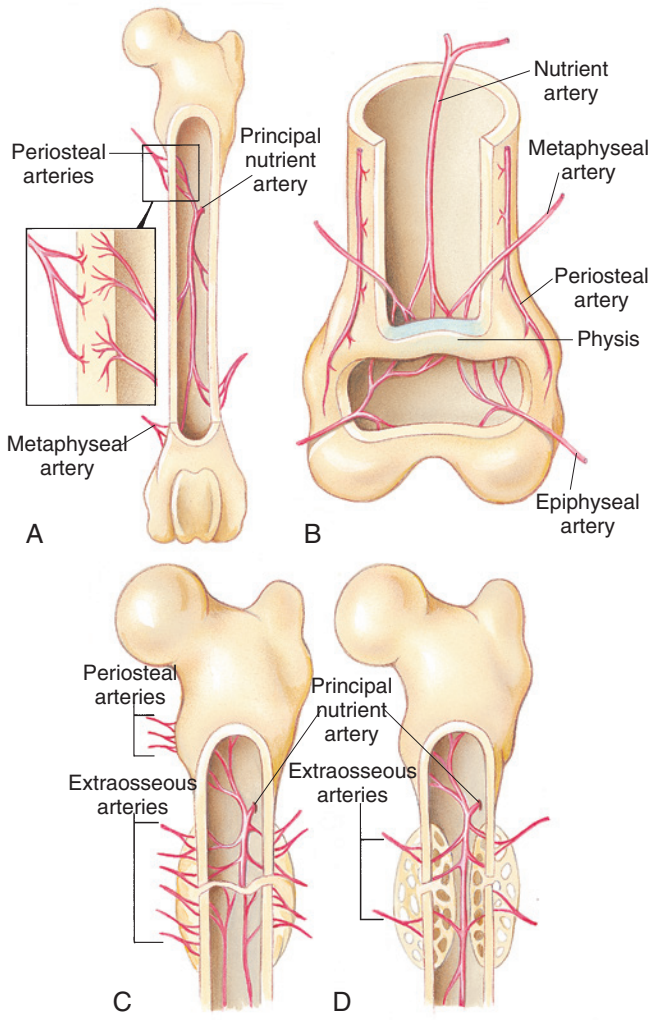


FIG. 32.57 Blood supply to bone. (A) Normal bone. (B) Immature bone. (C) Fractured bone (extraosseous blood supply). (D) Healing bone.

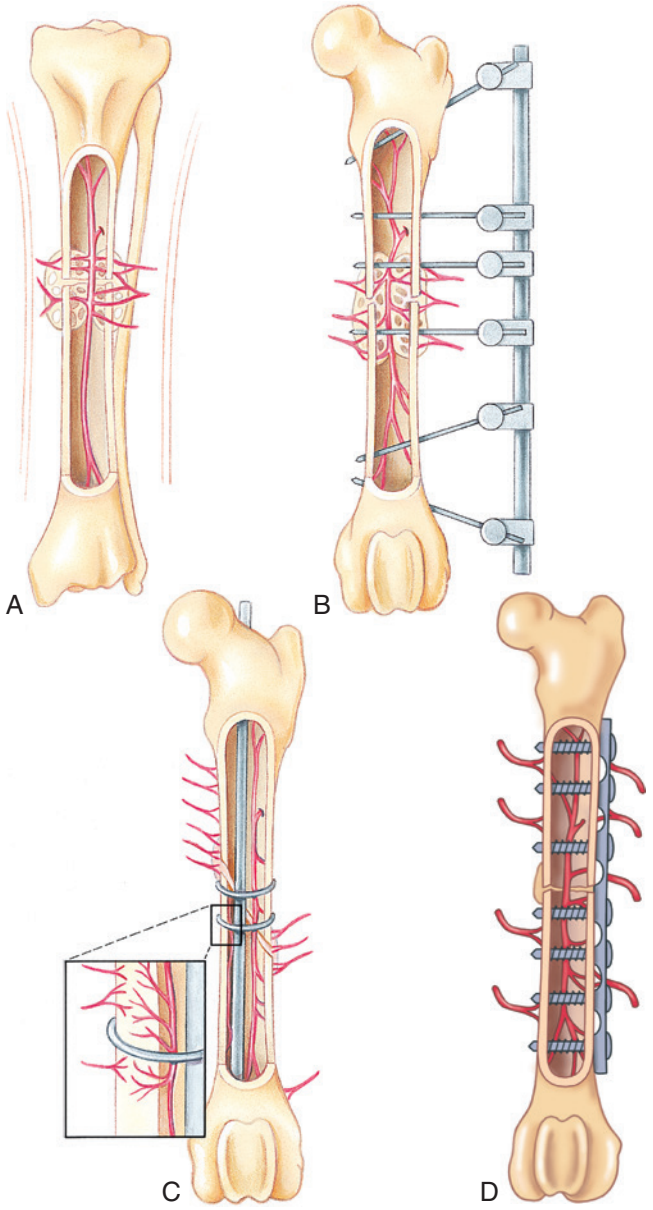


FIG. 32.58 Effect of fixation devices on circulation to fractured bone. (A) Cast. (B) External fixator. (C) intramedullar pin and cerclage wires. (D) Plate and screws. Note that closed fracture reduction and application of casts or external fixators cause the least disruption to surrounding soft tissue and newly formed extraosseous blood supply. (Modified from Fossum TW. *Small Animal Surgery*. 2nd ed. St. Louis: Mosby; 2002.)

periosteal callus (see Fig. 32.57C). As bone healing progresses and stability is restored, medullary blood supply is reestablished. Ultimately the extraosseous circulation diminishes and normal medullary centrifugal flow dominates (see Fig. 32.57D). Closed fracture reduction with application of casts or external fixators causes the least disruption to surrounding soft tissue and newly formed extraosseous blood supply (Fig. 32.58A, C), whereas open reduction disturbs developing extraosseous blood vessels and hinders reestablishment of medullary blood flow. Traumatic handling of surrounding soft tissue further impairs the extraosseous circulatory response. Insertion of any type of IM pin disrupts the medullary vasculature; pins that contact endosteal surfaces block medullary afferent flow. Stable implants allow new medullary circulation to develop, which supplies the adjacent endosteal surfaces (see Fig. 32.58C). Cerclage wire applied tightly to cortical surfaces does not significantly impair vasculature. Even in immature animals, circumferential wires seemingly do not significantly block periosteal blood flow. Although plate and screw application affords the greatest fracture stability and allows early reformation of medullary circulation, blood supply to outer cortical bone beneath plates may be impaired, causing affected cortices to remodel and become more porous. Some plate designs (e.g., LC-DCPs) minimize this phenomenon (see Fig. 32.58D). Because adequate blood supply is essential for bone healing, any circulatory impairment may delay healing. The motion of loose implants, especially cerclage wires, disrupts developing vasculature; excessive fracture motion discourages reestablishment of medullary vasculature. Excessive medullary reaming interferes with circulation of the inner cortex, resulting in intense bone remodeling. Large bone fragments denuded of soft tissue (and thus blood supply) can be used to reconstruct fractures anatomically; however, these fragments must be rigidly immobilized to

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encourage early revascularization. For comminuted fractures, it is preferable if the fracture is minimally disturbed and major bone segments are stabilized with a plate or external fixator. This "biological treatment" consists of indirect reduction techniques to preserve the surrounding soft tissue and optimal stabilization to promote rapid callus formation. In these cases, the fragments revascularize rapidly and incorporate into callus.

FRACTURE HEALING AND FRACTURE MANAGEMENT

Understanding fracture healing is paramount to interpretation of radiographic evaluations of fracture patients. Orthopedic surgeons must have a working knowledge of bone healing under various mechanical environments and the corresponding radiographic appearance of the fracture. Assuming adequate vascularity, the pathway of bone healing is primarily influenced by the amount of interfragmentary movement caused by the load on the fracture and modulated by the stability of the fracture fixation. The pathways of bone formation include *indirect bone union,* which is *endochondral* bone formation (bone formed on a cartilaginous precursor), *direct bone union* (bone formed without evidence of callus), or *intramembranous* bone formation (direct differentiation of MSCs into osteoblasts so bone forms without a cartilaginous precursor).

Sequential radiographs allow the evaluation of fracture healing. Generally, radiographs should be made postoperatively to assess fracture alignment and implant position, and they should be repeated in 6 to 8 weeks to assess healing (Box 32.18). Most fractures will demonstrate significant healing by 8 to 10 weeks postoperatively. In cases of inadequate healing, additional radiographs should be performed every 4 to 6 weeks. Follow-up radiographs should be compared with earlier studies to determine the dynamics of bone healing. Fractures should be evaluated for evidence of bone formation, and implant position should be appraised to detect instability.

Inflammatory Phase of Bone Healing

Immediately after fracture, an inflammatory phase of bone healing is initiated. The hematoma appears to signal molecules that have the capacity to initiate the inflammatory cascades of cellular responses critical to fracture healing. Inflammatory cells secreting cytokines, such as interleukin-1 and interleukin-6, may be important in regulating the early events in fracture healing. Platelets are likely to be the first source of platelet-derived growth factor and TGF-β1, both of which are important regulators of cell proliferation and differentiation. Inflammatory mediators, such as prostaglandins E1 and E2, may stimulate angiogenesis and may also be responsible for signaling early bone resorption by osteoclasts and proliferation of osteoprogenitor cells. Biological techniques for fracture stabilization emphasize minimal manipulation of the fracture environment to preserve these inflammatory mediators. Radiographically, there is minimal evidence of this phase of bone healing.

Indirect Bone Healing

Indirect bone healing is characterized by the formation of fibrous connective tissue and cartilage callus at the fracture site. This form of healing occurs in the unstable mechanical environment caused by motion of the bone segments. The amount of motion can range from the extremes of unrestricted motion of untreated fractures through the increasing levels of stability achieved with casts, IM pins, external fixators, interlocking nails, and bone plates. In general, as the motion at the fracture decreases, the amount of callus formation also decreases.

Motion at fracture sites affects the size of gaps between fragments. This motion is calculated as *strain,* which is the ratio between change in gap width to total gap width (Fig. 32.59). A given tissue will not proliferate under strain conditions that exceed that tissue's limits of deformation. Sequential formation of stiffer tissue in the fracture gap is a biological method of decreasing the motion and subsequently the strain. Hematoma and granulation tissues have a high strain tolerance and can survive in an initially mobile fracture site with strains up to 100%. As these tissues infiltrate the fracture site, there is less motion, and consequently less strain in the gap. In addition, resorption of bone occurs in excessive strain environments and results in widening of the fracture gap that in turn decreases the strain environment at the fracture site. The granulation tissue is gradually replaced by fibrous connective tissue and fibrocartilage that survive in strains of 20% and 10%, respectively, and in turn continue to stabilize the fracture site. As the fracture stabilizes, mineralization of the cartilage begins at the fragment surfaces and continues toward the center of the gap. Local resorption of the mineralized tissue occurs, followed by vascularization of the resorption cavity and the formation of lamellar bone within that cavity. Trabecular or cancellous bone is formed at the fracture site in this manner. Strain values of up to 10% may be tolerated by the three-dimensional configuration of woven bone. Formation of a wide cuff of periosteal callus increases the diameter of the

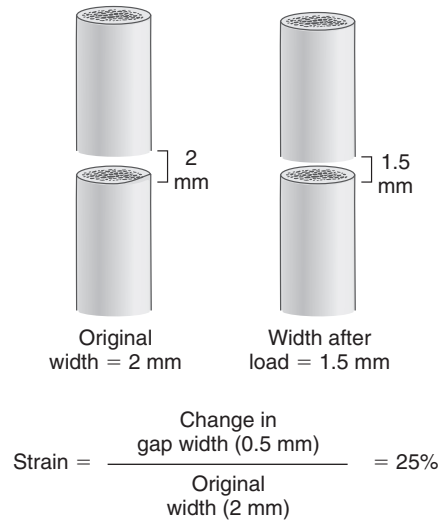


FIG. 32.59 Ratio between the change in gap width to total gap width is called *strain.*

BOX 32.18 Postoperative Fracture Management: Evaluation Schedule

- 1 week: phone
- 2 weeks: suture removal and/or physical examination
- 4 weeks: phone
- 6–8 weeks: radiographic and physical examination
- Repeat phone contact and radiographic and/or physical examination every 6 weeks until fracture is healed; intervals and duration vary with fracture assessment score

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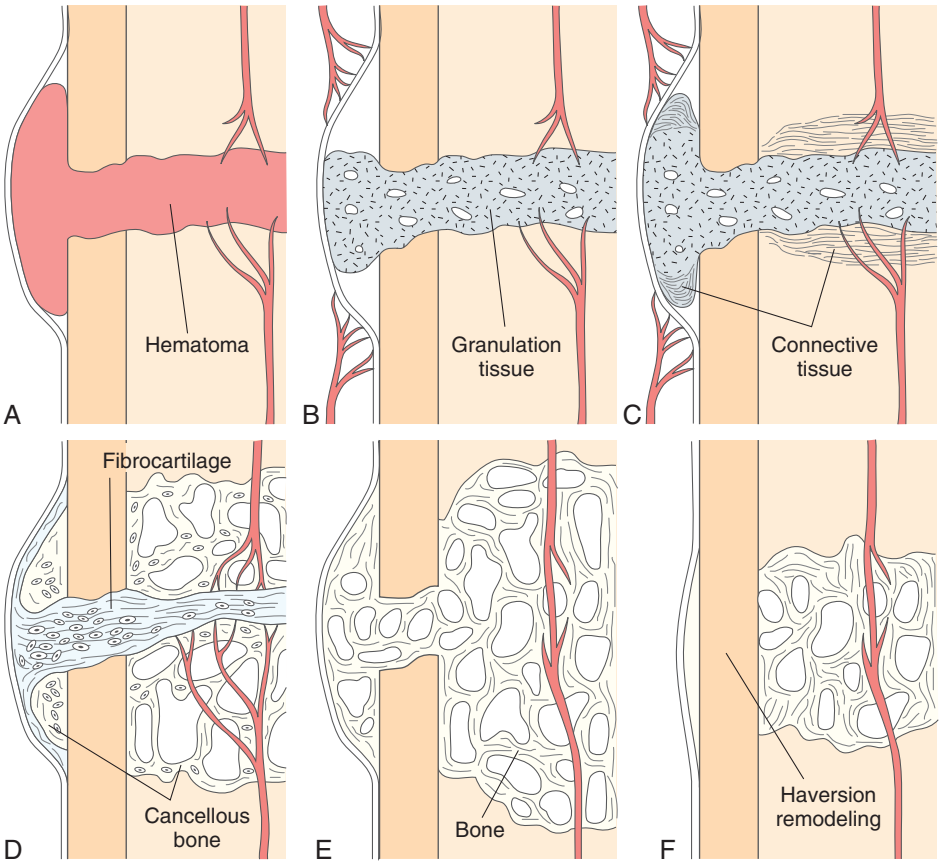


FIG. 32.60 Strain decreases with increased fracture rigidity as fractures are bridged first by more strain-tolerant tissue and later by less strain-tolerant tissue. (A) Defect is first filled by a hematoma, which is then (B) replaced by granulation tissue. (C) Connective tissue forms. (D) Fibrocartilage is mineralized, forming (E) cancellous bone and bone. (F) Haversian remodeling occurs to eliminate the callus.

bone at the fracture site. The stabilizing influence of a callus is a factor of its radius to the fourth power, thereby greatly increasing the ability of the bone to resist bending and torsional forces early in the fracture repair process. Formation and resorption of lamellar bone, which tolerates strain values up to 2%, result in remodeling of the callus to cortical bone (Fig. 32.60).

Radiographically, indirect bone union is visible as proliferation of endosteal and periosteal new bone forming a sclerotic area in both bone segments at the fracture surface. Initially, the fracture gap may increase in width as bone resorption occurs. The periosteal bone formation starts at a distance from the fracture surface and increases in width as it approaches the fracture. Eventually the callus resembles an arch spanning the fracture. The fracture gap will slowly become indistinct as the callus begins to overlay the fracture and as mineralization of the fibrocartilage occurs. After the callus bridges the fracture, stability is achieved and bone remodeling occurs. Radiographically, this is visible as a decrease in the callus bone density with reestablishment of the medullary canal and evidence of cortical bone at the fracture site (Fig. 32.61). The implant can be removed when there is bone bridging the fracture on all radiographic views.

Direct Bone Healing

Direct bone healing (bone formation directly at fracture sites, without an intermediate cartilage stage or visible callus) occurs when fixation devices maintain absolute fragment stability. For this to occur, the mechanical environment must be such that fracture motion is negligible and the fragments are in contact or separated by only small (150- to 300-μm) gaps. Where bone fragments are in contact under rigid fixation, there is a process of simultaneous union and reconstruction with haversian remodeling called *contact healing* (Fig. 32.62A–B). Direct bone formation occurring in the small gaps in the fracture line after rigid fixation is called *gap healing*. Initially, these gaps are filled with a network of fibrous bone, but within 7 to 8 weeks, this mechanically weak bone union begins to remodel (see Fig. 32.62C–D). The longitudinal reconstruction of fracture sites with haversian remodeling is the second stage of gap healing and provides a strong union between fracture fragments. *Haversian remodeling* begins with osteoclastic resorption of bone and formation of resorption cavities that penetrate longitudinally through fragment ends and newly formed bone in fracture gaps. The osteoclasts are followed by vascular loops, mesenchymal cells, and osteoblast precursors. Osteoblasts line resorption cavities and secrete osteoid, which is mineralized to bone. This lamellar bone is arranged along the bone's long axis, through the fragment ends and fracture gaps, and results in a strong union of bone fragments.

The radiographic appearance of direct bone union is one of slowly increasing density of the fracture line without bridging

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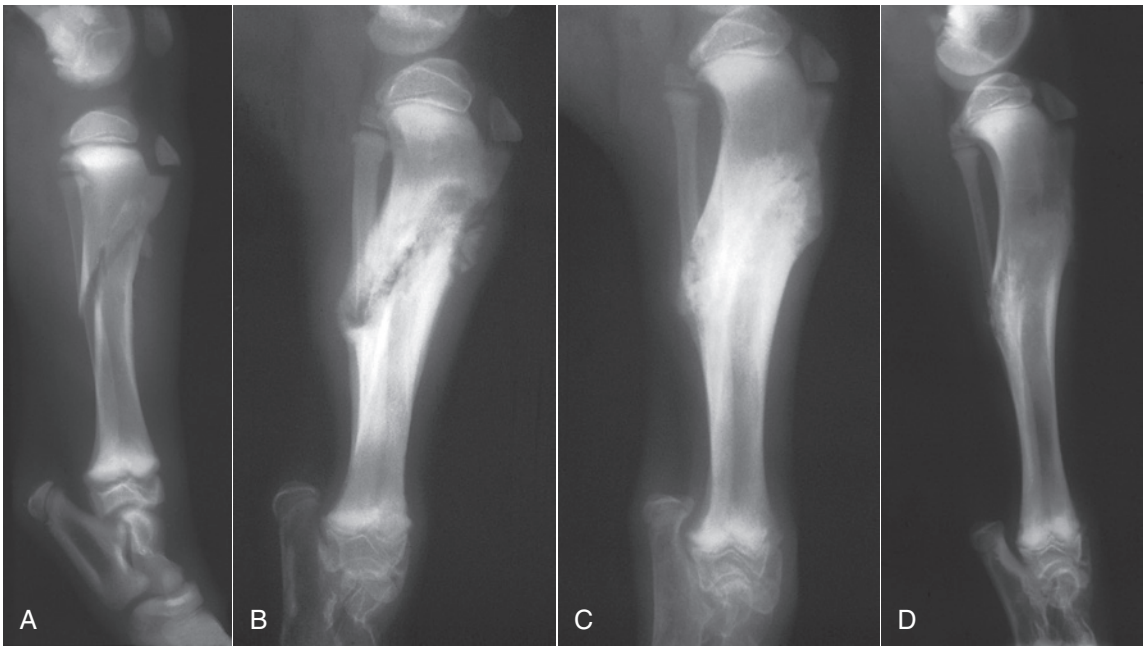


FIG. 32.61 (A) Indirect bone healing occurs in fractures stabilized with a cast. (B) Initially the fracture gap increases in width as bone resorption occurs. Periosteal callus starts at a distance from the fracture surface and increases in width as it approaches the fracture. (C) Eventually the callus resembles an arch spanning the fracture. The fracture gap slowly becomes indistinct as the callus begins to overlay the fracture and as mineralization of the fibrocartilage occurs. (D) After the callus bridges the fracture, stability is achieved and bone remodeling occurs.

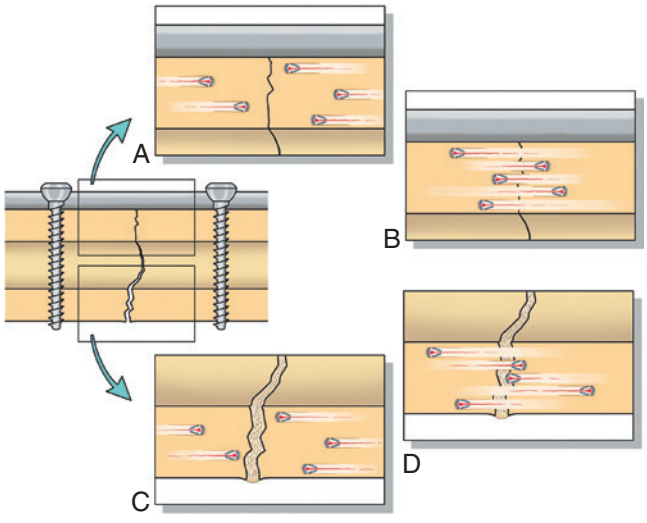


FIG. 32.62 Direct bone healing occurs in rigidly stabilized fractures. (A–B) When the bone fragments contact, simultaneous union and reconstruction with haversian remodeling occurs. When there are microscopic gaps, (C) first the gap fills with fibrous bone, and then (D) longitudinal reconstruction of bone with haversian remodeling occurs.

periosteal and endosteal callus (Fig. 32.63). Although the fracture line may be filled with bone density material within 6 to 8 weeks after fracture (reflecting the network of fibrous bone), the bone is not strong enough to withstand unprotected weight bearing until osteonal remodeling is complete.

Intramembranous Bone Healing

Intramembranous bone formation, a type of direct bone healing occurring where stability is optimal for direct differentiation of mesenchymal cells into osteoblasts, is possible in an environment of up to 5% strain. This type of bone formation is observed when bone is deposited directly on bone fragments at a distance from the fracture site or when bone bridging between comminuted bone fragments occurs to stabilize fragments after biological fixation techniques. Intramembranous bone formation is generally combined to a varying extent with indirect bone healing responses. The resultant periosteal callus may be smaller in comminuted fractures stabilized with bridging fixation techniques, with the endosteal and bridging callus supplying much of the fracture support. Again, resorption of woven bone and formation of lamellar bone at fracture sites result in remodeling of bony callus to cortical bone (Fig. 32.64).

Radiographically, endosteal bone appears first, followed by bone bridging between fragments with minimal periosteal callus formation. Bone bridging of the fragments is more evident. There is still minimal periosteal callus formation. Resorption of woven bone and formation of lamellar bone at the fracture sites result in remodeling of bony callus to cortical bone (Fig. 32.65). The implant can be removed when there is bone bridging the fracture on all radiographic views.

Distraction osteogenesis occurs when gradual traction is applied to cortical bone, such that sufficient stress is created to stimulate and maintain formation of new bone. This concept is used in external fixation techniques for limb lengthening, treatment of angular deformities, and transportation of cortical bone. To achieve bone formation during slow distraction after corticotomy or osteotomy, medullary and periosteal blood supply must be

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FIG. 32.63 (A) Fracture healing in a transverse radial fracture in a mature dog treated with a bone plate. (B–C) The radiographic appearance of direct bone union is one of slowly increasing density of the fracture line without bridging periosteal and endosteal callus.

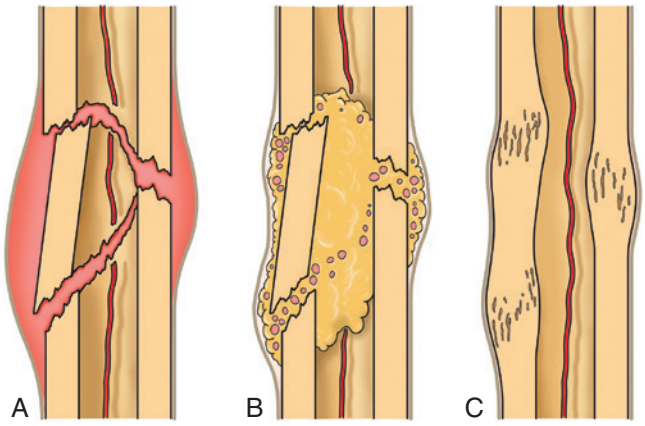


FIG. 32.64 (A) Comminuted fractures fixed with biological techniques of indirect reduction, major segment alignment, and optimal stabilization appear to heal with a combination of direct differentiation of mesenchymal cells to osteoblasts and endochondral ossification. (B) The fracture site fills with endosteal and bridging callus. (C) Resorption of woven bone and formation of lamellar bone at the fracture sites result in remodeling of bony callus to cortical bone.

preserved and major bone fragments optimally stabilized. The bone surface is covered with cells that may differentiate into osteoblasts or chondroblasts, depending on the cell's mechanical and biological environment. Within 3 to 7 days after osteotomy, these cells organize and begin to proliferate. The ideal rate of distraction is 1 mm per day divided into two to four distraction periods. Osteoid is laid down in parallel columns that extend from osteotomy surfaces centrally. Normally, lamellar bone develops within these columns; if there is sufficient instability at the fracture site, however, formation of an intermediate cartilaginous phase or fibrous tissue formation may occur. After the desired limb length is achieved, the fixator remains in place to allow remodeling of new cortical bone (Fig. 32.66).

Trabecular Bone Healing

Metaphyseal fractures involving trabecular or cancellous bone heal differently than similar fractures through cortical bone (Fig. 32.67A). Trabecular bone is inherently more stable than cortical bone and does not heal by periosteal callus formation unless there is significant instability. Increased osteoblastic activity occurs on either side of these fractures if they have been adequately immobilized (see Fig. 32.67B). New bone is deposited on existing trabeculae, and fracture gaps are filled with woven bone. Bridging between trabeculae occurs before union of the cortical *shell* (see Fig. 32.67C).

Trabecular bone healing in metaphyseal fractures appears radiographically as the formation of one or two dense bands at the fracture site. Gradual bridging of these bands occurs until the cortical shell is completely spanned by bone. Periosteal callus is not usually evident in metaphyseal fractures unless there is instability of fracture fragments (Fig. 32.68).

Physeal Healing

Physeal fractures generally occur because the physis is weaker than surrounding bone and ligaments (Fig. 32.69A). The mechanically weak portion of the physis is the junction of the hypertrophic zone where the cells are relatively large in comparison with the amount of matrix and the stronger zone of provisional calcification (see Fig. 32.69B). Avulsion or shearing forces can cause fractures through this zone that heal rapidly by continued growth of physeal cartilage and metaphyseal callus formation because the growing cells and adjacent vascularity are undamaged. Once fracture gaps are filled, normal endochondral ossification resumes and physeal function continues. If damage occurs to growing cells (reserve and proliferating zones), however, growth of physeal cartilage does not occur. Rather, endochondral ossification proceeds (see Fig. 32.69C), and bone formation in fracture gaps results in premature physeal closure. Malalignment of the fractured physis (where metaphyseal and epiphyseal bone are in contact) allows trabecular bone healing and physeal bridging. The bone bridge may prevent normal physeal function. Because physeal fractures most often heal with endochondral ossification, radiographically the physeal line is replaced with bone.

Implant Removal

Deciding if and when to remove fixation devices may be difficult. This decision is usually made after evaluating radiographs of healing fractures. Knowledge of radiographic appearance of bone healing associated with various fixation systems is mandatory for informed decision-making. In general, fixation systems can be removed when there is radiographic evidence of bone bridging all fracture lines on all views.

Fractures stabilized with casts heal by indirect bone formation; radiographically, this appears as bridging periosteal and endosteal callus at fracture sites (see Fig. 32.61). The large amounts of callus that generally form with this type of fixation serve as an internal support for bone remodeling. The cast should be removed once the callus has bridged the fracture lines on all radiographic views.

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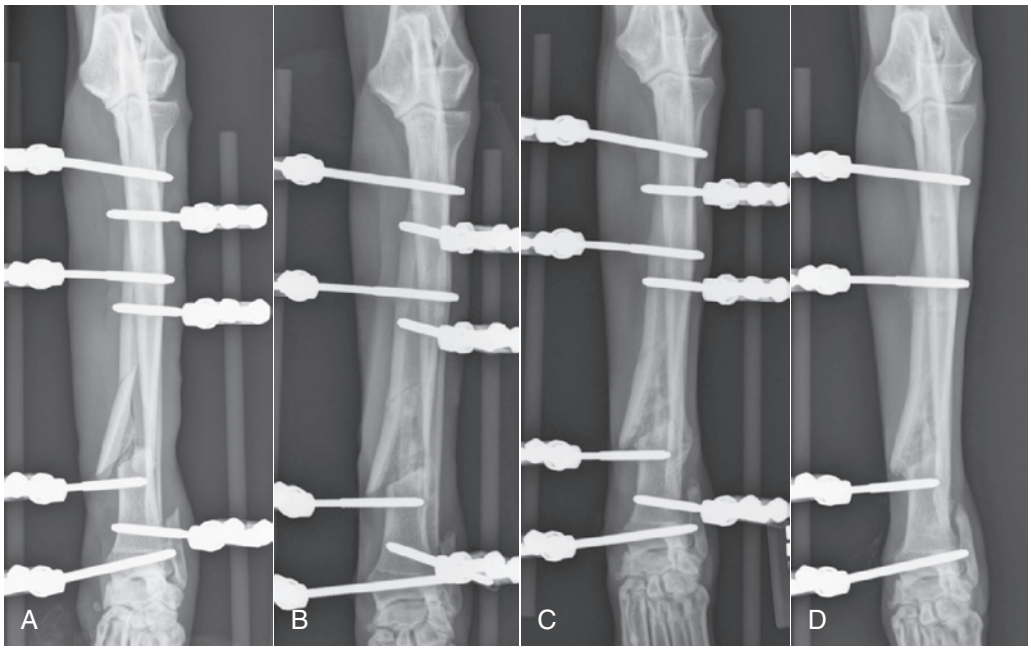


FIG. 32.65 (A) Fracture healing in comminuted nonreducible radial fracture treated with closed reduction and external fixation. (B–C) Radiographically endosteal bone appears first, followed by bone bridging between fragments with minimal periosteal callus formation. (D) Bone remodeling occurs after the fracture lines are bridged.

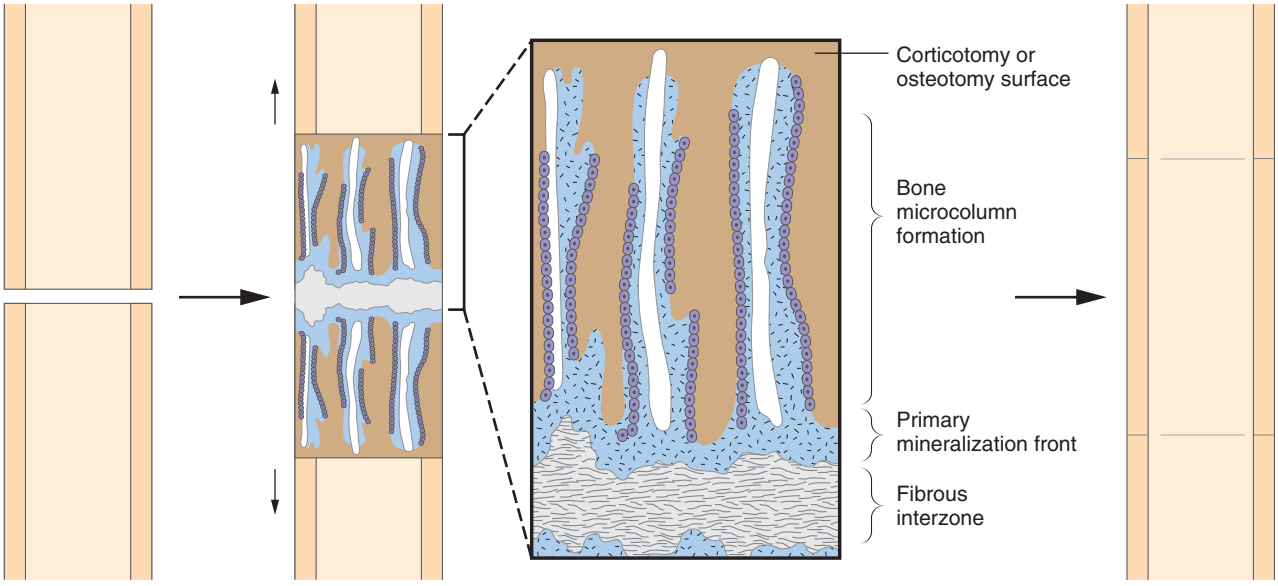


FIG. 32.66 During distraction osteogenesis, osteoid is laid down in parallel columns that extend from osteotomy surfaces centrally. Lamellar bone develops within these columns if the fracture is sufficiently stable.

Bone healing with external fixators may be direct, indirect, or somewhere between these extremes, depending on fracture type, mechanical environment afforded by external fixators, and degree of bone reconstruction. In general, fractures stabilized with external fixators have less periosteal callus formation compared with similar fractures treated with casts. In addition, fractures stabilized with external fixators develop more endosteal and bridging callus than periosteal callus. Radiographically, simple fractures that are anatomically reduced and rigidly stabilized with external fixators (e.g., bilateral external fixators with multiple pins) heal with minimal periosteal or endosteal callus. This is radiographically similar to the direct bone union response observed in fractures treated with plates and screws. As fixator stiffness decreases, increased callus is usually evident. When simple fractures are not anatomically reduced (e.g., with closed reduction) and fixators are not rigid, resorption of bone at fracture

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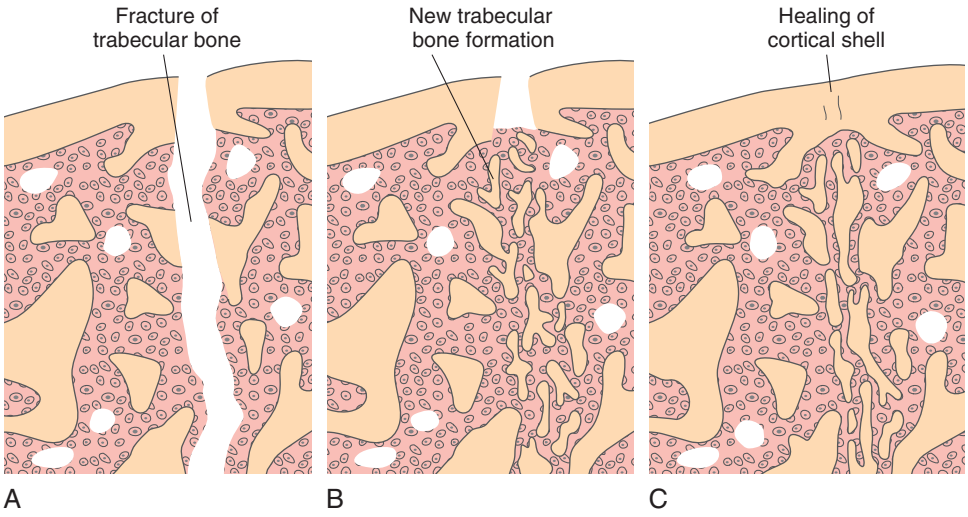


FIG. 32.67 (A) Trabecular bone responds to fracture by increasing osteoblastic activity. (B) New bone is deposited on existing trabeculae, and fracture gaps are filled with woven bone. (C) Bridging between trabeculae occurs before union of the cortical shell.



FIG. 32.68 Metaphyseal fracture healing after a tibial plateau leveling osteotomy, which is well stabilized, showing trabecular bone healing.

lines and callus formation are often evident. This occurs as a normal response of healing bone to high strain when the increased strain is concentrated in a single fracture line.

External fixators may be manipulated to provide optimal mechanical environment for bone formation and remodeling. In general, rigid fixation is recommended for the initial fixator to allow vascularization of the fracture and early bone formation. The external fixator frame may be destabilized approximately 6 weeks after surgery to increase the load on the healing fracture and stimulate bone formation. This should be based on radiographic evaluation for normal healing progression. Destabilization may be achieved by decreasing the planes of fixation or removing fixation pins. The external fixator is removed entirely when there is radiographic evidence of bone bridging all of the fracture lines.

The type of bone healing observed in comminuted fractures depends on how well the biological environment is preserved and on the rigidity of the fixation. When anatomic reconstruction and rigid fixation of all fragments are difficult or impossible in severely comminuted fractures, preservation of the biological environment is often best accomplished with closed reduction or limited exposure and rigid fixation (e.g., external fixators and bridging plates). Fractures treated by this method heal with endosteal bone formation and bone bridging between fragments. At 1 month, radiographs often show increased mineral density throughout the fracture site with minimal formation of periosteal callus. In most patients, bone formation is evident within 2 months, and remodeling of callus is evident 3 months after fixation (see Fig. 32.65). Computed tomography images show endosteal bone by 2 weeks and bridging endosteal and interfragmentary bone (which unites the bone fragments) by 12 weeks after fixation. There is minimal evidence of periosteal callus formation.

Fractures stabilized with pins and wires may heal by primary bone union if implants have rigidly stabilized the fractures (e.g., long oblique fractures managed with IM pins and multiple, appropriately applied cerclage wires). However, in most cases, rigid fixation is not achieved with pins and wires, and callus formation will be evident radiographically. Pins should be removed once bone has bridged the fracture lines in all views. Wires are not removed unless they cause problems through migration or interference with fracture healing.

Fractures that are anatomically reconstructed and rigidly stabilized (e.g., with plates and screws) heal by direct bone union. With direct bone healing, fracture lines disappear, and fractures appear devoid of bridging periosteal and endosteal callus (see Fig. 32.63). Because the implant is acting as callus to support the fracture during haversian remodeling, implant removal should generally be delayed until 6 to 12 months after surgery to allow adequate time for bone remodeling.

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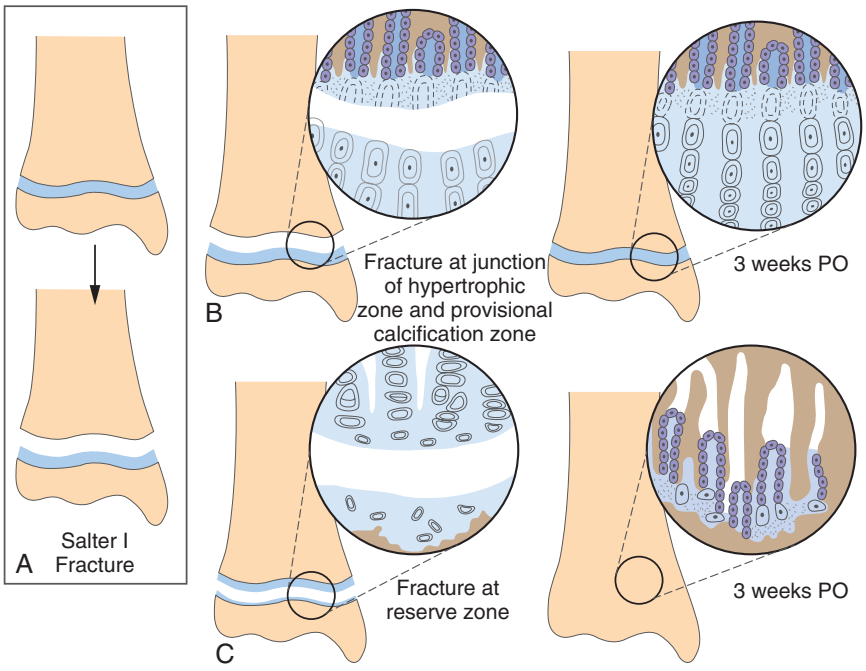


FIG. 32.69 Physeal cartilage healing. (A) Salter-Harris type I physeal fracture occurs through the hypertrophied zone of cartilage. (B) If reduced accurately, these physeal fractures heal by continued formation of cartilage. (C) If the fracture involves the reserve zone or if the germinal cells are damaged, healing occurs by endochondral ossification. *PO,* Postoperative.

COMPLICATIONS IN FRACTURE HEALING

Delayed Union

Fractures that heal more slowly than anticipated are classified as *delayed unions*. Most long bone fractures have radiographic evidence of bone bridging the fracture lines by 12 weeks, allowing confidence that bone healing will occur. With delayed unions, signs of progressive bone activity are visible on sequential radiographs, and bone union is anticipated, but not ensured. Factors contributing to delayed unions include the systemic status of the patient (e.g., malnutrition and anemia; see Chapter 10), nature of the trauma (e.g., high-energy fractured diaphysis with extensive soft tissue injury and possibly open fractures), local host postinjury response (e.g., inadequate cellular response for healing), fracture management (i.e., poor decision making, large fracture gaps, unstable implants or ones that are too rigid, radiation therapy), and pharmacologic factors (e.g., steroids, NSAIDs).

As long as implants are adequate and remain intact, the animal should have strictly supervised activity (physical rehabilitation; see Table 32.7 and Chapter 11) with no need for additional surgery. Cancellous bone autografts (see p. 991) may be added to speed healing before implant failure occurs. Loose or migrating implants should be removed, fractures stabilized appropriately, and cancellous bone autografts applied. Stable implants promote weight bearing, which accelerates fracture healing and the strength of the union.

Nonunion

Fracture *nonunion* is defined as an arrested fracture repair process, which requires surgical intervention to create an environment conducive to bone healing. Most nonunions are a result of poor decision making and technical failure on the part of surgeons, rather than biological failures attributable to the patient. Instability at the fracture site is the most common reason for nonunion. Common scenarios are distal radial diaphyseal fractures in toy-breed dogs stabilized with external coaptation, IM pins used with inadequate rotational and axial stability, external fixators of inadequate frame and pin size, loose cerclage wire that has migrated into the fracture site, and plates and screws that are inadequately sized for the patient. Poor biology at the fracture site, including fracture location, high-energy injury with extensive soft tissue destruction, and excessive surgical intervention, contributes to the development of nonunion. The risk factors for nonunion of fractures in cats are increased age, increased weight, open fractures, fracture of the proximal ulna and of the tibia, and the use of type II external fixators to stabilize tibial fractures.

Fracture nonunions are diagnosed when there is a lack of activity on sequential radiographs. A lucent line through fractures, representing cartilage and fibrous tissue, and ineffective callus formation at the fracture site are characteristic of the radiographic appearance of *vascular nonunions*. *Hypertrophic nonunions* are vascular nonunions with large amounts of nonbridging callus (Fig. 32.70). Hypertropic nonunions need stability to unite and are best treated by removal of loose implants and necrotic cortical bone pieces, joint alignment, and placement of a compression plate (see p. 1021). Cancellous bone grafts may be used, although the hypertrophic callus usually provides adequate cancellous bone for healing. If sequestered bone is removed, the resultant defects are filled with cancellous bone autografts, which may be placed during the plating procedure or after 5 to 7 days of open wound management. This delay allows formation of healthy granulation tissue beds before grafting. Samples for bacterial

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FIG. 32.70 Radiograph of dog with hypertrophic nonunion of femur. Notice formation of large periosteal callus that cannot bridge the fracture. The fracture was inadequately stabilized with an intramedullary pin and cerclage wires.

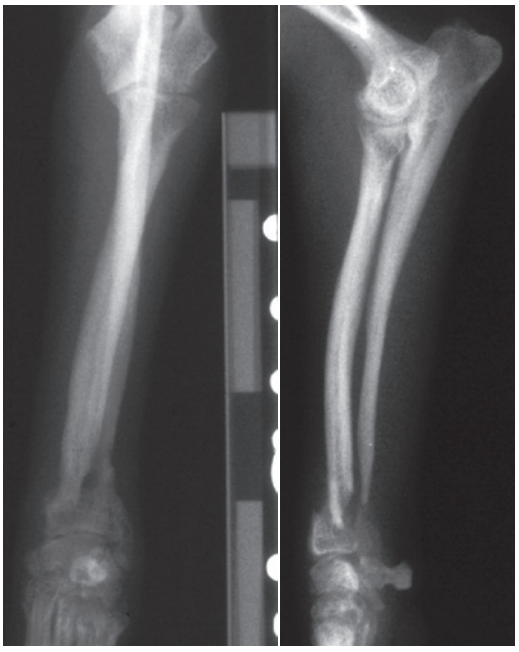


FIG. 32.71 Radiograph of dog with atrophic nonunion of the distal radius. Notice lack of callus formation at the fracture site.

culture and sensitivity should be obtained when nonunions are treated because concurrent osteomyelitis is common. If a radiographic or clinical diagnosis of osteomyelitis is made, treatment must include appropriate antimicrobial therapy. Plates should be removed after healing of infected nonunions because plates may serve as a nidus for continued infection.

Atrophic nonunions are biologically inactive pseudoarthroses. Radiographically, there is no evidence of bone reaction at fracture sites and the bone ends appear sclerotic (Fig. 32.71). Scintigraphy may confirm the lack of vascularity at the fracture. Histologically, fracture gaps are filled with fibrous tissue, necrotic bone, and cartilage. The medullary cavities are sealed with cortical bone. Atrophic nonunion requires surgery to remove fibrous tissue and open medullary canals. *En bloc* resection of the inactive bone and fracture site creates sufficient circumferential bony contact to allow excellent compression of the fracture with a bone plate. Plates and screws are generally the implants of choice to stabilize an atrophic nonunion, along with placement of autogenous cancellous bone graft. Swabs for bacterial culture and sensitivity should be obtained when atrophic nonunions are treated because concurrent osteomyelitis also occurs.

Osteomyelitis

Osteomyelitis is strictly defined as an inflammatory condition of bone and the medullary canal. In posttraumatic osteomyelitis associated with delayed union and nonunion, the most common cause is bacterial infection, promoted by instability at the fracture and the bone implant interfaces. Open fractures provide increased opportunity for osteomyelitis, as does vascular compromise and tissue ischemia. Posttraumatic osteomyelitis occurs with microbial colonization of the implants and adjacent damaged tissue. Biofilms occur with the formation of a conditioning film on the implant surface, which encourages adhesion of microorganisms. Additionally, bacteria produce a glycocalyx, which also assists the cell in adhering to the implant. The biofilm (bacteria, glycocalyx, and implant surface) protects the microorganism from antimicrobials and host defenses.

Radiographic evidence of osteomyelitis includes periosteal and endosteal bone formation, and bone resorption. There may also be loose or broken implants and nonviable bone fragments (sequestra) (Fig. 32.72). Radiographic and clinical evidence of osteomyelitis, coupled with a positive bacterial culture, provide the diagnosis. Treatment for posttraumatic osteomyelitis must include restoring a favorable fracture environment for healing (removal of loose implants and sequestered bone, disruption of the biofilm, stabilization of the fracture) and appropriate antimicrobial therapy for at least 4 to 6 weeks.

Malunion

Malunions are healed fractures in which anatomic bone alignment was not achieved or maintained during healing. Malunions may have a deleterious effect on function. Angular deformities are characterized by loss of correct parallel relationships between joints above and below the fractured bone. Deformities may be classified as *valgus, varus, internal or external rotation, translation, antecurvatum,* or *recurvatum* (Fig. 32.73). When severe, these deformities affect limb function and may precipitate a gait abnormality and osteoarthritis of adjacent joints. Shortening of affected bones may also rarely occur. A shortened bone in a single bone system (femur and humerus) can be compensated for by extension of adjacent joints; however, shortening of a single bone in a paired bone system (radius-ulna, tibia-fibula) causes incongruity in alignment of adjacent joints. Malunion should be treated with corrective osteotomy if it adversely affects the animal's ability to ambulate or is likely to lead to osteoarthritis.

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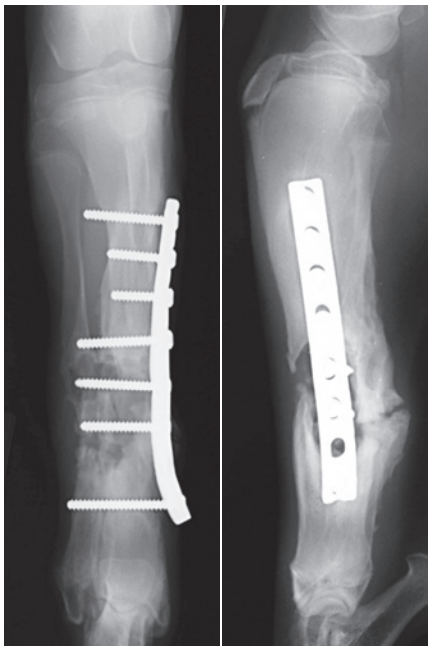


FIG. 32.72 Radiograph of dog's tibia with osteomyelitis of the tibia. Notice bone resorption around the screws indicating loose implants, increased bone proliferation, and the bone resorption around the sequestered lateral cortex.

OSTEOTOMIES

Osteotomies are procedures in which the bone is cut into two segments. *Apophyseal osteotomies,* such as trochanteric or acromial osteotomies, are performed to enhance surgical exposure of a joint (see pp. 1100 and 1049). *Corrective osteotomies* are elective procedures in which the diaphysis or metaphysis of the bone is cut, realigned, and stabilized until union occurs. The indications for corrective osteotomies are to (1) correct angular deformities caused by physeal trauma or fracture malunion by realigning joint surfaces, (2) establish adequate bone length to shortened bones, (3) correct torsional deformities caused by physeal trauma or fracture malunion, and (4) improve articular configuration. *Ostectomies,* or removal of a bone segment, are generally performed in immature dogs to allow unimpeded growth of paired long bones where one of that pair has suffered early physeal closure. They may also be used to improve joint congruity (Table 32.8).

The objective in performing corrective osteotomy is to return a limb to normal function by restoring normal alignment to the bone or joint. Presurgical planning is a prerequisite for success. The surgeon must determine the correct location(s) to cut the bone, whether the soft tissue can tolerate the strain of repositioning the bone, and the appropriate fixation to maintain stability until bone union occurs. Radiographs or computed tomography provide the most useful information for planning an osteotomy. Craniocaudal and mediolateral radiographs of the affected bone, including the proximal and distal adjacent

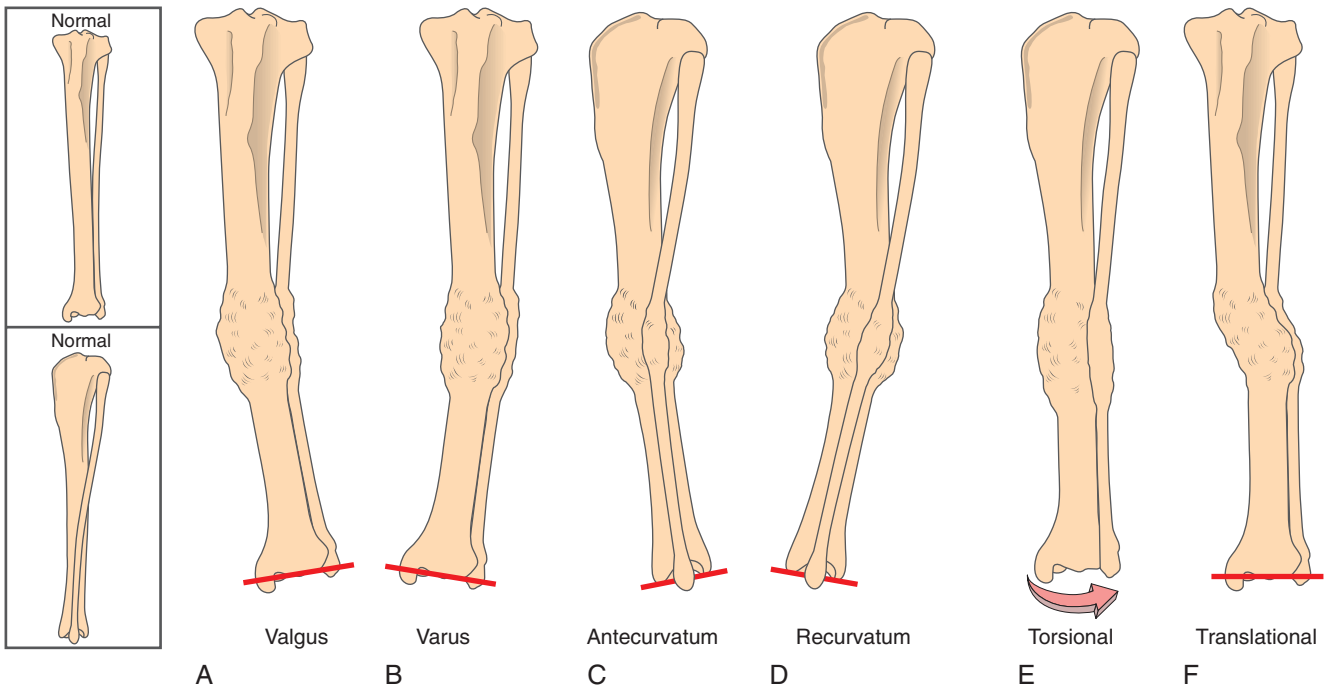


FIG. 32.73 Malalignment of bone. (A) Valgus is lateral angulation of distal segment of bone. (B) Varus is medial angulation. (C) Antecurvatum is caudal angulation. (D) Recurvatum is cranial angulation of distal segment of bone. (E) Torsion is medial or lateral twisting of distal segment of bone. (F) Translation is displacement of a distal segment of bone with the joint surfaces parallel.

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joints, are required to evaluate the deformity. Similar radiographs of the contralateral bone are used as a reference. Significant angular deformity in both views indicates that the deformity lies in an oblique plane. Oblique views may be needed to determine the plane of the deformity. The degree of angular deformity and length discrepancy can be determined from radiographs. Rotational deformity may be estimated from radiographs but is usually best determined from physical examination or computed tomography. Rotational deformity is measured directly on the dog by comparing the relationship of adjacent bones, such as the radius and metacarpal bones, during flexion and extension of the adjoining joint. Computed tomography permits three-dimensional reconstruction of the bones to further evaluate and measure deformity. For very complex cases, three-dimensional models may be generated from computed tomography data to enhance surgical planning.

Outcomes of improper osteotomy planning are incomplete correction of the deformity, implant failure, delayed union, infection, and nonunion.

TABLE 32.8 Indications for Corrective Osteotomy Procedures

Deformity	Osteotomy Technique
Angular	Opening wedge Closing wedge Reverse wedge Dome
Shortening	Transverse lengthening Stair step Continuous distraction
Rotational	Derotational transverse
Angular, shortening, and rotational	Opening wedge Continuous distraction
Incongruent joint	Triple pelvic Intertrochanteric Tibial plateau leveling Transverse lengthening Stair step Ulnar ostectomy Continuous distraction

Corrective Osteotomy for Deformities

Deformities may result from growth abnormalities (usually premature physeal closure) or fracture malunion. Immature animals with premature closure of the physis in the radius or ulna may be treated with a segmental ostectomy to release the restraint of the affected bone to allow the unaffected bone to grow normally (see p. 1081); however, in many cases the patient will have inadequate remaining growth to adequately correct the deformity. Deformities in mature animals are treated with corrective osteotomy. Rotational deformities can be corrected with a *transverse osteotomy,* after which the distal segment is rotated to restore proper bone alignment (Fig. 32.74A). Angular limb deformities are treated with *closing wedge* or *opening wedge*

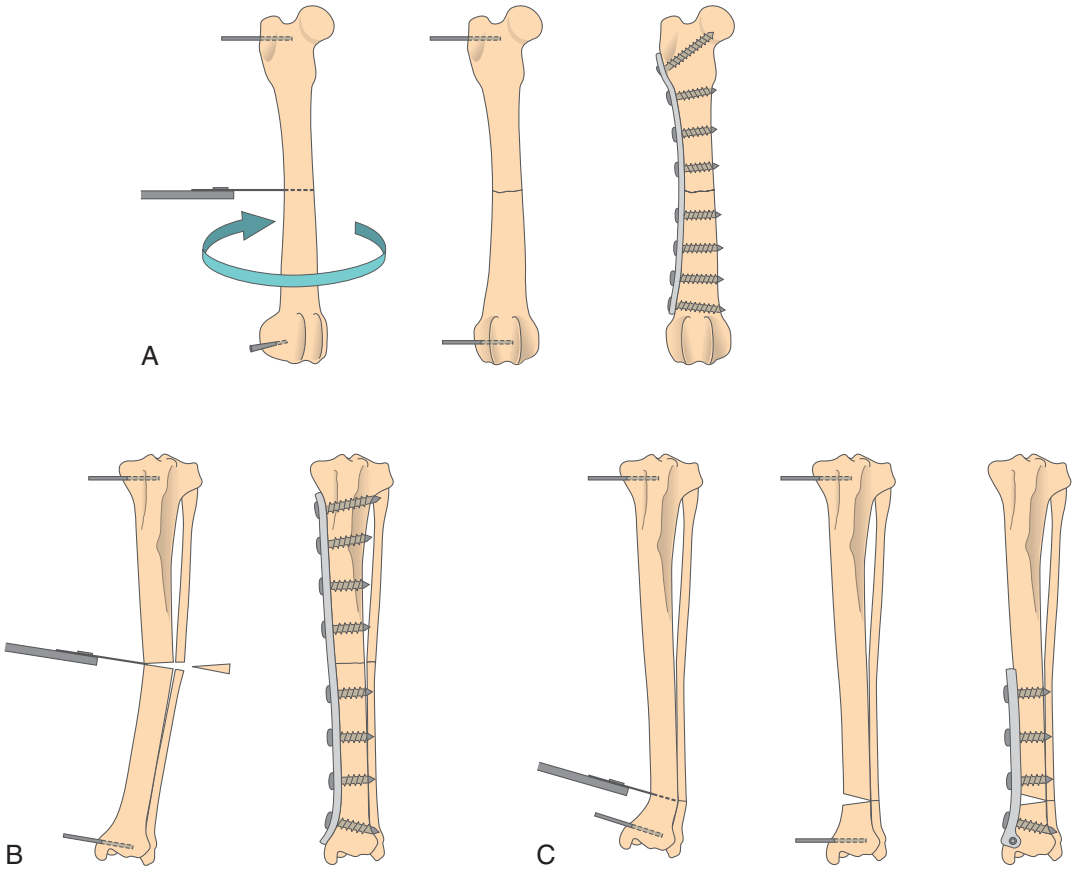


FIG. 32.74 Corrective osteotomies used to treat bone deformities. (A) Derotational osteotomy. (B) Closing wedge osteotomy. (C) Opening wedge osteotomy.

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osteotomy to restore the proper alignment of the proximal and distal joint surfaces (see Fig. 32.74B–C). Opening wedge osteotomy preserves bone length, whereas closing wedge osteotomy provides a more stable fracture surface. These osteotomies may be stabilized with bone plates or external fixators.

Deformities with significant shortening of the affected bone require a lengthening procedure to restore limb function. *Lengthening osteotomies* may be acute or progressive. The soft tissue often limits one-stage correction, whereas *progressive distraction* overcomes this limitation and provides for new bone formation during distraction osteogenesis (see p. 1005). Progressive distraction may also be used to treat growing dogs with complete physeal closure. Distraction osteogenesis is usually accomplished using a ring fixator apparatus.

Corrective Osteotomy for Joint Incongruity

Most frequently performed corrective osteotomies provide for realignment of an incongruent joint. Dogs with early evidence of hip dysplasia are candidates for *triple pelvic osteotomy* designed to rotate the dorsal acetabular rim to provide additional stability to the hip joint (see p. 1215) (Fig. 32.75A). *Release osteotomies* or *ostectomies* are performed when there is an incongruent elbow to allow the ulna to return to a more normal relationship with the distal humerus (see Fig. 32.74B). These procedures are also used to relieve the pressure on an ununited anconeal process and/or fragmented coronoid process (see p. 1182). If the incongruity is too great, a lengthening osteotomy may be indicated to restore elbow alignment. Dogs with cranial cruciate ligament rupture may be treated with a TPLO to improve the mechanics of the joint (see p. 1232) (see Fig. 32.75C). Occasionally, dogs with patellar luxation and associated femoral varus or valgus, or rotation, are treated with a corrective osteotomy to realign the femur.

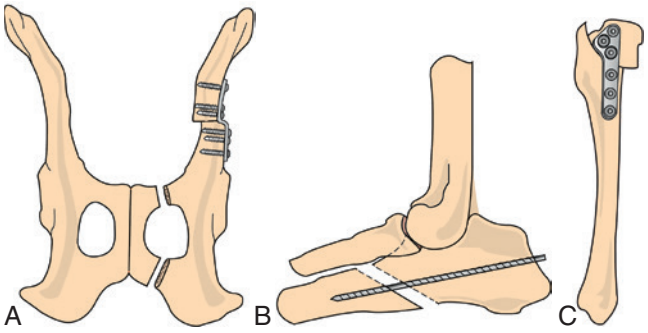


FIG. 32.75 Corrective osteotomies used to treat joint disease. (A) Triple pelvic osteotomy. (B) Dynamic ulnar osteotomy. (C) Tibial plateau leveling osteotomy.

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33

Management of Specific Fractures



MAXILLARY AND MANDIBULAR FRACTURES

DEFINITIONS

Maxillary fractures and **mandibular fractures** may result from trauma, severe periodontitis, or neoplasia. **Periodontitis** is an inflammatory reaction of the tissue surrounding a tooth that usually results from extension of gingival inflammation into the periodontium.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Maxillary and mandibular fractures are usually caused by head trauma, and concurrent injuries (e.g., upper airway obstruction, central nervous system trauma, pneumothorax, pulmonary contusions, and traumatic myocarditis) are often present. These abnormalities may be acutely life-threatening and may require prompt diagnosis and treatment. Definitive fracture repair must often be delayed until the animal has been appropriately stabilized. Mandibular fractures occasionally occur as a result of bone loss associated with severe periodontitis. Teeth extraction should be performed with care in older patients with severe periodontitis to prevent this complication. With severe periodontitis, bone healing may be impaired. Diseased teeth must be extracted before fracture stabilization in these patients. Pathologic fractures may also result from mandibular neoplasia (see p. 352). Histopathologic examination of bone from animals with mandibular fractures is warranted unless fractures are clearly caused by trauma. Mandibular fractures associated with neoplasia are treated by mandibulectomy rather than by definitive fracture repair (see p. 336).

The teeth occupy a large portion of the mandible and are integral components of normal mandibular structure (Fig. 33.1). Teeth involved in fractures should not be removed unless they are loose. This is especially important with fractures involving the caudal mandibular body because the large premolar and molar teeth occupy a substantial portion of this bone; they are essential contributors to fracture stability. Fractures extending along the tooth root from the alveolar crest to the apex of the tooth are at risk for pulpal necrosis and periapical bone resorption. Temporary or permanent root canal therapy at the time of fracture stabilization may promote bone healing. If affected teeth are not treated at surgery, periodic evaluation for evidence of infection or loosening is necessary postoperatively to determine whether endodontic therapy or extraction is indicated. The anatomy and location of tooth roots must be considered when implants are applied to the mandible. Damage to tooth roots from pins, wires, drill bits, and screws may cause sufficient damage that extraction becomes necessary after fracture healing.

DIAGNOSIS

Clinical Presentation

Signalment

Although traumatic mandibular or maxillary fractures may occur in dogs at any age, young dogs are at greater risk. However, because the canine population reflects the increase in small and toy breeds, as well as those in urban leashed populations, the mean age of dogs with mandibular fractures has increased. Pathologic fractures of the mandible are particularly common in geriatric, small breed, and toy breed dogs (e.g., poodles) that have not had regular dental prophylaxis and are fed soft food and table scraps.

History

Usually, a history of trauma (e.g., stepped on, hit by car, kicked by horse) is noted in animals with traumatic oral fractures. Mandibular symphyseal fractures and fractures of the hard palate may occur in cats that fall from great heights ("high-rise syndrome"). Tooth extraction may be associated with pathologic fractures of dogs with severe periodontitis.

Physical Examination Findings

Animals with mandibular fractures may drool excessively and exhibit pain on opening of the mouth, and they are often reluctant to eat. Saliva may be blood tinged, but profuse bleeding is uncommon. Crepitation and instability can often be palpated during careful oral examination; however, thorough inspection of these structures for mucosal wounds and crepitation often requires general anesthetic. Mandibular symphyseal fractures allow one hemimandible to be moved separately from the other. Less instability is usually present with maxillary fractures than with mandibular fractures. The teeth should be examined carefully for evidence of trauma. A biopsy should be taken of fractures associated with notable bony lysis or proliferation.

NOTE Use endodontic therapy to treat teeth fractured above the gumline; extract teeth that are fractured below the gumline.

Diagnostic Imaging

Radiographs of the mandible and maxilla generally require anesthesia. Thorough radiographic examination of the skull usually requires a minimum of four radiographic views: dorsoventral

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ABSTRACT

The application of the principles of fracture repair vary significantly depending on the specific bone involved and particular type of fracture. This chapter provides effective means of stabilization of the most common fractures reported in dogs and cats.

KEYWORDS

Fracture
articular
diaphysis
physeal
displacement
comminution
reduction
stabilization

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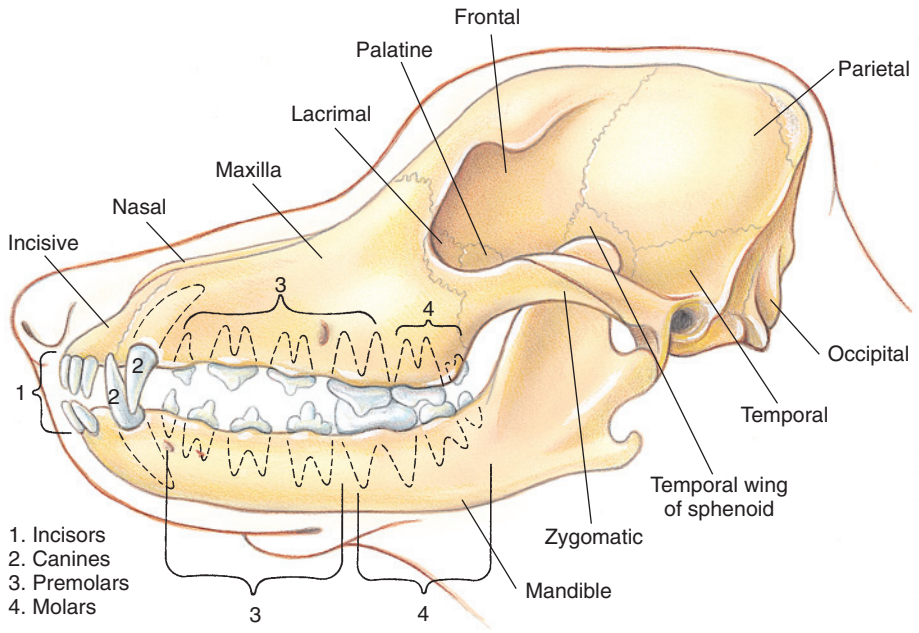


FIG. 33.1 Skull anatomy. Note that the tooth roots occupy a large portion of the mandible. Normal relationships of the maxillary and mandibular dental arcades are illustrated.

or ventrodorsal, lateral, and right and left lateral obliques. Skull radiographs are often difficult to interpret because of the presence of multiple overlying structures; therefore comparison with a normal skull is often helpful. Symmetry between the two sides is critical to interpretation, thus care in positioning is critical. Additional specialized views, such as intraoral views, may be necessary for complete evaluation. Computed tomography (CT) can help identify fractures in the caudal mandibular body, vertical ramus, and mandibular condyle that may be difficult to detect radiographically. CT is often more efficient than survey radiography for evaluating complex mandibular and maxillary fractures.

Laboratory Findings

Specific laboratory abnormalities are not present with mandibular or maxillary fractures from any cause. Traumatized animals should have sufficient bloodwork done to determine whether contraindications to anesthesia exist.

DIFFERENTIAL DIAGNOSIS

Animals with mandibular or maxillary fractures should be evaluated to determine whether fractures are the result of trauma or underlying pathology (e.g., periodontitis, neoplasia, metabolic disease; see previous discussion).

MEDICAL MANAGEMENT

A tape muzzle may be applied to support the mandible if minimal displacement of fracture fragments (usually fractures of the ramus) occurs, if dental occlusion is adequate, and if the fracture assessment score (see p. 985) is favorable and dictates early union. A muzzle can be made from two pieces of tape placed with the sticky sides together and fashioned into a circle that fits over the dog's nose. A similarly made piece of tape extends from the circle around the head and behind the ears (Fig. 33.2). The muzzle should allow the dog to open its mouth enough to lap water and eat gruel. To allow adequate healing, muzzles should remain in place for 6 weeks. Dogs with mandibular plus maxillary fractures may not tolerate tape muzzles because the muzzle may put pressure on the maxillary fracture. Because of the cat's short nose, tape muzzles are difficult to apply and maintain. Similarly, it may be difficult to maintain a tape muzzle in brachycephalic breeds.



FIG. 33.2 Stabilization of a minimally displaced mandibular fracture with a tape muzzle. Tape muzzles can also be used preoperatively to support the mandible.

INTERDENTAL STABILIZATION TECHNIQUES

Oral stabilization techniques incorporating wire ligatures and/or dental acrylics attached to the teeth allow closed reduction of mandibular and maxillary fractures, preserving the periosteal attachments and blood supply. These techniques are especially useful for fractures rostral to the first mandibular molars and

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FIG. 33.3 Stabilization of a mandibular fracture in a dog with a wire-reinforced acrylic splint. (Courtesy Dr. Sandra Manfra Marretta and Dr. Robert Ulbricht.)



FIG. 33.4 Stabilization of a caudal mandibular fracture in a cat with acrylic bonding of the maxillary and mandibular canine teeth. (Courtesy Dr. Sandra Manfra Marretta.)

for comminuted fractures in which anatomic reduction may not be possible. Maxillomandibular fixation may also be used to stabilize temporomandibular joint luxations.

Application of Interdental Fixation

Interdental fixation is limited to use in mandibular and maxillary fractures occurring between intact canine and carnassial teeth. In dogs, interdental wire ligatures are combined with acrylic intraoral splints to provide stable fixation. In cats, acrylic intraoral splints may be used without metallic reinforcement. Acrylic materials with a low curing temperature are recommended for intraoral use.

The crowns of the mandibular teeth are cleaned, pumiced, and acid-etched to improve acrylic resin bonding. The mandible is aligned using interdigitation of the teeth as a landmark for accurate reduction. Interdental wire ligatures (0.3- to 0.5-mm wire) are applied to initially stabilize the fracture(s). Dental acrylic is applied to the prepared teeth to stabilize the fracture (Fig. 33.3). The splints are removed when radiographic evidence reveals bone bridging the fracture by sectioning the splint interdentally and removing the splint in sections.

Application of Maxillomandibular Fixation

Bonding the maxillary and mandibular canine teeth together in anatomic alignment is an alternative to the tape muzzle for conservative fracture treatment. The canine teeth are cleaned, pumiced, acid-etched, and aligned with dental composite, leaving the mouth open approximately 1 cm. Although simple, the technique requires that all four canine teeth be intact and healthy. This technique can be used in dogs and cats (Fig. 33.4).

NOTE Warning: Animals treated by limiting their ability to open their mouths are at risk for hyperthermia if placed in a warm environment and for aspiration pneumonia if vomiting occurs.

SURGICAL TREATMENT

The appropriate method of treating mandibular and maxillary fractures is determined on the basis of the fracture assessment score (see p. 985) and fracture location. Conservative treatment with a tape muzzle or dental composite bonding may be appropriate for some fractures (see previous discussion). Internal fixation systems applicable for mandibular fractures include orthopedic wire, Kirschner wire (K-wire), and plates and screws. Intramedullary (IM) pins are contraindicated because the mandibular canal contains the mandibular alveolar artery and the inferior alveolar nerve. External fixators (both standard and acrylic frames) are very effective for nonreducible comminuted fractures. Many maxillary fractures are nondisplaced and require only conservative therapy; however, segmental maxillary fractures or depressed fracture lines may require repositioning and stabilization. Mandibular or maxillary fractures that alter normal occlusion should be reduced and stabilized. Maxillary fractures that result in nasal malpositioning or instability should be reduced and stabilized. Interfragmentary wires may be used to stabilize some maxillary fractures where only a few large bone fragments are involved and stable anatomic reduction is possible (Fig. 33.5), especially with K-wires incorporated into the fixation to prevent overriding of the fragments. Maxillofacial miniplates are more effective for restoring anatomic contour to comminuted and depressed maxillary fractures involving thin portions of the skull.

Application of Interdental Wires

Interdental wires are placed around teeth adjacent to fracture lines. *Position the wires securely in the bone around the tooth's neck to prevent them from sliding off the crown. Place the wires through guide holes that are drilled between the teeth and through the superficial cortical bone surface. Pass the wire through the guide holes, circling the teeth, and tighten. Bend the ends of the wire into the mucosa (Fig. 33.6).*

Application of Interfragmentary Wires

Interfragmentary wires are ideal for stabilizing relatively simple, reconstructible mandibular and maxillary fractures. Large-gauge wire (18- to 22-gauge), properly applied, provides adequate fracture support (Table 33.1); however, wires may be difficult to position and tighten unless certain application guidelines are followed. The largest gauge wire that can be manipulated should

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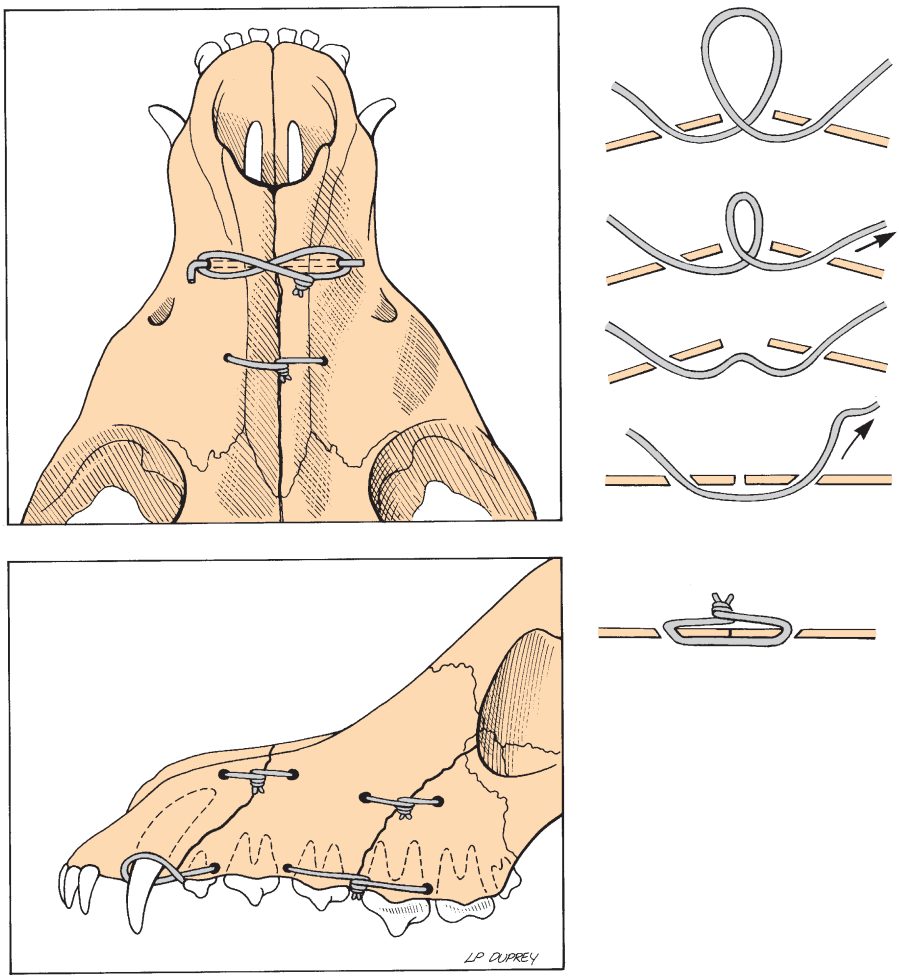


FIG. 33.5 Stabilization of maxillary fractures with K-wire and orthopedic wire. Wires are applied perpendicular to the fracture and are tightened to compress it. K-wires are used to prevent collapse of fragments. Occasionally a canine tooth can be used to secure the wire. To facilitate passing cerclage wire, angle the drill holes toward the fracture. Loop a long piece of wire under the bone and through the second hole. Pull the wire until kinks caused by looping are beyond incorporation into the final cerclage, and tighten the wire.

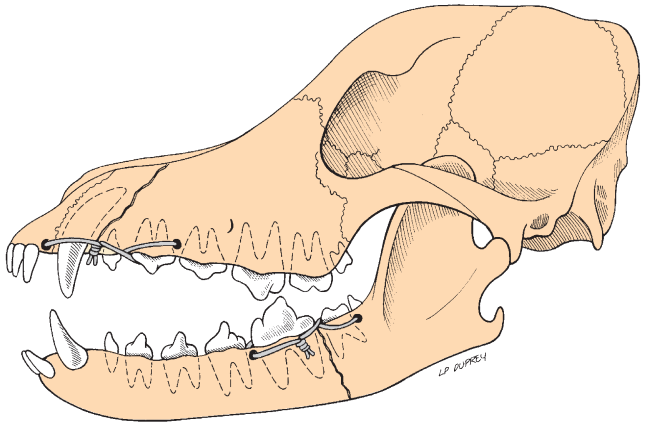


FIG. 33.6 Interdental wires may be placed through holes drilled in the maxillary or mandibular bone to prevent the wire from slipping.

TABLE 33.1 Comparable Wire Sizes for Fracture Stabilization

Gauge	Millimeter	Inch
16	1.25	.049
18	1.00	.040
20	0.80	.035
		.032
		.030
22	0.60	.028
24		.020

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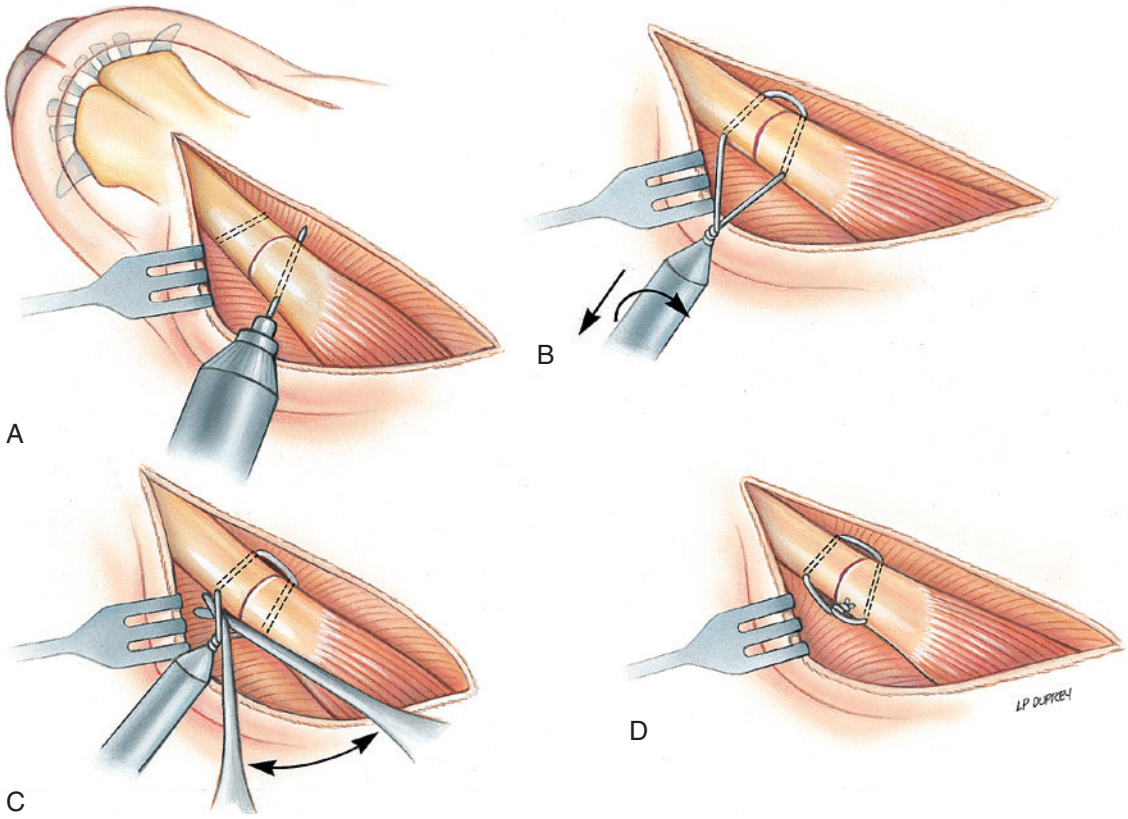


FIG. 33.7 (A) To place an interfragmentary wire on the mandible, angle the drill holes toward the fracture. (B) Place the wire and begin the twist while placing traction on the wire. (C) To ensure that the wire is in contact with the far cortex, insert a forceps between the twist and the near cortex, and lever the wire away from the near cortex. (D) Finish tightening the wire, cut the twist, and bend it into the bone.

be used. *Use K-wires to drill holes in the bone, 5 to 10 mm from the fracture line (Fig. 33.7A). Position these holes so that the wire will be perpendicular to the fracture line when it is tightened. Slope the drill holes toward the fracture line; this results in obtuse angles on the bone side opposite to where the wire knot will be tightened. This positioning allows the wire to slide into position easily and enhances tightening efforts. Use long segments of wire to facilitate wire passage and allow manipulation of any wire kinks away from the area of wire that is to be tightened. Tighten the wire using a twist knot or a tension loop (see p. 1014). Twist it in such a manner that equal tension is applied to both strands (Fig. 33.7B). Use a periosteal elevator or large tissue forceps to lever the orthopedic wire under the twist and eliminate slack (Fig. 33.7C). When tightened, bend the wire perpendicular to the wire surface and away from the gingival margin. Cut the wire, twist the ends, and bend them into the bone surface (Fig. 33.7D).* Optimally, wires should be located near the oral margin to neutralize forces that tend to disrupt fractures. Because this margin also contains the teeth, care must be used to position drill holes between teeth or tooth roots. When multiple wires are used, all holes should be drilled and wires positioned before any wires are tightened. Wires are tightened beginning at the caudal fracture line and working toward the symphysis. If bending or slippage is encountered with oblique or transverse fractures, K-wires with figure-eight–patterned orthopedic wire may be used. The K-wire helps prevent sliding or bending while the orthopedic wire is tightened.

Application of Bone Plates and Screws

Bone plates can be used to stabilize single or comminuted mandibular fractures (see p. 1016). Miniplates, often made of titanium, provide excellent stabilization, rapid healing, excellent long-term function, and few complications.[1] Plates are applied to the ventrolateral mandibular surface. Care must be used to contour the plate correctly because the mandible aligns with the plate as the screws are tightened; malalignment results in malocclusion. Screws are positioned to avoid tooth roots. Maxillofacial miniplates may offer easier contouring and screw placement in mandibular and maxillary fractures and may be combined with standard straight plates and reconstruction plates (Fig. 33.8). Locking screws and plates may be particularly useful in maxillary and mandibular fractures as the increased stiffness of these implants provides greater stability in these regions of limited bone stock when compared to nonlocking implants.

Application of External Skeletal Fixators

External Fixators

External fixators (see p. 998) can be used to stabilize mandibular body fractures if sufficient bone is present to hold fixation pins. Fixation pins are placed percutaneously through the mandibular body, avoiding tooth roots. Type I fixators are applied to the ventrolateral mandibular surface with at least two pins inserted on either side of the fracture line. Positive-profile, end-threaded

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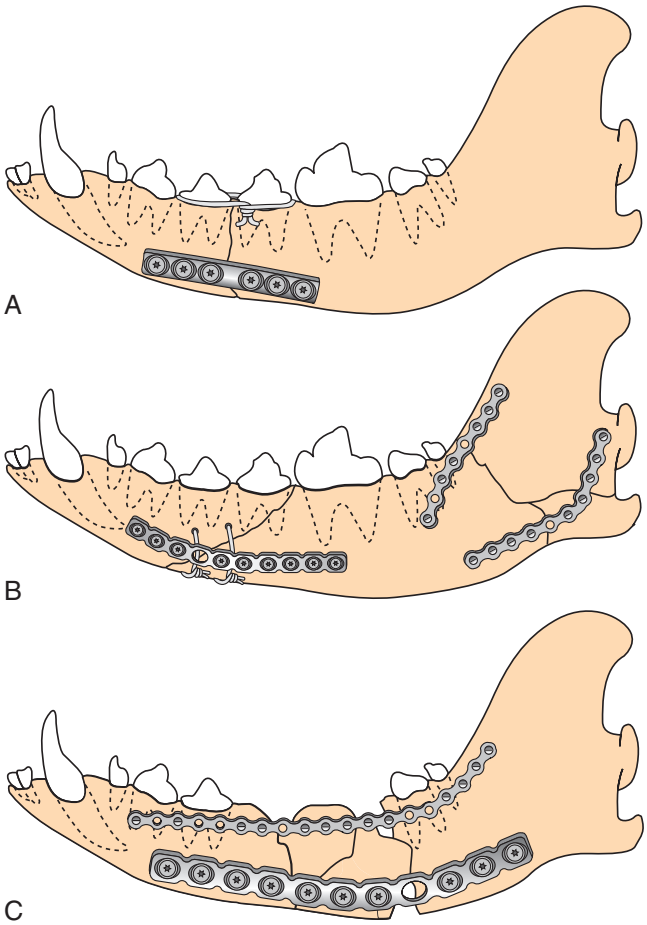


FIG. 33.8 (A) Compression plates are applied to the lateral mandibular surface to stabilize transverse fractures. Avoid penetrating the tooth roots with screws. An interdental wire may function as an additional tension band fixation. (B) Oblique fractures may be stabilized with hemicerclage wires and a plate. Fractures of the vertical ramus may be stabilized with miniplates applied to the thicker rostral and ventral borders of the bone. (C) Comminuted fractures of the mandible may be bridged with a reconstruction plate. Concurrent miniplate fixation adds stabilization to the tension surface of the mandible. (Modified from Johnson AL, Houlton JEF, Vannini R, eds. *AO Principles of Fracture Management in the Dog and Cat*. New York: Thieme; 2005.)

pins are used to increase the fixator's holding power. For bilateral mandibular body fractures, a type II fixator may be constructed using bilateral and unilateral fixation pins. A connecting bar is placed on either side of the dog's jaw (Fig. 33.9).

Acrylic Fixators

Dental acrylic, used to replace clamps and connecting bars, is a versatile rigid fixation for mandibular fractures, especially severely comminuted fractures. After pins have been placed in appropriate locations in the mandible (use fixation pins of appropriate size for the bone area), the ends are bent parallel to the skin (Fig. 33.10A). To evaluate dental occlusion and reduce fractures, the animal's mouth is closed. If necessary, Kirschner clamps and a connecting bar can be placed to hold the fracture temporarily in reduction while the acrylic is being mixed and molded (see Fig. 33.10B). Soft tissue is protected by laying wet sponges beneath the pins. After the acrylic is mixed, it is molded over the bent pins to form a connecting bar (see Fig. 33.10C). Alternatively, plastic tubing may be impaled over the pins (do not bend them) and then used as a mold for the liquid-phase acrylic. The acrylic splint can be curved around the rostral portion of the mandible.

NOTE Acrylic bars are versatile because they can accommodate various sizes of fixation pins placed in multiple planes.

Animals with mandibular fractures that have high fracture assessment scores of 8 to 10 (e.g., simple fractures in young animals; see p. 985) may be treated with tape muzzles, interdental fixation, interdental wiring, or interfragmentary wiring techniques, depending on fracture location. Interfragmentary wire is usually sufficient to stabilize simple, bilateral, mandibular fractures if they can be anatomically reconstructed and if fracture assessment scores indicate rapid healing (Box 33.1). It has the advantage of providing internal fixation with minimal aftercare. With moderate fracture assessment scores of 4 to 7 (e.g., larger or older dogs with longer healing times) and if bilateral fractures can be anatomically reconstructed, interfragmentary wires, interdental fixation, external fixation, or bone plates and screws may be used for fracture fixation. A cancellous bone autograft can be used to promote rapid bone union in these patients. With low fracture assessment scores of 0 to 3 and comminution, bone loss, or severe soft tissue damage, mandibular fractures are best treated with closed reduction and external fixator application or bridging plate application. Closed reduction techniques preserve the biological environment, but external fixators require intensive aftercare, and the pin-bone interface loosens over time. Mandibular alignment is determined by observing dental occlusion. Plate and screw fixation can be used to stabilize mandibular fractures with cortical bone loss; however, the plate must be carefully contoured to the normal shape of the fractured bone. If open reduction is performed in patients with low fracture assessment scores, autogenous cancellous bone or demineralized bone matrix with cancellous bone allograft can be used to facilitate healing.

BOX 33.1 Implant Use for Maxillary and Mandibular Fractures According to Fracture Assessment Score (FAS)

FAS 0 to 3
Closed reduction and external skeletal fixation
Bone plate and screws
Maxillomandibular fixation for vertical ramus

FAS 4 to 7
Interdental fixation techniques
Bone plates and screws
External skeletal fixation

FAS 8 to 10
Tape muzzle
Interdental fixation techniques
Interfragmentary wiring
Cerclage wires (symphyseal fractures)

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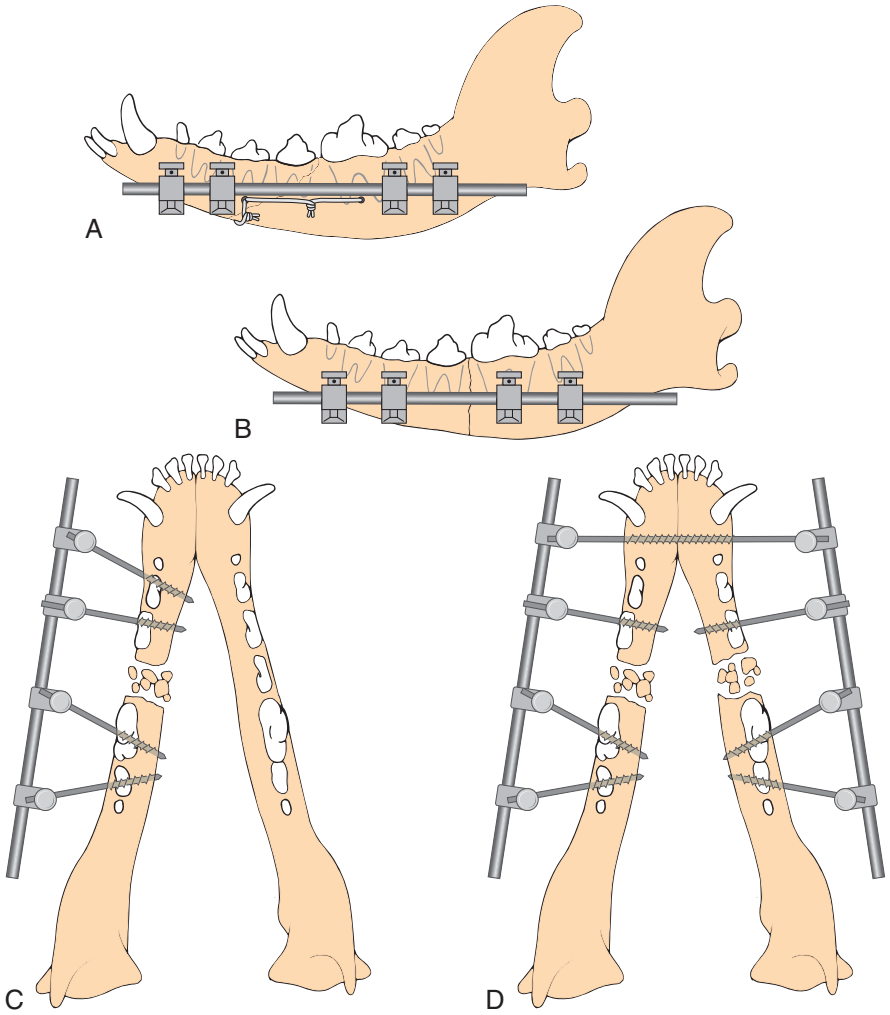


FIG. 33.9 (A) An external fixator may be combined with interfragmentary wire to stabilize oblique mandibular fractures or (B) may be used by itself for transverse fractures. (C–D) When placing a type I or II external fixator to stabilize comminuted mandibular fractures, use positive-profile, end-threaded pins if the fracture assessment score indicates prolonged healing. Avoid penetrating tooth roots with fixation pins.

Dogs with severely comminuted fractures of the vertical ramus may be treated with a tape muzzle, relying on the heavy masseter muscle to maintain fragment alignment. In some cases, maxillomandibular fixation can be used to maintain mandibular and maxillary alignment. Although most cats tolerate maxillomandibular fixation well, dogs may be more difficult to manage postoperatively.

Preoperative Management

After the status of the animal has been determined, mandibular fractures in most dogs may be gently reduced and temporarily held in position with a tape muzzle until a definitive stabilizing procedure can be performed (see Fig. 33.2). However, mandibular fractures in cats and brachycephalic dogs cannot be easily stabilized with tape muzzles and are often not treated until surgery. Because the oral cavity contains numerous bacteria, prophylactic antibiotics are recommended. Infections are rare, however, because of the extensive vasculature supplying this area. If infection is likely and if open reduction is performed, bacterial cultures can be submitted during surgery. Analgesics should be provided to posttraumatic animals (see Chapter 13).

Anesthesia

Anatomic reconstruction or realignment of mandibular cortices is mandatory to provide proper dental occlusion. Simple mandibular fractures can be anatomically reconstructed using bone cortex as a guide. With complex fractures or cortical bone loss, however, dental occlusion must be used to guide mandibular realignment. Because mandibular and maxillary teeth closely interdigitate, precise alignment of upper and lower arcades is necessary. If precise dental occlusion cannot be determined with the endotracheal tube in place, it may be repositioned through a pharyngotomy incision (Fig. 33.11) or a temporary tracheostomy. This allows the mouth to be completely closed during surgery, which facilitates determination of adequate dental occlusion. After surgery, the endotracheal tube is removed, and the pharyngotomy incision is allowed to granulate closed. If a temporary tracheostomy is performed, it is closed primarily.

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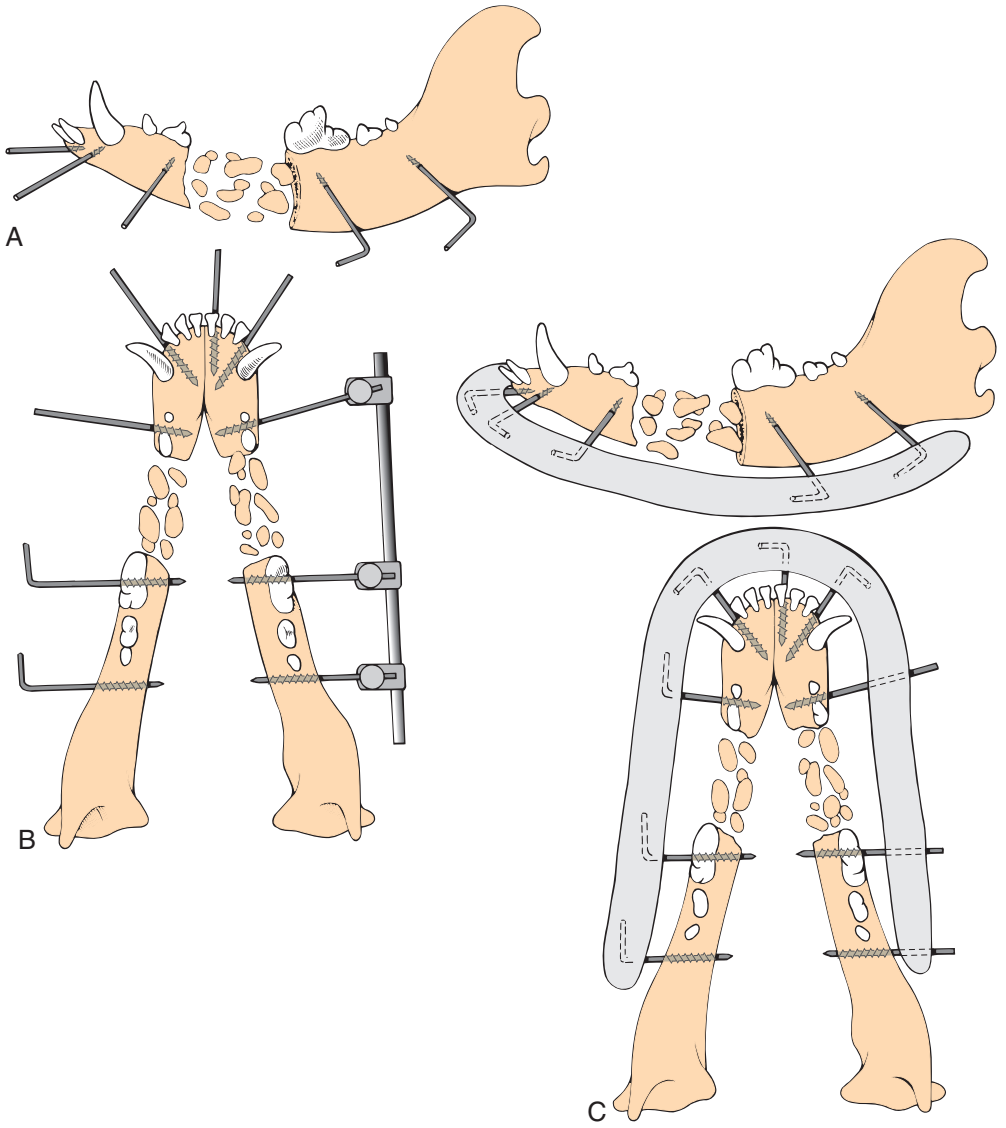


FIG. 33.10 Acrylic splints may be used to accommodate fixation pins of various sizes that are placed in multiple planes. (A) Place the fixation pins where there is adequate bone. Use positive-profile, end-threaded pins when the fracture assessment score indicates prolonged healing. (B) Use a temporary fixation bar to maintain mandibular alignment. (C) Bend the fixation pins, and apply the acrylic over them while it has a doughy consistency.

Surgical Anatomy

Bones of the maxilla and mandibular body are easily palpated and surgically approached through skin and subcutaneous tissue (Fig. 33.12). The maxillary nerve (branch of the trigeminal nerve that innervates cutaneous muscles of the head, nasal and oral cavities, and muscles of mastication) passes rostrally through the alar canal and can be injured by maxillary fracture. The mandibular alveolar nerve, which is sensory to the teeth of the mandible, passes through the mandibular canal along with the mandibular alveolar artery. These structures are frequently damaged by mandibular fracture, although clinical signs are seldom evident. Tooth roots must be avoided when implants are placed in the maxilla or mandible. The approach to the ramus and the temporomandibular joint involves dissection and elevation of the masseter muscle (Fig. 33.13). The parotid duct and gland and the facial nerve are dorsal and superficial to the masseter muscle and should be avoided.

NOTE The shape of the mandibular canal and the presence of major vessels and nerves preclude intramedullary pin fixation of mandibular fractures.

Positioning

Animals are positioned in ventral recumbency for treatment of maxillary fractures and in dorsal or lateral recumbency for treatment of mandibular fractures. The surgical field, including the mouth, is aseptically prepared for surgery. If an autogenous cancellous bone graft is required, animals may be positioned in dorsal recumbency with forelimbs tied caudally. The skin over

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the proximal humerus is also prepared for aseptic surgery. This positioning allows simultaneous access to the proximal humeral metaphysis and the oral cavity. Because this is an uncommon limb position for obtaining a bone graft, care must be taken to orient oneself before the procedure. Additionally, the graft should be harvested first to avoid bacterial contamination of the donor site. Draping may be performed to include access to the oral cavity. A lap sponge or surgical sponge may be placed in the laryngeal region to block blood from the larynx and esophagus. A notation of the number of sponges should be made to ensure their removal at the conclusion of the procedure.

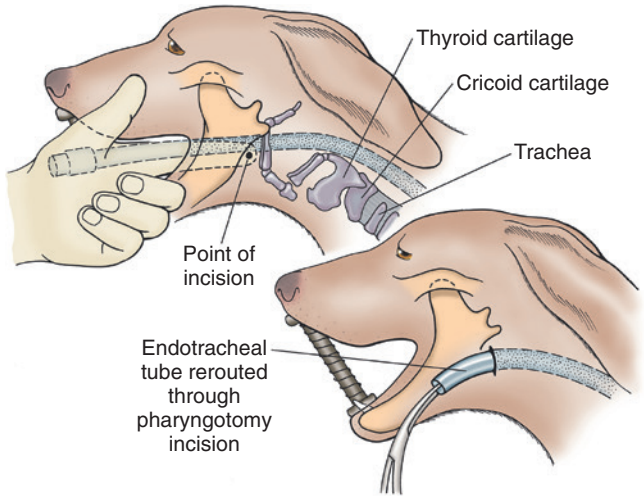


FIG. 33.11 To place an endotracheal tube through a pharyngotomy incision, insert an index finger into the oral cavity and locate the pharyngeal area immediately cranial to the hyoid bones. Incise skin, subcutaneous tissue, and mucous membrane to create a passage for the endotracheal tube. Place a forceps through the surgically created passage to grasp the endotracheal tube (with connector removed), and reroute it.

SURGICAL TECHNIQUE

Open Reduction of Mandibular Fractures

With bilateral mandibular fractures, make either a ventral midline incision in the skin between the mandibles or ventral skin incisions over each hemimandible. When a single incision is performed, move this incision in either direction to expose both mandibles. If only one mandible is involved, make a ventral skin incision directly over that mandible. Elevate soft tissue from the mandibles to expose the fracture(s). Maintain the digastricus muscle attachment (see Fig. 33.12). Reduce and stabilize the fracture. If a segmental fracture of the mandibular body occurs, stabilize the caudal fracture first. Because little musculature is present around the mandibular body, reduction is usually easily accomplished. Open reduction of the mandibular cortex will realign the teeth. *Evaluate the oral cavity for open wounds. If large wounds are present, lavage the wounds and close the mucosa. Close the surgical wound by suturing apposing layers.*

Open Reduction of Fractures of the Vertical Ramus and Temporomandibular Joint

Make a skin incision over the ventrolateral border of the caudal mandibular body, and separate the platysma muscle to expose the digastricus muscle. Elevate the masseter muscle from the ramus to expose its lateral mandibular surface and angular and coronoid processes (see Fig. 33.13). Reduce the fracture and stabilize it with interfragmentary wires or bone plates. Close the surgical wound by suturing apposing layers. Most of the vertical ramus is very thin bone that does not provide adequate implant purchase. Many of these fractures should be managed conservatively because loose implants will lead to significant complications. Fractures

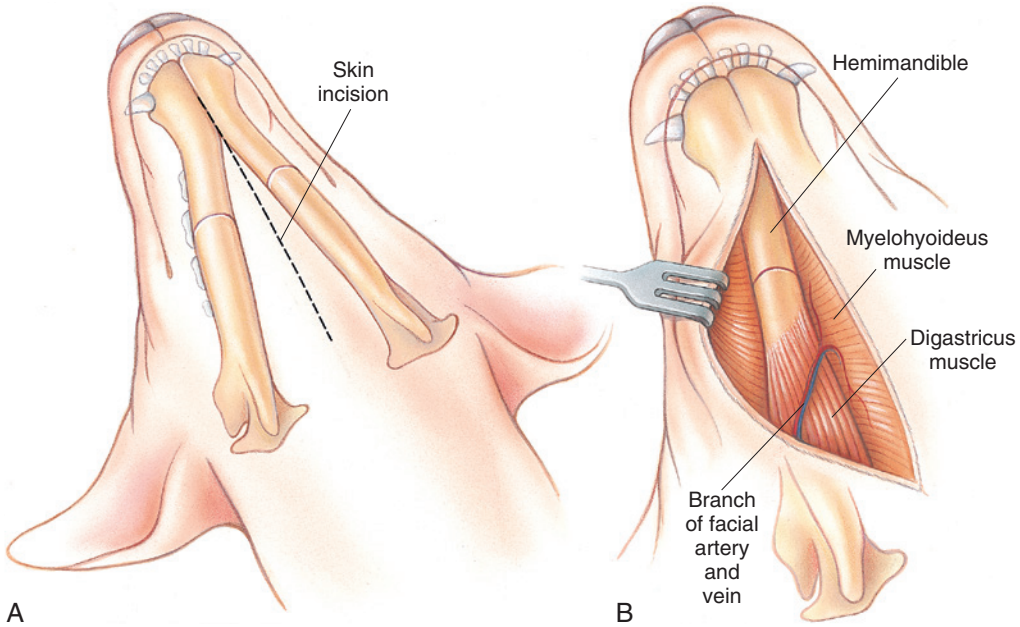


FIG. 33.12 (A) For a ventral approach to the mandible, make a ventral midline incision in the skin between the mandibles. (B) Elevate soft tissue from the mandibles to expose the fracture(s), but maintain the digastricus muscle attachment.

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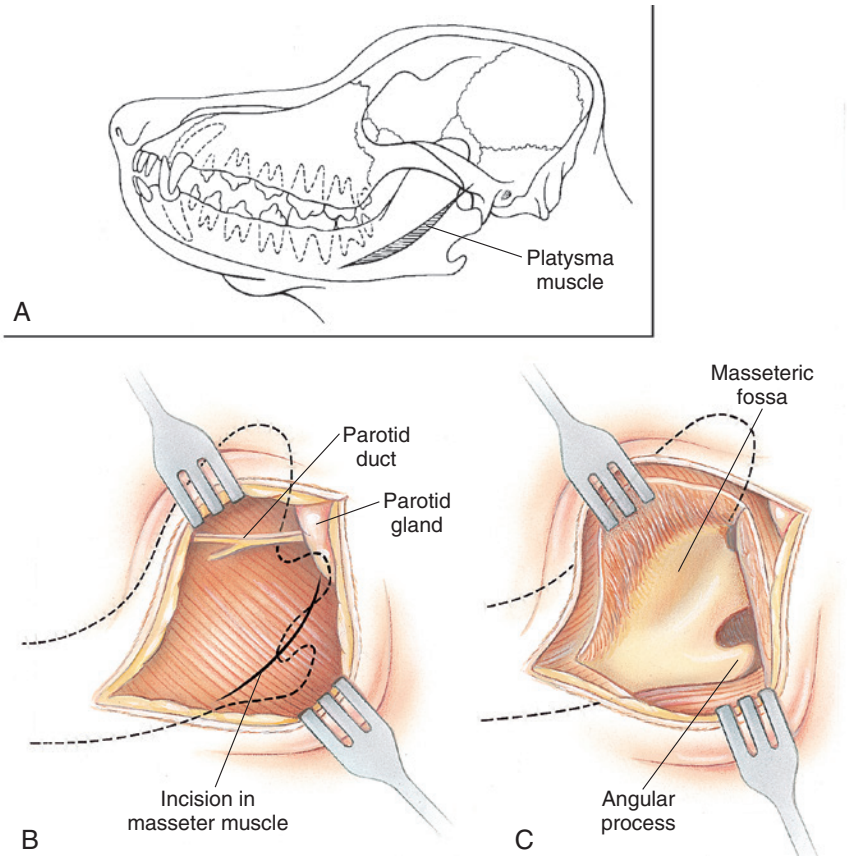


FIG. 33.13 (A) For a lateral approach to the ramus of the mandible, make a skin incision over the ventrolateral border of the caudal mandibular body, and separate the platysma muscle to expose the masseter muscle. (B–C) Incise and elevate the masseter muscle from the ramus to expose its lateral mandibular surface and angular and coronoid processes.

of the temporomandibular joint are rare and difficult to repair. Resection of the mandibular condyle may be indicated.

Open Reduction of Maxillary Fractures

Make a skin incision over the fracture(s), and gently elevate the soft tissue from the bone. Reduce the fracture and stabilize it with interfragmentary wires or bone plates. Repair large, open wounds to the oral cavity.

Stabilization of Mandibular Symphyseal Fractures

Symphyseal fractures are best treated with cerclage wire. A single wire serves as effective treatment for symphyseal fractures. Symphyseal cerclage wires encircle the mandible caudal to the canine teeth. The wire can be removed once the fracture has healed (generally 6–8 weeks) by cutting it with wire scissors where it is exposed behind the canine teeth. *Make a small nick in the skin overlying the ventral aspect of the symphysis. Insert a 16- or 18-gauge hypodermic needle through this nick and along one lateral mandibular surface (under the subcutaneous tissue). Exit the needle in the oral cavity caudal to the canine tooth, and thread an 18-g or 20-gauge wire through the needle. Reposition the needle on the opposite side of the mandible, curve the wire across and behind the canine teeth, and reinsert it through the hypodermic needle. Exit the wire from the skin incision at the original insertion point. Once the fracture is reduced, tighten the wire. Leave the ends of the wire exposed through the skin incision, and bend them to decrease the possibility of injury to the owner (Fig. 33.14).*

Stabilization of Mandibular Transverse Fractures

Transverse fractures should be realigned and compressed. *Apply one or two interfragmentary wires perpendicular to the fracture line to achieve compression (Fig. 33.15A–C). Alternatively, if the patient's fracture warrants more rigid fixation, use an external fixator or compression bone plate.*

Stabilization of Oblique Fracture Lines

Oblique fractures may override when parallel wires are tightened so that alternative wire patterns may be required. *Stabilize caudal to rostral (or vice versa) oblique fracture lines with two wires placed at right angles to each other.* More than one wire may be placed through a single drill hole. *Stabilize medial to lateral (or vice versa) oblique fracture lines with two wires placed perpendicular to each other in two perpendicular planes.* In both cases, the second wire prevents overriding of the fracture as the wires are tightened (see Fig. 33.15D–F).

Stabilization of Comminuted Fractures

Stabilize comminuted fractures that have long fracture lines and can be anatomically reconstructed with interfragmentary wires (see Fig. 33.15G). Support the reconstructed fracture with a bone plate or external fixator. Bridge comminuted fractures that cannot

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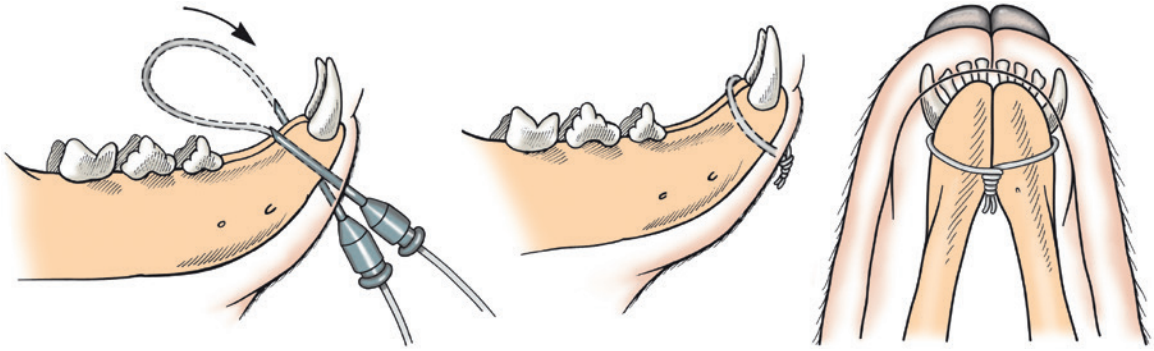


FIG. 33.14 To stabilize mandibular symphyseal fractures, use a 16- or 18-gauge hypodermic needle to place the cerclage wire.

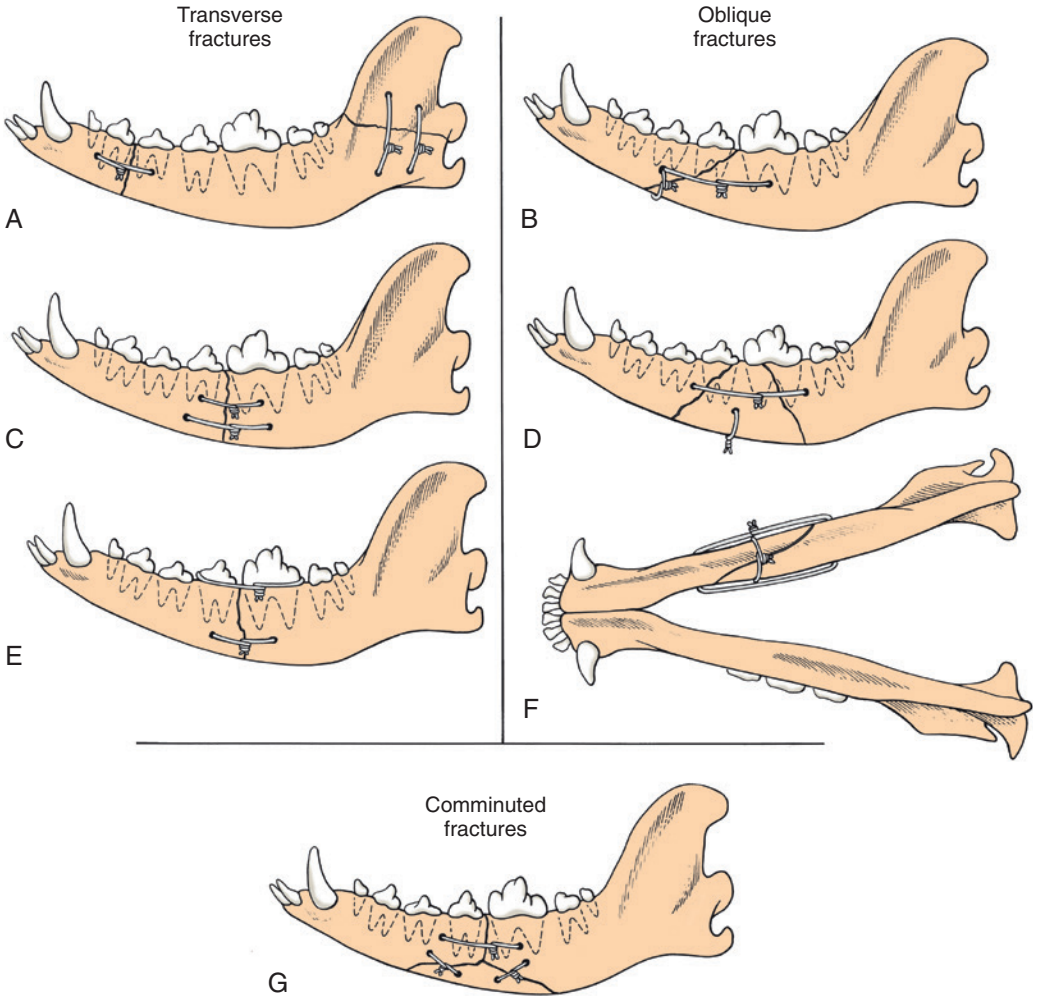


FIG. 33.15 Interfragmentary wire may be used to stabilize mandibular fractures. (A–C) For transverse fractures, wires may be placed perpendicular to the fracture line. (D) Stabilize caudorostral, long, oblique fracture lines with two wires placed at right angles to each other. Wire around the ventral mandibular border prevents the caudal segment from displacing rostrally when the interfragmentary wire is tightened. (E–F) To stabilize long, oblique, slab fractures, place wire through both bone segments and around the ventral mandibular border to prevent overriding of bone segments. (G) Secure large butterfly fragments with interfragmentary wires.

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be reconstructed with a bone plate or external fixator. Pay careful attention to ensure that dental occlusion is appropriate.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

A low-revolutions per minute (RPM) power drill is helpful for placing fixation pins. A pin chuck or power drill is used to drill holes in the bone for wires and to place K-wires. Dental resin composite is needed for canine tooth bonding. Polymethylmethacrylate or dental acrylic may be used to form the acrylic splint. Bone-plating equipment is needed for plates.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiographs should be evaluated for alignment, apparatus, and apposition. Occlusion of the teeth takes precedence over accurate fragment reduction. Dental occlusion is more readily determined by physical examination than with radiographs. Occasionally a tape muzzle can be used postoperatively to support the fixation (see Fig. 33.2). When muzzles are used as primary fixation or support for internal fixation, the skin on the ventral surface of the mandible may become irritated. The skin should be carefully cleaned and treated with a soothing ointment. External fixators should be evaluated postoperatively to ensure that the clamps or acrylic is not too close to the skin.

The animal should be fed soft food until the fracture heals; chew toys should be avoided. Patients with maxillomandibular fixation who have difficulty with oral feeding may benefit from an esophageal feeding tube (see p. 95). Owners should be instructed to not allow the animal to chew on rocks or sticks or to play tug-of-war. Clients should be instructed to clean the skin beneath a tape muzzle daily and to clean the area around external fixation pins. Oral chlorhexidine rinses twice daily are recommended for patients with interdental splints to minimize gingivitis associated with entrapped debris. The animal should be reevaluated 2 weeks postoperatively and sutures removed. Radiographs should be obtained after 6 weeks to evaluate healing and repeated every 6 weeks thereafter until healing is complete. Dental composite used for tooth bonding may break prematurely and require reapplication if healing is not complete. Dental composite, intraoral wires, and external fixators should be removed when fractures are healed; interfragmentary wires and bone plates and screws are not removed unless they cause problems. Sectioning the interdental splint and removing it segmentally helps avoid fracturing of teeth during this maneuver.

COMPLICATIONS

Malocclusion is the most common serious complication. Many animals learn to compensate for malocclusion; however, temporomandibular arthritis, impaired mastication, abnormal tooth wear, plaque and tartar accumulation, and periodontitis are possible sequelae. Mild malocclusion associated with interference of teeth can be treated by remodeling the involved teeth to allow clearance. Severe malocclusion may require tooth extraction or corrective osteotomy. Osteomyelitis (see p. 1039) and bone sequestration should be treated by sequestrectomy, removal of loose implants, mandibular stabilization (if necessary), cancellous bone autografts, culture/sensitivity, and appropriate antibiotics. Nonunions are treated with appropriate stabilization and cancellous bone autografts.

PROGNOSIS

The prognosis for healing of mandibular and maxillary fractures is generally excellent if proper techniques of fracture management are observed.

NOTE Mandibular and maxillary fractures generally heal without a large callus.

SCAPULAR FRACTURES

DEFINITION

Scapular fractures may occur through the body, spine, acromion, neck, supraglenoid tuberosity, and glenoid cavity.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Scapular fractures are relatively uncommon in dogs and cats because the large muscles surrounding the scapula protect it from direct injury. Common concurrent injuries include thoracic injuries (e.g., pulmonary contusions, pneumothorax, rib fractures, traumatic cardiomyopathy) and nerve injury (e.g., brachial plexus and suprascapular nerve contusions). Thus cardiorespiratory parameters and neurologic function of the limb should be determined preoperatively in animals presenting with scapular fractures. It is difficult to determine the status of the suprascapular nerve preoperatively.

Scapular fractures are classified according to location (e.g., body, spine, acromion, neck, supraglenoid tubercle, glenoid cavity), involvement of the articular surface, and stability. Scapular body and spine fractures may be minimally displaced and stable, requiring only conservative therapy. However, transverse fractures of the scapular body and spine may allow the scapula to fold on itself, resulting in a poor cosmetic appearance if the fractures are not reduced and stabilized. Similarly, comminuted fractures may be unstable and may be candidates for internal fixation. Avulsions of the supraglenoid tuberosity occur in immature dogs and are physeal separations subjected to the pull of the biceps muscle, which should be stabilized with internal fixation methods.

NOTE Fractures of the scapular neck (if displaced and unstable) and glenoid cavity (intraarticular fractures) may affect scapulohumeral joint function and thus should be anatomically reduced and stabilized with internal fixation methods.

DIAGNOSIS

Clinical Presentation

Signalment

Although traumatic scapular fractures may occur in animals at any age, young, large animals are at increased risk.

History

Affected animals usually have a history of trauma.

Physical Examination Findings

Most affected animals present with non–weight-bearing lameness. Swelling may occur over the scapula, and crepitation may be obvious when this region is palpated.

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Diagnostic Imaging

Radiographs of the scapula should include lateral and caudocranial views. Lateral views may be obtained by placing the scapula dorsal to the vertebral column (preferred) or by superimposing the scapula over the cranial thorax. To avoid superimposing scapulae, the contralateral forelimb should be retracted away from the affected limb during the lateral projection. A distal-proximal or axial projection provides a skyline view of the scapular spine and cranial and caudal scapular borders. Because of the manipulation necessary for diagnostic radiographs, some animals may require sedation (see Tables 31.2 and 31.3).

Laboratory Findings

Specific laboratory abnormalities are not present in animals with scapular fractures. Traumatized animals undergoing surgery should have sufficient bloodwork done to determine the optimal anesthetic regimen.

DIFFERENTIAL DIAGNOSIS

Animals with forelimb lameness attributable to scapular fractures should be carefully evaluated before surgery for concurrent nerve damage (brachial plexus, spinal cord trauma). Fractures are generally evident radiographically. Special attention should be given to identifying concurrent thoracic injuries.

MEDICAL MANAGEMENT

Conservative treatment with cage rest, analgesics, and limited exercise are appropriate for most closed, minimally displaced fractures of the scapular body and spine in dogs that will heal rapidly. A Velpeau sling (see p. 1167) may be applied but may lead to varus deformity of the bone and may lead to loss of range of motion of the elbow joint and contracture of the carpus. Spica splints (see p. 1166) may also be used for scapular fractures and eliminate the risk of varus deformity, but are difficult to apply and maintain. Fractures of the articular surface must be treated with open reduction, anatomic alignment, and rigid fixation (see later discussion).

SURGICAL TREATMENT

Surgical treatment is indicated for unstable extraarticular fractures and for intraarticular fractures. Fixation systems applicable for scapular fractures consist of plates and screws, orthopedic wire, and K-wires. The most appropriate fixation method should be determined on the basis of the fracture assessment score and fracture location; however, bone plates and screws offer the most stable fixation (Boxes 33.2 and 33.3).

NOTE Plates and screw fixation are preferred to ensure optimal outcome for limb function.

Preoperative Management

The overall health of the animal should be determined before surgery. Thoracic radiography and electrocardiographic analysis are warranted before anesthetic induction. Analgesics should be provided to posttraumatic animals (see Chapter 13).

BOX 33.2 Implant Use for Fractures of the Scapular Body and Spine According to Fracture Assessment Score (FAS)

FAS 0 to 3
Conservative therapy
Bone plate and screws

FAS 4 to 7
Conservative therapy
Bone plate and screws

FAS 8 to 10
Conservative therapy
Interfragmentary wires
Bone plate and screws

BOX 33.3 Implant Use for Scapular Articular Fractures According to Fracture Assessment Score (FAS)

FAS 0 to 3
Lag screws

FAS 4 to 7
Lag screws

FAS 8 to 10
Lag screws
Tension band wire

Anesthesia

Refer to Tables 32.1 and 32.2 for anesthetic management of patients with fractures.

Surgical Anatomy

Palpable landmarks of the scapula are the spine; acromial process; and cranial, dorsal, and caudal borders. The body and spine of the scapula are easily approached with dissection and elevation of muscle. The neck of the scapula is surrounded by muscles and tendons that support the scapulohumeral joint (Fig. 33.16). Osteotomy of the acromial process allows reflection of a portion of the deltoideus muscle and visualization of the joint. The suprascapular nerve and artery course over the scapular notch and under the acromial process, and care should be taken to avoid these structures (see Fig. 33.16). The axillary artery and nerve are located immediately caudal to the joint but are not usually visualized with routine approaches.

Positioning

The animal is placed in lateral recumbency with the affected side up. The entire scapular region should be prepared for aseptic surgery. For maximal maneuverability, drape out the forelimb. The most accessible site for a cancellous bone graft is the ipsilateral proximal humerus. If a bone graft is necessary (e.g., comminuted fracture with a fracture assessment score less than 3), the draped area should encompass the proximal humerus.

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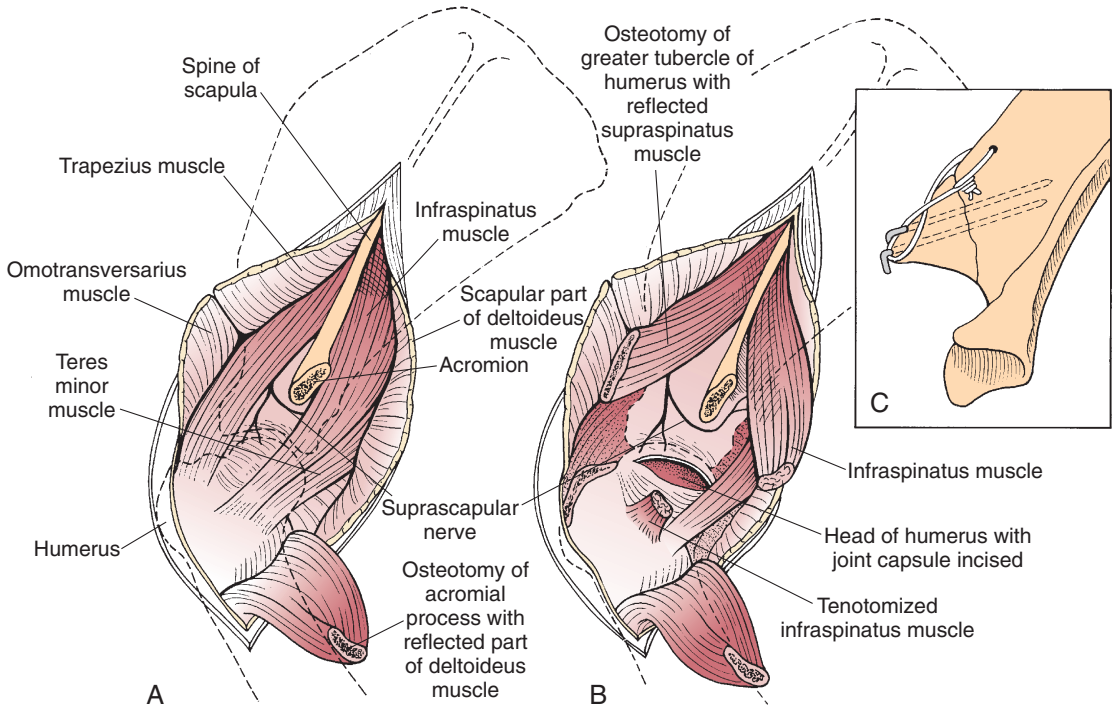


FIG. 33.16 (A) For a lateral approach to the scapular neck, make a lateral skin incision from the midportion of the scapular spine distally to the shoulder joint. Incise scapular attachments of the omotransversarius and trapezius muscles and the scapular head of the deltoid muscle. Osteotomize the acromial process and reflect it distally with the acromial head of the deltoideus muscle. (B) For additional exposure, tenotomize the infraspinatus muscle, and, if necessary, osteotomize the greater tubercle of the humerus. Incise the joint capsule to observe the articular surface during reduction of fractures involving the glenoid cavity. (C) Repair the osteotomy(ies) of the acromial process (and greater tubercle) with a tension band wire.

SURGICAL TECHNIQUE

Approaches to the scapulohumeral joint are described on p. 1158. Excision arthroplasty and shoulder arthrodesis are discussed on p. 1163.

Approach to the Scapular Spine and Body

Make a lateral skin incision extending the length of the spine distally to the shoulder joint (Fig. 33.17A). Transect the omotransversarius muscle from the spine, and reflect it cranially. Incise the trapezius and the scapular parts of the deltoideus muscles from the spine and reflect them caudally. Incise the supraspinatus and infraspinatus muscular attachments to the spine, and elevate these muscles from the scapular body (see Fig. 33.17B).

Approach to the Scapular Neck and Glenoid Cavity

Make a lateral skin incision from the middle portion of the scapular spine, and extend it distally to the shoulder joint. Expose the acromion process by incising attachments of the omotransversarius, the trapezius, and the scapular head of the deltoideus muscles to the scapula. Osteotomize the acromial process, and reflect it distally with the acromial head of the deltoideus muscle. Reflect the supraspinatus and infraspinatus muscles away from the scapular spine and neck. Take care to identify and protect the suprascapular nerve. If needed for complete joint exposure, tenotomize the infraspinatus muscle. Incise the joint capsule to observe the articular surface during reduction of fractures involving the glenoid cavity (see Fig. 33.16B). For additional exposure, osteotomize the greater tubercle of the humerus and reflect the supraspinatus muscle. Close the joint capsule with interrupted sutures of 3-0 absorbable material. Reappose the infraspinatus tendon with a tendon suture (i.e., three-loop pulley, Bunnell suture, or locking loop; see p. 1283) and support it with interrupted 0 or 2-0 nonabsorbable sutures. Repair the acromial osteotomy with a tension band wire (see Fig. 33.16C). Repair the humeral osteotomy with lag screws. Suture deep fascia, subcutaneous tissue, and skin separately.

Application of Orthopedic Wire

Orthopedic wire may be used as interfragmentary wire for fractures of the scapular spine and body (Figs. 33.18 and 33.19) or in conjunction with K-wires as a tension band for avulsions of the supraglenoid tuberosity (Fig. 33.20) in animals with high fracture assessment scores. Larger gauge (18 to 22) wire is used for interfragmentary fixation, whereas smaller gauge (20 to 24) wire is used in a figure-eight pattern (see Table 33.1). Large-gauge wire may be difficult to position and tighten unless application guidelines are followed (see pp. 1014–1016 for general principles of wire application).

NOTE Portions of the scapula are thin, and large-gauge wire may pull through when tightened.

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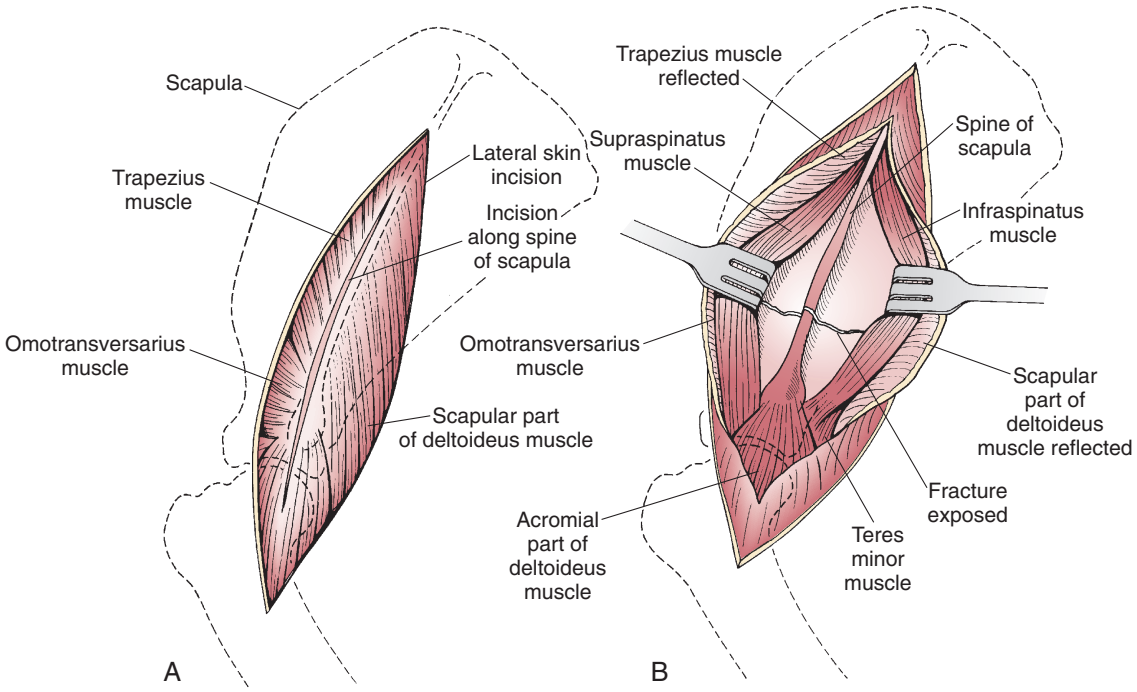


FIG. 33.17 (A) For a lateral approach to the scapula, make a lateral skin incision extending the length of the spine distally to the shoulder joint. (B) Incise the omotransversarius muscle, trapezius muscle, and scapular parts of the deltoideus muscle from the spine. Elevate the supraspinatus and infraspinatus muscles from the scapular body to expose the fracture.

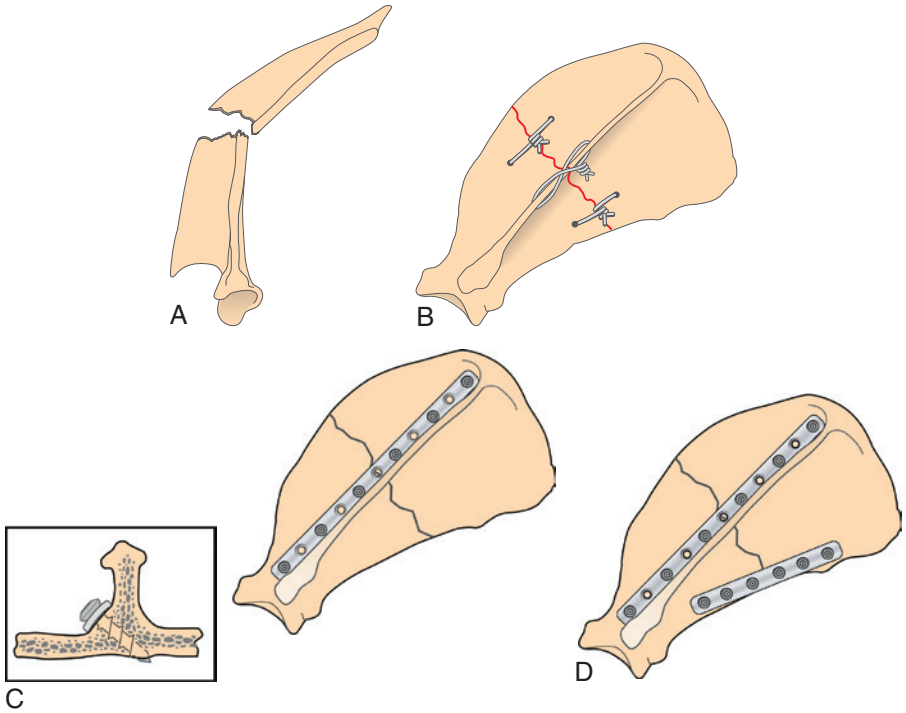


FIG. 33.18 (A) Transverse fracture of the scapular body may cause unsightly folding deformities. (B) Orthopedic wire may be used to repair these fractures in some patients with a high fracture assessment score. (C–D) The same fracture in most patients should be stabilized with one or two veterinary cuttable plates. (B–D, Modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat.* St. Louis: Elsevier; 2005.)

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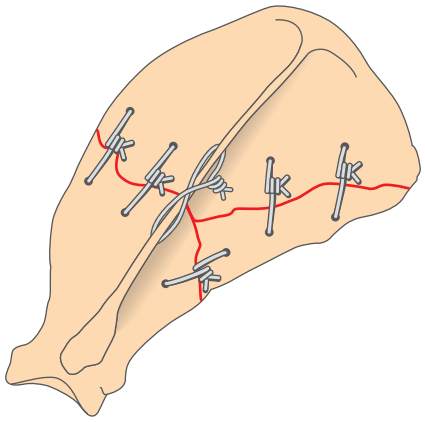


FIG. 33.19 Comminuted fractures of the scapular body can be reconstructed with orthopedic wire in patients with high fracture assessment scores.

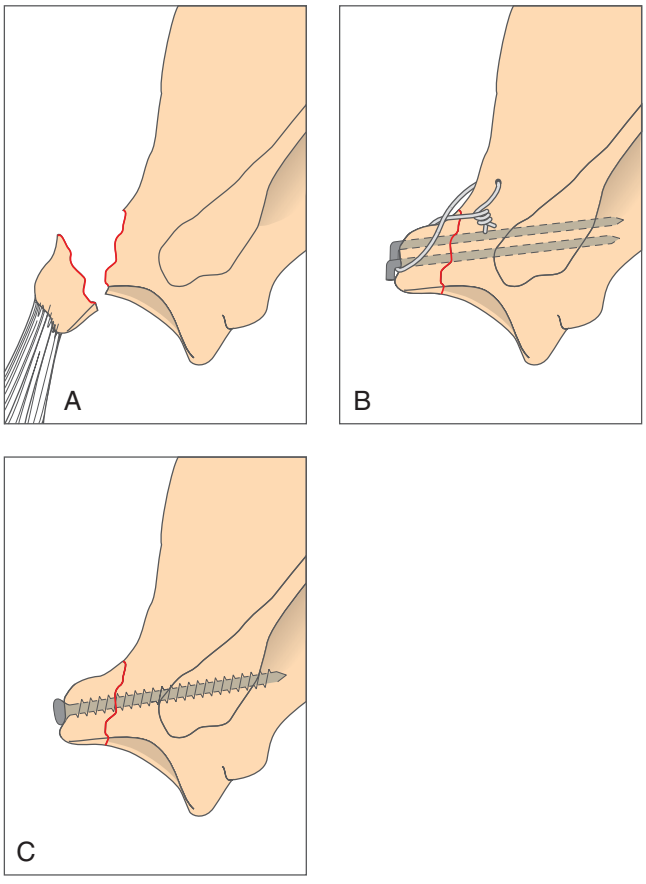


FIG. 33.20 (A) To overcome the pull of the biceps brachii muscle, avulsions of the supraglenoid tuberosity are treated with (B) a tension band wire in patients with a high fracture assessment score, or with (C) a lag screw in patients with a medium to low fracture assessment score.

Application of K-wires

K-wires can be used as crossed pins to stabilize transverse fractures of the scapular neck (Fig. 33.21) in animals with high fracture assessment scores (see p. 985). K-wires with figure-eight orthopedic wire may also be used for tension band fixation of avulsion fractures or repair of acromial osteotomies (see Fig. 33.16C) (see pp. 1008 and 1015 for general principles of K-wire and tension band application).

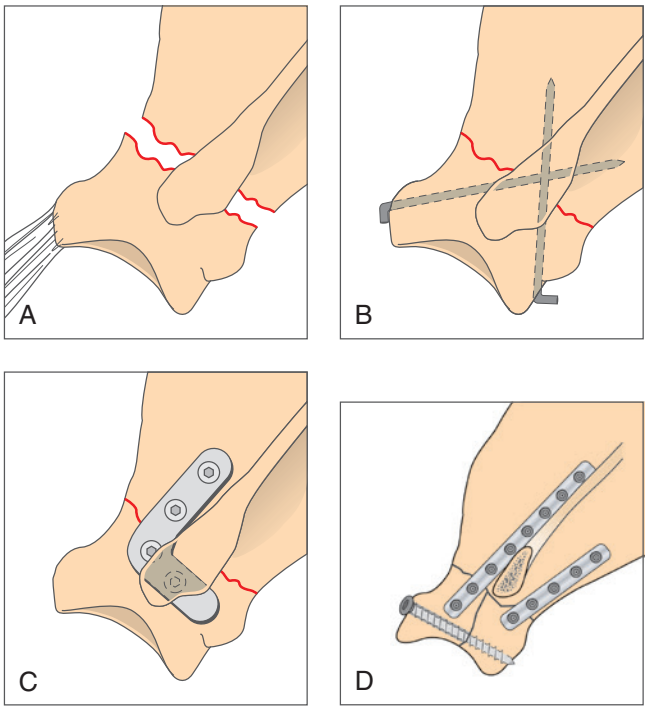


FIG. 33.21 (A) Transverse fractures of the scapular neck are best treated with internal fixation. (B) Crossed K-wires may be used in some patients with high fracture assessment scores. (C) Angle plates may be used in those with medium to low fracture assessment scores. (D) Articular fractures must be anatomically reconstructed and stabilized with a lag screw. The T fracture is then stabilized with a veterinary cuttable plate or plates. (D, Modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat.* St. Louis; Elsevier; 2005.)

Application of Bone Plates and Screws

Veterinary cuttable plates may be used to stabilize fractures of the scapular body and neck (see Fig. 33.18). Veterinary cuttable plates permit more screws per unit of plate length, making them particularly useful in scapular fractures. Plates applied to the body of the scapula must be secured with screws placed into the relatively thick bone that is under the spine. Additionally, the caudal border of the scapula offers thicker bone stock. A small (2.7 and 2.0 mm) angle and T plates may be used to stabilize neck fractures (see Fig. 33.21). With neck fractures, plates are placed under the suprascapular nerve, and the nerve must be protected from trauma while the plate is positioned. Cancellous and cortical bone screws are used as lag screws for stabilizing avulsion fractures of the supraglenoid tuberosity and T fractures of the neck (see Figs. 33.20 and 33.21) (see p. 1016 for general principles of plate application).

NOTE Articular fractures are best treated with anatomic reconstruction and lag screw compression.

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STABILIZATION OF SPECIFIC FRACTURES

SCAPULAR BODY AND SPINE FRACTURES

If the patient fracture assessment (see p. 985) indicates rapid healing (score of 8 to 10), conservative therapy may be used for stable scapular body and spine fractures. If these fractures are grossly displaced or have resulted in folding of the body, open reduction and stabilization with veterinary cuttable plates or interfragmentary wiring (see Fig. 33.18) may be more appropriate. If the fracture assessment score is 4 to 7 (e.g., older or larger dogs, in which healing may be delayed), open reduction and plate and screw fixation are indicated. Fracture assessment scores less than 3 indicate prolonged healing (see Box 33.2). If severe angulation of the joint is not present, severely comminuted fractures of the scapular body and spine that cannot be reconstructed may be treated conservatively. If the animal has multiple limb trauma or must bear weight on the limb, however, bridging the scapula with one or two bone plates will enhance postoperative mobility.

AVULSION FRACTURES OF THE SUPRAGLENOID TUBEROSITY AND FRACTURES OF THE SCAPULAR NECK AND ARTICULAR SURFACE

Avulsion fractures of the supraglenoid tuberosity are treated with open reduction and placement of a lag screw or tension band wire. Simple fractures of the scapular neck are treated with open reduction and stabilization with crossed K-wires, or with a small plate or plates. Severely comminuted fractures involving the neck are buttressed with a small plate or plates. Cancellous bone autografts are used in conjunction with open reduction to promote healing in these patients.

Articular fractures must be anatomically reconstructed (see Box 33.3). Lag screws with plate fixation is the most appropriate method of fixation.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Army-Navy, Myerding, and Hohmann retractors are useful for retracting muscles. A high-speed drill is necessary for application of a plate and can be used to drill holes for placement of orthopedic wire or to drive K-wires. Wire cutters are needed. Plating equipment is necessary for lag screw and plate placement.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiographs should be taken to evaluate alignment, apparatus, and apposition (see p. 1023). Postoperative pain management may be indicated (see Boxes 32.1 and 32.2 and Table 32.4). Radiographs should be repeated every 6 weeks to evaluate healing activity until fractures are healed. If concern arises about implant stability during full weight bearing, a Velpeau sling (see p. 1167) may be applied for a short time postoperatively; however, early return to function is preferred for joint fractures. Activity should be restricted to leash walking and physical rehabilitation until the fracture has healed. Physical rehabilitation (Table 33.2; see also Chapter 11) encourages controlled limb use and optimal limb function after fracture healing. Care must be taken to develop customized protocols for each patient depending on location of the fracture, stability and type of fracture fixation, potential for healing, abilities and attitudes of the patient, and willingness or ability of the client to participate in the animal's care. After fractures have healed, implant removal may be considered, but removal is usually unnecessary. If a Velpeau sling is applied, it should be kept clean and dry, and the owner should observe it daily for evidence of slippage or irritation.

COMPLICATIONS

Most scapular fractures heal without complication. Potential complications of scapular fracture repair include iatrogenic infection (with open reduction), malunion, delayed union, and secondary degenerative joint disease after articular fracture. K-wires will migrate if the fracture is unstable. Nonunions are uncommon after repair of scapular fractures because of the large muscle mass and good regional blood supply.

PROGNOSIS

The prognosis is generally excellent if proper fracture management procedures are followed.

HUMERAL FRACTURES

HUMERAL DIAPHYSEAL AND SUPRACONDYLAR FRACTURES

DEFINITIONS

Humeral diaphyseal fractures result in disruption of the continuity of the diaphyseal cortical bone. **Supracondylar fractures** are fractures of the distal diaphysis that involve the supracondylar foramen.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

High-velocity injuries (e.g., motor vehicle accidents, gunshot injuries, blunt trauma) are a common cause of humeral fractures in veterinary patients. When evaluating a patient subjected to a high-velocity injury, it is important not to focus on the obvious fracture but to complete a thorough examination of all body systems to rule out any concurrent injury. Common injuries associated with fractures of the humerus include chest wall trauma, pneumothorax, and pulmonary contusion. Radiographs should be taken to assess the degree of thoracic injury before anesthesia. The radial nerve courses from medial to lateral in the musculospiral groove of the distal humerus. Radial nerve injury may occur with fractures involving the distal humerus; therefore a careful assessment of the animal's neurologic status is essential. Reflexes and proprioception may be difficult to assess owing to muscle trauma and tissue swelling associated with the fracture. However, superficial pain sensation should be easily elicited on the dorsum of the paw if the radial nerve is functional.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, sex, or breed of dog or cat may be affected.

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TABLE 33.2 Sample Rehabilitation Protocol for Patient With a Diaphyseal Fracture (Internal Fixation or External Fixator)

Treatments/ Modalities	Day 1–7	Day 7– 21	3–4 Wk	5–7 Wk	8–12 Wk
Pain medications	As directed	As directed	PRN	PRN	PRN
Cryotherapy	10–15 min 3 times daily before walks or exercises First session immediately after surgery	Use after exercise for 15 minutes	PRN	PRN	PRN
Heat therapy		Apply heat to the muscles before exercise or if dog is stiff	PRN	PRN	PRN
Massage	5 min 3 times daily before starting exercises- massage from toes toward heart	Continue twice daily	Twice daily	Twice daily	Only if desired
Passive range of motion	10 repetitions 3 times daily, all joints; affected limb—start above and below knee	Continue twice daily; if dog is using leg well, then decrease	Discontinue if dog is walking well and range of motion is normal		
Laser therapy	Daily or every other day for first week	Every other day for first week then 2 times weekly	Twice weekly	Twice weekly	Discontinue
Walks	5 min supported leash walk 2–3 times daily	Increase each walk by 2–3 min each week	Increase by 5 min each week	Increase by 5 min each week	15–20 min walks 2–3 times daily
Neuromuscular electrical stimulation	10 min twice daily	10 min twice daily	Discontinue if dog is doing well		
Balancing		5 min twice daily	5 min twice daily	One-leg standing up to 5 min	Replace with hill walks
Cavalettis/obstacles/ sit to stand		5 min twice daily	5 min twice daily	5 min twice daily	5 min twice daily
Weaving/circles		2–3 min once daily	5 min twice daily	5 min twice daily	5 min twice daily
Hills				Zig zag low slow hills 5 min up and down[a]	Increase to 10 min twice daily
Underwater treadmill		10 min 3 times weekly after sutures are removed[b]	10 min 3 times weekly[b]	15–20 min twice weekly[b]	15–30 min twice weekly until released from rehab[b]
Swimming					8 wk post internal fixation for 5–10 min if all healed[c]

[a]Wait until after 8 wk for external fixators.
[b]For external fixators, wait until device is removed at 14+ wk to start water therapy.
[c]Wait until after 4 wk after external fixators are removed.
PRN, as needed.

History

Motor vehicle accidents cause most cases of humeral diaphyseal fracture. Other modes of injury include gunshots and falls.

Physical Examination Findings

Patients with humeral diaphyseal fractures are usually non–weight-bearing and exhibit varying degrees of limb swelling. Pain and crepitus may be elicited on limb manipulation. Affected animals often drag the limb when walking and may not lift the paw when proprioception is checked. This may cause the examiner to assume that neurologic injury is present. However, similar findings may occur because of the orthopedic injury alone if pain and swelling make the patient reluctant to move the limb when proprioception is evaluated.

Diagnostic Imaging

Most of these animals are in pain and require sedation or general anesthesia for proper positioning to obtain quality radiographs (see Tables 31.2 and 31.3). Caudocranial or craniocaudal and lateral views are necessary to assess the extent of bone and soft tissue injury. Radiographs can be taken under anesthesia just before surgery, but this decreases the time available for planning the surgical procedure. If the fracture is comminuted and if bone plate fixation is contemplated, radiographs of the contralateral limb are useful for assessment of bone length and

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shape. These radiographs assist in proper contouring of the bone plate.

Laboratory Findings

Complete blood count and serum chemistry evaluation should be done to assess the status of the animal for anesthesia. Consistent laboratory abnormalities are not present.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include shoulder or elbow luxation, severe soft tissue contusion, and pathologic fractures secondary to neoplasia.

MEDICAL MANAGEMENT

Medical treatment of animals with humeral fractures includes analgesics for posttraumatic pain (see Chapter 13) and antibiotics to treat open fractures. Casts or splints are contraindicated for repair of humeral fractures because the scapulohumeral joint cannot be effectively immobilized.

SURGICAL TREATMENT

IM pins and orthopedic wire, interlocking nails, IM pins plus external skeletal fixation (ESF), external skeletal fixators alone, and bone plates may be used to repair humeral fractures. The implant system chosen should reflect the patient fracture assessment score (Box 33.4).

Preoperative Management

Before surgery, a spica splint (see p. 1166) can be applied to increase patient comfort and protect soft tissue from further injury induced by bone fragments. Because these fractures result from trauma, all affected animals should be examined for concurrent injury and stabilized if necessary before surgery. Analgesics should be provided to posttraumatic animals (see Chapter 13).

Anesthesia

Refer to Tables 32.1 and 32.2 for anesthetic management of patients with fractures.

BOX 33.4 Implant Use for Fractures of the Humeral Diaphysis According to Fracture Assessment Score (FAS)

FAS 0 to 3

Bone plate and intramedullary (IM) pin
Bone plate and screws
Interlocking nail
Modified type Ib external fixator with IM pin

FAS 4 to 7

External skeletal fixation with or without IM pin
Bone plate and screws
Interlocking nail

FAS 8 to 10

IM pin with cerclage wires or external fixator
Flexible bone plate and screws

Surgical Anatomy

Although the humerus may be approached from all four anatomic directions, most often craniolateral or medial exposures are used to approach the humeral diaphysis. Proximal diaphyseal fractures are best exposed with the craniolateral approach; distal diaphyseal fractures are best exposed by a medial approach. The distal aspect of the humerus is straighter on the medial aspect than on the lateral aspect, making applications easier on the distal medial surface. When using a lateral approach, the radial nerve must be identified and protected during fracture reduction and stabilization. The radial nerve lies superficial to the brachialis muscle and deep to the lateral head of the triceps. During the medial approach to the humerus, care must be taken to isolate and protect the median, musculocutaneous, and ulnar nerves, as well as the brachial artery and vein. The canine humerus has a normal cranial-to-caudal curvature that positions the long axis of the marrow cavity cranial to the shoulder joint. This facilitates pin placement by ensuring that an IM pin will exit cranial to the joint. However, distally the supratrochlear foramen is in direct line with the long axis of the marrow cavity, which makes distal placement of the IM pin into the cancellous bone more difficult. The anatomy of the feline humerus is similar to that of the canine. However, less cranial-to-caudal curvature is noted, and the diameter of the marrow cavity is more uniform. In many cats, the medullary canal ends at the level of the supratrochlear foramen. Care must be taken to avoid entering the supratrochlear foramen as a result of the presence of the median nerve.

NOTE Be sure to identify and protect the major nerves during surgical approaches to the humerus.

Positioning

For the craniolateral approach to the humerus, the animal is positioned in lateral recumbency with the affected leg up. For the medial approach to the humerus, the animal is positioned in dorsal recumbency. A hanging-leg preparation facilitates limb manipulation during surgery. The limb should be prepped from the dorsal midline to the carpus. If the fracture location permits, the proximal humerus may be used as a donor site for cancellous bone graft. If not, the ipsilateral iliac wing should be prepared for surgery.

SURGICAL TECHNIQUE

Surgical Approach to the Humeral Diaphysis

The proximal and central humeral diaphysis is most easily exposed through a craniolateral approach. *Make a skin incision from the cranial border of the tubercle of the humerus to the lateral epicondyle distally (Fig. 33.22A). Follow the normal curvature of the humerus. Incise the subcutaneous fat and brachial fascia along the same line, taking care to isolate and protect the cephalic vein (Fig. 33.22B). If necessary, ligate the cephalic vein to achieve the desired exposure. Incise the brachial fascia along the border of the brachiocephalicus muscle and lateral head of the triceps. Use caution when incising the fascia along the cranial border of the triceps overlying the brachialis muscle until the radial nerve is visualized (Fig. 33.22C). Once the nerve is isolated, reflect the brachiocephalicus and superficial pectoral muscles cranially and the brachialis muscle caudally to expose the proximal and central*

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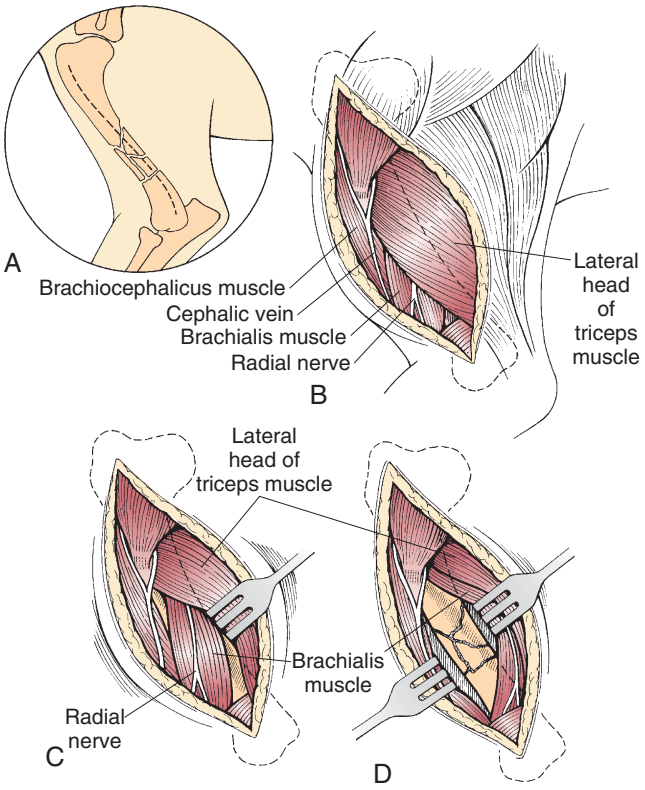


FIG. 33.22 (A) To expose the mid-humeral diaphysis, make a skin incision from the cranial border of the humeral tubercle to the lateral epicondyle distally. (B) Incise subcutaneous fat and brachial fascia along the same line, being careful to isolate and protect the cephalic vein. (C) Visualize the radial nerve when incising fascia along the cranial border of the triceps overlying the brachialis muscle. (D) Retract the brachiocephalicus muscle cranially and the brachialis muscle caudally to expose the humerus.

humeral shaft (Fig. 33.22D). To gain further exposure of the distal humeral shaft, reflect the brachialis muscle cranially and the lateral triceps muscle caudally. Release the origin of the extensor carpi radialis muscle from the ridge of the lateral epicondyle for maximum exposure. To close, suture the brachiocephalicus muscle and the superficial pectoral muscles to the fascia of the brachialis muscle. Suture the subcutaneous tissue and skin using standard methods. The distal one-half of the humerus is accessible through a medial exposure, which is the choice of some surgeons when a bone plate is used as the fixation method. *Make an incision from the greater tubercle proximally to the medial epicondyle distally. Incise the deep brachial fascia along the caudal border of the brachiocephalicus muscle (Fig. 33.23A). Take care distally to preserve and isolate the neurovascular structures (i.e., median, musculocutaneous, and ulnar nerves and brachial artery and vein) (Fig. 33.23B). Reflect the brachiocephalicus muscle cranially, and incise through the insertion of the superficial pectoral muscle. For exposure of the midportion of the humerus, reflect the superficial pectoral muscle cranially and the biceps brachii and neurovascular structures caudally (Fig. 33.23C). For exposure of the distal humerus, reflect the biceps brachii, neurovascular structures, and superficial pectoral muscle cranially. To close, suture the superficial pectoral to the brachiocephalicus fascia. Suture the remaining deep fascia, subcutaneous tissue, and skin routinely.*

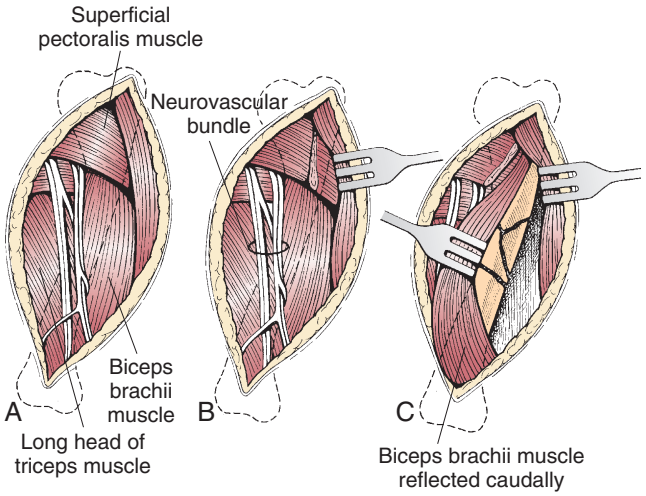


FIG. 33.23 (A) To expose the medial surface of the distal third of the humerus, incise deep brachial fascia along the caudal border of the brachiocephalicus muscle. (B) At the distal aspect, take care to preserve and isolate the median, musculocutaneous, and ulnar nerves and the brachial artery and vein. (C) Reflect the biceps brachii and neurovascular structures caudally and the superficial pectoral muscle cranially.

Implant Application Techniques Specific to the Humerus

Application of an Intramedullary Pin

An IM pin can be used to stabilize humeral mid-diaphyseal fractures, providing excellent resistance to bending but no resistance to rotational forces or axial loading. Additional implants must be used to provide rotational and axial support for most fractures (for general principles of IM pin application, see p. 1008). *Place an IM pin in a retrograde or normograde manner in the humerus. To retrograde the IM pin, drive it proximally from the fracture surface toward the shoulder joint. To ensure that the pin exits at the proper site proximally, press the shaft of the pin against the medial and caudal surface of the marrow cavity to force the point of the pin to glide along the craniolateral cortex and exit craniolateral to the shoulder joint.* This maneuver also "presets" the pin in the proximal fragment so that the distal pinpoint is directed toward the caudomedial cortex when the fracture is reduced and the pin is driven into the distal fragment. *To normograde the pin, insert it at the craniolateral aspect of the greater tubercle and drive it in line with the medullary canal to exit at the fracture. With either technique, reduce the fracture and drive the pin distally. Seat a large pin at the isthmus of the marrow cavity just proximal to the supracondylar foramen, or drive a smaller pin into the medial portion of the condyle (Fig. 33.24).* The medullary canal ends in the supracondylar area in many cats, and passage of the IM pin into the medial portion of the condyle is impossible. Pin placement is easier in cats because the marrow cavity has a uniform diameter, the bone has less curvature, and the soft tissue envelope covering the bone is less than in dogs. Care must be taken not to enter the supratrochlear foramen of a cat because the median nerve is in this area. IM pins may be placed down the medial epicondyle in distal humeral fractures; however, the pin must be of a narrow diameter to avoid splitting the epicondyle. Pins may be placed

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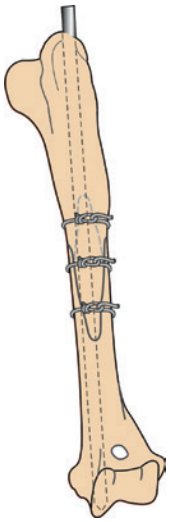


FIG. 33.24 Humeral fractures may be stabilized with an intramedullary pin, which may be wedged into the narrowest part of the marrow cavity (isthmus) or guided through the epicondyloid ridge to seat into the medial epicondyle. Additional implants, such as cerclage wires, are needed to provide mechanical support. (Modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat,* St. Louis: Elsevier; 2005.)

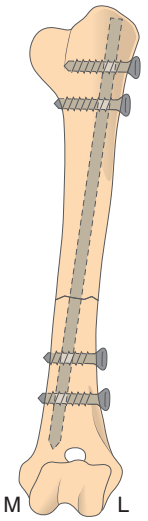


FIG. 33.25 Position of an interlocking nail in the humerus.

normograde or retrograde. With retrograde placement, care must be taken to avoid damage to the articular surface of the elbow joint.[2]

NOTE Estimate pin size from the preoperative radiograph.

NOTE To align the pin properly with retrograde placement, press the shaft against the caudomedial cortex. This causes the pin to exit craniolateral to the shoulder joint.

Application of an Interlocking Nail

Interlocking nails are used to stabilize both single and comminuted mid-diaphyseal humeral fractures (Fig. 33.25). The interlocking nail provides resistance to bending, rotational, and axial loading forces and can effectively bridge a nonreducible fracture (for general principles of interlocking nail application, see p. 1011). *Ream the medullary canal using a normograde or retrograde technique. Insert the interlocking nail in a normograde manner, starting on the ridge of the greater tubercle. Make a craniolateral approach to the shoulder (see p. 1160) to expose the point of insertion. Flex the shoulder to facilitate nail insertion.*

Application of External Skeletal Fixation

The surrounding muscle mass and proximity of the chest wall and motion of the elbow make external fixator application to the humerus challenging. When external fixators are applied to the humerus as the sole means of fixation, stress on fixation pins is high owing to the long distance from the external bar to their entrance into the bone and the inability to use stronger bilateral frames. Thoughtful preoperative planning and strict

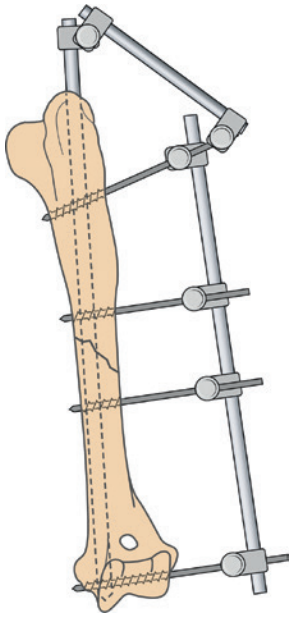


FIG. 33.26 To increase the strength of the fixation system, connect the intramedullary pin to an external fixator frame to make a tie-in configuration. (Modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat.* St. Louis: Elsevier; 2005.)

adherence to principles of application are necessary to prevent fixator-related complications and unacceptable patient morbidity. In general, type Ia and modified type Ib frames combined with IM pins are used to stabilize humeral fractures (Figs. 33.26 and 33.27). Combining the bending support of an IM pin with axial and rotational support from an external fixator is useful in controlling all weight-bearing forces in the humerus. The frame, number, and type of fixation pins vary with the rigidity of fixation desired and the length of time the fixator must remain in place (for general principles of ESF application, see p. 998). *Insert an IM pin that occupies 50% to 60% of the medullary cavity in a normograde or retrograde manner. Apply a type Ia fixator to the lateral surface of the humerus. Center the distal fixation pin within*

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the humeral condyle by inserting it 1 to 2 mm cranial and distal to the lateral epicondylar prominence. Predrill the condyle because this area is composed of dense cancellous bone. Tie the IM pin into the frame to enhance stability (see Fig. 33.26).

To construct a modified type Ib external skeletal fixator, place an additional fixation pin in the cranial portion of the proximal half of the humerus. Use a transfixation pin through the condyle that exits the skin on the medial aspect. Connect the cranial pin to the medial aspect of the condylar transfixation pin with a connecting bar (see Fig. 33.27). Connect the external bars with articulating external bars. Tie the IM pin into the frame.

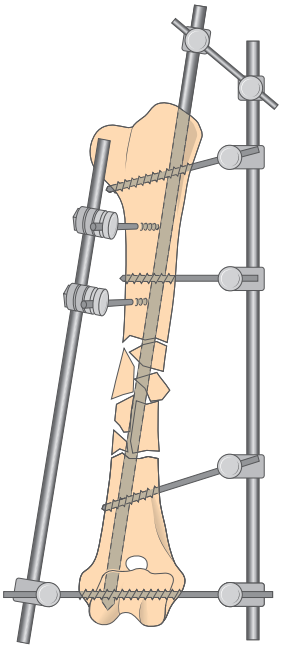


FIG. 33.27 External skeletal fixator (modified type Ib shown here) may be used with an intramedullary pin or as the sole means of stabilizing a comminuted fracture.

Additional fixation pins can be added to the craniomedial or lateral external bars to achieve enhanced strength and stiffness. If one or more fixation pins are placed in the distal third of the humerus, care must be taken when making the soft tissue tunnel and placing the fixation pins to prevent radial nerve injury.

Application of Bone Plates and Screws

Bone plates provide needed stability and allow early return to function when used for complex or stable humeral fractures. Factors that influence plate sizes are its intended function (i.e., compression, neutralization, or bridging plate) and patient size (see p. 1016 for general principles of plate application). *Place an appropriately sized and contoured plate on the cranial, lateral, caudolateral, caudomedial, or medial surface of the humerus (Fig. 33.28). Combine the plate with an IM pin for additional stability for comminuted fractures (Fig. 33.29).* A locking plate applied as an internal fixator to stabilize comminuted fractures does not need to anatomically conform to the shape of the bone.

Cranial plate application is easiest with proximal and midshaft humeral fractures. Lateral plate application is easiest with proximal and midshaft fractures but can be used with distal fractures. Medial, caudomedial, and caudolateral placement is easiest with distal fractures.

Fixation Techniques for Specific Fractures

Midshaft Transverse or Short Oblique Fractures

Stabilization of a transverse or short oblique fracture requires rotational and bending support; short oblique fractures also require axial support. This can be achieved with bone plates, an interlocking nail, an IM pin with an external fixator, or an external fixator alone.

Fixation systems that are useful with a low fracture assessment score include a bone plate and screws inserted to function as a compression plate, an interlocking nail, or a six-pin modified type Ib external skeletal fixator with raised-threaded transfixation pins "tied in" with an IM pin. With a moderate fracture assessment score, a compression plate, an interlocking nail, or an IM pin tied in with a three-pin type Ib external skeletal fixator would be functional. Transverse or short oblique fractures are common

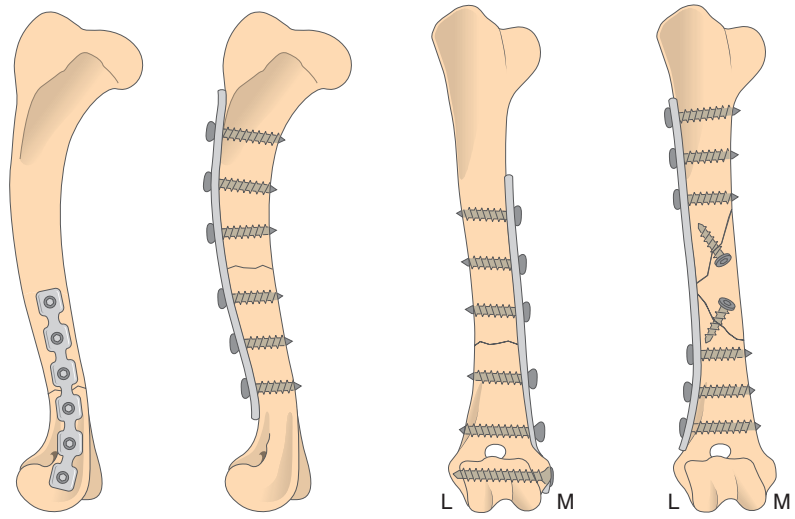


FIG. 33.28 Bone plates are applied as compression plates to the lateral cranial or medial humeral surface to stabilize transverse diaphyseal fractures. Oblique or comminuted reducible fractures are stabilized with lag screws and neutralization plates.

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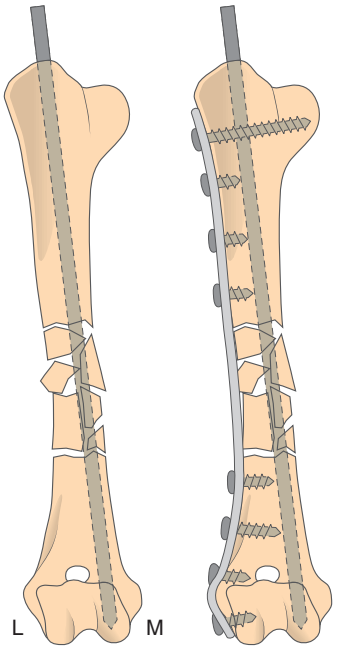


FIG. 33.29 Application of the plate-pin combination for stabilization of comminuted humeral fracture. The intramedullary pin reduces cyclic bending stress in the bone plate.

in puppies and kittens because their owners often step on them. These patients have a high fracture assessment score and can be stabilized with an IM pin with a two-pin type Ia external skeletal fixator (Fig. 33.30).

Midshaft Long Oblique Fractures or Reducible Comminuted Fractures With a Large Butterfly Fragment

These fractures can be anatomically reduced and interfragmentary compression applied with cerclage wire or lag screws. Axial, rotational, and bending forces generated by weight bearing are neutralized with bone plates, interlocking nails, IM pins with cerclage wires, or external fixators (Fig. 33.31).

Fixation systems that are useful with a low fracture assessment score include neutralization plates, interlocking nails, and type Ib external fixators. With a moderate fracture assessment score, a neutralization plate, an interlocking nail, or an IM pin plus cerclage wire for interfragmentary compression combined with a type Ia external skeletal fixator may be used. With a high fracture assessment score, an IM pin combined with cerclage wire for interfragmentary compression is a useful method to stabilize the fracture.

Midshaft Comminuted Fractures With Multiple Fragments

These fractures are realigned using indirect reduction techniques (see p. 990). Because no load sharing occurs between the implant and the bone until biological callus forms to provide support, these fractures need rigid axial, rotational, and bending support.

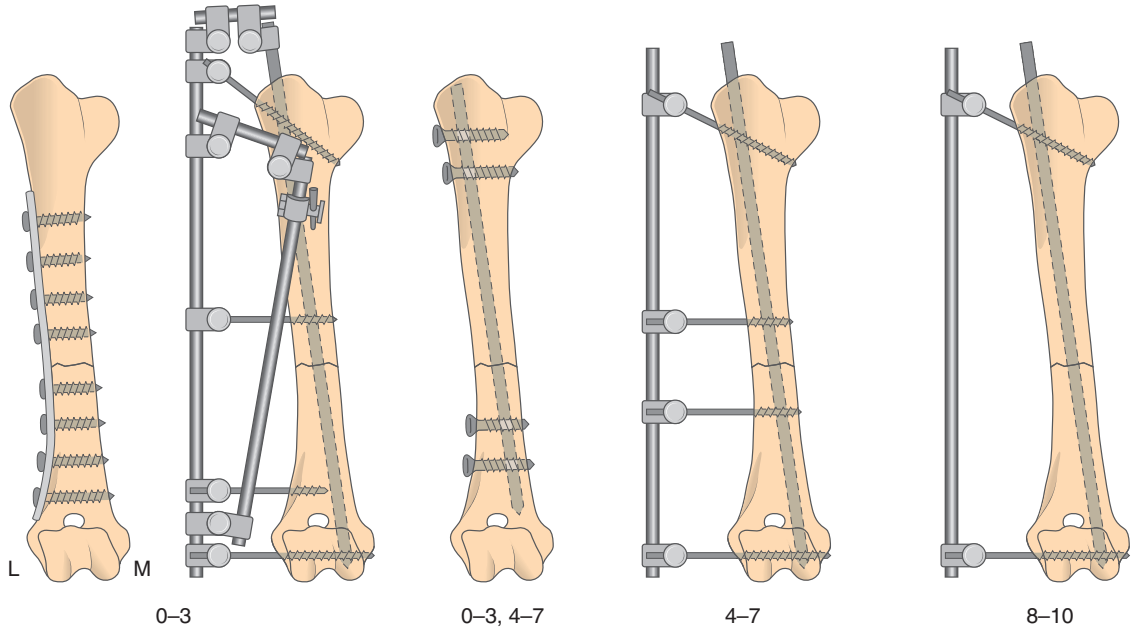


FIG. 33.30 Recommended methods of stabilizing transverse or short oblique humeral fractures based on fracture assessment score. If the fracture assessment score is 0 to 3, a compression plate, an external skeletal fixator plus intramedullary (IM) pin (tie-in configuration), or an interlocking nail may be used. If the fracture assessment score is 4 to 7, an interlocking nail or an external skeletal fixator with IM pin may be used. The IM pin may be tied in for extra stability. With fracture assessment scores of 8 to 10, an external skeletal fixator plus IM pin provides necessary stability.

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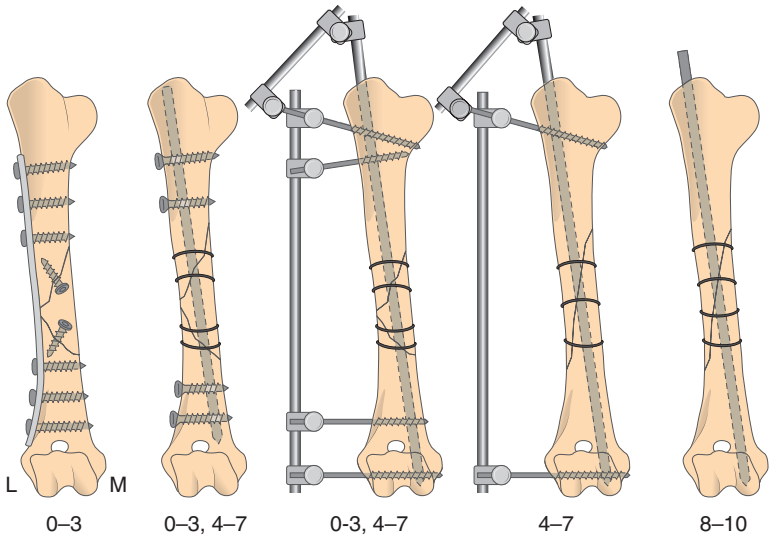


FIG. 33.31 Recommended methods for stabilizing long oblique or reducible comminuted humeral fractures (single large fragment) based on fracture assessment score. If the fracture assessment score is 0 to 3, a neutralization plate, an interlocking nail, or a four-pin external skeletal fixator plus intramedullary (IM) pin (tie-in configuration) and cerclage wires may be used. If the fracture assessment score is 4 to 7, a two-pin external skeletal fixator with IM pin (tie-in configuration) and cerclage wire may be used. With fracture assessment scores of 8 to 10, an IM pin plus cerclage wire provides necessary stability.

Very high stresses will be imposed on the implant and its connection to the bone. If the biological assessment is favorable, the imposed stresses will be of short duration, reducing the likelihood of implant failure. If the biological assessment is not favorable, however, imposed stresses will act on the implant for an extended period, making implant failure more likely.

Fixation systems useful in managing patients with a low fracture assessment score include a bone plate-pin combination, an interlocking nail, or a modified type Ib external skeletal fixator tied into an IM pin. Patients with a moderate fracture assessment score may be treated with a bone plate functioning as a bridging plate (lengthening plate or broad plate with or without an IM pin) or an interlocking nail. An external skeletal fixator, with or without a tie-in to the IM pin, may also be used. A patient with this type of fracture would have a high fracture assessment score only when the biological assessment is extremely favorable (e.g., 4- to 5-month-old animal with a closed, single-limb injury). These animals may be treated with bridging osteosynthesis with an IM pin, with or without a tie-in to a type Ia or type Ib external skeletal fixator (Fig. 33.32).

Supracondylar Fractures

Supracondylar fractures are usually transverse or short oblique fractures. Occasionally a comminuted fracture with multiple small fragments will be seen. When comminution is present, however, the length of bone involved is usually limited to a small area.

Stabilization of supracondylar fractures depends on the fracture configuration and the patient fracture assessment (Fig. 33.33). Transverse or short oblique fractures require rotational and bending support, whereas comminuted fractures require axial, rotational, and bending support. In patients with a low fracture assessment score, a caudolateral plate combined with a caudomedial plate or a caudolateral plate combined with a medial IM pin is preferred. The plate functions as a compression plate with transverse fractures and as a buttress plate with highly fragmented fractures. In patients with a moderate fracture assessment score, a medial plate, a caudolateral plate, or an IM pin supported with an external skeletal fixator is suggested. The external skeletal fixator frame and the number of pins depend on the fracture assessment. Closed reduction and application of a modified type Ib external fixator can be used to preserve the biology of comminuted nonreducible fractures. If the fracture is transverse and has a high assessment score, an IM pin seated into the medial portion of the condyle and a laterally positioned pin or K-wire crossing the fracture may provide the necessary stability to allow for fracture union.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Equipment necessary for pin and wire placement includes retractors, bone-holding forceps, reduction forceps, a Jacobs pin chuck, IM pins, K-wires, orthopedic wire, wire cutters, and pin cutters. Additional equipment needed for external fixation includes a drill bit, a drill guide, a low-RPM power drill, and external fixation clamps and bars. Interlocking nail equipment is needed for nail application. Plating equipment and a high-speed drill are necessary for application of plates and screws.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiographs are made to evaluate alignment, apparatus, and apposition. Postoperative pain management may be indicated (see Boxes 32.1 and 32.2 and Table 32.4). Activity

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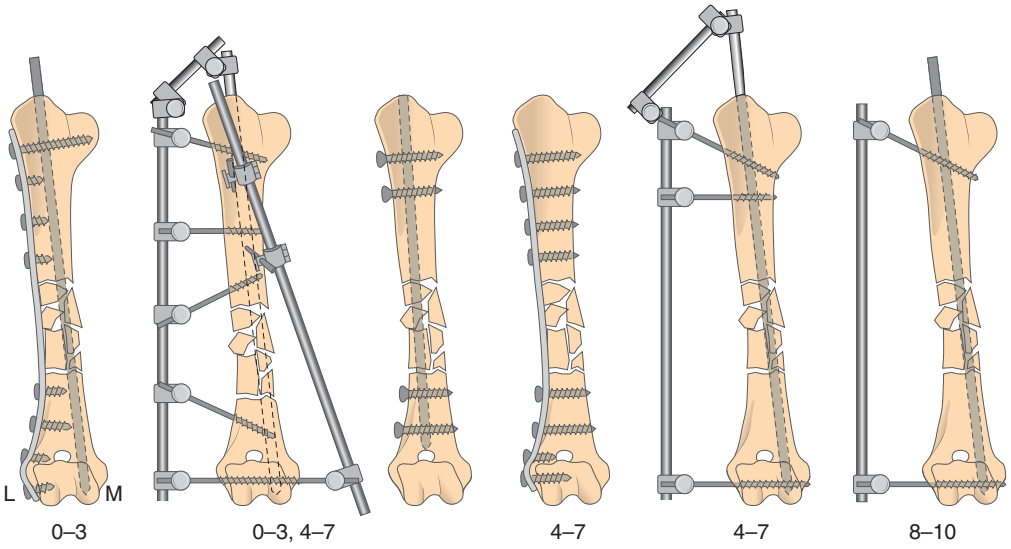


FIG. 33.32 Recommended methods for stabilizing nonreducible comminuted humeral fractures (multiple fragments) based on fracture assessment score. If the fracture assessment score is 0 to 3, a plate-rod combination, an interlocking nail, or an external skeletal fixator plus intramedullary (IM) pin (tie-in configuration) may be used. If the fracture assessment score is 4 to 7, a buttress plate or an external skeletal fixator with IM pin (tie-in configuration) may be used. With fracture assessment scores of 8 to 10, a two-pin external skeletal fixator plus IM pin provides necessary stability.

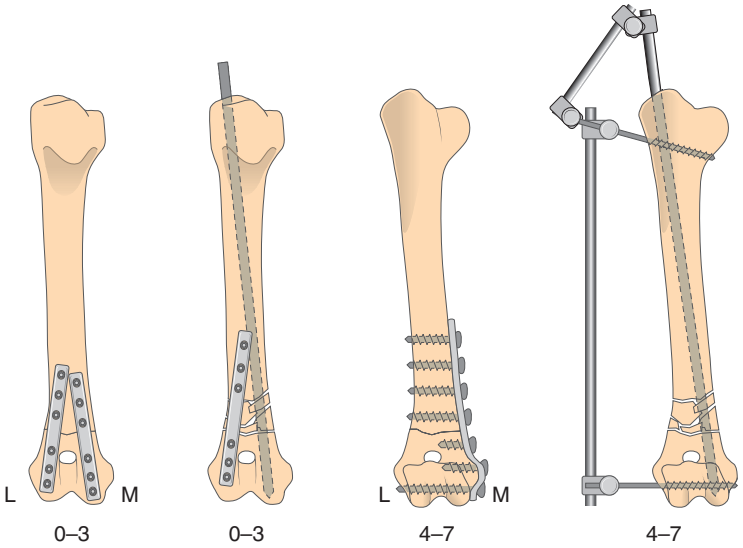


FIG. 33.33 Recommended methods for stabilizing supracondylar fractures based on fracture assessment score. If the fracture assessment score is 0 to 3, two plates or a plate-rod combination may be used. If the fracture assessment score is 4 to 7, a medial compression plate or a two-pin external skeletal fixator plus intramedullary (IM) pin (tie-in configuration) may be used. With fracture assessment scores of 8 to 10, an IM pin seated into the medial condyle and a laterally placed crossed pin may be used to stabilize a transverse fracture *(not illustrated)*.

should be restricted to leash walking and physical rehabilitation until the fracture has healed. Physical rehabilitation encourages controlled limb use and optimal limb function after fracture healing. Care must be taken to develop customized protocols for each patient depending on location of the fracture, stability and type of fracture fixation, potential for healing, abilities and attitudes of the patient, and willingness of the client (see Table 33.2 and Chapter 11). External fixator management includes daily pin care and pin packing as needed. Radiographs should be evaluated in 6 weeks. External fixators may be destabilized at this time if healing is progressing satisfactorily. Radiographs should be repeated at 6-week intervals until fracture bridging is observed.

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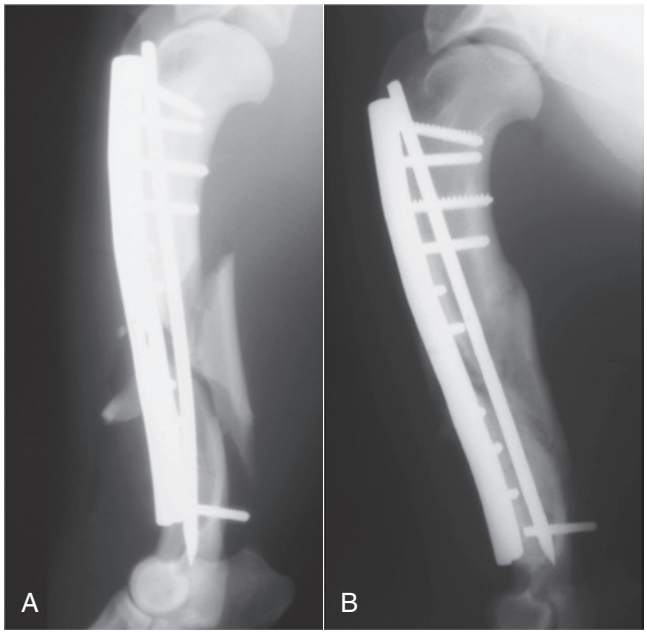


FIG. 33.34 Postoperative radiographs. (A) Stabilization of comminuted fracture with intramedullary pin and plate. (B) Fracture healing 12 weeks later.

Time to bone union depends on the patient fracture assessment. IM pins and ESFs should be removed when healing occurs; cerclage wire, interlocking nails, and bone plates and screws are generally not removed unless a problem is associated with their presence.

COMPLICATIONS

Complications include delayed union, nonunion, malunion, osteomyelitis, pin tract infection, and fixation failure. Poor decision making regarding implant choice relative to the fracture assessment is the most common reason for complications (see p. 1031 for treatment of complications).

PROGNOSIS

The prognosis is generally excellent if proper fracture management procedures are followed (Fig. 33.34).

HUMERAL ARTICULAR, PHYSEAL, AND METAPHYSEAL FRACTURES

DEFINITIONS

Articular fractures involve the joint surface. **Physeal fractures** involve the growth plates in immature animals. **Epiphyseal fractures** and **metaphyseal fractures** occur in the trabecular bone at the proximal or distal end of the humerus.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Fractures of the proximal humeral metaphysis and epiphysis are uncommon but occasionally occur through the proximal humeral physis in young animals. Fractures through the physis may result from minimal external force and exhibit only slight displacement. Careful scrutiny of the lateral radiograph and comparison with a radiograph of the contralateral limb may be necessary to correctly diagnose these fractures. Gunshot fractures can cause severe comminution of the proximal or distal humerus. Fractures of the distal humeral condyle (elbow) are common and include fractures of the lateral or medial portions of the condyle, or both, known as a *T fracture* or *Y fracture* of the condyle. These fractures can also occur with comminuted supracondylar fractures. Lateral condylar fractures predominate over medial condylar fractures for two reasons. First, the radial head articulates with the lateral portion of the condyle, transmitting weight-bearing forces primarily through the lateral portion of the condyle. Second, the anatomic position of the lateral portion of the condyle is eccentric to the bony column, causing weight-bearing forces to be transmitted through the weaker epicondyloid ridge to the humeral diaphysis. Fractures of the lateral portion of the condyle are frequently diagnosed in young, toy breed dogs that fall or jump from furniture or the owner's arms with the elbow extended. When the animal lands, high loads are transmitted through the radial head-lateral condylar axis, resulting in separation of the lateral portion of the condyle. The fracture line passes between the lateral and medial portions of the condyle, crosses the physis, and exits through the metaphysis. Because the physis is involved, the fracture is classified as a Salter IV fracture (see p. 984). Careful evaluation of the craniocaudal radiograph is essential because minimal displacement of the intercondylar fracture can occur. Adult animals also sustain lateral condylar fractures through the mechanism already described. In some breeds, particularly spaniels, incomplete ossification between the medial and lateral portions of the condyle predisposes to condylar fracture and may cause forelimb lameness. Incomplete ossification causing lameness (e.g., incomplete humeral condylar fractures) is best treated with preemptive placement of a large lag screw. Incomplete ossification often occurs bilaterally.

NOTE Evaluate radiographs carefully to avoid missing intercondylar fractures that are minimally displaced.

Isolated medial condylar fractures are not common but do occur in both immature and mature patients. T and Y fractures of the elbow are more common and represent an intracondylar fracture combined with a transverse (T) or oblique (Y) fracture through both medial and lateral epicondyloid ridges.

DIAGNOSIS

Clinical Presentation

Signalment

Lateral condylar fractures are frequently diagnosed in young, toy breed dogs; however, physeal fractures may occur in any juvenile dog or cat of any breed or sex that has open growth plates. Adult animals of any breed or sex may sustain a proximal epiphyseal or distal epiphyseal (elbow) fracture. Spaniel breeds appear to be predisposed to lateral condylar fractures (see previous discussion).

History

Physeal fractures usually result from a fall but also may be caused by automobile accidents. Elbow fractures or proximal humeral

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fractures in adult animals are usually associated with vehicular trauma or gunshot wounds.

Physical Examination Findings

Most affected animals have non–weight-bearing lameness. Swelling of the affected limb is usually obvious if the fracture occurs secondary to an automobile accident. Pain and crepitus can be elicited with limb manipulation.

Diagnostic Imaging

Craniocaudal and lateral radiographs are usually sufficient to make a diagnosis. Because of the manipulation necessary for diagnostic radiographs, some animals may require sedation (see Tables 31.2 and 31.3). In spaniels, if a fracture of the intercondylar articular surface or incomplete ossification is suspected but not evident, oblique radiographic views of both elbows are recommended. CT may be used to obtain definitive diagnosis of incomplete ossification.

Laboratory Findings

Complete blood count and serum chemistry evaluation should be done to assess the status of the animal for anesthesia. Consistent laboratory abnormalities are not present.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include ligament injury of the shoulder or elbow, scapular fractures, and proximal radial or ulnar fractures.

MEDICAL MANAGEMENT

Fractures involving or close to the joint should not be managed with conservative treatment.

SURGICAL TREATMENT

Treatment of humeral metaphyseal and epiphyseal fractures depends on the animal's age and overall health and the fracture configuration. The fracture assessment score (see p. 985) is used to help determine the rigidity of stabilization necessary for the fracture to heal. Surgical treatment of Salter I and II physeal fractures consists of anatomic reduction and stabilization with K-wires or small pins that are smooth so as not to interfere with physeal function. Salter III fractures of the proximal humerus may also be treated with multiple K-wires or small pins. In animals that are close to maturity, threaded implants may be used to compress the fractured physis. Anatomic reduction and rigid fixation with a lag screw are critical for optimal outcome with Salter IV fractures of the humeral condyle (Box 33.5).

BOX 33.5 Implant Use According to Fracture Assessment Score (FAS) for Humeral Condylar Fractures

FAS 4 to 10
Lag screw plus K-wires
Self-compressing pin

FAS 0 to 3
Lag screw plus plate

Preoperative Management

Patients that have sustained trauma should be stabilized before anesthesia and fracture treatment. Analgesics should be provided to posttraumatic animals (see Chapter 13).

Anesthesia

Refer to Tables 32.1 and 32.2 for anesthetic management of patients with fractures.

Surgical Anatomy

Depending on the severity of injury, the normal anatomy and surgical landmarks of this region may be distorted by soft tissue bruising and swelling. Starting the surgical dissection in an area with less swelling or bruising and using bony landmarks are helpful. Proximally, the greater tubercle and acromion of the scapula are readily palpable; distally the medial and lateral epicondyles are easy to identify. The cephalic vein courses within the subcutaneous tissue along the craniolateral surface of the limb. The radial nerve lies beneath the lateral head of the triceps near the distal third of the humerus. This nerve must be identified as it courses superficial to the brachialis muscle before the brachialis muscle is reflected from the humeral diaphysis. To visualize the nerve, dissection should be initiated adjacent to the cranial edge of the lateral head of the triceps, near the lateral epicondyle. The tissue plane between the brachiocephalicus and triceps muscles must be carefully dissected to prevent injury to this nerve. When performing an olecranon osteotomy, take care to avoid the ulnar nerve along the cranial edge of the medial head of the triceps muscle.

NOTE To make identification of landmarks easier, begin the surgical dissection proximal or distal to the area of bruising.

Positioning

The patient is positioned in lateral recumbency for all lateral approaches and for olecranon osteotomy. The patient is positioned in dorsal recumbency for medial or combined medial and lateral approaches to the elbow. A hanging-leg preparation will facilitate manipulation of the limb during surgery. The region from just dorsal to the scapula proximally to the carpus distally should be clipped and prepared for aseptic surgery.

SURGICAL TECHNIQUE

Surgical Approach to the Proximal Epiphysis

Make an incision over the craniolateral region of the proximal humerus (Fig. 33.35A). Begin the incision 2 to 3 cm proximal to the greater tubercle, and extend it distally to a point near the midshaft of the humerus. Incise through the subcutaneous tissue along the same line to expose deep fascia along the lateral border of the brachiocephalicus muscle and the insertion of the deltoid muscle (Fig. 33.35B). Elevate and reflect the brachiocephalicus muscle from the cranial surface of the bone. Elevate the deltoid muscle, and retract it caudally to expose the insertions of the teres minor and infraspinatus muscles. Make an incision through the insertions of these two muscles to expose the lateral surface of the proximal humerus (Fig. 33.35C). If increased exposure of the craniomedial surface of the proximal humerus is needed, release the insertion of the superficial pectoral muscle deep to the brachiocephalicus muscle. To close, suture the tendons of the teres minor and infraspinatus muscles.

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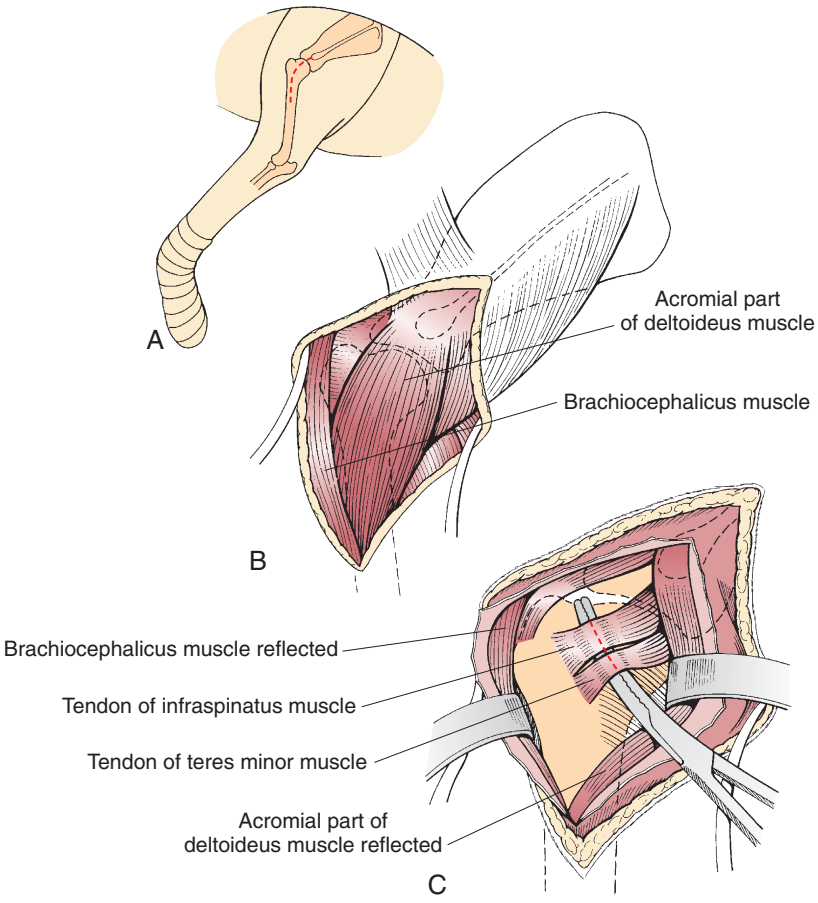


FIG. 33.35 (A) To expose the proximal humerus, make an incision over its craniolateral region. (B) Expose deep fascia along the lateral border of the brachiocephalicus muscle and the insertion of the deltoid muscle. (C) Make an incision through the insertions of the infraspinatus and teres minor muscles to expose the lateral surface of the proximal humerus.

Then suture the fascia of the superficial pectoral muscle to the fascia of the deltoid muscle. Appose the fascia of the brachiocephalicus muscle, then suture subcutaneous tissue and skin.

Surgical Approach to the Lateral Portion of the Humeral Condyle and Epicondyle

Make a lateral incision beginning over the distal third of the humerus and extending to a point 4 to 5 cm distal to the elbow overlying the ulna (Fig. 33.36A). Incise the subcutaneous tissue to expose the deep brachial fascia. Incise the deep fascia along the cranial border of the lateral triceps muscle, and continue this incision across the joint over the extensors (Fig. 33.36B). Incise the intermuscular septum between the extensor carpi radialis and the common digital extensor muscles, and continue the incision proximally through the periosteal origin of the extensor carpi radialis muscle (Fig. 33.36C). Retract the muscle cranially to expose the joint capsule and underlying lateral condyle. For further exposure of the epicondyle, incise through the anconeus muscle at its origin on the epicondylar ridge (Fig. 33.36D). Incise the joint capsule with an L-shaped incision to visualize the humeral condyle. To close the incision, suture the joint capsule and close the intermuscular septum with interrupted sutures. Suture the origins of the external carpi radialis and anconeus muscles together with interrupted sutures, and then suture subcutaneous tissue and skin.

Surgical Approach to the Elbow via Olecranon Osteotomy

Make an incision from the distal third of the humerus to the proximal third of the ulna. Center the incision at the level of the olecranon process over the caudolateral region of the leg. Undermine the subcutaneous tissue such that the caudal skin margin can be reflected over the olecranon process to expose the medial epicondyle. Laterally, free the cranial border of the lateral head of the triceps near its tendinous insertion at the olecranon (Fig. 33.37A).

Next, flex the elbow and palpate the ulnar nerve as it courses in the deep fascia along the cranial edge of the medial head of the triceps. The nerve should be isolated and protected during the osteotomy procedure. *Incise the fascia along the cranial border of the medial head of the triceps near its insertion at the olecranon. Pass a Gigli wire through the lateral fascial incision so that it exits through the medial fascial incision (Fig. 33.37B). Pull the wire caudally next to the olecranon beneath the tendon of the triceps (Fig. 33.37C). Make sure the ulnar nerve is free from the wire, and then osteotomize the olecranon process with the Gigli wire. Incise and retract the anconeus muscle from the lateral and medial epicondylar ridges (Fig. 33.37D), then reflect the olecranon process and triceps muscle proximally to visualize the caudal surface of the elbow joint. For closure, reduce and stabilize the olecranon process with a tension band wire (see p. 1015). Suture the border of the triceps to the deep fascia on the medial and lateral sides of the leg, and close the subcutaneous tissue and skin.*

Stabilization of Proximal Epiphyseal and Metaphyseal Fractures

Patients with gunshot injuries or injuries resulting from high-velocity motor vehicle accidents generally have comminuted fractures and significant soft tissue injury. A buttress plate or a buttress plate-rod construct is used for patients with a low fracture

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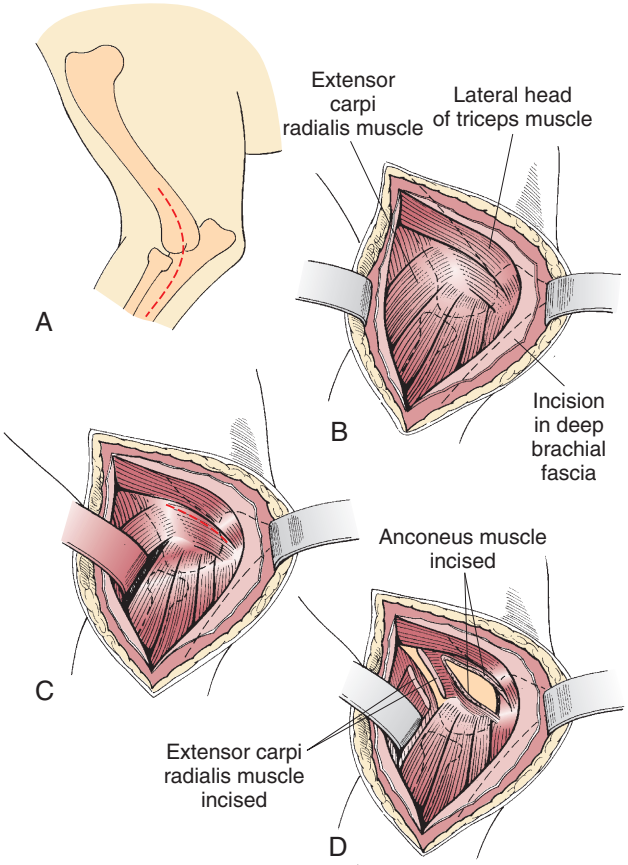


FIG. 33.36 (A) To expose the distal humeral condyle, make a lateral incision over the distal third of the humerus, extending 4 to 5 cm distal to the elbow joint. (B) Incise the deep fascia to expose the extensor muscles. (C) Incise the intermuscular septum between the extensor carpi radialis and common digital extensor muscles. Continue the incision proximally through the periosteal origin of the extensor carpi radialis muscle. (D) Retract the muscle cranially to expose the joint capsule and underlying humeral condyle. For further exposure of the epicondyle, incise through the anconeus muscle at its origin on the epicondylar ridge.

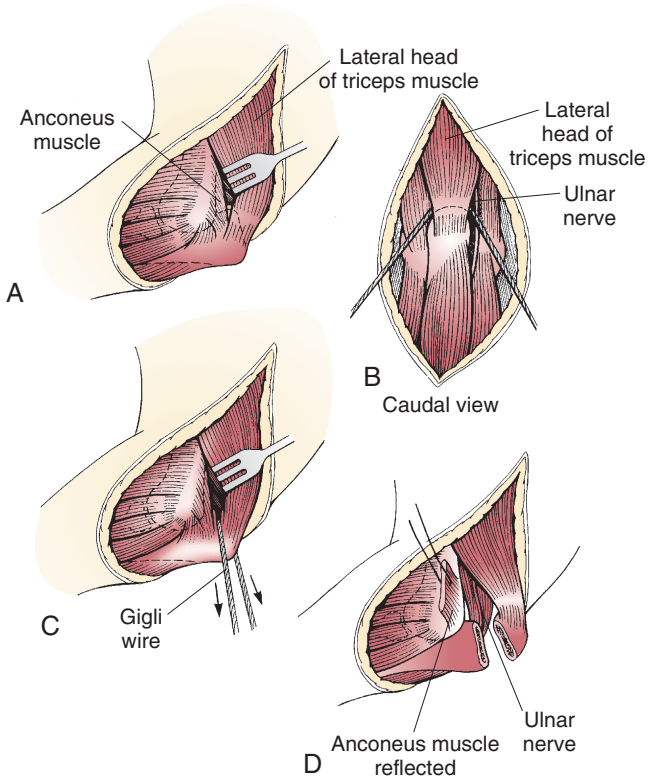


FIG. 33.37 (A) For osteotomy of the olecranon, free the cranial border of the triceps lateral head near its tendinous insertion at the olecranon. (B) Pass a Gigli wire through a lateral fascial incision to exit through a medial fascial incision. (C) Pull the wire caudally next to the olecranon beneath the triceps tendon. (D) Incise and retract the anconeus muscle from the lateral and medial epicondylar ridges.

assessment score. Comminuted fractures in patients with a moderate fracture assessment score may be treated with a buttress plate or a modified type Ib external skeletal fixator, with an IM pin tie-in. Patients with a low or moderate fracture assessment score and a simple fracture pattern (i.e., transverse or oblique fractures) may be treated with a compression plate or neutralization plate. Alternatively, use an IM pin combined with cerclage wire and K-wire (in an oblique fracture) and supported with a type Ia external skeletal fixator.

Stabilization of Proximal Physeal Fractures

Salter I or II physeal fractures of the proximal humerus may be stabilized with diverging K-wires or small Steinmann pins. *Reduce the fracture and maintain reduction with a pointed reduction forceps. Insert two pins or K-wires into the bone proximally at the greater tubercle, and drive them distocaudally to cross the fracture line and seat in the caudal metaphysis (Fig. 33.38A).*

Salter III fractures of the proximal physis are also stabilized with divergent K-wires. *Attach the humeral head to the proximal humerus with two K-wires, then reduce the greater tubercle and secure it to the humerus with two K-wires (see Fig. 33.38B).*

Stabilization of Lateral or Medial Condylar Fractures

Condylar fractures are usually stabilized after open reduction and inspection of the joint reconstruction. Fluoroscopically guided closed reduction with internal fixation with screws and K-wires or small pins is an alternative. In most cases, stabilization is best attained with an intercondylar lag screw or a self-compressing pin combined with a K-wire, a small pin, or a plate bridging the fracture of the epicondyloid crest. Condylar fractures stabilized with a transcondylar screw and K-wire have a higher complication rate than those stabilized with a transcondylar lag screw and supracondylar screw or bone plate.[3] Lateral humeral condylar fractures may also be repaired using multiple transcondylar convergent or parallel K-wires.[4] *Reflect the lateral condyle fragment laterally, and drill a glide hole retrograde from the center of the fracture surface to exit cranially and distally to the lateral epicondyle before reducing the fracture. Reduce the fracture and secure the reduction with pointed reduction forceps. Place a K-wire perpendicular to the fracture surface and parallel to the glide hole (Fig. 33.39A). Place an appropriately sized drill sleeve in the glide hole, and drill, measure, then tap the thread hole to accept the appropriate screw. Use a washer with the screw to prevent subsidence of the screw head in soft bone. Place a K-wire across the lateral epicondyloid*

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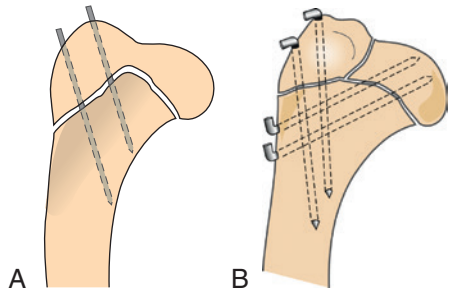


FIG. 33.38 (A) Stabilize a proximal humeral Salter I fracture with two K-wires or small Steinmann pins. (B) Stabilize a proximal humeral Salter III fracture with multiple K-wires or small Steinmann pins. (B, Modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat.* St. Louis: Elsevier; 2005.)

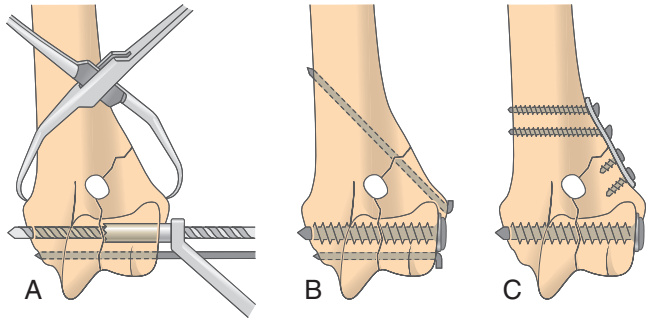


FIG. 33.39 (A) Reduce the lateral portion of the condyle with the predrilled glide hole and secure it with pointed reduction forceps and a K-wire. Drill the medial portion of the condyle to prepare for the lag screw. (B) Stabilize the fracture with a lag screw and K-wires. (C) In older dogs with potentially long healing times, stabilize the lateral condylar fracture with a lag screw and plate. (Modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat.* St. Louis: Elsevier; 2005.)

crest to provide additional stability for short oblique or transverse fractures (Fig. 33.39B). In patients with suspected incomplete ossification and subsequently prolonged healing time, bridge the epicondyloid crest with a small plate to protect the lag screw from shearing forces (Fig. 33.39C) (see Box 33.5).

Stabilization of T or Y Fractures of the Elbow

These elbow fractures most often result from motor vehicle accidents and falls from a significant height. Occasionally, these fractures occur after minimal trauma because incomplete ossification of the condyle is the underlying pathology. The intercondylar fracture is accompanied by a transverse, oblique, or comminuted fracture through the medial and lateral epicondyloid crests. Open reduction is necessary for accurate alignment of the intercondylar fracture. A combined medial and lateral approach to the humeral condyle (see Figs. 33.29 and 33.36), or an osteotomy of the olecranon (see Fig. 33.37) may be used to expose the fracture. The method of stabilization depends on the patient's fracture assessment score. Those with a low score or an intermediate score should be treated with plates and screws or with a plate and rod combination.

For elbow fractures in patients with a favorable fracture assessment score, an intercondylar lag screw and two IM pins may be sufficient. *Perform medial and lateral approaches to the elbow joint and distal humerus (Fig. 33.40A). Reduce the articular fracture and maintain reduction with pointed reduction forceps across the condyles. Stabilize the articular fracture with a lag screw (Fig. 33.40B), then reduce the humeral metaphyseal fracture. Place an IM pin up the medial portion of the condyle and across the fracture line to help maintain reduction. Place a plate across the lateral fracture line. Cut the pin short at the distal humerus. Alternatively, stabilize the medial and lateral fractures by applying one plate to the medial surface of the humerus and a second plate to the caudolateral epicondyloid crest.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Helpful instruments for fracture repair include a battery-powered or air-driven drill with a wire-driving attachment, self-retaining retractors, pointed reduction forceps, and wire cutters. A plating system is required for screws and bone plates.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiographs are taken to evaluate alignment, apparatus and apposition. Postoperative pain management is indicated (see Boxes 32.1 and 32.2 and Table 32.4). Activity should be restricted to leash walking and physical rehabilitation until the fracture has healed. Physical rehabilitation encourages controlled limb use and optimal limb function after fracture healing and is particularly important for fractures involving the joint. Care must be taken to develop customized protocols for each patient depending on location of the fracture, stability and type of fracture fixation, potential for healing, abilities and attitudes of the patient, and willingness of the client (Table 33.3; see also Chapter 11). External fixator management includes daily pin care and pin packing as needed. Radiographs should be evaluated in 6 weeks. External fixators may be destabilized at this time if healing is progressing satisfactorily. Radiographs should be repeated at 6-week intervals until fracture bridging is observed. IM pins and external fixators should be removed when the fracture has healed. Lag screws and plates are left in place unless they cause problems.

COMPLICATIONS

Proximal physeal fractures of the humerus generally heal quickly; however, premature closure of the physis generally occurs and may affect bone length in immature dogs. K-wires and small pins may migrate. Damage to the physis from Salter IV fractures of the lateral portion of the condyle and subsequent fixation does not appear to cause shortening of the humeral diaphysis or distortion of the condyle. Intraarticular fracture may result in postoperative degenerative joint disease, although this is minimized with careful reduction and rigid fixation. Supracondylar and condylar fractures of the humerus may have prolonged healing periods, with the implant subjected to moderate stresses for extended periods. Bone resorption and implant loosening may eventually result. Fatigue breakage of implants can occur (Box 33.6). A high complication rate is associated with olecranon osteotomy repair. Distal condylar fractures generally heal quickly

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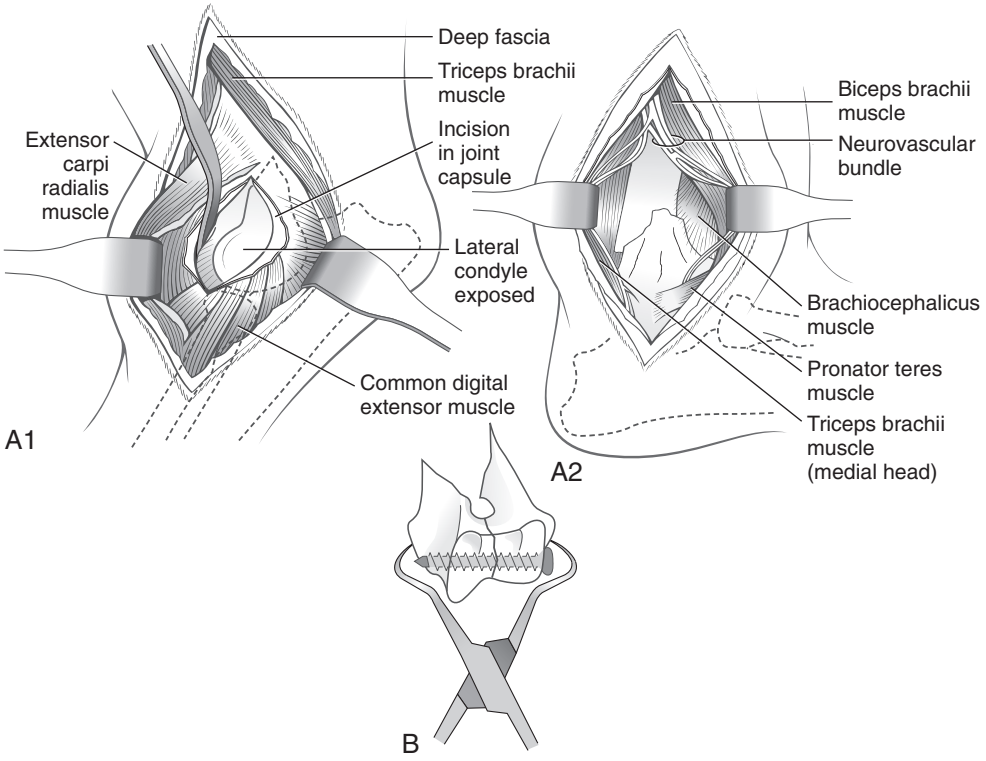


FIG. 33.40 (A) Perform medial and lateral approaches to the elbow joint and distal humerus. (B) Stabilize a Y or T fracture with a lag screw across the condylar fracture, an intramedullary pin through the medial condyle driven up the medullary canal, and a plate on the lateral condylar surface. Alternatively, apply a plate to the medial surface. (Modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat.* St. Louis: Elsevier; 2005.)

in immature dogs, but healing is often delayed in older dogs. Decreased range of motion of the elbow often occurs after surgery.

PROGNOSIS

The prognosis is generally excellent if proper fracture management procedures are followed (Fig. 33.41).

RADIAL AND ULNAR DIAPHYSEAL FRACTURES

DEFINITIONS

Radial diaphyseal fractures and **ulnar diaphyseal fractures** represent disruption of the continuity of the diaphyseal cortical bone. **Open fractures** (wounds through the skin over the bone) may occur because of sparse soft tissue coverage.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Diaphyseal fractures usually involve the middle to distal diaphysis of both radius and ulna. Because these fractures usually occur secondary to trauma, the animal should be closely evaluated to detect concurrent injuries (e.g., pulmonary contusions, pneumothorax, rib fractures, traumatic myocarditis). The paucity of soft tissue increases the possibility of open fracture and potentially decreases the extraosseous blood supply, both of which can delay bone healing. Minimal soft tissue coverage of bone plates results in tissue irritation and cold hypersensitivity but is advantageous for placing an external fixator.

Toy breed dogs often fracture the radius and ulna after apparently minimal trauma of jumping or falling. These fractures are associated with a high complication rate. Biomechanical instability related to the short oblique nature of these fractures, a paucity of distal diaphyseal vasculature compared with large breed dogs, and limited surrounding soft tissue for the extraosseous vasculature may contribute to the high frequency of delayed unions and nonunions.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, breed, or sex of dog or cat may be affected. Young animals sustain vehicular trauma more frequently.

History

Affected animals usually have non–weight-bearing lameness after trauma. Toy breed dogs are often brought in after apparently minimal trauma of jumping or falling.

Physical Examination Findings

Because of the traumatic nature of radial and ulnar fractures, the entire animal must be assessed to detect abnormalities of

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TABLE 33.3 Sample Rehabilitation Protocol for Patient With an Articular Fracture

Treatments/Modalities	Day 1–7	Day 7–21	3–8 Wk	8–12 Wk and Beyond
Pain medications	As directed	As directed	PRN	PRN
Cryotherapy	10–15 min 3 times daily before walks or exercises First session immediately after surgery	Use after exercise for 15 min	PRN after exercises	PRN after exercises
Heat therapy		Apply heat to the adjacent muscles 10 min before PROM or exercise	As before 10 min twice daily	PRN
Massage	Twice daily for edema from toes toward heart	Continue twice daily	Massage adjacent muscles before active exercise	Massage adjacent muscles before active exercise
PROM	Gentle joint flexion/extension 10 reps, 3–4 times daily Elicit flexor reflex by toe pinching	Continue flexion and extension with mild resistance 10–15 reps 3 times daily	Continue PROM as previously described up to 4 wk	
Laser therapy	Daily or every other day for first week	Every other day for first week then twice weekly	PRN	
Walks	At 5 d start slow controlled leash walks for 5 min twice daily to encourage active ROM	Increase each walk by 1–3 min each week	Gradually increase to 10–15 min twice daily	20– to 30-min walks twice daily including 10 min of incline work as long as healing has taken place
Balancing	Gentle balancing exercises on foam pad to encourage weight bearing 5 min twice daily	5 min three times daily After 10 d increase the instability of the surface to challenge the muscles	10 min 2–3 times daily Consider balancing on a ball or inflatable disc with assisted support	Incorporate challenging surfaces to increase stifle flexion: sand, snow, tall grass
Underwater Treadmill[a]		10–15 min once daily after day 10—incision healed	15–20 min daily	20–30 min 3 times weekly
Stairs[a]			Likely 6+ weeks after surgery Start with one flight and add one flight per week	Work up to 5 flights twice daily
Cavalettis[a]			5–10 reps over 5 rails once daily	10–15 reps over 5 rails twice daily
Swimming[a]			10–15 min with breaks daily	20–30 min once to twice daily

Please see special instructions for Distal Femoral Physeal Fractures regarding bandaging and rehabilitation.
[a]Should only be done after radiographic evidence of healing has occurred and implant integrity is verified by surgeon.
PRN, As needed; *PROM,* passive range of motion; *ROM,* range of motion.

BOX 33.6 Common Errors in Humeral Condylar Fracture Fixation

- Failure to reduce a joint surface anatomically results in degenerative joint disease and loss of function.
- Failure to use a large lag screw and support the fracture with a plate can result in implant failure in mature animals with condylar fractures initiated by incomplete ossification.

other body systems. Palpation of the limb reveals swelling, pain, and crepitation. The fracture may be open: loss of or damage to adjacent soft tissue may be substantial. Affected animals often appear to have abnormal proprioceptive responses because they are reluctant to move the limb.

NOTE A careful neurologic examination will help differentiate between true neurologic injury and apparent neurologic injury associated with pain.

Diagnostic Imaging

Craniocaudal and lateral radiographs of the affected radius and ulna (which include the elbow and carpus) are required to assess the extent of bone and soft tissue injury. Fractious or animals in extreme pain may require sedation (see Tables 31.2 and 31.3) or general anesthesia for radiography after it has been determined that no contraindications (e.g., shock, hypotension, or severe dyspnea) to administration of sedatives or anesthetics exist. Thoracic radiography should be performed to evaluate for thoracic trauma.

Laboratory Findings

Complete blood count and serum chemistry evaluation should be done to evaluate the status of the animal for anesthesia and to determine whether concurrent injury or damage to the renal or hepatobiliary systems has occurred.

DIFFERENTIAL DIAGNOSIS

Animals that have radial and ulnar fractures should be evaluated to determine whether the fractures are the result of trauma or underlying pathology (e.g., neoplasia).

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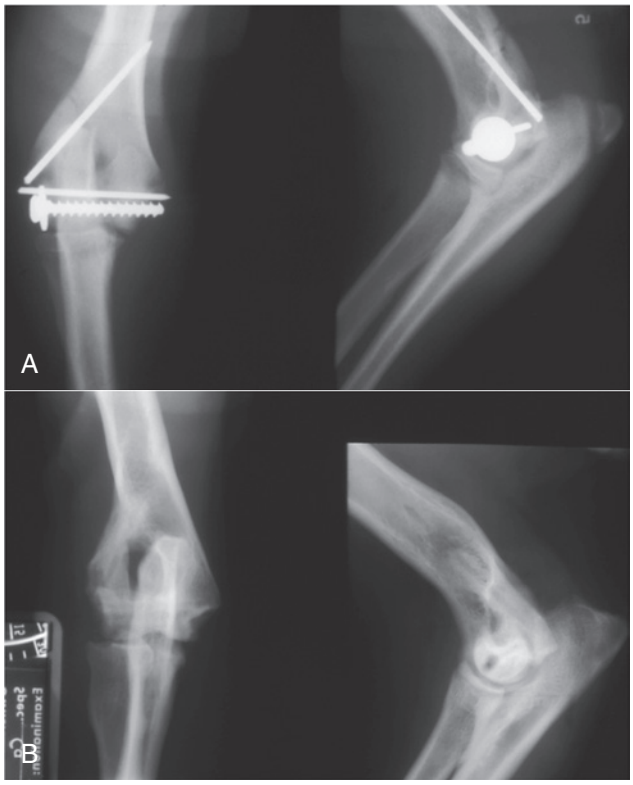


FIG. 33.41 Postoperative radiographs. (A) Stabilization of a Salter IV fracture of the lateral portion of the humeral condyle with a lag screw and two K-wires. (B) Implants were removed 5 months later. Note the lack of callus and minimal degenerative joint disease.

BOX 33.7 Decision Making for Open or Closed Reduction for Radial Diaphyseal Fractures

Open Reduction
- Displaced reducible fractures with plate application

Limited Open Reduction
- Displaced reducible fractures with external skeletal fixation
- Comminuted fractures for cancellous bone graft placement

Closed Reduction
- Nondisplaced fractures with external coaptation or external skeletal fixation
- Comminuted nonreducible fractures with external skeletal fixation

BOX 33.8 Implant Use for Fractures of the Radial Diaphysis According to Fracture Assessment Score (FAS)

FAS 0 to 3
Bone plate and screws
Type II external skeletal fixation

FAS 4 to 7
Type Ib or type II external skeletal fixation
Bone plate and screws

FAS 8 to 10
Type Ia or type Ib external skeletal fixation
Cast

MEDICAL MANAGEMENT

Medical treatment of animals with radial and ulnar fractures includes analgesics for posttraumatic pain (see Chapter 13) and antibiotics to treat open fractures. Conservative management of radial and ulnar diaphyseal fractures consists of splints and casts and is reserved for closed, nondisplaced, or greenstick fractures in immature animals. Cast fixation is appropriate for these fractures because the joint above and below the fractured bone (elbow and carpus) can be immobilized, and the fractures heal rapidly. Casts and splints are contraindicated for treatment of distal diaphyseal fractures in miniature and toy breed dogs owing to the high incidence of nonunion in these animals.

NOTE When choosing cast fixation, consider whether the animal will be able to bear weight on the other three limbs.

SURGICAL TREATMENT

External skeletal fixators and bone plates are the implants of choice to repair radial fractures. The implant system chosen should reflect the patient fracture assessment score (Box 33.7). The decision to perform an open or a closed reduction of radial and ulnar diaphyseal fractures is made on the basis of fracture configuration, fracture assessment score, and the implant system chosen (Box 33.8). Simple or moderately comminuted fractures with large fragments that can be anatomically reconstructed to establish the bone column are candidates for open reduction and stabilization with internal fixation, limited open reduction and ESF, or a combination of techniques. Severely comminuted fractures that cannot be completely reconstructed are candidates for closed reduction and ESF or open reduction and application of a bridging plate and a cancellous bone autograft (see p. 991), or minimally invasive percutaneous osteosynthesis. Minimally invasive plate application has similar results as open reduction and fixation in terms of reduction, alignment, and time to union.[5] Whether the fracture is open or closed is less important than the potential of the fracture to be anatomically reconstructed. Advantages and disadvantages of open and closed reduction need to be weighed to determine the best approach for each individual fracture. The ulna is usually supported indirectly by radial stabilization; however, stabilization of the ulna is indicated when doing so will add support to a comminuted radial fracture, when additional support is needed for a large dog, and when anatomic reduction of the radius and ulna is essential to future performance of an athlete.

Preoperative Management

Damage to or loss of tissue in the area of the fracture may be extensive. Open wounds should be managed initially by carefully clipping surrounding hair, cleaning the wound, and taking a swab for bacterial culture and susceptibility testing. The antebrachium should be temporarily stabilized with a Robert Jones bandage (see p. 981) to immobilize fragments, decrease or prevent soft tissue swelling, protect or prevent open wounds, and enhance

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patient comfort until surgery can be performed. Perioperative pain management should also be instituted (see Chapter 13). Concurrent injuries should be managed before anesthetic induction for fracture fixation.

Anesthesia

Refer to Tables 32.1 and 32.2 for anesthetic management of patients with fractures.

Surgical Anatomy

The craniomedial surface of the radius and the caudal lateral surface of the ulna are not covered by muscle and can be easily palpated to serve as landmarks for location of the incision. Extensor muscles are located cranial to and flexor muscles caudal to the radius and can be retracted to expose the bone. The cephalic vein crosses the medial portion of the distal radius. The lateral radial head is palpable beneath the extensor muscles of the forearm.

Positioning

The limb should be clipped and prepared for aseptic surgery from the shoulder to the carpus. If cancellous bone graft harvest is anticipated, a donor site (e.g., proximal humerus) should also be prepared. If closed reduction or limited open reduction with ESF is chosen, positioning the animal with the affected leg suspended from the ceiling facilitates visualization of correct joint alignment (see p. 990). If open reduction with plate fixation is performed, the animal may be positioned in dorsal recumbency and the limb draped out and pulled laterally to expose the craniomedial aspect of the radius, or positioned with the affected leg draped out while suspended from the ceiling.

SURGICAL TECHNIQUE

Craniomedial Approach to the Radius

Palpate the radius directly under the skin and subcutaneous tissue on the craniomedial surface of the limb. Make an incision through the skin and subcutaneous tissue to expose the radial diaphysis. Extend the incision distally, and elevate the extensor tendons to expose the cranial surface of the distal metaphysis of the radius (Fig. 33.42).

Implant Application Techniques Specific to the Radius

Application of Casts

Casts can be applied as the sole method of fixation for stable fractures in young dogs or cats when the fracture will maintain adequate reduction and will heal quickly. *Position the animal in lateral recumbency with the fractured leg against the table. Have the assistant hold the limb in adduction and slight carpal flexion. Apply stockinette, cast padding, and cast material (for general principles of cast application, see p. 995) (Fig. 33.43). To construct a bivalve cast, use extra cast padding while applying the cast. When the cast is hard, cut it longitudinally along the medial and lateral surfaces, and tape it in position.*

The bivalve cast does not offer as rigid a fixation as a cylinder cast, but it is easily changed to allow wound management.

NOTE Warning: Radial fractures in toy breed dogs treated with casts and splints have a high complication rate; these fractures should be managed with plate fixation.

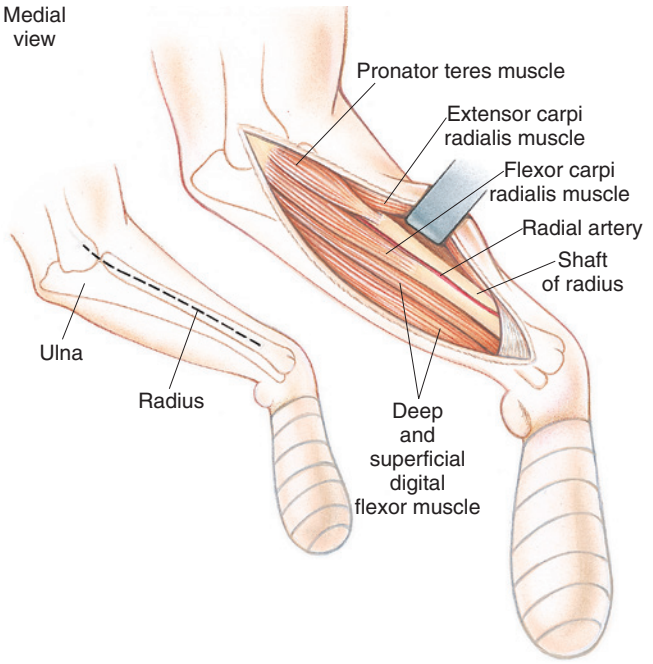


FIG. 33.42 For a craniomedial approach to the radial diaphysis, make an incision through skin and subcutaneous tissue to expose the radial diaphysis. Retract the extensor carpi radialis muscle laterally to expose the diaphysis.

Application of Intramedullary Pins

IM pins are difficult to use in the radius because of the narrow configuration of the radial medullary canal and the necessity of invading the carpal joint to position the pin. Complications associated with IM pin placement in the radius include rotation, osteomyelitis, delayed union, nonunion, and degenerative joint disease of the carpus.

NOTE IM pins and interlocking nails are contraindicated as treatment for radial fractures because of the narrow configuration of the radial medullary canal and the necessity of invading the carpal joint to position the pin.

An IM pin can be used to align the ulna, stabilize a simple ulnar fracture, and add support to the primary fixation of a comminuted radial fracture (i.e., external fixator or plate). *Insert the IM pin into the medullary canal from the proximal surface of the olecranon, and drive it in a normograde manner to the fracture surface. Keep the lateral cortex of the ulna parallel to the pin to maintain the pin within the medullary canal. Reduce the fracture and drive the pin distally as far as possible without penetrating the cortex. Cut the excess proximal pin below the level of the skin, over the proximal ulna (for general principles of IM pin application, see p. 1008).*

Application of External Skeletal Fixators

ESF is particularly useful for treating a wide variety of radial diaphyseal fractures. The stiffness of the fixator can be increased in animals with low fracture assessment scores by adding fixation pins and using bilateral and biplanar frames. Radial fractures

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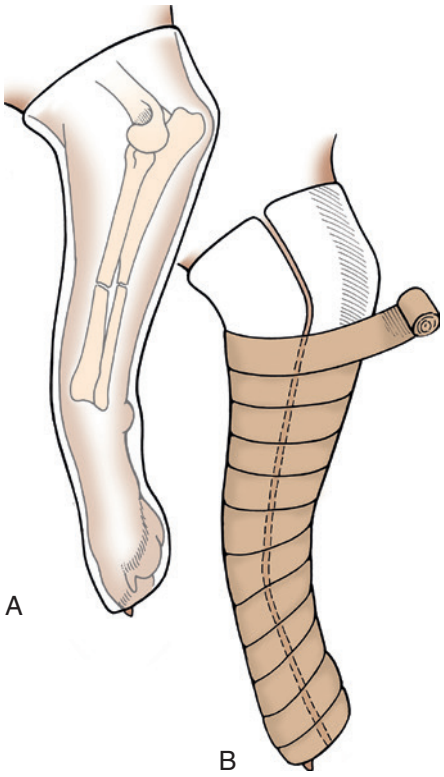


FIG. 33.43 Casts are used to stabilize closed, nondisplaced radial-ulnar fractures in patients with fracture assessment scores of 8 to 10. (A) A full cylinder cast, which immobilizes the elbow and carpus, is placed with the limb positioned in slight carpal flexion and varus angulation. (B) The cast can be bivalved by placing the cast material over multiple layers of padding, cutting it on the lateral and medial aspects, and securing it with elastic tape.

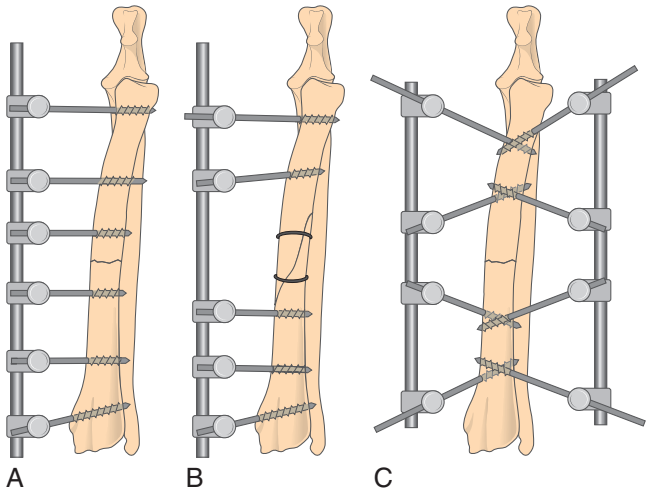


FIG. 33.44 Type Ia and Ib external fixators are used to treat radial fractures in patients with high and moderate fracture assessment scores. (A) A type Ia external fixator is placed on the craniomedial surface of the radius to stabilize a transverse fracture. (B) Anatomic reconstruction of a long oblique fracture with cerclage wire restores the bony column and allows load sharing with the external fixator. (C) A type Ib fixator is applied to patients with moderate fracture assessment scores to provide additional stability.

are frequently open, making use of external fixation attractive for avoiding invasion of the fracture site with metal implants. With open fractures, implant removal is desirable after fracture healing and is easily accomplished with external fixation. *Position the dog in dorsal recumbency and drape the leg while it is hanging from the ceiling. Use limited open reduction for reducible fractures and closed reduction for nonreducible fractures.* If open reduction of the fractured radius is performed, consideration should be given to harvesting autogenous cancellous bone to enhance bone healing. The most accessible site for cancellous bone harvest is the ipsilateral proximal humerus (see p. 991). *Apply a type Ia frame to the cranial medial surface of the radius (Fig. 33.44A–B) (see p. 998 for general principles of external fixator application).* Applying the frame at this location avoids penetration of the major muscle masses with fixation pins and decreases the morbidity associated with the pins. *Apply a type Ib fixation splint to the cranial medial and cranial lateral surfaces of the radius (Fig. 33.44C). Add articulation and diagonal connecting bars for enhanced stability as needed.*

NOTE Construct the fixator to suit the patient and the fracture. A lower fracture assessment score requires a stiffer fixator.

Although penetration of major muscle masses is unavoidable with type II frames, they may be used because of their increased stiffness, and because the proximal and distal transfixation pins can be used as guide pins for limb alignment.

NOTE Some dogs have very narrow radii when viewed from the lateral radiographic projection, making type II frame application hazardous. Scrutinize the radiographs before planning a type II frame.

To apply a type II frame, insert transfixation pins into the proximal and distal metaphyses of the radius. These pins should be centered in the bone in the frontal plane and parallel to the respective joint surfaces. Reduce the fracture and secure the pins with medial and lateral connecting bars. Fill out the frame by placing at least two pins (preferably three) proximal and distal to the fracture (Fig. 33.45A). Often the cranial curve of the radius precludes the placement of bilateral transfixation pins in the center of the fixator, and unilateral pins are used instead (Fig. 33.45*B*). *Correct valgus or varus malalignment by loosening the clamps and distracting the appropriate side of the limb until the joint surfaces are parallel. Correct mild rotation by reversing the position of the clamps on the appropriate side of the pins in the distal fragment. Make every effort to correctly align the limb before the procedure is completed.* External fixation with fixation pins and methylmethacrylate connecting bars is a versatile alternative to the standard external fixation system (see p. 1002 for general principles of acrylic frame application). Fixation pins of any size can be directed at angles that optimize the purchase of good quality bone, without regard for clamp compatibility or uniplanar pin placement. This technique may be used to stabilize distal diaphyseal fractures in toy breed dogs.

Circular external fixators may be used effectively for radial diaphyseal fractures, especially comminuted nonreducible

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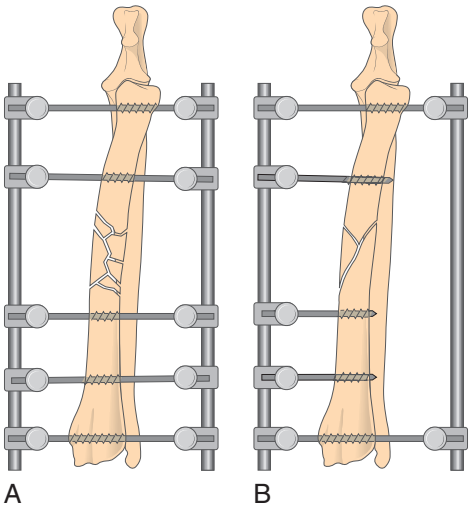


FIG. 33.45 (A) A maximal type II external fixator frame consists of all transfixation pins. (B) Because of the cranial curve of the radius, a minimal type II frame is often constructed using unilateral fixation pins to fill out the frame.

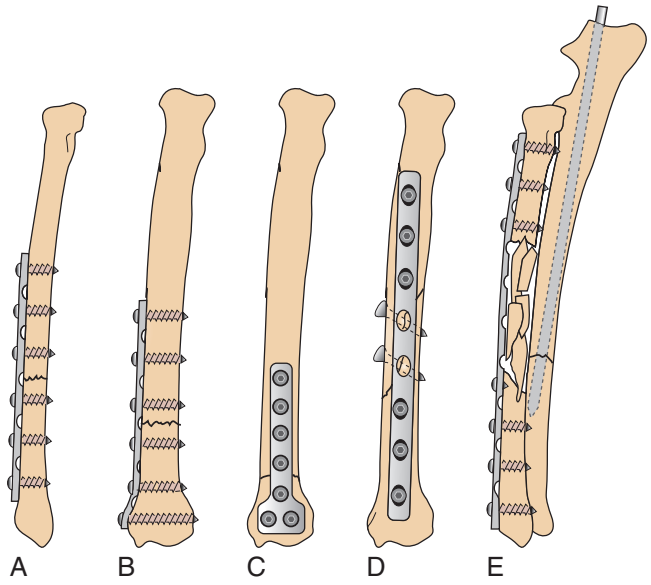


FIG. 33.46 (A) and (B) A compression plate is used to stabilize transverse fractures of the radial diaphysis and can be applied to the cranial or medial surface of the bone. (C) A T plate is used to stabilize distal radial diaphyseal fractures where a small distal bone segment is present. (D) Long oblique fractures may be stabilized with lag screws and a neutralization plate. (E) Comminuted nonreducible radial diaphyseal fractures are bridged with a plate. Additional support may be gained by supporting the ulna with an intramedullary pin. (Modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat*. St. Louis: Elsevier; 2005.)

fractures treated with closed reduction. The fixation frame may be manipulated postoperatively to correct angular deformities in both cranial caudal and medial lateral planes. Circular fixators allow controlled axial micromotion of stabilized bone segments, promoting rapid bone union. However, circular fixators require considerable preoperative planning and frame construction to shorten surgical time, as well as optimal aftercare to minimize implant-related complications (see p. 1003 for general principles of circular fixator application). Combining a distal ring and wires with a linear connecting rod and fixation pins placed proximally, a hybrid construct, is useful in stabilizing complex radial fractures with short distal bone segments (see p. 1008 for general principles of hybrid frame application).

Application of Bone Plates and Screws

Bone plates are an excellent method of stabilizing radial and ulnar diaphyseal fractures (see p. 1016 for general principles of plate and screw application). Mini T plates and veterinary cuttable plates are particularly useful for stabilizing fractures in miniature and toy breed dogs. *Apply bone plates to the wide, flat, cranial surface of the radius or, alternatively, to the medial surface of the distal radius (Fig. 33.46).* Cranially applied locking plates are usually larger than medially applied locking plates for stabilization of radial fractures due to the surface area of the bone. In spite of the size difference, each has distinct biomechanical advantages with the cranial plate having greater stiffness but the medial plate being superior in cyclic loading.[6]

Stabilization of Midshaft Transverse or Short Oblique Radial Fractures

Stabilization of a transverse or short oblique fracture requires rotational and bending support; short oblique fractures also require axial support. This can be achieved with bone plates or an external fixator.

Useful fixation systems in toy breed dogs (in which the complication rate is high with these fractures) and in animals with a low fracture assessment score include a bone plate and screws inserted to function as a compression plate for transverse fractures, a neutralization plate for short oblique fractures, or with a type II acrylic pin external skeletal fixator. In animals with a moderate fracture assessment score, a compression plate, a minimal type II external skeletal fixator, or a type Ib external skeletal fixator may be used. Transverse or short oblique fractures in patients with a high fracture assessment score may be stabilized with a six-pin type Ia external skeletal fixator or a cast if the fracture is greenstick or nondisplaced (Fig. 33.47).

Stabilization of Midshaft Long Oblique Radial Fractures or Comminuted Fractures With a Large Butterfly Fragment

These fractures can be anatomically reduced and interfragmentary compression applied with cerclage wire or lag screws. Axial, rotational, and bending forces generated by weight bearing are neutralized with bone plates or external fixators. Fixation systems that are useful with a low fracture assessment score consist of lag screws to reconstruct the cylinder of bone and achieve interfragmentary compression, supported by neutralization plates or with a type II ESF. Fractures with a moderate fracture assessment score may be stabilized by combining lag screws or cerclage wire for interfragmentary compression with a neutralization plate or type II or Ib ESF. Fractures with a high fracture assessment score may be stabilized with cerclage wire for interfragmentary compression and a type Ia external skeletal fixator (Fig. 33.48).

Stabilization of Midshaft Comminuted Radial Fractures With Multiple Fragments

These fractures are realigned using indirect reduction techniques (see p. 990). Because no load sharing occurs between the implant

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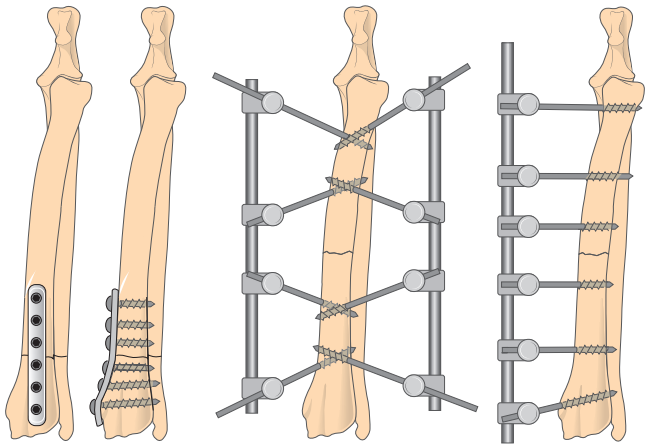


FIG. 33.47 Recommended methods of stabilizing transverse or short oblique radial fractures based on fracture assessment score. If fracture assessment score is 0 to 3, a bone plate and screws are the implants of choice, but external fixators may be used. If fracture assessment score is 4 to 7, a bone plate and screws or an external skeletal fixator may be used. With fracture assessment scores of 8 to 10, an external skeletal fixator is preferred, or, if the fracture is nondisplaced, a cast may be used.

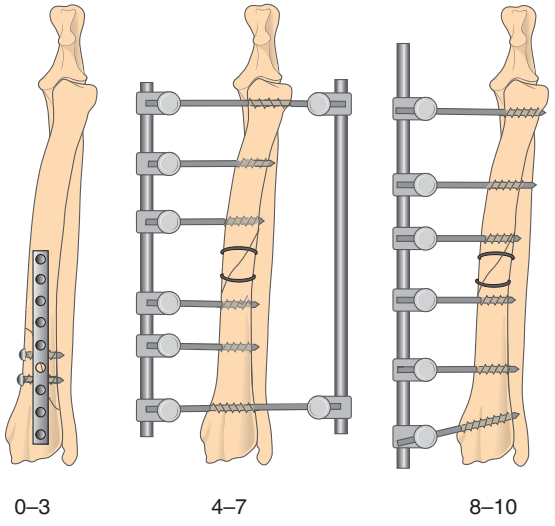


FIG. 33.48 Recommended methods for stabilizing long oblique or reducible comminuted radial fractures (single large fragment) based on fracture assessment score. If fracture assessment score is 0 to 3, a neutralization plate is preferred. If fracture assessment score is 4 to 7, a type II or type Ib external fixator and cerclage wire may be used. With fracture assessment scores of 8 to 10, a type Ia external fixator plus cerclage wire provides necessary stability.

and the bone until biological callus forms to provide support, these fractures need rigid axial, rotational, and bending support. Very high stresses will be imposed on the implant and its connection to the bone. If the biological assessment is favorable, the imposed stresses will be of short duration, reducing the likelihood of implant failure. If the biological assessment is not favorable, however, imposed stresses will act on the implant for an extended period, making implant failure more likely. Fractures in patients with low fracture assessment scores may be stabilized with a bone plate used in a bridging fashion or with a type II external skeletal fixator. Fractures in patients with a moderate fracture assessment score may be stabilized with a bone plate functioning as a bridging plate or a minimal type II or Ib external skeletal fixator. A patient with this type of fracture would have a high fracture assessment score only when the biological assessment is extremely favorable (e.g., 4- to 5-month-old animal with a low-velocity, closed, single-limb injury). Fractures in these animals may be stabilized with a type Ib external skeletal fixator (Fig. 33.49).

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Equipment necessary for pin and wire placement includes retractors, bone-holding forceps, reduction forceps, a Jacobs pin chuck, IM pins, K-wires, orthopedic wire, and wire cutters. Additional equipment needed for external fixation includes a low-RPM power drill, external fixation clamps, and bars or acrylic. Plating equipment and a high-speed drill are necessary for application of plates and screws.

POSTOPERATIVE CARE AND ASSESMENT

Postoperative radiographs should be taken to document alignment, apparatus, and apposition. Postoperative pain management may be indicated (see Boxes 32.1 and 32.2 and Table 32.4). Activity should be restricted to leash walking and physical rehabilitation until the fracture has healed. Physical rehabilitation (see Table 33.2 and Chapter 11) encourages controlled limb use and optimal limb function after fracture healing. Care must be taken to develop customized protocols for each patient depending on location of the fracture, stability and type of fracture fixation, potential for healing, abilities and attitudes of the patient, and willingness or ability of the client to participate in the animal's care.

Casts require intensive management by owners and frequent evaluations by the veterinarian. Abrasions and wounds often are caused by pressure from the cast. Management involves removal of the cast or replacement with a bivalve cast and wound treatment. Early destabilization of the fracture to treat wounds can cause delayed unions and nonunions.

After open reduction with ESF, the incision should be covered. Gauze sponges are opened and stuffed to fill the space between the fixation bar and the skin around the pins, then are secured in place with a bandage around the fixator. Care should be taken to bandage the paw as well during the immediate postoperative period to prevent swelling. Open wounds should be treated daily with wet-to-dry dressings until a granulation bed has formed. Wounds are then covered with a nonadhesive pad and the bandage changed as needed. The animal should be released to the owner with instructions to limit exercise and avoid fixator snares. If the external fixator bandage is maintained, it should be changed weekly. If a bandage is not used, daily hydrotherapy can be administered with a handheld shower massage. Checkups should be scheduled at 2 weeks for suture removal and fixator evaluation, and every 6 weeks for radiographic evaluation.

Although a rigidly stabilized fracture site is optimal for initial bone formation, subsequent bone remodeling is enhanced by increasing the load through the fractured bone. Removal of

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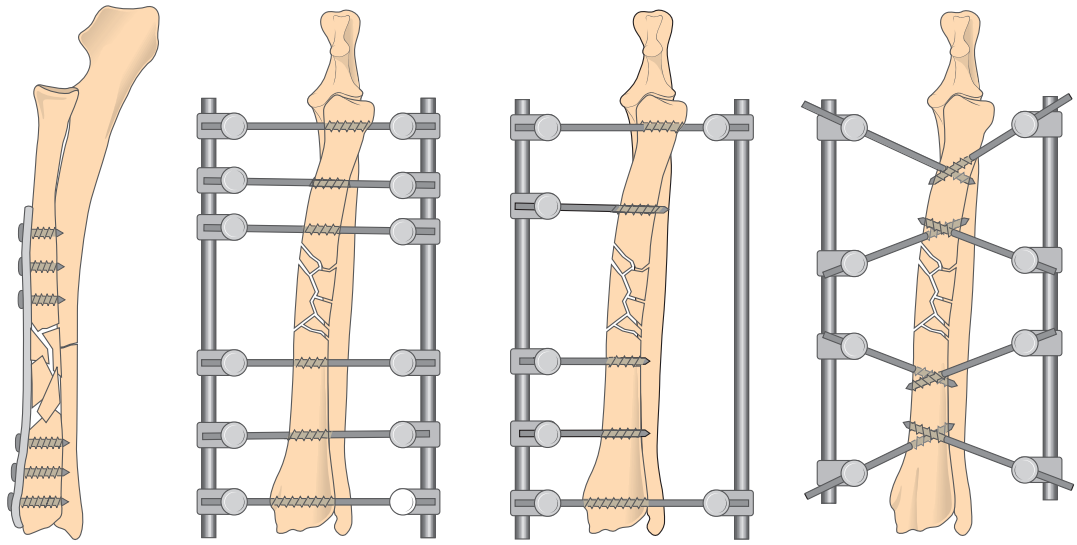


FIG. 33.49 Recommended methods for stabilizing nonreducible comminuted radial fractures (multiple fragments) based on fracture assessment score. If fracture assessment score is 0 to 3, a bridging plate or a maximal type II external fixator may be used. If fracture assessment score is 4 to 7, a bridging plate or minimal type II may be used. With fracture assessment scores of 8 to 10, a type Ib external fixator provides necessary stability. (Second and third images modified from Fossum TW. *Small Animal Surgery,* 2nd ed. St. Louis: Mosby; 2002.)

one-half of a type Ib external fixation splint (converting to a type Ia frame) or removal of selected transfixation pins from a type I or type II splint decreases the rigidity of the fixator, allowing greater load on the remodeling bone while still protecting the healing fracture. Destabilization is usually done at 6 weeks after surgery, depending on the fracture assessment. Early bone formation and bridging of the fracture site should be radiographically evident before destabilization is done. The external fixation splint can be removed completely when radiographic evidence of complete bone bridging of the fracture lines is noted. A support bandage or a splint may be used to protect the healing bone for a few weeks after fixator removal.

After placement of a plate on a diaphyseal fracture, the limb should be supported for a few days with a soft-padded bandage to reduce swelling. The animal will usually be fully weight-bearing within 2 to 3 weeks. Confinement is recommended until radiographic signs of bone union are evident. Although long-term plate application does not appear to have an effect on radial cortical bone density, plate removal may be recommended after bone union because tissue irritation and cold sensitivity have been associated with minimal soft tissue coverage over the plate.

COMPLICATIONS

Complications with radial fractures include malunion, delayed union, and nonunion in animals treated with external coaptation (Box 33.9). These problems are common in toy breed dogs sustaining fractures of the distal radial and ulnar diaphyses that have been inadequately stabilized. Complications are more prevalent in toy dogs treated with external fixators compared with large dogs treated similarly. Complications requiring revision surgery may occur in approximately 25% of cats with combined radial and ulnar fractures. Complications after open reduction include osteomyelitis, implant migration, irritation of soft tissue, malunion, delayed union, and nonunion (see p. 1031 for treatment of complications). Complications encountered with external fixation of the radius include pin loosening and pin or wire tract drainage; usually, neither problem is severe enough to warrant implant removal until the fracture has healed. Rarely, severe hemorrhage occurs from the medial exit hole of a proximal pin after the pin has eroded through an artery. Removal of the pin, with ligation of the vessel in some cases, is necessary to control hemorrhage.

BOX 33.9 Common Errors in Radial and Ulnar Diaphyseal Fracture Fixation

- IM pin placement through the carpus causes degenerative joint disease and loss of function.
- Inadequate rotational stabilization for transverse or short oblique fractures causes delayed union or nonunion, especially in toy breed dogs.
- Premature removal of fixation from slowly healing distal radial fractures contributes to the formation of nonunions.
- Valgus deformities after comminuted fracture fixation occur because of malalignment with the external fixator.

PROGNOSIS

The prognosis is generally good if proper fracture management procedures are followed.

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RADIAL AND ULNAR METAPHYSEAL AND ARTICULAR FRACTURES

DEFINITIONS

Metaphyseal fractures and **epiphyseal fractures** occur in trabecular bone. **Articular fractures** disrupt the joint surface. A **Monteggia fracture** is a fracture of the ulna coupled with a dislocation of the radial head.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Fracture of the proximal ulna may occur singularly or in combination with luxation of the radial head (Monteggia fracture). The fracture may involve the articular surface of the trochlear notch. The pull of the triceps muscle displaces the proximal segment of the ulna and must be neutralized by internal fixation to achieve bone union.

Fractures of the proximal radius are rare because the radial head is well protected by surrounding musculature. Fractures of the distal radius may be extraarticular or intraarticular. Intraarticular fractures usually disrupt the medial epicondyle and result in loss of ligamentous support for the carpus. The ligamentous attachment on the epicondyle causes displacement of the fragment that must be neutralized with internal fixation. Fractures of the styloid process of the ulna cause similar disruptions to the lateral aspect of the carpus.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, breed, or sex of dog or cat may be affected. Young animals more frequently sustain vehicular trauma.

History

Affected animals usually present with non–weight-bearing lameness after trauma. Owners may be unaware that trauma has occurred.

Physical Examination Findings

Because of the traumatic nature of this injury, the entire animal must be assessed to detect abnormalities of other body systems. Palpation of the limb reveals swelling, pain, crepitation, and apparent instability of the adjacent joint. Degloving injuries of the carpus may be associated with fractures of the distal radius. Although no major nerves are present in the immediate area, dogs often have abnormal proprioceptive responses because they are reluctant to move the limb.

Diagnostic Imaging

Craniocaudal and lateral radiographs of the affected radius and ulna (which include the proximal and distal joints) are required to assess the extent of bone and soft tissue injury. Animals that are fractious or in extreme pain may require sedation (see Tables 31.2 and 31.3) or general anesthetic for radiography after it has been determined that no contraindications (e.g., shock, hypotension, severe dyspnea) to administration of sedatives or anesthetics exist. Thoracic radiography should be performed to evaluate for thoracic trauma.

Laboratory Findings

Complete blood count and serum chemistry evaluation should be done to evaluate the advisability of anesthesia and to determine whether concurrent injury or damage to the renal or hepatobiliary system has occurred.

DIFFERENTIAL DIAGNOSIS

Animals presenting with distal radial fractures should be evaluated to determine whether fractures are caused by trauma or underlying pathology (e.g., neoplasia, metabolic disease). Joint luxations can be differentiated from fractures radiographically.

MEDICAL MANAGEMENT

Medical treatment of animals with radial and ulnar metaphyseal and epiphyseal fractures includes analgesics for posttraumatic pain (see Chapter 13) and antibiotics to treat open fractures.

SURGICAL TREATMENT

Proximal ulnar fractures require implant systems that resist the pull of the triceps muscle. Appropriate implants include tension band wire techniques and plates and screws. Monteggia fractures also require reduction and stabilization of the radial head. Articular fractures require anatomic reduction and compression with a lag screw or plate. Radial and ulnar styloid fractures require tension band wire techniques to resist the pull of the collateral ligaments.

> **NOTE** Failure to reduce a joint surface anatomically results in degenerative joint disease and loss of function.

Preoperative Management

Damage to or loss of tissue in the area of the fracture may be extensive. Open wounds should be managed initially by carefully clipping surrounding hair, cleaning the wound, and sampling deep tissue for microbial culture and susceptibility testing. The antebrachium should be temporarily stabilized with a Robert Jones bandage (see p. 981) to immobilize fragments, decrease or prevent soft tissue swelling, protect or prevent open wounds, and enhance patient comfort until surgery can be performed. Perioperative pain management should be instituted (see Chapter 13) and concurrent injuries managed before anesthetic induction for fracture fixation.

Anesthesia

Refer to Tables 32.1 and 32.2 for anesthetic management of patients with fractures.

Surgical Anatomy

Landmarks for the approach to the proximal ulna include the olecranon and the palpable caudal border of the ulna. The articular surface of the trochlear notch can be exposed surgically by muscle elevation. The ulnar nerve courses over the medial aspect of the elbow, caudal to the medial epicondyle. The antebrachial carpal joint is supported by the short radial collateral ligaments, which arise from the medial styloid process of the

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radius, the dorsal radiocarpal ligament arising from the dorsal surface of the distal radius, and the short ulnar collateral and radioulnar ligaments arising from the ulnar styloid process. The extensor tendons lie cranial to the antebrachial carpal joint and may need to be retracted to expose the joint surface.

Positioning

The limb should be prepared from shoulder to carpus. If cancellous bone graft harvest is anticipated, a donor site should also be prepared. The animal is positioned in lateral recumbency and the limb draped out for proximal radial and ulnar fractures. Placing the animal in dorsal recumbency allows greater flexibility in visualizing distal radial and ulnar fractures.

SURGICAL TECHNIQUE

Caudal Approach to the Ulna

Palpate the caudal border of the ulna directly under the skin and subcutaneous tissue on the caudal surface of the limb. Make an incision through the skin and subcutaneous tissue along the ulnar diaphysis (Fig. 33.50A). Elevate the flexor carpi ulnaris and deep digital flexor muscles medially and the ulnaris lateralis muscle laterally to expose the bone surface (Fig. 33.50B). Reflect the origin of the flexor carpi ulnaris muscle to expose the trochlear notch (Fig. 33.50C).

Approaches to the Proximal and Distal Radius and Ulna

Refer to p. 1078 under radial physeal fractures for approaches to the proximal radius, and to p. 1204 for approaches to the carpus and extend the approach proximally.

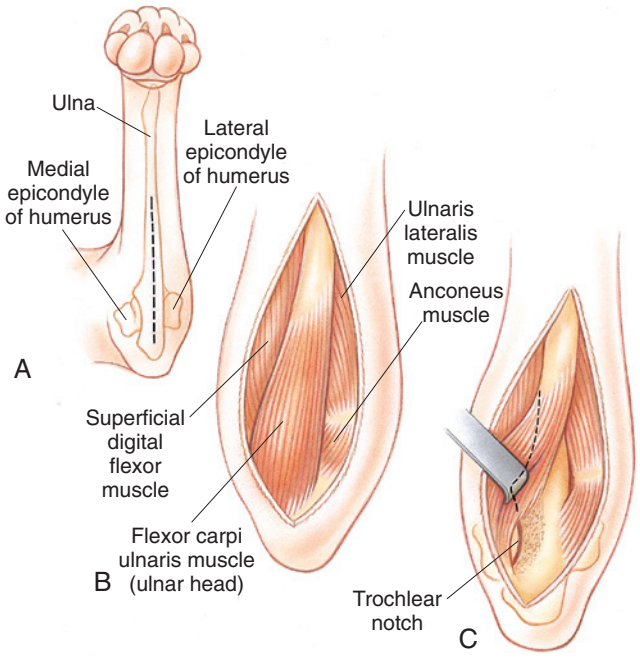


FIG. 33.50 (A) For a caudal approach to the proximal ulna, make an incision through skin and subcutaneous tissue over the caudoproximal ulna. (B) Elevate the flexor carpi ulnaris and the deep digital flexor muscles to expose the bone surface. (C) Reflect the origin of the flexor carpi ulnaris muscle to expose the trochlear notch.

Stabilization of Proximal Radial and Ulnar Fractures

If the fracture assessment score is 8 to 10, pins and wires are usually sufficient to stabilize the fracture. Extraarticular fractures of the proximal and distal radius can often be stabilized with crossed K-wires or small IM pins in animals with fracture assessment scores of 4 to 7. Severely comminuted, open fractures of the proximal ulna or fractures in dogs with anticipated prolonged healing times (fracture assessment scores of 0 to 3) should be stabilized with a plate functioning as a buttress plate. In addition to plating the ulna, Monteggia fractures are treated by reducing the radial head, reconstructing the annular ligament, and securing the radius to the ulna with bone screws or fiberwire. Articular fractures must be anatomically reduced and stabilized with tension band wires or lag screws to restore joint continuity and function and to limit the development of degenerative joint disease. *To place a tension band wire, reduce the fracture (proximal ulna, styloid process of ulna, medial epicondyle of radius) and start two K-wires in the fragment. Drive the wires across the fracture line to lodge in the major bone segment. Place a transverse drill hole in the major bone segment, pass a figure-eight wire through the hole and around the K-wires, and tighten (Fig. 33.51).* Bone plates provide an excellent method of stabilizing proximal ulnar fractures (see p. 1016). *Apply the plate to the caudal surface to function as a tension band plate, compressing the fracture (Fig. 33.52A). In some comminuted fractures, apply the plate to the caudal or lateral surface of the proximal ulna to function as a buttress plate (Fig. 33.52B).*

Stabilization of Distal Radial and Ulnar Fractures

Distal radial and ulnar fractures can be stabilized with a lag screw or tension band wire (Box 33.10 and Fig. 33.53). *To place a fully threaded cortical or cancellous lag screw, reduce the fracture and hold it in place with a K-wire. Drill a glide hole (equal to the diameter of the threads on the screw) into the epicondylar fragment (see p. 1019). Insert a drill sleeve into the gliding hole, and drill a smaller hole (equal to the core diameter of the screw) across the radius. Measure and tap the hole, then select and place in it a screw of appropriate length.* Compression of the fracture should occur. The K-wire may be left to provide rotational stability.

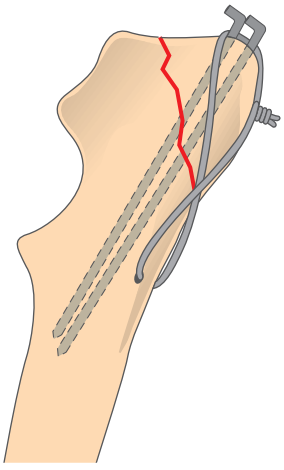


FIG. 33.51 Fractures (or osteotomies) of the olecranon can be treated with a tension band wire technique.

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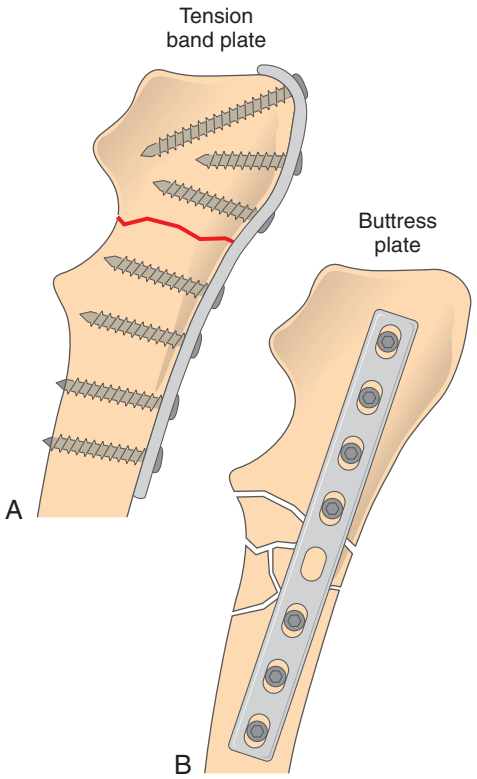


FIG. 33.52 (A) Transverse articular fractures should be anatomically reduced and stabilized with a compression plate in patients with moderate or low fracture assessment scores. (B) Severely comminuted fractures are bridged with a buttress plate.

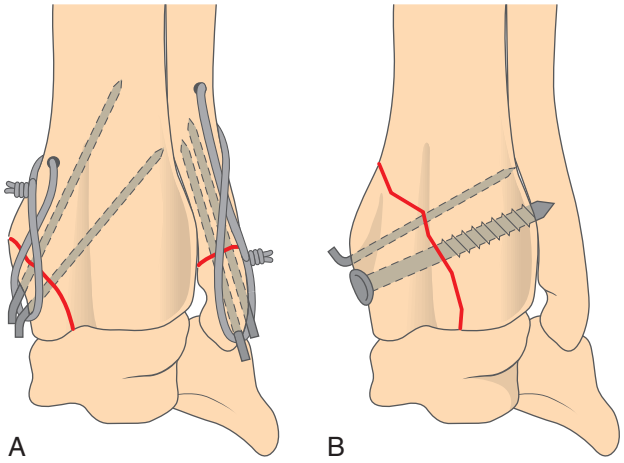


FIG. 33.53 (A) Fractures of the styloid processes can be stabilized with tension band wire in patients with high and moderate fracture assessment scores. Intraarticular fractures must be anatomically reduced and rigidly stabilized. (B) Lag screws are used in patients with moderate and low fracture assessment scores. K-wire is used as an antirotational implant.

BOX 33.10 Implant Use for Distal Radial and Ulnar Fractures According to Fracture Assessment Score (FAS)

FAS 0 to 3
Lag screw

FAS 4 to 7
Lag screw
Tension band wire

FAS 8 to 10
Tension band wire

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Plating equipment is necessary for plate and lag screws. Other equipment needed for the procedures described here includes a high-speed power drill, K-wires, IM pins, orthopedic wire, and reduction forceps.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiographs should be taken to document alignment, apparatus, and apposition. Postoperative pain management may be indicated (see Boxes 32.1 and 32.2 and Table 32.4). After internal fixation, a soft-padded bandage should be applied for a few days to control swelling and to support soft tissue. Distal radial and ulnar fracture repairs are often supported by a splint for 6 weeks. Activity should be restricted to leash walking and physical rehabilitation until the fracture has healed.

Physical rehabilitation (see Table 33.3 and Chapter 11) encourages controlled limb use and optimal carpal function after fracture healing. Care must be taken to develop customized protocols for each patient depending on location of the fracture, stability and type of fracture fixation, potential for healing, abilities and attitudes of the patient, and willingness and ability of the client to participate in the animal's care. Physical rehabilitation for patients with fractures supported by splints for 6 weeks generally begins after the splint has been removed. Open wounds are treated daily with wet-to-dry dressings until a granulation bed has formed. The wounds are then covered with a nonadhesive pad, and the bandage is changed as necessary. Daily hydrotherapy aids in cleaning open wounds and reducing postoperative swelling. Rechecks should be scheduled at 2 weeks for suture removal and every 6 weeks for radiographic evaluation. Implant removal may be necessary after bone healing if the implants interfere with or irritate soft tissue.

COMPLICATIONS

Degenerative joint disease (see p. 1143) may occur after articular fractures and may be severe if anatomic reduction and rigid fracture fixation are not achieved. Delayed union and nonunion (see p. 1031) can occur if the fracture is not adequately stabilized because of the constant tensile stress exerted on the fracture line from the pull of the triceps muscle or collateral ligaments.

PROGNOSIS

The prognosis is generally excellent if proper fracture management procedures are followed. Trabecular bone heals quickly with minimal formation of callus, although comminuted proximal ulnar fractures may require a long healing period.

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RADIAL AND ULNAR PHYSEAL FRACTURES

DEFINITIONS

Physeal fractures may occur through the cartilaginous growth plates of the proximal or distal radius or ulna in immature animals. Physeal fractures are also referred to as *epiphyseal plate fractures* or *slipped physes*.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The cartilaginous physis is weaker than surrounding bone and ligaments, making it more susceptible to injury. The weakest portion of the physis is the junction of the zone of hypertrophying cells with the zone of ossification. The zone of hypertrophying cells has a large cell-to-matrix ratio, which results in a relatively weak structure. Additionally, stress concentration is created when two areas with different mechanical properties (weak hypertrophying zone and stronger zone of ossification) are located adjacent to each other. Consequently, when a physis is fractured, separation should occur through the zone of hypertrophying cells. A fracture in this location does not affect proliferating cells and will not compromise potential growth. However, when severe trauma causes physeal fractures, the fracture line can occur anywhere within the physis and can damage growing cells. Trauma resulting in compression of the zone of proliferating cells and destruction of the chondrocytes causes premature physeal closure. This often results from trauma to the V-shaped distal ulnar physis in dogs, and trauma that would normally result in physeal separation instead compresses the zone of growing cells.

Fractures of the proximal and distal radial physis are usually radiographically classified as Salter-Harris types I and II (see p. 984). Occult type V fractures of the radial physes can occur and are diagnosed after closure of the physis has altered forelimb growth. The most common fracture of the distal ulnar physis is the Salter-Harris type V, which is traumatic and may be associated with a radial fracture. Because they are nondisplaced and are confined to the cartilage, these fractures are not visible radiographically until 2 to 3 weeks after trauma, when premature closure of the physis is observed (see p. 984).

DIAGNOSIS

Clinical Presentation

Signalment

Radial and ulnar physeal fractures occur in immature dogs and cats with open physes.

History

Affected animals usually have non–weight-bearing lameness after trauma. Owners may be unaware that trauma has occurred.

Physical Examination Findings

Because of the traumatic nature of radial or ulnar physeal fracture, the entire animal must be assessed to detect abnormalities of other body systems. Palpation of the limb reveals swelling, pain, crepitation, and apparent instability of the adjacent joint. Dogs often appear to have abnormal proprioceptive responses because they are reluctant to move the limb.

Diagnostic Imaging

Craniocaudal and lateral radiographs of the affected radius and ulna (which include the proximal and distal joints) are required to diagnose Salter I to Salter IV fractures. Animals that are fractious or in extreme pain may require sedation (see Tables 31.2 and 31.3) or general anesthesia for radiography after it has been determined that no contraindications (e.g., shock, hypotension, severe dyspnea) to administration of sedatives or anesthetics exist. Thoracic radiography should be performed to evaluate for thoracic trauma. Radiographs made at the time of injury do not give information about Salter V fractures (e.g., crushing injuries to physis) or damage to the physeal blood supply. It is therefore difficult to give an accurate prognosis for growth at the time of injury.

NOTE Compare radiographs of the contralateral bone to detect subtle changes in the physis.

Laboratory Findings

Complete blood count and serum chemistry evaluation should be done to evaluate the status of the animal for anesthesia and to determine whether damage to the renal or hepatobiliary system has occurred.

DIFFERENTIAL DIAGNOSIS

Physeal fractures can be differentiated from joint luxations and soft tissue trauma radiographically.

MEDICAL MANAGEMENT

Medical treatment of animals with radial and ulnar physeal fractures includes analgesics for posttraumatic pain (see Chapter 13) and antibiotics to treat open fractures.

SURGICAL TREATMENT

Most physeal fractures are classified as having assessment scores of 8 to 10 because affected animals are young and physeal fractures heal quickly (see p. 985). Therefore the implant system chosen need not function for a long time. Nondisplaced radial physeal fractures may be adequately stabilized with a cast. Surgical treatment of displaced physeal fractures consists of anatomic reduction and stabilization with K-wires or small pins that are smooth, so as not to interfere with residual physeal function. In animals that are close to maturity, threaded implants may be used to compress the fractured physis.

NOTE Use smooth implants when crossing the physis.

Preoperative Management

The antebrachium should be temporarily stabilized with a Robert Jones bandage (see p. 981) to immobilize the fragments, decrease or prevent soft tissue swelling, protect or prevent open wounds, and enhance patient comfort until surgery can be performed. Analgesics should be provided to posttraumatic animals (see

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Chapter 13). Concurrent injuries should be managed before anesthetic induction for fracture fixation.

Anesthesia

Refer to Tables 32.1 and 32.2 for anesthetic management of patients with fractures.

Surgical Anatomy

The lateral radial head is palpable beneath the extensor muscles of the forearm. The radial nerve lies deep to the extensor carpi radialis muscle. The craniomedial surface of the distal radius can be easily palpated to serve as a landmark for location of the incision. Extensor tendons are located cranial to and flexor tendons caudal to the distal radius. The cephalic vein crosses the medial portion of the distal radius.

Positioning

The limb should be prepared from shoulder to carpus. The animal is positioned in lateral recumbency with the limb draped out for proximal radial physeal fractures. Placing the animal in dorsal recumbency allows greater flexibility in visualizing distal radial and ulnar physeal fractures.

SURGICAL TECHNIQUE

Approach to the Proximal Radial Physis

Make a skin incision over the lateral humeral condyle, extending over the proximal third of the radius (Fig. 33.54A). Continue the incision through subcutaneous tissue and brachial and antebrachial fascia (Fig. 33.54B). Identify and separate the lateral digital extensor and ulnaris lateralis muscles to expose the proximal radius (Fig. 33.54C). Close the wound by suturing fascia, subcutaneous tissue, and skin in separate layers.

Approach to the Distal Radial Physis

Use the craniomedial approach to the radius (see p. 1069) to expose the distal radial physis.

Approach to the Distal Ulnar Physis

Use the approach for ulnar ostectomy (see p. 1075) to expose the distal ulnar physis.

Stabilization of Nondisplaced Physeal Fractures

Fractures of the radial physes and the distal ulnar physis that are nondisplaced can be treated with a cast. *Cast the limb from the toes to above the elbow with the carpus held in slight flexion and varus (inward angulation) until the cast hardens (see p. 1069).*

Stabilization of Displaced Physeal Fractures With Crossed K-wires or Steinmann Pins

Physeal fractures should be reduced carefully so as to avoid crushing or injuring the physeal cartilage. *For proximal radial physeal fractures, drive a K-wire from the lateral surface of the proximal radial epiphysis across the physis, into the radial metaphyses, and through the medial cortex. Then drive a second wire from the lateral proximal radial metaphysis across the fracture into the epiphysis (Fig. 33.55A). Take care to avoid penetrating the articular surface. For distal physeal fractures, drive a K-wire from the medial styloid process across the physis, into the radial metaphyses, and through the lateral cortex. Drive the second wire from the lateral aspect of the distal radial epiphysis, across the fracture into the metaphysis, and through the medial cortex (Fig. 33.55B), avoiding the articular surface.*

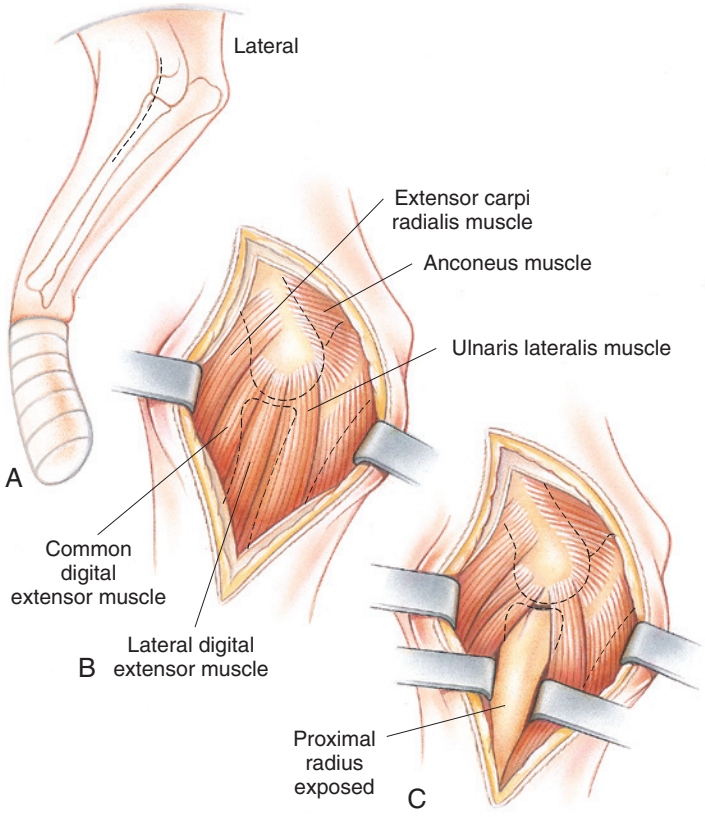


FIG. 33.54 (A) For a lateral approach to the radial head, make a skin incision over the lateral humeral condyle and extend it over the proximal third of the radius. (B–C) Identify and separate the lateral digital extensor and ulnaris lateralis muscles to expose the proximal radius.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Necessary equipment includes K-wires, small IM pins, a Jacobs pin chuck, and reduction forceps.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiographs should be taken to document alignment, apparatus, and apposition. Postoperative pain management may be indicated (see Boxes 32.1 and 32.2 and Table 32.4). After internal fixation, a soft-padded bandage should be applied for a few days to control swelling and to support soft tissue. Activity should be restricted to leash walking and physical rehabilitation until the fracture has healed. Physical rehabilitation (see Chapter 11) encourages controlled limb use and optimal joint function after fracture healing. Care must be taken to develop customized protocols for each patient depending on location of the fracture, stability and type of fracture fixation, potential for healing, abilities and attitudes of the patient, and willingness of the client. Rechecks should be scheduled at 2 weeks for suture removal and at 4 to 6 weeks for radiographic evaluation of fracture healing. Radiographs of the injured bone and the contralateral bone can be made and compared for length as early as 2 to 3 weeks after the

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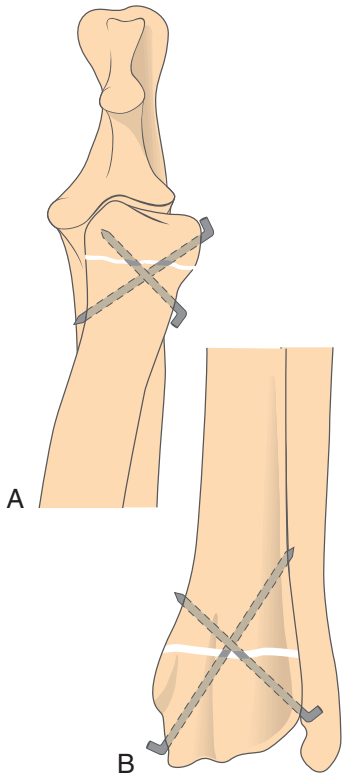


FIG. 33.55 (A) For proximal radial physeal fractures, K-wires can be driven from the lateral surface of the proximal radial epiphysis and from the lateral proximal radial metaphysis. (B) For distal physeal fractures, K-wires can be driven from the medial styloid process and from the lateral aspect of the distal radial epiphysis.

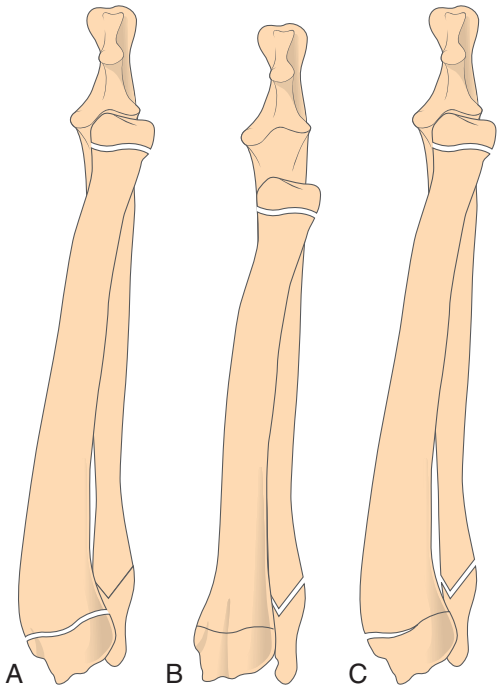


FIG. 33.56 (A) Premature closure of the distal ulnar physis causes shortening, cranial bowing, external rotation, and valgus angulation of the radius. (B) Complete premature closure of the distal (or proximal) radial physis causes shortening of the radius and subluxation of the radial humeral joint. (C) Partial distal radial physeal closure causes angular deformity of the radius.

injury to determine physeal function. Cartilage physes that heal in a manner that allows continued function appear radiographically as a lucent line. Additionally, increased bone length should be apparent. If the physeal line appears as a bone density, endochondral ossification has occurred and continued physeal function is likely. Implant removal is indicated after physeal healing (4 weeks) to allow bone growth (if the physis is functional after the trauma).

COMPLICATIONS

The most common complication associated with radial and ulnar physeal fractures is premature physeal closure with resulting growth deformities. The severity of the deformity depends on the animal's age when physeal closure occurs and the location and extent of the physeal closure. Younger animals with greater growth potential have more severe sequelae. Premature closure of the distal ulnar physis interferes with the normal development of the radius by acting as a restraint. This results not only in a shortened ulna but also in shortening, rotation, and angulation of the radius. Additionally, asynchronous growth of the paired bones results in incongruity of the elbow and carpal joints, leading to degenerative joint disease. Asymmetric or partial closure of the distal radial physis results in angular deformity of the bone and may affect the anatomy of adjacent joints, again resulting in degenerative joint disease (Fig. 33.56; see also p. 1143).

PROGNOSIS

Although the prognosis for fracture healing with physeal fractures is excellent, the prognosis for continued function or growth of the physis depends on the severity of damage sustained by the zone of proliferating cells. Most traumatically induced physeal fractures have a guarded prognosis for growth.

RADIAL AND ULNAR GROWTH DEFORMITIES

DEFINITIONS

Growth deformities involve abnormal conformation of the limb after premature closure of a physis. These deformities in the forelimb are also referred to as **angular limb deformities** or **radius curvus**. The **frontal plane** is a vertical plane that divides the bone into cranial and caudal sections. The **sagittal plane** is a vertical plane that divides the limb into lateral and medial sections. **Corrective osteotomy** is a planned osteotomy of the bone followed by restoration of normal alignment of the bone and rigid fixation. **Ostectomy** is the removal of a portion of bone. **Acute distraction** or **acute correction** is repositioning of bone segments in alignment during surgery. **Continuous distraction** or **continuous correction** is repositioning of bone segments over time using the appropriate fixator frame.

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GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Synchronous growth of the radius and ulna in the dog is essential for the development of a normal forelimb. The radius receives 40% of its length from the proximal physis and 60% from the distal physis, whereas 85% of the ulnar length comes from the distal physis, with the proximal physis contributing only 15%. In most dogs, growth accelerates rapidly during the fourth to sixth months and tapers off in the ninth or tenth month. This period varies depending on the breed of dog (smaller dogs mature faster than larger dogs). Premature closure of the physes in the radius or ulna may lead to deformity. The severity of the deformity depends on the physis affected and the growth potential of the dog.

The cone-shaped distal ulnar physis frequently sustains a Salter-Harris type V crushing injury during forelimb trauma, resulting in complete physeal closure. Sequelae include shortening of the ulna with cranial bowing, external rotation, and shortening of the radius with valgus angulation of the carpus. Varying amounts of elbow and carpal incongruity can occur (see Fig. 33.56A).

Symmetric, complete closure of the proximal or distal radial physis results in a shortened but straight radius, elbow incongruity, and a varus angulation of the carpus (see Fig. 33.56B). The limb deformity seen with asymmetric or partial physeal closure of the distal radius varies depending on the location of the closure. The most common deformity is a caudolateral closure of the physis, resulting in valgus deformity of the carpus (see Fig. 33.56C). The normal anatomy of the carpus may be disrupted.

DIAGNOSIS

Clinical Presentation

Signalment

Young dogs are affected. Growth deformities are rare in cats.

History

The animal may have a history of fracture of the radius and ulna or an obscure history of trauma.

Physical Examination Findings

Dogs with premature closure of the distal ulnar physis show varying degrees of lameness, cranial bowing and shortening of the forelimb, and valgus deviation of the carpus. Dogs with symmetric closure of the radial physes may have minimal angular deformity but may have pain on palpation of the elbow. Dogs with asymmetric closure of the distal radial physis may show an angular deformity depending on the location of the partial closure of the physis.

Diagnostic Imaging

Craniocaudal and lateral radiographs of the affected radius and ulna, which include the elbow and the carpus, are required to assess the deformity. Fractious animals may require sedation (see Tables 31.2 and 31.3). The normally functioning physis is radiolucent. A physis that has slowed or ceased functioning will still be radiolucent until endochondral ossification is complete. A closed physis is seen as bone density. Radiographs of the contralateral limb are taken to obtain a control for determining normal radial and ulnar length and normal forelimb anatomy. In early cases of premature closure of the distal ulnar physis, a discrepancy in ulnar lengths may be seen before obvious radiographic signs of physeal closure or forelimb deformity are noted. Bone length and angular limb deformity should be measured from the radiographs to establish a preoperative standard against which the results of treatment may be compared.

NOTE Always obtain radiographs of the contralateral radius and ulna for comparison. Make length measurements from lateral radiographs.

Laboratory Findings

These animals are usually young and healthy. No consistent laboratory findings are associated with growth deformities after premature physeal closure.

DIFFERENTIAL DIAGNOSIS

Growth deformities caused by premature physeal closure must be differentiated from poor conformation and laxity or contracture of supporting soft tissue structures, such as ligaments and tendons. Diagnosis of premature closure of the radial or ulnar physes is made with physical and radiographic examinations of the forelimb.

MEDICAL MANAGEMENT

No medical therapy for growth deformities is available.

SURGICAL TREATMENT

Surgical treatment of dogs with growth potential is aimed at allowing unrestricted growth of the normal physes of the radius and ulna to obtain maximal limb length and in some cases correction of the angular deformity. Immature dogs are treated with an ostectomy of the affected bone coupled with placement of a free autogenous fat graft to prevent premature union of the segment. Immature dogs with partial premature closure of the distal radial physis may be treated by resecting the bone-bridged area of the physis, followed by placement of a free autogenous fat graft in the defect to prevent reestablishment of the bone bridge. Immature dogs with complete premature closure of the distal ulnar and radial physes are treated with radial and ulnar osteotomy and continuous distraction to mimic growth (see p. 1005). This procedure is complex and requires constant postoperative monitoring by both veterinarians and clients.

Surgical treatment of mature dogs with angular limb deformities caused by premature closure of the distal ulnar physis is aimed at correcting angular and rotational deformities while preserving limb length and improving joint congruity. Dogs may be treated with an oblique radial and ulnar corrective osteotomy stabilized with a type II external fixator when limb length discrepancy is not significant and cranial caudal angulation is mild. Alternatively, hybrid fixators may be used for stabilizing the osteotomy. Bone plates may be used if there is adequate space on the distal aspect for three screws. Dome osteotomies with plate fixation have been described to treat dogs with both uniapical and biapical deformities. Continuous distraction with the circular fixator may be used to treat dogs with severe limb angulation and length discrepancy (see p. 1005).

The goal of treatment of mature dogs with premature closure of the proximal or distal radial physis is to improve limb function by reestablishing normal radial length and elbow congruency.

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One approach used to treat elbow incongruity caused by a short radius in dogs with minimal radial length discrepancies is transverse ostectomy of the proximal ulna above the interosseous ligament.

NOTE Ostectomies release restraint so that functional physes can grow normally. The animal must have growth potential for an effective ostectomy.

Preoperative Management

Preoperative management of the dog consists of obtaining a complete blood count and chemistry profile to confirm that the dog is healthy. Preoperative counseling of the owners is imperative. Owners must understand the goals and expectations of the surgeon, the limitations of the procedures, and the potential for complicated aftercare and additional procedures.

Preoperative Planning

Preoperative planning is essential for successful corrective osteotomies. Radiographs are scrutinized to plan for an oblique osteotomy of the radius and ulna stabilized with an external fixator. The degree of varus or valgus deformity is determined from cranial caudal radiographs. In the normal limb, the joint surface of the proximal radius approximately parallels the joint surface of the distal radius. To determine the angulation and the point of greatest curvature of the radius, a line is drawn along the proximal radial joint surface and a perpendicular line dropped from it down the center of the proximal radius. Another line is drawn along the distal radial joint surface and a perpendicular line extended from it up the center of the distal radius. The intersection of the perpendicular lines indicates the location of the osteotomy. Cranial caudal angulation is determined similarly from lateral radiographs. Measurements do not have to be precise because intraoperative and postoperative correction is possible with an external fixator. Rotational deformity is difficult to measure from radiographs. Instead, the rotational deformity may be estimated by flexing the carpus and the elbow of the affected limb and measuring the degree of angulation of the metacarpal bones in relation to the radius and ulna.

More precise planning using determination of the centers of rotation of angulation to prepare for dome osteotomy and plate fixation has been described. This technique is based on determining the joint orientation and the mechanical or anatomic axes. The elbow joint orientation angle in the frontal plane is determined by drawing a line that connects points located at the medial and lateral extents of the distal humeral condyle or the coronoid and lateral radial head. The carpal joint orientation angle in the frontal plane is determined by drawing a line that connects points located at the lateral distal radial articular face and the medial articular face, ignoring styloid processes or, alternatively, two points located tangentially across the proximal radial carpal bone. The normal radial anatomic axis in the frontal plane is defined as the straight mid-diaphyseal line along the length of the radius. The medial proximal radial angle is measured between the proximal anatomic axis and the elbow joint orientation angle. The lateral distal radial angle is measured between the distal anatomic axis and the carpal joint orientation angle. In deformed bone, the place where the axes intersect is the center of rotation of angulation of the deformity. If the joint segment reference axes do not have contact with one another or contact outside of the bone, an anatomic axis line is drawn in the central diaphysis, and the points where that line intersects the proximal and distal anatomic axis are the sites of a biapical deformity. This technique is repeated on lateral radiographs to identify sagittal deformities. Severe rotational deformity may cause difficulties in localizing points of greatest curvature, or centers of rotation of angulation, because simultaneous images of the entire proximal and distal portions of the radius cannot be obtained on a single film. Independent views of the proximal and distal portions of the radius may be useful. Alternatively, CT three-dimensional image reconstructions can be used to evaluate the deformity and to create three life-size stereolithographic models for use in preparing for the procedure.

Anesthesia

Refer to Tables 32.1 and 32.2 for anesthetic management of orthopedic patients.

Surgical Anatomy

Surgical anatomy of the radius is discussed on p. 1069. The distal ulna in the immature dog is large and is easily palpated on the lateral aspect of the limb. The flexor tendons of the carpus border the distal ulna. Immature dogs have a thick layer of periosteum that must be identified and resected during ostectomies. The interosseous artery lies between the radius and the ulna and is often encountered when an ostectomy is performed.

Positioning

For an ulnar or radial ostectomy, both the affected forelimb and the ipsilateral flank are prepared for aseptic surgery. The animal is positioned in lateral recumbency. For an oblique osteotomy, or a transverse lengthening osteotomy stabilized with an external fixator and possible cancellous bone graft harvest, the forelimb is prepared from the dorsal border of the scapula to below the carpus. The dog is positioned in dorsal recumbency with the affected limb suspended securely from the ceiling (see p. 990). For a circular fixator, the limb may be suspended from the ceiling, but plans must be made to release the limb and place the preconstructed circular fixator.

SURGICAL TECHNIQUE

Ulnar Ostectomy and Autogenous Fat Graft

Make a lateral skin incision extending over the middle to the distal ulna (Fig. 33.57A). Incise subcutaneous tissues, and identify and separate the lateral digital extensor muscle from the extensor carpi ulnaris muscle to expose the ulna distal metaphysis. Isolate 1 to 2 cm of the ulna metaphysis immediately proximal to the physis by elevating surrounding musculature and fascia. The ostectomy should be below the interosseous ligament to maintain elbow stability. *Ensure that all of the periosteum, with its osteogenic potential, remains with the segment of bone to be resected.*

Failure to remove all periosteum causes premature bone bridging of the ostectomy. *Resect a 1 cm to 2 cm segment of ulna with bone cutters or an oscillating bone saw cooled with a saline flush. Remove the segment of bone and associated periosteum (Fig. 33.57B). If the interosseous artery is cut, achieve hemostasis by clamping the vessel if possible, or by applying pressure for 5 minutes. To harvest the fat graft, make a 2 cm to 3 cm skin incision in the ipsilateral flank to expose the subcutaneous fat (Fig. 33.57C). Using sharp dissection, free a large single piece of fat, and place it in the ostectomy gap (Fig. 33.57D). Close the flank wound by suturing*

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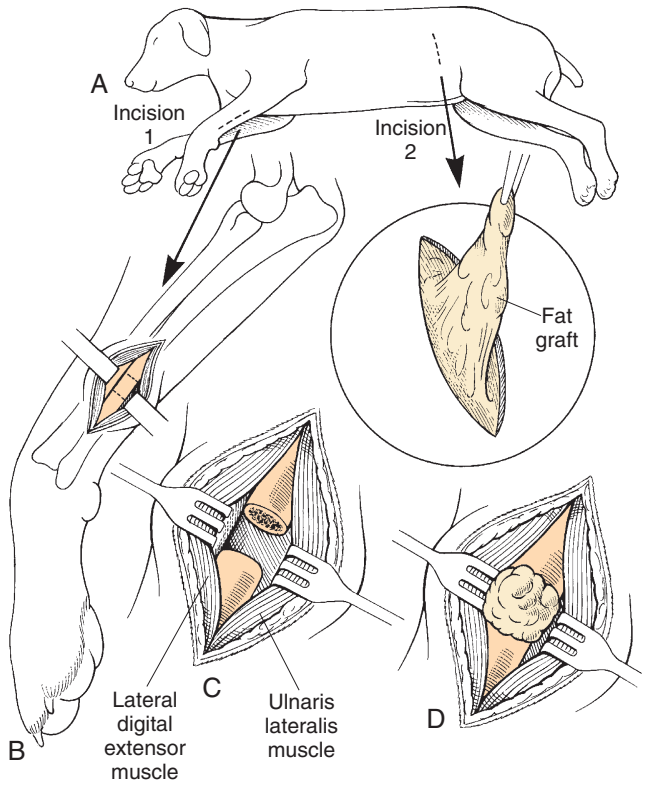


FIG. 33.57 (A) For ulnar ostectomy with a free autogenous fat graft, prepare two sites for surgery (*incision 1* and *incision 2*). Make a lateral skin incision extending over the middle to distal ulna. Separate the lateral digital extensor muscle from the ulnaris lateralis muscle to expose the ulnar distal metaphysis. (B) Resect a 1- to 2-cm segment of the ulna. (C) Make a 2- to 3-cm skin incision in the ipsilateral flank area to expose subcutaneous fat. (D) Free a large single piece of fat and place it in the ostectomy gap.

subcutaneous tissues and skin. Close the limb wound over the fat graft by suturing adjacent soft tissues. Close subcutaneous tissue and skin separately.

Partial Physeal Resection

Surgically expose the closed and bridged portion of the distal radial physis. Determine the limitations of the bony bridge by exploring the area with a hypodermic needle (Fig. 33.58A). The cartilage of the normal physis is easily penetrated by a needle, unlike the resistance felt when the bone bridge or adjacent metaphyseal or epiphyseal bone is probed. *Remove the bony bridge with a curette or a high-speed burr (Fig. 33.58B).* Curettage is complete when normal physeal cartilage is observed or probed with the needle. *Harvest free autogenous fat from the flank, as described previously, and place it within the physeal defect (Fig. 33.58C). Close soft tissue and skin over the transplanted fat.*

Radial Ostectomy and Free Autogenous Fat Graft

Expose the mid-diaphysis of the radius using a craniomedial approach (see p. 1069). Isolate 1 to 2 cm of the radial diaphysis by elevating surrounding musculature and fascia. Ensure that all periosteum, with its osteogenic potential, remains with the segment of bone to be resected. Resect a 1 cm to 2 cm segment of the radius with bone cutters or an oscillating bone saw cooled with a saline

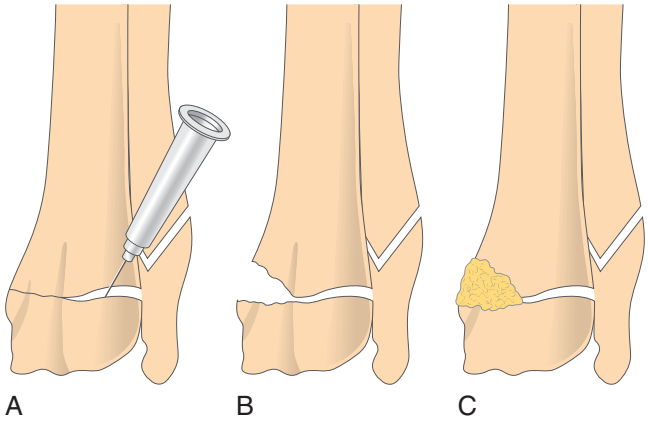


FIG. 33.58 (A) During resection of a partial physeal closure/fat graft, determine the limitations of the bone bridge by exploring the area with a hypodermic needle. (B) Remove the bone bridge with a curette or a high-speed burr. (C) Harvest a free autogenous graft from the flank and place it in the physeal defect.

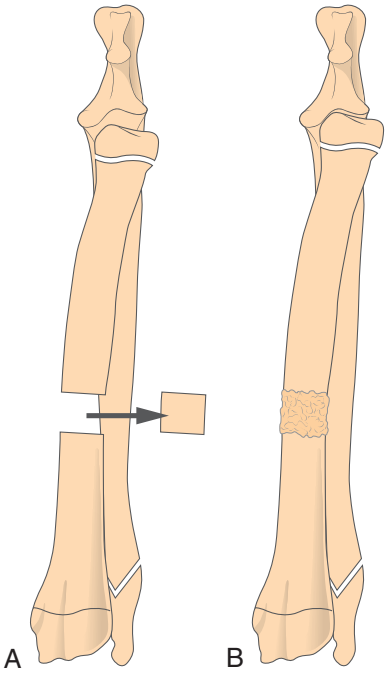


FIG. 33.59 (A) To perform a radial ostectomy and fat graft, resect a 1- to 2-cm segment of the radius. (B) Harvest a free autogenous graft from the flank and place it in the physeal defect.

flush. Remove the segment of bone and associated periosteum (Fig. 33.59A). If the interosseous artery is cut, achieve hemostasis by clamping the vessel if possible, or by applying pressure for 5 minutes. Harvest a free autogenous fat graft from the flank and place it in the defect to prevent bone union (Fig. 33.59B). Close the wound over the fat graft.

Oblique Radial and Ulnar Osteotomy

Place a centrally threaded, positive-profile fixation pin through the proximal radius from the lateral aspect (Fig. 33.60A). The pin should be parallel to the proximal radial articular surface and should be within the frontal plane of the proximal radius. *Place*

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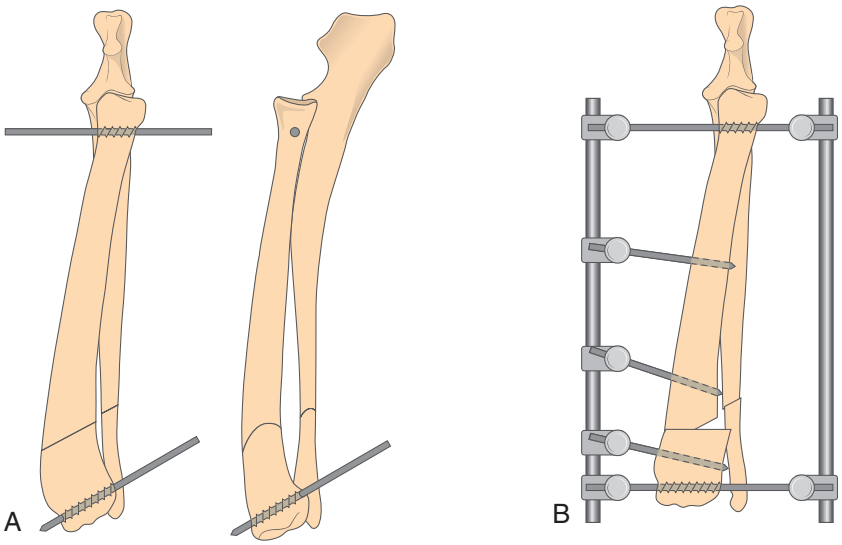


FIG. 33.60 (A) For an oblique osteotomy, place transfixation pins through the frontal planes of the proximal and distal radial metaphyses parallel to the respective joint surfaces. (B) Perform an osteotomy at the point of greatest curvature of the radius, which is parallel to the distal articular surface in both frontal and sagittal planes. Perform an ulnar osteotomy. Realign the radius using the fixation pins as guides to eliminate angular and rotational deformities, and stabilize it with the external fixator.

another pin through the distal radius from the lateral aspect. The pin should be parallel to the distal radial articular surface and should be within the frontal plane of the distal radius. The transfixation pins serve as landmarks for reestablishing limb alignment. *Make a lateral approach to the distal ulna, and cut the bone with an oscillating saw.* It may be necessary to remove a 1- to 2-cm segment of ulna in order to accurately realign the radius. *Make a craniomedial approach to the distal radius at its point of greatest curvature. Perform an oblique osteotomy of the radius with an oscillating bone saw, directing the osteotomy line parallel to the distal radial articular surface in both frontal and sagittal planes. Lower the operating table so that the weight of the animal distracts the distal radius and helps to align the joint surfaces. Realign the radius and ulna using proximal and distal transfixation pins to eliminate any angular or rotational deformity. Place a connecting bar with single fixation clamps on the medial aspect of the limb, and a connecting bar without clamps on the lateral aspect of the limb (Fig. 33.60B). Drive additional fixation pins through single clamps placed on the medial bar. Place at least one additional pin in each radial segment. Close the wounds by suturing the subcutaneous tissue and skin separately. Tighten the clamps, and cut the fixation pins to the desired length.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Instruments needed include an oscillating saw, a low-RPM power drill, fixation pins, external fixation clamps, and connecting bars or acrylic.

POSTOPERATIVE CARE AND ASSESSMENT

After an ostectomy or a partial physeal resection, postoperative radiographs are taken to document the location and length of the gap. Postoperative pain management may be indicated (see Boxes 32.1 and 32.2 and Table 32.4). A soft-padded bandage or splint may be used to protect the limb for 2 weeks if a radial ostectomy or bilateral ulnar ostectomies are done. Owners should be instructed to limit activity and to return for monthly reexaminations. Radiographs should be taken monthly and compared with immediate postoperative radiographs for radial (or ulnar) growth, correction of angular deformity, and ostectomy gap patency. Restoration of normal elbow configuration due to release of the appropriate bone may be noted. Reevaluations can be discontinued when the animal is skeletally mature.

NOTE If ostectomies prematurely bridge with bone, a second ostectomy may be needed.

After corrective osteotomy, postoperative radiographs should be taken to document the correction obtained and the position of the implants. The radial joint surfaces should be parallel and the cranial surfaces of the proximal and distal radial segments located in the frontal plane. If they are not, some correction may be obtained by readjusting an external fixator (see p. 998). Postoperative pain management may be indicated (see Boxes 32.1 and 32.2 and Table 32.4). Activity should be restricted to leash walking and physical rehabilitation until the fracture has healed. If the external fixator bandage is maintained, it should be changed weekly. If a bandage is not used, daily hydrotherapy can be administered with a handheld shower massage. Checkups should be scheduled at 2 weeks for suture removal and fixator evaluation and every 6 weeks for radiographic evaluation. Physical rehabilitation (see Table 33.2 and Chapter 11) encourages controlled limb use and optimal limb function after fracture healing.

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COMPLICATIONS

Complications include delayed union, nonunion, osteomyelitis, pin tract infection, and fixation failure (see p. 1031 for treatment of complications).

PROGNOSIS

Prognosis for normal appearance and function is guarded in immature animals after ostectomies. Much of the outcome depends on the growth potential of the remaining open physes. With favorable conditions, a dog treated with an ulnar ostectomy may achieve normal limb length and some correction of valgus angulation; however, any rotation deformity will persist. The prognosis is guarded after partial physeal resection until radiographic evidence of physeal function is obtained. Premature bone healing may be an indication for reoperation. A corrective radial and ulnar osteotomy may be indicated if the angular deformity has not corrected when the dog has reached maturity.

The prognosis is good for bone union at the osteotomy site. Function of the limb depends on the amount of correction achieved and the presence of degenerative joint disease.

CARPAL AND TARSAL FRACTURES

DEFINITIONS

Carpal fractures and **tarsal fractures** may cause loss of weight-bearing support if the integrity of these bones is disrupted. A **plantigrade stance** occurs when the foot is positioned such that the plantar surface of the calcaneus contacts the ground. A **valgus position** of the foot is an outward deviation; a **varus position** is an inward deviation.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Although carpal or tarsal fractures are rare in companion animals, such fractures are often disabling because the carpal and tarsal joints serve a major weight-bearing function. If injuries involving these joints are not treated, joint incongruity and subsequent development of osteoarthritis often lead to severe lameness. Fractures of the distal row of carpal bones may occur as compression fractures with carpal hyperextension injuries. Carpal fractures in racing dogs may occur as avulsion of bone fragments by ligamentous attachments or as compression, which results in slab or stress fractures. Carpal fractures in other breeds may have a different mechanism. Radial carpal bone fractures are the most frequently diagnosed injury involving fracture of the carpal bones in companion animals. Radial carpal bone fracture in these animals often involves chronic lameness without history of trauma and may occur bilaterally. Idiopathic radial carpal bone fractures are identified most commonly in boxers. Three common fracture patterns occur, and it is speculated that these fractures are the result of incomplete ossification of the radial carpal bone. Accessory carpal bone fractures occur in racing greyhounds and sled dogs but are rare in other companion animals. Most accessory carpal bone fractures occur in the right leg because of the counterclockwise direction of racing. Accessory carpal bone fractures are considered avulsion injuries because they occur at tendinous or ligamentous insertions and are classified according to the site of injury on the accessory carpal bone.

Fractures of the tarsus are seen regularly in working breeds of dogs but are rare in companion animals, with the exception of a fractured calcaneus. Because calcaneal fractures are distracted by the pull of the gastrocnemius muscle, preventing bone contact between fragments and interfering with healing, treatment methods must resist tensile forces. Talar neck fractures occur in cats and occasionally in dogs, and condylar fractures also occur. As with all articular fractures, anatomic reduction and rigid fixation are needed for optimal outcome. Reconstruction may be difficult owing to the small size of the trochlea and the degree of comminution. If reconstruction is not feasible, primary arthrodesis may be performed (see p. 1204).

Racing greyhounds often incur fractures of the central tarsal bone; however, this injury is rarely seen in companion animals. Central tarsal fractures are graded according to fracture type and degree of fragment displacement. Once the buttress effect of the central tarsal bone is lost, fractures of the fourth tarsal bone and calcaneus may occur. Repair requires accurate placement of one or more small lag screws.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, breed, or sex of dog or cat may be affected. Racing greyhounds most often develop fractures of the central tarsal bone and accessory carpal bones. Boxers and sporting breeds most often develop chronic radial carpal bone fractures.

History

Affected animals usually have acute onset of non–weight-bearing lameness after injury. Dogs with radial carpal bone fractures may have chronic forelimb lameness.

Physical Examination Findings

Patients with acute fractures of the carpus or tarsus usually have non–weight-bearing lameness; attempts to place weight on the limb cause the carpus and/or the tarsus to collapse in a plantigrade stance. Patients with chronic radial carpal bone fracture have weight-bearing lameness (which may be intermittent), reduced carpal range of motion, and soft tissue swelling. If the calcaneus is fractured, the animal may walk plantigrade on the limb or may be non–weight bearing. Pain, swelling, and crepitus are present in the affected limb. Varus or valgus deviation of the foot is usually present.

Diagnostic Imaging

Most of these animals are in pain and require sedation or general anesthesia for proper positioning to obtain quality radiographs (see Tables 31.2 and 31.3). High-detail radiographs using dorsopalmar (dorsoplantar), medial lateral, and oblique projections are usually sufficient to make the diagnosis. Bilateral carpal radiographs and/or CT may be helpful for diagnosing occult radial carpal bone fractures.

Laboratory Findings

Consistent laboratory abnormalities are not present. Traumatized animals undergoing surgery should have sufficient bloodwork done to assess the risk of anesthesia and surgery.

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DIFFERENTIAL DIAGNOSIS

Fractures of the carpus and/or tarsus must be differentiated from ligamentous injury, which may occur concurrently with fractures. Calcaneal fractures must be differentiated from lacerations or rupture of the Achilles tendon. Acute lacerations have an open wound, and soft tissue swelling is limited to an area proximal to the calcaneal tuberosity. Fractures of the calcaneus exhibit swelling caudal to the tarsus, and crepitation may be elicited on palpation.

MEDICAL MANAGEMENT

Medical or conservative management is not indicated. Anatomic reduction and rigid fixation are necessary for optimal outcome in animals with intraarticular fractures of the carpus or tarsus; conservative treatment with casts or splints is not effective. External coaptation is also not appropriate for calcaneal fractures because bandaging or splint application is ineffective in countering tensile forces produced by the Achilles muscle-tendon unit.

SURGICAL TREATMENT

The radial carpal bone is a major weight-bearing structure. Congruity of the articular surface between it and the distal radius is necessary for optimal long-term function. Small chip fragments that cannot be stabilized should be removed; large fragments, however, should be anatomically reduced and rigidly stabilized with lag screws or a combination of lag screws and K-wires (Fig. 33.61). Dogs with chronic radial carpal bone fractures, severely comminuted fractures, osteoarthritis, luxation, bone loss, or infection are best treated with carpal arthrodesis. With calcaneal fractures, the pull of the gastrocnemius muscle must be resisted with a tension band wire (see p. 1015), lag screws, or a plate (Fig. 33.62). Articular fractures of the talus must be anatomically reduced and rigidly stabilized for optimal outcome (Fig. 33.63). If preoperative assessment indicates that fracture repair is not feasible, arthrodesis of the tarsocrural joint should be considered.

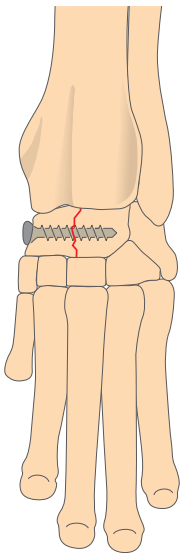


FIG. 33.61 Reduction of a radial carpal bone fracture with a lag screw.

Preoperative Management

Because these fractures result from trauma, all affected animals should be examined for concurrent injury and stabilized if necessary before surgery. Patients with comminuted fractures of the carpus and/or tarsus may be brought in with open wounds. These wounds should be cleaned and protected from further damage and contamination. A Mason metasplint (see p. 982) or a bivalve cast (see p. 995) can be applied to enhance patient comfort and protect soft tissue from further contamination or injury induced by bone fragments. Analgesics should be provided to posttraumatic animals (see Chapter 13).

Anesthesia

Refer to Tables 32.1 and 32.2 for anesthetic management of patients with fractures. Racing greyhounds should be anesthetized with special care.

Surgical Anatomy

The carpus is composed of the proximal and distal row of carpal bones. The radial carpal bone articulates primarily with the radius and serves as the major weight-bearing area in the joint. The calcaneus is the largest of the tarsal bones. The distal half of the bone has two facets and two processes that articulate with the talus to form a stable joint. Proximally, the tuber calcaneus forms a sturdy prominence to accommodate insertion of the Achilles tendon. The talus is the second largest of the tarsal bones. It articulates proximally with the tibia and the fibula and distally with the central tarsal bone. The body of the talus is divided into medial and lateral trochleae that articulate with the tibia and the fibula proximally and a base that articulates with

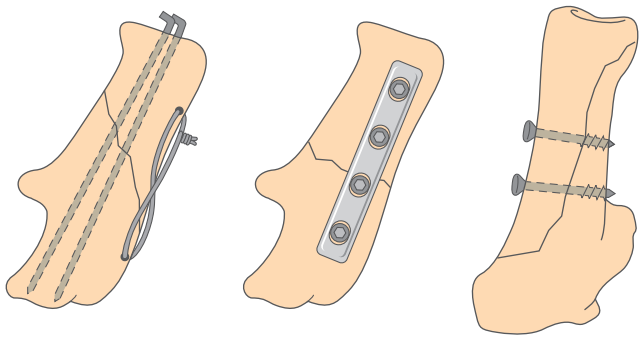


FIG. 33.62 Transverse calcaneal fractures can be stabilized with a tension band wire or plate. Oblique or slab fractures can be stabilized with lag screws.

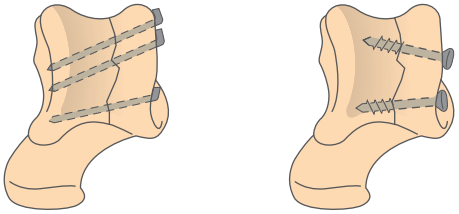


FIG. 33.63 Fractures of the articular surface of the talus may be stabilized with diverging K-wires or lag screws.

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the central tarsal bone distally. The sides of the trochleae articulate with the medial and lateral malleoli.

Positioning

For carpal fractures, the animal is placed in dorsal recumbency. The limb should be clipped and prepared for aseptic surgery from the elbow to the digits. A hanging-limb preparation facilitates limb manipulation during surgery. Animals are placed in lateral recumbency with the affected limb uppermost for calcaneal fractures and in dorsal recumbency for fractures of the talus. With tarsal injuries, the limb should be clipped and surgically prepared from the stifle joint to the digits.

SURGICAL TECHNIQUE

Stabilization of Radial Carpal Bone Fractures

Make a craniomedial incision beginning 3 to 4 cm proximal to the radiocarpal joint (see Chapter 34). Extend the incision distally to the mid-metacarpus, and incise subcutaneous tissue along the same line. Continue deep dissection medial to the extensor carpi radialis tendon to expose the joint capsule. Incise the capsule and identify the fracture plane through the radial carpal bone. Reduce the fracture and stabilize fragments with one or more lag screws (see Fig. 33.61) Close the joint capsule and subcutaneous tissue with absorbable suture, and close the skin with nonabsorbable suture.

Stabilization of Transverse Calcaneal Fractures

Make an incision along the lateral surface of the calcaneus. Begin the incision along the common calcaneal tendon, just proximal to the tuber calcanei. Continue the incision distally to the level of the tarsometatarsal joint. Incise superficial and deep fascia overlying the caudal border of the calcaneus. Identify the lateral aspect of the superficial digital flexor tendon, and make an incision parallel to this border. Retract the tendon medially to expose the caudal surface of the calcaneus. Reduce the fracture fragments, and place two small pins or K-wires to maintain reduction. Drill a hole from lateral to medial in the distal fragment to accept orthopedic wire. Proximally, pass the wire around the ends of the pins or through a second predrilled hole positioned through the body of the calcaneus proximal to the fracture. Tighten the wire to complete the tension band procedure. Alternatively, use a bone plate (see Fig. 33.62). Replace the superficial digital flexor tendon, and suture surrounding deep fascia with absorbable material to maintain the position of the tendon. Then suture superficial fascia and skin using standard techniques. Calcaneal fracture repairs may have a high rate of complications. Fractures repaired with bone plates and screws have a lower risk of complication than fractures repaired with pin and tension band.[7]

Stabilization of Talar Trochlear Fractures

Exposure of the trochlea and visualization of the fracture are best accomplished with an osteotomy of the medial malleolus. To expose the lateral condyle of the talus, a distal fibula transverse osteotomy is performed. *Center the skin incision over the medial malleolus. Begin 5 cm proximal to the malleolus, and carry the incision distally to the tarsometatarsal joint. Incise through superficial and deep fascia to identify the long component of the medial collateral ligament. Make a transverse caudal-to-cranial incision through the joint capsule overlying the distal tibia to allow visualization of landmarks for completion of an osteotomy of the medial malleolus. Make the osteotomy deep enough to include most of the origin of the collateral ligament, but do not interfere with the weight-bearing articular surface. Retract the malleolus and attached ligaments to expose the talus. Reduce fracture fragments and stabilize each with a lag screw or K-wire (see Fig. 33.63). Once fracture reduction and stabilization are satisfactory, reduce and stabilize the medial malleolus with a tension band (see p. 1015). Suture superficial and deep fascia with absorbable suture, and close the skin with nonabsorbable sutures.*

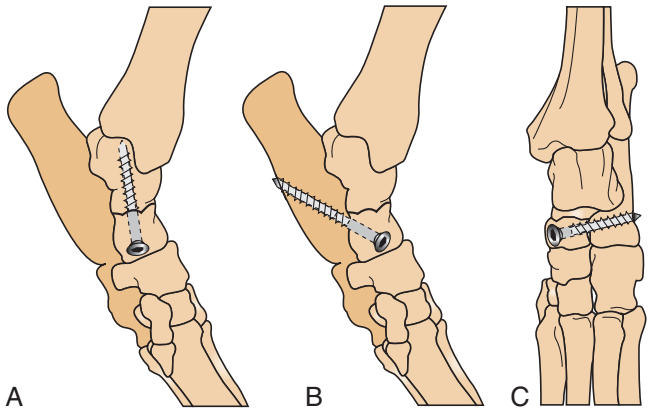


FIG. 33.64 Fractures of the neck of the talus may be stabilized with (A) a lag screw angled from the caudal medial surface of the head of the talus into the trochlea of the talus, or (B–C) the screw may be placed from the caudal medial base of the talus into the calcaneus. (Modified from Johnson AL, Dunning D. *Atlas of orthopedic surgical procedures of the dog and cat.* 1st ed. St. Louis: Saunders; 2005.)

Stabilization of Talar Neck Fractures

A talar neck fracture may be stabilized with a lag screw angled from the caudal medial surface of the head of the talus into the trochlea of the talus, or, alternatively, the screw may be placed from the caudal medial base of the talus into the calcaneus. If reduction can be maintained with forceps, the screw may be placed as a position screw; if not, it should be placed as a lag screw (Fig. 33.64). *Incise skin, subcutaneous tissue, and deep fascia along the medial malleolus to the tarsometatarsal joint. Elevate the fascia to expose the talus. Reduce the distal fragment of the talus by manipulating it with pointed reduction forceps, and stabilize the fracture. Suture superficial and deep fascia with absorbable suture, and close the skin with nonabsorbable sutures.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Necessary equipment includes pointed reduction forceps, osteotome and mallet or bone saw, K-wire, small IM Steinmann pins, orthopedic wire, pin and wire cutters, bone screws, and instrumentation for insertion of lag screws. An air-driven or battery-operated drill is helpful.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiographs are taken to evaluate alignment, apparatus, and apposition. Postoperative pain management may be indicated (see Boxes 32.1 and 32.2 and Table 32.4). Coaptation postoperatively with a soft-padded bandage controls bleeding and swelling. Coaptation with a splint for up to 6 weeks is

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indicated for most dogs. Activity should be restricted to leash walking and physical rehabilitation until the fracture has healed. Short leash walks should be used initially to help maintain strength and joint mobility. The distance walked can be gradually increased. Passive flexion and extension of the carpus and/or tarsus can be performed to maintain joint motion, enhance patient comfort, and improve synovial nutrient presentation to the articular cartilage (see Table 33.3). Pins used in application of the tension band for stabilization of calcaneal fractures may irritate soft tissue and should be removed after healing. Screws used for reconstruction of the radial carpal bone and the talus are not removed unless they cause a problem.

COMPLICATIONS

Degenerative joint disease (see p. 1143) may occur after articular fractures and may be severe if anatomic reduction and rigid fracture fixation are not achieved. Delayed union and nonunion (see p. 1031) can occur if the calcaneal fracture is not adequately stabilized because of constant tensile stress exerted on the fracture line from the pull of the gastrocnemius muscle.

PROGNOSIS

Prognosis with calcaneal fractures is excellent for return to normal activity. Return to function with carpus and/or tarsus fractures is fair to good, depending on the degree of articular cartilage damage and whether the articular surface can be reconstructed.

METACARPAL, METATARSAL, PHALANGEAL, AND SESAMOLD FRACTURES AND LUXATIONS

DEFINITION

Sesamoid bones are small, round or oblong bones found adjacent to joints.

GENERAL CONSIDERATIONS AND CLINCIALLY RELEVANT PATHOPHYSIOLOGY

Metacarpal and metatarsal bone fractures are common in dogs and cats. They may result from a direct blow or force to the paw or from hyperextension injuries. Complete metacarpal and metatarsal fractures in greyhounds occur as a result of fatigue or from the relatively normal bone being loaded beyond its yield strain. Metacarpal and metatarsal fractures are classified according to location (e.g., base or proximal end of the bone, shaft, or diaphysis; head or distal end of the bone). Avulsion fractures of the base occur most often on the second and fifth bones because of their ligamentous insertions. Phalangeal fractures occur similarly in dogs and cats; however, fragments are often smaller and more difficult to secure. Two palmar or plantar sesamoid bones are present per metacarpophalangeal or metatarsophalangeal joint, numbered one through eight beginning on the medial side. Sesamoid fractures occur after excessive tension on the digital flexor tendons; sesamoid bones two and seven of the forelimb are most often affected. Luxations of the metacarpophalangeal joints or interphalangeal joints typically occur in working dogs or racing greyhounds. Early surgical repair yields better results than closed reduction and splintage because chronic instability leads to degenerative joint disease and less than optimal function.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, breed, or sex of dog or cat may be affected. Racing greyhounds develop stress fractures of the second metacarpal and third metatarsal bones of the right foot and luxations of the distal interphalangeal joints. Sesamoid fractures are most common in large breed dogs.

History

A history of trauma is generally reported. Dogs with sesamoid fractures may have a history of acute lameness that subsided but recurred with exercise.

Physical Examination Findings

Animals with metacarpal, metatarsal, or phalangeal fractures have non–weight-bearing lameness of the affected limb. Soft tissue surrounding the fracture is swollen, crepitation can be palpated, and deformity of the paw may be noted. The animal will be in pain when the area is palpated. Cats presenting with metacarpal or metatarsal fractures from trauma often have concomitant injuries to the head and/or thorax and additional fractures of the limbs, pelvis, or spine. Dogs with sesamoid fractures, especially chronic fractures, usually have weight-bearing lameness. Mild swelling may be evident, and affected animals have pain on deep palpation over the bone. Dogs with joint luxations have lameness, swelling over the affected joint, medial or lateral deviation of the digit, joint instability, and pain on palpation.

NOTE Most fractures in the paw cause non–weight-bearing lameness, but sesamoid fractures cause less obvious lameness.

Diagnostic Imaging

Fractious or extremely painful animals may require sedation (see Tables 31.2 and 31.3) or general anesthesia for radiography after it has been determined that no contraindications (e.g., shock, hypotension, or severe dyspnea) to administration of sedatives or anesthetics exist. Detailed radiographs should include dorsopalmar/plantar and mediolateral views extending from the carpus or the tarsus to the ends of the digits. Oblique views, with the digits spread, or a lateral view with the affected digit pulled cranially with tape or gauze, may be necessary to isolate individual bones. Stress radiographs taken while the distal digit is displaced may be necessary to show joint instability.

Laboratory Findings

Specific laboratory abnormalities are not present with these fractures or luxations. Traumatized animals should have sufficient bloodwork done to assess the risk of anesthesia and surgery.

DIFFERENTIAL DIAGNOSIS

Animals presenting with fractures of the metacarpal, metatarsal, and phalangeal bones should be carefully evaluated for concurrent ligamentous injury in the carpus, tarsus, and distal joints of the paw. Radiographs help to differentiate between fractures and luxations caused by ligamentous injury.

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MEDICAL MANAGEMENT

Medical treatment of animals with metacarpal and metatarsal fractures includes analgesics for posttraumatic pain (see Chapter 13). Conservative treatment with a fiberglass bivalve cast or a metasplint (see p. 1143) is appropriate for treating closed, nondisplaced metacarpal and metatarsal diaphyseal fractures affecting one or two bones, especially the second and fifth bones. Coaptation is useful for cats with comminuted nonreducible fractures. This treatment is also appropriate for most phalangeal fractures and acute sesamoid bone fractures. The cast or splint should not be removed until radiographic evidence indicates that the fracture has bridged with bone (usually 4–8 weeks).

Chronic sesamoid fractures causing lameness may not respond to conservative therapy. Acute luxations can be treated conservatively with a cast or splint, but are best treated surgically in working or racing dogs. Chronic luxations causing lameness do not respond to conservative care and are usually treated with arthrodesis or amputation.

SURGICAL TREATMENT

Metacarpal or metatarsal fractures occurring in athletic or racing dogs usually require anatomic reduction and rigid stabilization (plates and screws) for optimal return to racing form (Fig. 33.65). Large avulsed fragments from the base of the second and fifth metacarpals and metatarsals generally require open reduction and internal fixation because their ligamentous insertions cause fragment distraction (Fig. 33.66). Fractures in cats may be treated with dowel pinning. Fracture of the shaft of one or two metacarpal or metatarsal bones can be treated with external coaptation because the unaffected bones form an internal splint to prevent deformity (Box 33.11). Fractures affecting three or four metacarpal or metatarsal bones may require internal fixation for optimal alignment and outcome (Fig. 33.67).

Fractures of the phalanges occur less frequently but are handled similarly to metacarpal and metatarsal bone fractures. Fractures of the proximal sesamoid bones of the metacarpophalangeal joints and metatarsophalangeal joints causing chronic lameness are generally treated by removing the fragments.

Acute luxations are treated surgically with open reduction and suturing of the joint capsule and collateral ligaments. Failure of the initial surgical stabilization and chronic joint luxations are best treated with amputation (second or fifth toe) or arthrodesis (middle weight-bearing third and fourth digits) (Box 33.12).

Fixation systems applicable for metacarpal, metatarsal, and phalangeal fractures include orthopedic wire, IM pins, external fixation, and plates and screws. The most appropriate fixation method should be determined on the basis of fracture assessment score (see p. 985) and fracture location (Box 33.13). If the fracture assessment indicates rapid healing (score of 8 to 10), conservative therapy is indicated; however, if three or four bones are affected, resulting in gross displacement or folding of the paw, open reduction and stabilization with IM pins should be considered. If athletic function is desired, accurate reduction and plate and screw fixation should be considered. Nondisplaced avulsion

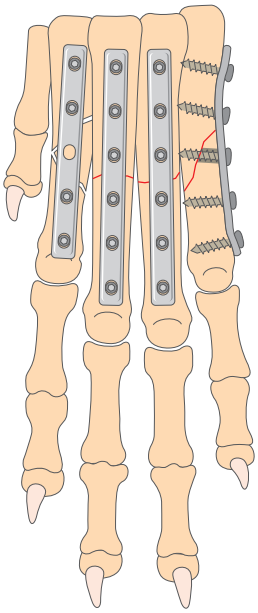


FIG. 33.65 Plate fixation of metacarpal/metatarsal fractures. Plate fixation is used when the fracture assessment score is low or when athletic function is desired. A bridging plate has been used to span and support a comminuted fracture (digit 2); compression plates have been applied to transverse fractures (digits 3 and 4); and lag screw compression of an oblique fracture line was protected by a neutralization plate (digit 5).

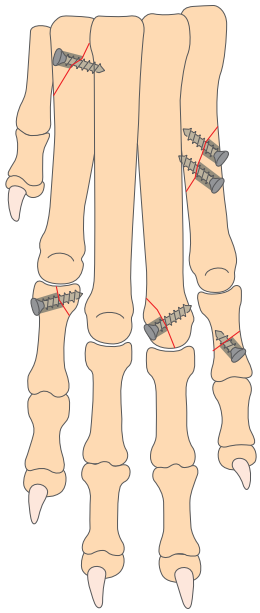


FIG. 33.66 Lag screw fixation for avulsion fractures. Lag screws are used to counteract the pull of adjacent ligaments or to compress oblique fractures.

BOX 33.11 Treatment Considerations for Metacarpal/Metatarsal Fractures

- Fractures of one or two metacarpal or metatarsal bones can be treated with a splint or cast.
- Fractures of three or four metacarpal or metatarsal bones should be treated with internal fixation.
- Large displaced avulsion fractures should be treated with a lag screw.
- A splint or bivalve cast should be applied after internal fixation until radiographic evidence of bone healing is noted.

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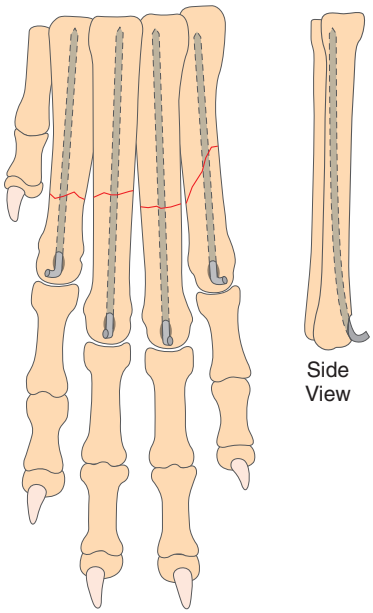


FIG. 33.67 Intramedullary pinning technique used to treat multiple transverse or short oblique fractures in patients with high fracture assessment scores. A slot is developed in the distal metaphysis, and the pin is directed through the slot (proximally across the fracture line) and is seated in the proximal metaphysis of the metacarpal or metatarsal bone.

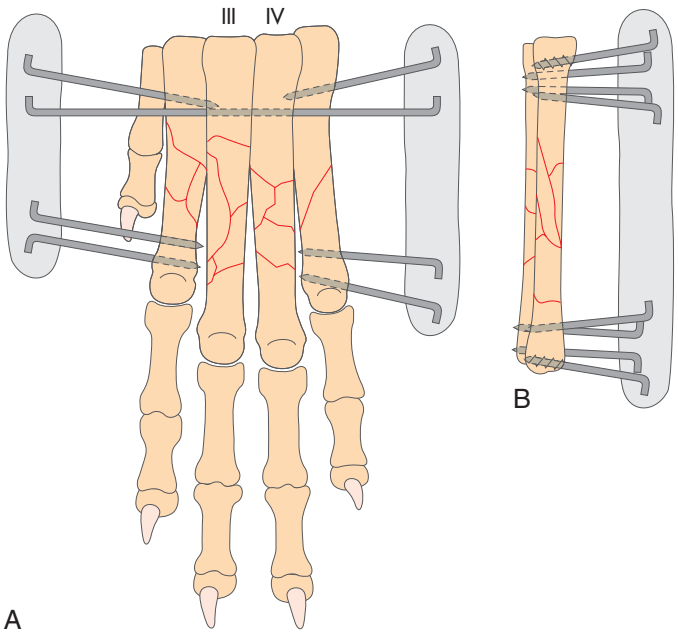


FIG. 33.68 (A) Connect fixation pins with acrylic bars for rigid stabilization of comminuted fractures. This fixation does not interfere with open wound treatment. (B) Alternatively, place pins in a type Ib configuration and connect with acrylic.

BOX 33.12 Treatment Considerations for Luxations

- Acute luxations in working or racing dogs are best treated with open reduction and suturing of the joint capsule and collateral ligaments.
- Chronic luxations of the second or fifth toe can be treated with amputation.
- Arthrodesis of metacarpophalangeal and interphalangeal joints can result in good function and pain relief.

BOX 33.13 Implant Use for Metacarpal/Metatarsal/Phalangeal Fractures According to Fracture Assessment Score (FAS)

FAS 0 to 3

Bridging plates[a]
External fixators[a]
Lag screws for avulsion fractures

FAS 4 to 7

Bone plates and screws[a]
Intramedullary (IM) pins[a]
Lag screws for avulsion fractures

FAS 8 to 10

Splint or cast[a]
IM pins[a]
Tension band wire for avulsion fractures

[a]Choice depends on number of bones fractured, comminution, displacement, number of limbs injured, and desired function of animal (see p. 985).

fractures of the base or head of these bones can be treated with a splint or a cast, but some displacement of the fracture usually occurs during the healing process. Open reduction and placement of a lag screw or tension band wire offer the best chance for return to normal function. If the fracture assessment score is 4 to 7 (e.g., older or larger dogs, in which healing may be delayed; displaced, comminuted diaphyseal fractures of three or four metacarpal or metatarsal bones; multiple limb fractures), treatment with open reduction and plate and screw fixation should be considered. Fracture assessment scores less than 4 indicate prolonged healing. Severely comminuted fractures or open fractures with degloving injuries can be treated with bridging plates or external fixation with small pins and acrylic (Fig. 33.68). Cancellous bone autografts are indicated in conjunction with open reduction to promote healing in these patients.

Preoperative Management

The paw should be temporarily stabilized with a soft-padded bandage and a metasplint (see p. 982) to immobilize fragments, decrease or prevent soft tissue swelling, protect or prevent open wounds, and enhance patient comfort until surgery can be performed. Open wounds should be initially managed by carefully clipping surrounding hair, cleaning the wound, obtaining a sample for microbial culture and sensitivity testing, and administering broad-spectrum bactericidal antibiotics. Concurrent injuries should be managed before anesthetic induction for fracture fixation. Perioperative pain management should also be instituted (see Chapter 13).

Anesthesia

Refer to Tables 32.1 and 32.2 for anesthetic management of patients with fractures.

Greyhounds must be anesthetized with special care.

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Surgical Anatomy

The primary weight-bearing bones are the third and fourth digits. The superficial dorsal metacarpal or metatarsal artery courses over the dorsal aspect of the paw. The extensor tendons course down the dorsal aspect of each digit. The flexor tendons and the superficial and deep metacarpal or metatarsal artery and vein lie on the palmar or plantar aspect of the digits. Each joint has a medial and a lateral collateral ligament. The paired proximal sesamoid bones are located caudal to the metacarpophalangeal or metatarsophalangeal joints and have firm ligamentous attachments. With minimal soft tissue coverage, the bones and the joints can be easily palpated. Skin incisions are generally made on the dorsal surface of the paw, directly over the fracture or luxation. The extensor tendons and ligaments of the dorsal surface of the paw need to be retracted to expose the bones or joints. Ventral approaches to the digits are made only to expose the proximal sesamoid bones.

NOTE A tourniquet is useful to control hemorrhage from the paw.

Positioning

The entire distal limb should be prepared for aseptic surgery. The animal is placed in dorsal recumbency with the affected paw isolated for draping. The most accessible site for harvesting a cancellous bone graft for the forelimb is the proximal humerus; for the hindlimb, the site is the proximal tibia or distal femur (see p. 991). These areas should be prepared and draped if a bone graft is needed (e.g., comminuted fractures with fracture assessment score <4).

SURGICAL TECHNIQUE

Stabilization of Transverse Metacarpal or Metatarsal Fractures

Multiple, simple transverse (or very short oblique) metacarpal bone fractures in animals with fracture assessment scores of 8 to 10 and 4 to 7 may be repaired with IM pins. The medullary diameters of the second and fifth metacarpal and metatarsal bones are smaller than the third and fourth, affecting pin diameter selection. *Incise skin over the dorsal surface of the third and fourth bones. Incise subcutaneous tissue, and elevate and retract extensor tendons to expose the fractures. Introduce the pin into the distal, dorsal surface of the bone to avoid the joint (use a high-speed burr to develop a slot in the bone). Blunt the tip of the pin to prevent it from penetrating the intact opposite cortex. Drive the pin through the slot and proximally across the fracture line, and seat it in the proximal bone segment. Bend the distal end of the pin to prevent migration and simplify removal. Repeat the procedure for at least the third and fourth metacarpal or metatarsal bones (and in some cases all four bones; see Fig. 33.67). Protect the fixation with a splint or a cast for 4 to 6 weeks.* Cat metacarpals and metatarsals are small in diameter and may fracture when pins are inserted through the distal cortex. Dowel pinning is an alternative treatment. *Grasp the larger bone segment with mosquito forceps and drive a K-wire that almost or completely fills the medullary canal as far as possible into the medullary canal without penetrating the bone end. Cut the wire so that approximately 8 to 14 mm protrudes from the fracture. Ream the medullary canal of the opposing bone segment by advancing and retracting a similar-diameter K-wire. Grasp both segments as far away from the fracture site as possible, and distract them until the shorter fragment can be placed on the free end of the K-wire. Reduce the fracture by sliding the bone segments together.* If more rigid fixation is required in animals with low fracture assessment scores (e.g., large dogs with multiple-limb injury), veterinary cuttable plates or small dynamic compression plates are useful in stabilizing the fracture (see Fig. 33.65).

Stabilization of Avulsion Fractures and Oblique Diaphyseal Fractures

The size, location, and degree of displacement of an avulsion fracture will dictate whether repair or removal is optimal. Small, avulsed, minimally displaced fragments associated with other fractures may not require stabilization. Basilar fractures with significant displacement require primary repair. *Repair avulsion fractures of the base or head and intraarticular fractures of the metacarpal, metatarsal, or phalangeal bones using lag screws (see p. 1019). Place lag screws after anatomic reconstruction of these fractures to counteract the pull of attached ligaments (see Fig. 33.66). Use orthopedic wire with K-wires as tension band wire (see p. 1015) in avulsion fractures in animals with fracture assessment scores of 8 to 10. Reconstruct simple oblique fractures of the diaphyses with lag screws, and support the repair with a splint or a bivalve cast; the screws only minimally interfere with function. If diaphyseal fractures require additional support, consider applying veterinary cuttable plates as neutralization plates.*

NOTE Use anatomic reconstruction with lag screws for best results in athletic dogs.

Stabilization of Comminuted Diaphyseal Fractures

Repair severely comminuted fractures of the diaphyses of the metacarpal and metatarsal bones with small, dynamic compression plates (2.7 or 2.0) or veterinary cuttable plates (see p. 1016), which bridge the comminuted portions of the fracture (see Fig. 33.65). Leave fragments undisturbed, and attach the plate to the proximal and distal segments. Apply plates to two to four bones, depending on fracture configuration and the number of bones fractured. Use a splint or a cast to support the fixation after surgery. Alternatively, insert multiple K-wires or IM pins through the proximal and distal segments of the bones, and connect with acrylic to form an external fixator (see Fig. 33.68).

NOTE Acrylic fixator bars are versatile; they can accommodate various sizes of pins placed in multiple locations.

Sesamoid Bone Excision

Incise the skin adjacent to the large central pad directly over the ventral aspect of the affected joint. Continue the incision through subcutaneous tissue, and identify the fractured sesamoid. Sharply dissect the sesamoid fragments from their ligamentous attachments. If the fragment involves less than one-third of the sesamoid bone, remove only the fragment. If the fragment is larger, remove the entire sesamoid bone. Suture subcutaneous tissue and skin separately.

Suture Repair of Luxations

Incise skin and subcutaneous tissue dorsally over the affected joint to expose the torn joint capsule and collateral ligaments.

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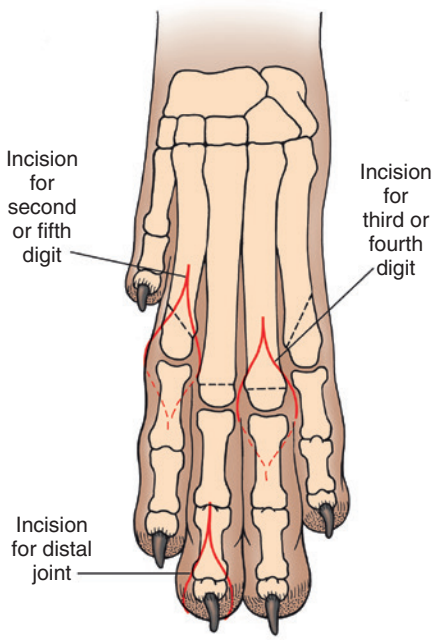


FIG. 33.69 Suggested skin incision lines and osteotomy lines for proximal and distal digit amputation.

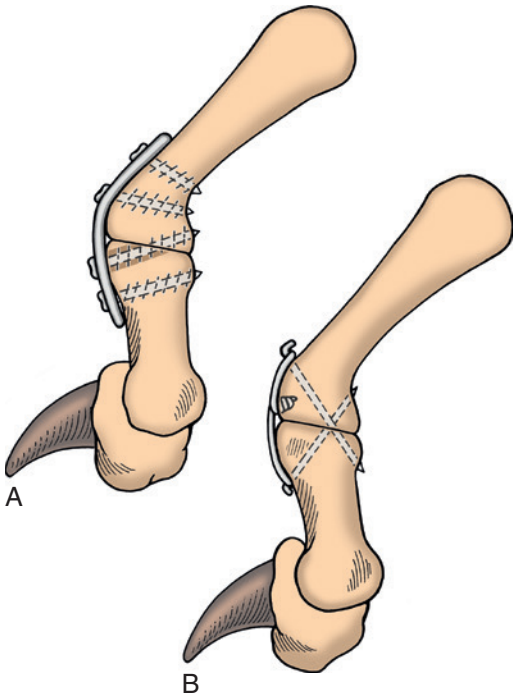


FIG. 33.70 Arthrodesis of proximal interphalangeal joint with (A) plate and lag screws or (B) K-wires and tension band wire.

Use multiple absorbable horizontal mattress sutures to repair the joint capsule and ligament. Continue to imbricate (tighten the joint capsule with mattress sutures) the capsular tissue until the joint is stable. Repair the capsule bilaterally if necessary. In addition, inspect the extensor tendon retinaculum for tears, and stabilize it with interrupted sutures. Suture subcutaneous tissue and skin.

Digit Amputation

Make an elliptical skin incision paralleling the long axis of and around the digit, starting proximodorsally and ending distally on the palmar or plantar surface (Fig. 33.69). Preserve the pad if amputating at the interphalangeal joints. Disarticulate the joint with sharp dissection. Remove the sesamoid bones (metacarpophalangeal or metatarsophalangeal joint amputation). Resect the distal end of the remaining proximal bone. Make a transverse osteotomy line for the interphalangeal joints and the third and fourth metacarpal or metatarsal bones, but bevel the osteotomy distally toward the midline for the second and fifth metacarpal or metatarsal bones. Suture soft tissue and skin, taking care to obtain a cosmetic closure.

Arthrodesis

Expose the joint using the approach described for suturing joint luxations. Open the joint capsule, and remove the articular cartilage with rongeurs. Contour the surfaces to obtain good contact at a functional angle (determined by observing adjacent toes). Temporarily hold the bones in position by driving small K-wires from the dorsal surface of each bone across the joint surface. Contour a small plate (2.0) to the dorsal surface of the bones. Attach the plate using at least one lag screw across the joint surface (Fig. 33.70A). Alternatively, stabilize the arthrodesis with the small K-wires and a tension band wire (see Fig. 33.70B) in small dogs or cats when rapid healing is anticipated.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Equipment that should be available for management of fractures or luxations of the distal extremity includes IM pins, K-wires, orthopedic wire, a Jacobs pin chuck, a wire tightener, plating equipment, acrylic, and rongeurs, bone cutters, or an osteotome. A tourniquet applied to the distal limb is useful for controlling hemorrhage during surgery but should be maintained for less than 1 hour.

POSTOPERATIVE CARE ASSESSMENT

Postoperative radiographs should be taken to evaluate alignment, apparatus and apposition, reduction of luxations, and alignment of arthrodeses. Postoperative pain management may be indicated (see Boxes 32.1 and 32.2 and Table 32.4). Limited activity is advised until radiographic evidence of fracture bridging with bone is obtained. Radiographs should be repeated every 6 weeks until the fracture(s) or the arthrodesis is healed. The foot should be supported in a splint or a bivalve cast (even after internal fixation) until radiographic evidence indicates bone healing. After digit amputation, the paw should be supported in a soft bandage for 1 to 2 weeks. After fracture healing, IM pins, plates, and external fixators should be removed, but dowel pins, orthopedic wire, and screws may be left in place as long as no implant-associated complications arise.

Physical rehabilitation (see Chapter 11) may be indicated to ensure optimal limb function after bone healing. Care must be taken to develop customized protocols for each patient depending on location of the fracture, stability and type of fracture fixation, potential for healing, abilities and attitudes of the patient, and willingness or ability of the client to provide for the animal's care. Physical rehabilitation for patients with fractures supported by splints for 6 weeks generally begins after the splint has been

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removed (see Table 33.2). Owners should be instructed to confine animals to a small area (preferably a cage). The cast or the splint should be kept clean and dry, and the owner should observe it daily for evidence of slippage or irritation.

COMPLICATIONS

Most metacarpal and metatarsal fractures progress to healing. Fractures of the diaphyses of three or four metacarpal or metatarsal bones treated with a metasplint may develop delayed union or malunion and may become nonunions (see p. 1031 for treatment of complications). Other potential complications include iatrogenic infection (with open reduction) and secondary degenerative joint disease after articular fracture.

PROGNOSIS

Metacarpal and metatarsal fractures have good to excellent prognosis with good long-term results.[8] More severe cases require surgery and often develop synostoses, whereas in most cases there is no difference in outcome between surgical and medical management. Chronic lameness associated with sesamoid fragmentation may resolve or improve with conservative therapy. The prognosis after sesamoid removal is fair for early resolution of the lameness, but degenerative joint disease progresses and lameness may recur with heavy use.

The prognosis for function after surgical repair of joint luxations depends on the stability of the repair. Unstable joints will develop progressive degenerative joint disease, causing lameness. Function after amputation of the second and fifth digits is usually good. Function after an arthrodesis procedure is generally good.

PELVIC FRACTURES

SACROILIAC LUXATIONS AND FRACTURES

DEFINITIONS

Sacroiliac luxation results from disruption of the articulation between the wing of the sacrum and the iliac wing. **Sacral fracture** may accompany sacroiliac luxation.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHSYIOLOGY

The sacroiliac joint is often injured when the pelvis is fractured. Displacement of the pelvic girdle after a fracture requires bilateral separation of the sacroiliac joints, fracture of the pelvic bones at three sites, or a combination of these injuries. One of these points of fracture may be a separation of the sacroiliac articulation. Displacement of the wing of the ilium is generally cranial and dorsal, with medial movement of the ilium to compromise the pelvic canal. The femoral and sciatic nerves are close to the sacroiliac joint, and concurrent nerve damage may occur. Preoperative attention to neurologic status is necessary before treatment. Neurologic deficits are common when sacral fractures traverse the spinal canal or the sacral foramina.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, breed, or sex of dog or cat may be affected.

History

Sacroiliac luxations and fractures are most often caused by motor vehicle accidents.

Physical Examination Findings

Patients are usually non–weight-bearing or minimally weight-bearing on the affected limb. If contralateral long bone or pelvic injury is present, however, the animal may be required to bear weight on the limb with sacroiliac luxation. Instability may be difficult to palpate. Animals with severe displacement of the ilium may have severe pain when movement occurs. When the patient is sedated, dorsoventral movement of the ilium may be detected.

Diagnostic Imaging

Most of these animals are in pain and require sedation or general anesthesia for proper positioning to obtain quality radiographs (see Tables 31.2 and 31.3). Ventrodorsal and lateral radiographs must be taken to assess the degree of injury to the hemipelvis and to delineate fracture planes. Sacroiliac luxation is diagnosed when there is a visible step at the sacroiliac joint and is usually identified on the ventrodorsal view. Care should be taken when positioning the animal to obtain as much symmetry as possible. Special attention should be paid to identifying sacral fractures because they are often missed. CT may be helpful in identifying sacral fractures. The width of the pelvic canal should be ascertained.

Laboratory Findings

Consistent laboratory abnormalities are not present. Traumatized animals undergoing surgery should have sufficient bloodwork done to assess the risks of anesthesia and surgery.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include fractures of the hemipelvis (i.e., ilium and acetabulum) and sacral fractures. These fractures can usually be differentiated with appropriate radiographs or on CT evaluation.

MEDICAL MANAGEMENT

Medical treatment of animals with sacroiliac luxation includes analgesics for posttraumatic pain (see Chapter 13). Most patients function normally with asymmetric craniocaudal positioning of the hip joints. Conservative treatment is indicated for minimal patient discomfort and minimal displacement of the hemipelvis, for very small patients or subacute luxations, or when financial limitations preclude surgery. In very small patients, minimal loads enable rapid healing, whereas surgical stabilization has an increased risk of implant malposition within the sacral canal and possible neurologic injury. Subacute (more than 1 week) luxations are difficult to reduce without significant dissection of scar tissue that may increase pain and discomfort and risk neurologic injury. Most patients treated conservatively for a sacroiliac luxation regain normal function. However, lameness may persist for up to 12 weeks, and malunion with pelvic narrowing can occur. Conservative therapy should not be elected if there is significant pelvic canal narrowing that would be corrected by surgical reduction and stabilization.

Conservative therapy should include enforced rest for the first 3 weeks after injury. After 3 weeks, supervised activity on a

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leash should be encouraged for an additional 3 weeks, with the amount of exercise gradually increased. Nonsteroidal antiinflammatory drugs can be used to control inflammation and pain (see p. 981), but the animal must be observed for adverse side effects (e.g., vomiting, melena, renal insufficiency). The animal should be maintained on well-padded bedding that is changed frequently to prevent decubital ulcers and urine scalding. Bowel movements should be monitored and stool softeners given if necessary. Warm water sponge baths may be used to decrease odor and improve patient comfort. Physical rehabilitation should be performed to prevent joint and muscle contracture (see Chapter 11).

SURGICAL TREATMENT

Surgical stabilization of a sacroiliac fracture-separation is useful for restoring the weight-bearing arch and encouraging early return to function. Surgery is indicated with significant narrowing of the pelvic outlet, which could lead to constipation or obstipation. This may be done with a standard open approach and bone screws or with fluoroscopically assisted reduction and implant placement. A transiliac bolt may be helpful in preventing undue forces on the bone screws and medial displacement of the ilium.

Preoperative Management

Patients with sacroiliac injuries should be adequately stabilized before fracture treatment. The animal's neurologic status must be determined preoperatively. Because urinary trauma may be associated with pelvic fractures, the integrity and function of the lower urinary tract should be determined before surgical repair of the sacroiliac fracture. Analgesics should be provided to posttraumatic animals (see Chapter 13).

Anesthesia

Refer to Tables 32.1 and 32.2 for anesthetic management of patients with fractures.

Surgical Anatomy

The sacroiliac joint has two distinct components: a semilunar, crescent-shaped synovial joint and a fibrocartilaginous synchondrosis. The joint's strength is derived from dorsal and ventral ligaments and from the fibrocartilaginous synchondrosis (Fig. 33.71).

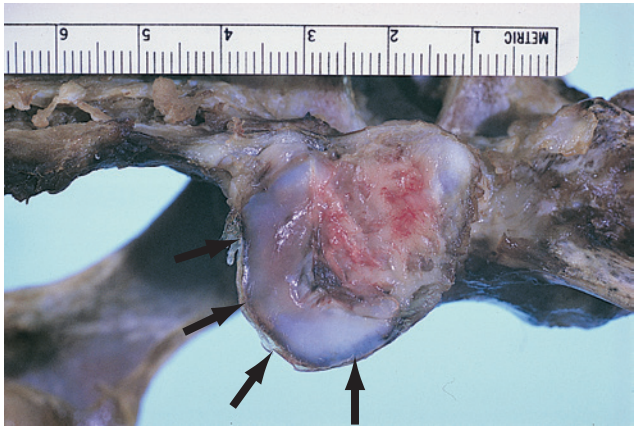


FIG. 33.71 Lateral surface of sacroiliac joint. Note C-shaped zone of articular cartilage *(arrows)*.

Positioning

The animal should be positioned in lateral recumbency with the dorsal midline raised 45 degrees from the table. The prepared area should extend from the dorsal midline to the stifle joint and from a point 10 cm cranial to the iliac crest to the tail head caudally.

SURGICAL TECHNIQUE

Surgical Approach

Make a skin incision beginning over the dorsal iliac crest and continuing caudally, parallel to the spine, to a point even with the hip joint (Fig. 33.72). Incise subcutaneous tissue and pelvic fat along the same line to expose the iliac crest. Incise through the periosteal origin of the middle gluteal muscle on the lateral ridge of the iliac crest. Make a second incision through the periosteal origin of the sacrospinalis muscle on the medial ridge of the iliac crest. The incisions merge caudally, where it may be necessary to incise through fibers of the superficial gluteal muscle. Supporting ligamentous tissues between the sacrum and the ilium are usually separated with the impact of the original trauma. Therefore incision of the lumbar fascia allows lateral reflection of the ilium and exposure of the sacroiliac joint. *Ventrally displace*

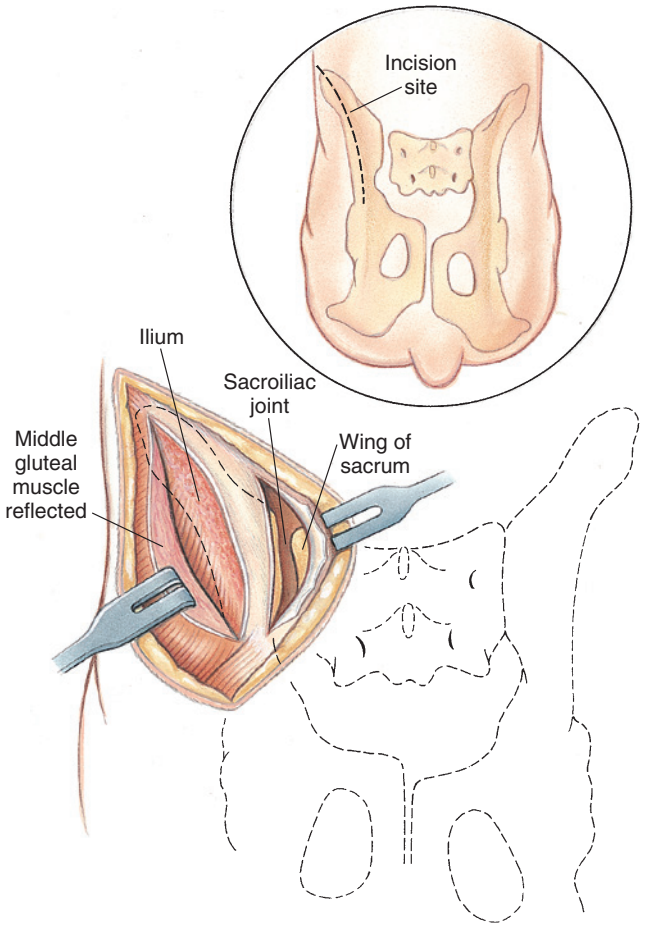


FIG. 33.72 To expose the sacroiliac joint through a dorsal exposure, make an incision over the iliac crest. Reflect the gluteal muscles laterally and the sacrococcygeus dorsalis lateralis muscle medially.

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the ilium with bone-holding forceps, and remove the fibrous debris to obtain a clear view of the surface of the sacral wing. Elevate the middle gluteal muscle from the lateral surface of the ilium to expose the ilium further for placement of implants. To close the incision, suture fasciae of the middle gluteal and sacrospinalis muscles with an absorbable suture in an interrupted pattern. Then suture subcutaneous tissue and skin using standard methods.

Stabilization Using a Screw

Once the joint is exposed, position a blunt Hohmann retractor between the ilium and the ventral bony shelf of the sacrum. Reflect the ilium ventrally with the retractor. This maneuver exposes the crescent-shaped articular cartilage and the fibrocartilaginous joint surfaces of the sacrum. To position a screw properly in the sacrum, direct visualization of the lateral surface of the sacral wing is preferred. *Use an appropriately sized drill bit to drill a thread hole 2 mm cranial and 2 mm proximal to the center of the crescent-shaped articular cartilage (Fig. 33.73A).* The depth of the thread hole in the sacral body should be such that the screw tip will extend a minimum of 60% of the distance across the sacral body. *Determine the proper location of the glide hole in the ilium by palpating the articular prominence on the medial surface of the iliac wing. Drill the glide hole at the predetermined position with the appropriately sized drill bit. A glide hole is not needed if a partially threaded cancellous screw is used. Advance the proper-length screw through the glide hole until the tip appears on the medial surface of the ilium. Bring the ilium caudally in alignment with the articular surface of the sacroiliac joint. Visually guide the screw tip into the prepared thread hole in the sacrum, and tighten the screw (Fig. 33.73B). Add a second screw placed immediately dorsal and cranial to the initial screw if space permits.* This screw must be shorter to avoid the spinal canal. Large or overweight dogs may benefit from the placement of a transiliac bolt. A small Steinmann pin may be placed through the iliac wings and over the dorsal surface of L7. *Bend the smooth end of the pin, and place a nut on the threaded end to prevent migration (Fig. 33.73C).*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Instruments required for placement of a lag screw are needed. A Hohmann retractor and a drill sleeve facilitate implant insertion.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiographs are taken to evaluate sacroiliac reduction and implant location. Postoperative pain management may be indicated (see Boxes 32.1 and 32.2 and Table 32.4). Activity

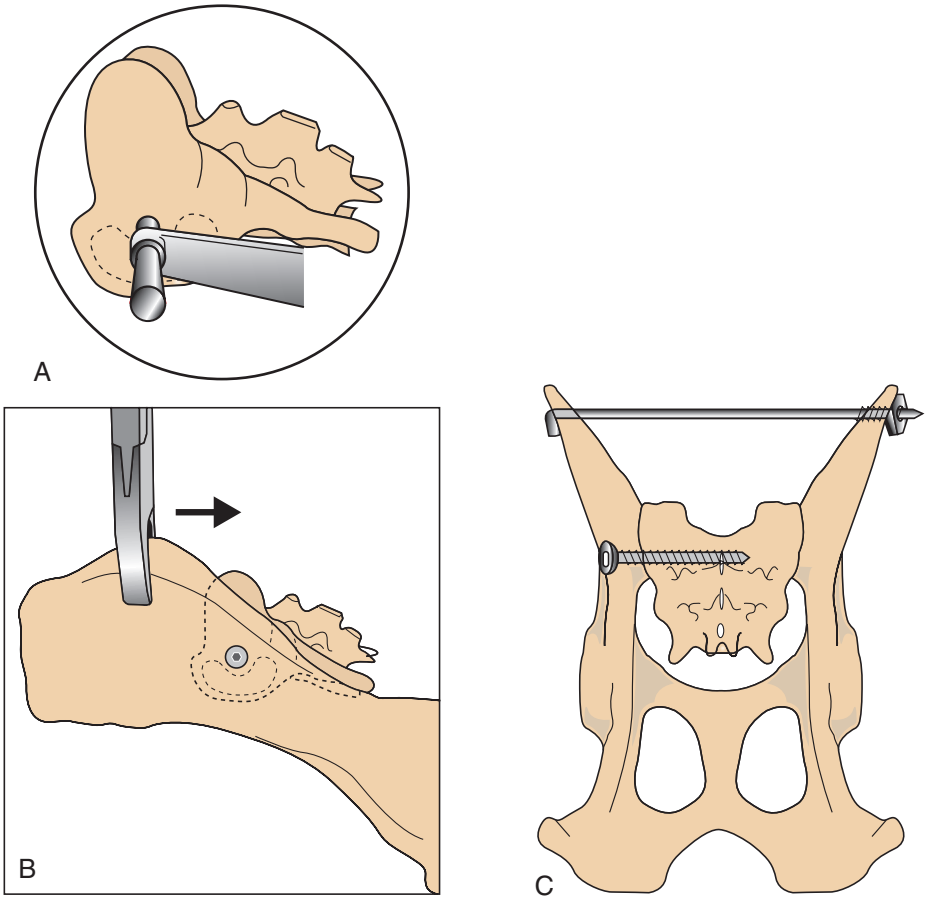


FIG. 33.73 To place a lag screw, lever the iliac body ventrally with a Hohmann retractor to expose the lateral surface of the sacroiliac joint. (A) Drill the threaded hole in the sacrum directly. (B) Then drill the glide hole in the iliac wing, reduce the fracture/luxation, and secure it with the lag screw. (C) For additional stability, add a transiliac bolt. (Modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat.* St. Louis: Elsevier; 2005.)

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should be restricted to leash walking and physical rehabilitation until the fracture has healed. Physical rehabilitation (see Chapter 11) encourages controlled limb use and optimal limb function after fracture healing. Care must be taken to develop customized protocols for each patient depending on location of the fracture, stability and type of fracture fixation, potential for healing, abilities and attitudes of the patient, and willingness and ability of the client to provide for the animal's care. Radiographs should be evaluated in 6 weeks to ascertain pelvic width after healing. Implants do not need to be removed unless they cause a problem.

COMPLICATIONS

Pelvic canal narrowing may be present after sacroiliac luxation healing and is related to the preoperative width of the pelvic canal. Severe pelvic canal narrowing can result in pelvic canal stenosis, which can cause obstipation and dystocia. Malpositioning of a screw within the sacral canal can cause serious neurologic deficits including fecal and/or urinary incontinence. Implant migration may occur with screws that are malpositioned or too short.

PROGNOSIS

The prognosis for return to normal activity is excellent after surgical stabilization of sacroiliac luxations and fractures. Animals treated with surgical stabilization usually return to limb function within 6 weeks. Longer rehabilitation times can be expected without surgery. Factors that influence the prognosis include accuracy of implant placement and depth of the implant. Screws placed in the sacral body that in depth exceed 60% of the width of the sacrum are less likely to have screw loosening than those that are placed more shallowly.

ILIAC, ISCHIAL, AND PUBIC FRACTURES

DEFINITIONS

Fractures may occur through the body or wing of the ilium. Fractures of the ischium and pubis may occur through the ischial body, floor, or pubis.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The pelvis is a boxlike structure, and generally, the hemipelvis must be fractured at three different sites to displace bone fragments. Typically, the ilium, ischium, and pubis are fractured simultaneously, resulting in loss of weight transfer from the affected limb to the spine, along with instability and pain. Iliac fractures are most often long oblique fractures of the body of the ilium. Transverse fractures and comminuted fractures also occur. The caudal fragment is usually displaced medially and cranially, compromising the pelvic canal. Because soft tissue injury may accompany obvious bony injury, careful patient assessment is necessary. Bladder and urethral tears may occur concurrent with pelvic fracture, particularly if the bladder is full at the time of impact (see p. 698). Muscle separation or avulsion of the bony insertion of the rectus abdominis muscle and herniation of abdominal viscera may also occur (see p. 519). Herniation of abdominal viscera may result in strangulation and necrosis of tissue if early diagnosis and treatment are not initiated. Impaired sensory and motor functions of the lumbosacral plexus or sciatic nerve may result from an iliac fracture. Tearing of the rectum is rare with pelvic fractures, but when it occurs, it is often fatal due to septic peritonitis and septicemia. A rectal examination should be performed to gently palpate the colon and observe for frank blood or hematochezia.

NOTE Because concurrent injuries (e.g., bladder and urethral tears, hernias, peripheral nerve injuries) are common in patients with ischial, iliac, or pubic fractures, a complete physical examination should always be performed.

Isolated ischial or pubic fractures are rare. When ischial or pubic fractures occur in combination with other pelvic fractures, reduction and stabilization of the primary weight-bearing segment usually result in acceptable reduction and stabilization of the pubis and ischium. The most common reason for surgical intervention of a pubic or ischial fracture is associated soft tissue herniation. Herniation of abdominal viscera can result from separation of the pubic symphysis or from avulsion of the cranial pubic ligament. Rarely, herniation may occur caudal to the acetabulum.

DIAGNOSIS

Clinical Presentation

Signalment

Any breed, age, or sex of dog or cat may be affected.

History

Vehicular accidents are the usual cause of iliac, ischial, and pubic bone injuries, but any type of blunt trauma may result in these pelvic fractures.

Physical Examination Findings

Affected animals usually have non–weight-bearing lameness, with minimal displacement and soft tissue injury; however, they may be partially weight-bearing. Bruising is common in patients sustaining pelvic fractures, but urethral trauma should be suspected if ventral abdominal bruising is severe or progresses. The integrity of the abdominal musculature should be carefully assessed and the function of the sciatic nerve determined before surgery.

Diagnostic Imaging

Most of these animals are in pain and require sedation or general anesthesia for proper positioning to obtain quality radiographs (see Tables 31.2 and 31.3). Ventrodorsal and lateral radiographs should be taken to assess the degree of injury to the hemipelvis and to delineate fracture planes (Fig. 33.74). Additional diagnostic tests (e.g., cystography, urethrography) may be needed to confirm the presence or absence of soft tissue injury.

Laboratory Findings

Consistent laboratory abnormalities are not found. Bladder or urethral rupture may result in azotemia and hyperkalemia (see p. 678). Traumatized animals undergoing surgery should have sufficient bloodwork done to assess the risks of anesthesia and surgery.

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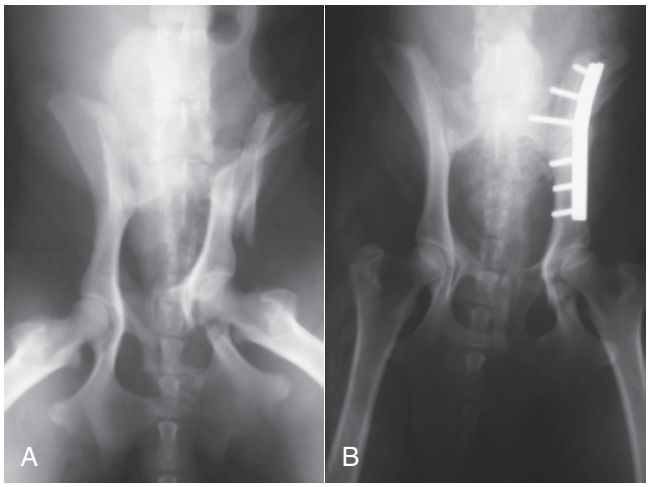


FIG. 33.74 (A) Preoperative and (B) postoperative radiographs showing reduction and placement of a bone plate and screws to stabilize an iliac body fracture.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include fracture and/or separation of the sacroiliac joint, acetabular fracture, and coxofemoral luxation.

MEDICAL MANAGEMENT

Medical treatment of animals with pelvic fractures includes analgesics for posttraumatic pain (see Chapter 13). Conservative treatment is indicated with iliac fractures that are minimally displaced and relatively stable. Conservative treatment may be considered in patients if the owners are unable to afford surgery (see p. 1092) or in cases when other medical conditions preclude anesthesia or surgery. The pelvic girdle is unstable when fractured, and excessive weight bearing may cause further medial displacement of the hemipelvis, resulting in continued pain, further compromise of the pelvic canal, and malunion of the ilium with malalignment of the coxofemoral joint. Conservative treatment is usually appropriate for isolated ischial and pubic fractures. If they occur in combination with other pelvic fractures, reduction and stabilization of the primary weight-bearing segment (i.e., ilium and acetabulum) result in acceptable reduction and stabilization of the pubis and ischium.

SURGICAL TREATMENT

Surgery is indicated to restore the weight-bearing arch of the pelvis when moderate to severe displacement and instability occur. Animals that have sustained bilateral pelvic limb injury benefit from surgery because they are able to walk more quickly and require less intensive postoperative care. Surgery is indicated for notably displaced ischial fractures and those associated with soft tissue herniation, or for reestablishment of integrity in the pelvic girdle of intact female dogs or cats that will be bred. Surgery is also indicated for pubic fractures associated with soft issue herniation.

NOTE Inform owners that although many fractures heal adequately with conservative therapy, surgery will prevent malunion and will shorten the rehabilitation time.

Application of Plates and Screws

Bone plates are the only implants that can be curved to mimic the shape of the lateral surface of the ilium, and once applied, they will maintain reduction of the ilium in that shape. Restoring the curve of the ilium prevents collapse of the pelvic canal. The dynamic compression plate is used most frequently (see Fig. 33.74). Reconstruction plates can be used when concurrent iliac body and ipsilateral acetabular fractures are present. T plates, tibial plateau leveling osteotomy plates, and other specialty plates may be appropriate for ilial shaft fractures depending upon the specific orientation of the fracture. Iliac fractures with a long oblique configuration may also be stabilized with lag screws. Combining a lateral plate with the application of a ventrally placed veterinary cuttable plate will gain additional screw purchase and will support comminuted fractures, or fractures in large or obese dogs.

Preoperative Management

Urination and bowel movements should be monitored before surgery (see previous discussion). Respirations should be monitored, and chest radiographs are indicated. An electrocardiogram is useful to determine cardiac rhythm, particularly if peripheral pulses appear weak or irregular. Analgesics should be provided to posttraumatic animals (see Chapter 13).

Anesthesia

Refer to Tables 32.1 and 32.2 for anesthetic management of patients with fractures.

Surgical Anatomy

The ilium is formed by the iliac wing and the iliac body. The iliac wing is located cranially and is recognized by the palpable iliac crest dorsally. The bone curves medially to allow for the middle and deep gluteal muscles; the bone is thin in this area and may not hold implants well. The sacroiliac joint is located medially. The body of the ilium is rectangular and is located between the wing of the ilium cranially and the acetabulum caudally. The iliac body holds implants well because of its cortical dimensions. The sciatic nerve is located medial to the body, along its dorsal longitudinal plane. Reestablishment of iliac integrity is required for weight transfer from the limb to the axial skeleton. With iliac fractures, the caudal fragment is often displaced medially and cranially to the wing of the ilium. For orientation purposes, it is often helpful to identify the ventral border of the iliac wing. The deep gluteal muscle is usually torn and lies between the two fragments. Because the sciatic nerve may be sandwiched between the dorsal borders of the two fragments, fragment manipulation should be done carefully.

NOTE Use caution when dissecting near the dorsal iliac border (or when placing bone-holding forceps over the dorsal border) to prevent injury to the sciatic nerve.

The ischium is formed by the lesser ischiatic notch cranially, the ischial floor medially, and the ischial tuberosity caudally. Caution must be exercised with surgical dissection of the ischial notch because of the presence of the sciatic nerve. Identification of the nerve is necessary for exposure and implant application. The sciatic nerve is protected once the insertion of the external rotators is incised and reflected.

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Herniation of soft tissue is the most common indication for repair of pubic fractures. The surgical area is often bruised and swollen, making identification of normal anatomic landmarks difficult. The key for surgical dissection is to begin cranial to the injury, where the ventral midline is visible. Surgical dissection can be continued caudally along the midline to expose the fractured pubis. The obturator foramen is located caudal to the cranial pubic brim. Hernia repair is discussed on p. 521.

Positioning

For iliac and ischial fractures, the patient is positioned in lateral recumbency. The surgical site should be prepared from the dorsal midline to the stifle joint and from a point 10 cm cranial to the iliac crest to the tail head caudally. Animals with pubic fractures should be positioned in dorsal recumbency and the ventral midline prepared from the umbilicus to the perineal region.

SURGICAL TECHNIQUE

Approach to the Iliac Body

Make an incision from the cranial extent of the iliac crest to 1 to 2 cm beyond the greater trochanter caudally. Center the incision over the ventral third of the iliac wing. Incise subcutaneous tissue and gluteal fat along the same line to visualize the intermuscular septum lying between the middle gluteal muscle and the long head of the tensor fasciae latae muscle (Fig. 33.75A). Visualize the intermuscular septum between the superficial gluteal and the short part of the tensor fasciae latae muscle caudally. Continue the incision to separate the tensor fasciae latae and middle gluteal muscles cranially and the tensor fasciae latae and superficial gluteal muscles caudally. Sharply dissect cranially to separate the middle gluteal muscle and the long head of the tensor fasciae latae muscle (Fig. 33.75B). Palpate the ventral border of the ilium, and make an incision at the ventral border of the middle gluteal muscle. Isolate and ligate the iliolumbar vessel, and reflect the deep and middle gluteal muscles from the lateral surface of the ilium (Fig. 33.75C). If additional exposure is needed, incise the branch of the cranial gluteal nerve that innervates the tensor fasciae latae muscle. Also dissect the origin of the middle gluteal from the cranial wing of the ilium to achieve additional exposure and to ease reduction.

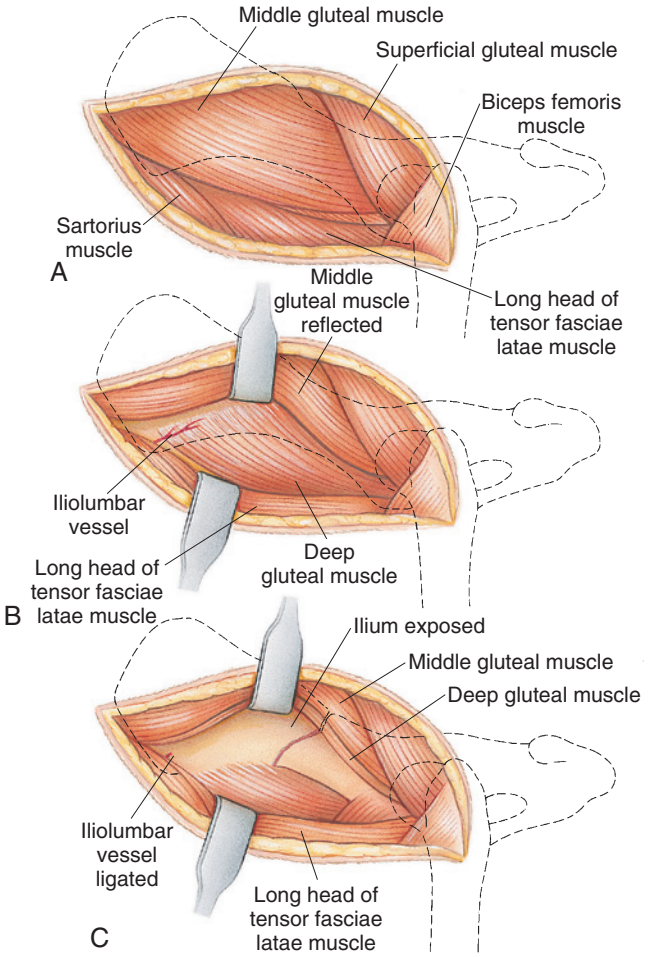


FIG. 33.75 (A) To expose the ilium, dissect between the middle gluteal muscle and the long head of the tensor fasciae latae muscle. (B) Separate these muscles. (C) Elevate the deep gluteal muscle to expose the fracture. Incise the insertion of the gluteal muscles from the iliac wing for additional exposure.

NOTE To enhance exposure when inserting lag screws or applying a ventral plate, incise through the origin of the iliacus muscle along the ventral border of the iliac body.

Stabilization of the Ilium With a Bone Plate

Reduce the fracture by placing bone-holding forceps over the dorsal edge of the caudal iliac fragment and retracting it caudally and then laterally. Be careful not to injure the sciatic nerve when manipulating the fracture fragments. Contour a plate to fit the normal curvature of the lateral surface of the bone. Use a ventrodorsal radiograph of the opposite ilium as a guide for plate contouring. Attach the plate to the caudal fragment first to allow the contour of the plate to assist in reduction of the cranial fragment. Reduce the caudal fragment by aligning the fracture and lifting the caudal fragment laterally. Clamp the cranial portion of the plate to the cranial fragment of the ilium, then place screws in the cranial fragment (Fig. 33.76A). Caudal distraction of the iliac fragment

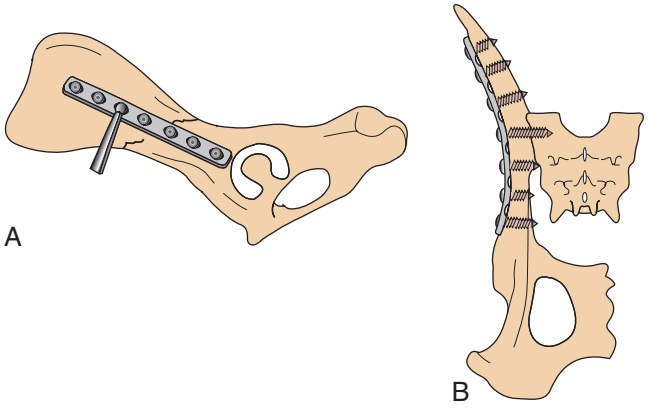


FIG. 33.76 Application of a bone plate and screws to the iliac wing. (A) The fracture is reduced by clamping the cranial portion of the plate to the iliac wing before inserting the screws. (B) The curve of the lateral plate restores the contour of the ilium. (Modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat.* St. Louis: Elsevier; 2005.)

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is readily achieved, but lateral distraction of the caudal fragment can be difficult.

Use the contour of the plate to aid in mediolateral reduction of the fracture. The shape of the precontoured plate helps bring the caudal fragment into position. Place at least three plate screws in the cranial fragment and two or three in the caudal fragment. Secure the sacrum with a long screw through the cranial plate when possible (Fig. 33.76B). For additional support, contour a veterinary cuttable plate to the ventral surface of the ilium, and secure it with bone screws. To close the incision, place sutures between the fascia of the middle gluteal muscle and the tensor fasciae latae muscle cranially and the superficial gluteal muscle and the tensor fasciae latae muscle caudally. Approximate deep gluteal fat, subcutaneous tissue, and skin routinely.

Stabilization of the Ilium With Lag Screws

Reduce the fracture as described previously, and temporarily stabilize it with bone-holding forceps. Rotate the hemipelvis to visualize the ventral surface of the body of the ilium, and insert two small Kirschner pins from ventral to proximal. To achieve additional stability, place two lag screws ventral to proximal (Fig. 33.77).

Approach to and Stabilization of Ischial Fractures

Make a skin incision adjacent to the caudal border of the greater trochanter. Reflect the biceps femoris muscle caudally to expose the sciatic nerve and external rotators as they insert into the trochanteric fossa (see previous discussion of surgical anatomy). Incise and reflect the insertions of the external rotators caudally to expose the ischial body. Reduce and stabilize the fragments with a small reconstruction bone plate and screws (Fig. 33.78).

Approach to and Stabilization of Pubic Fractures

Make a skin incision along the ventral midline (in the male dog adjacent to the penile sheath). Visualize the midline cranial to the pubic brim, and incise through the tissue overlying the pubic symphysis. If herniated tissue is present, be careful to avoid inadvertently incising vital structures. Replace the herniated tissue into the abdominal cavity. Use a periosteal elevator to reflect adductor muscles from the pubis. Reduce the fragments and drill holes in adjacent fragments for placement of orthopedic wire (Fig. 33.79). Place and tighten the wire to stabilize the fragments.

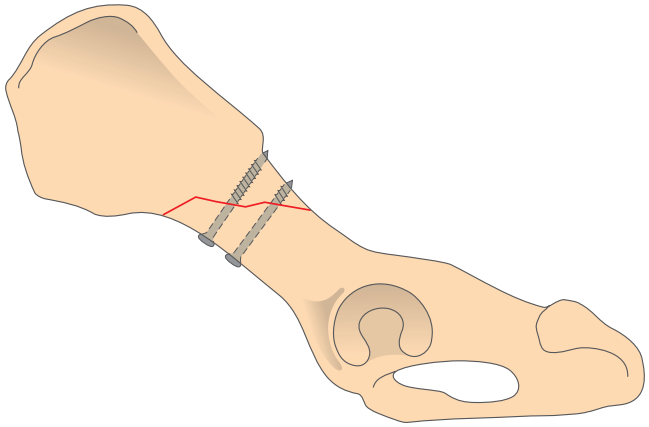


FIG. 33.77 Stabilization of oblique fracture of the iliac wing with lag screws. The screws have been inserted in a ventrodorsal direction, across the fracture line.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Instruments are needed for implantation of bone plates and screws. Bone-holding forceps and self-retaining retractors are beneficial. For pubic fractures, the symphysis can be stabilized with 20-gauge orthopedic wire in small and medium-sized dogs and with 18-gauge wire in larger dogs.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiographs are taken to evaluate alignment, apparatus and apposition. Postoperative pain management may be indicated (see Boxes 32.1 and 32.2 and Table 32.4). Activity should be restricted to leash walking and physical rehabilitation until the fracture has healed. Physical rehabilitation (see

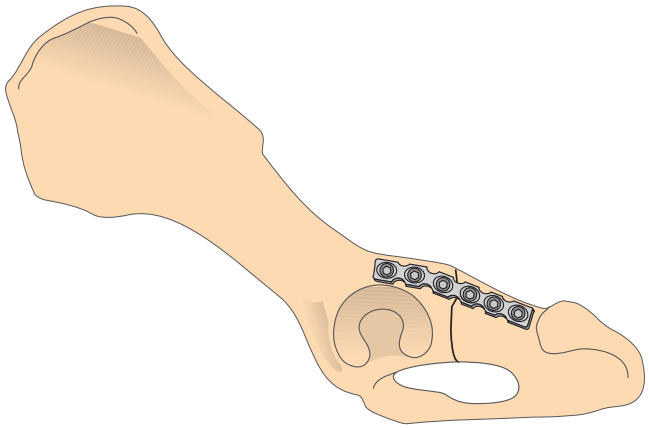


FIG. 33.78 Stabilization of ischial fracture with bone plate and screws. (Modified from Johnson AL, Houlton JEF, Vannini R, eds. *AO Principles of Fracture Management in the Dog and Cat.* New York: Thieme; 2005.)

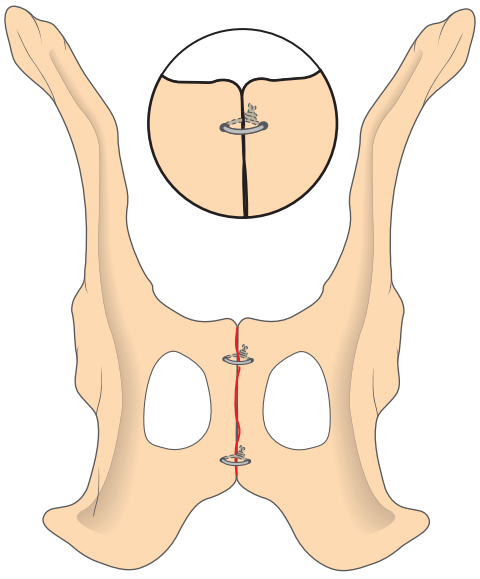


FIG. 33.79 Pubic fractures can be stabilized with orthopedic wire. The wire serves to align the bone fragments.

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Chapter 11) encourages controlled limb use and optimal limb function after fracture healing. Care must be taken to develop customized protocols for each patient depending on location of the fracture, stability and type of fracture fixation, potential for healing, abilities and attitudes of the patient, and willingness and ability of the client to provide for the animal's care. Radiographs should be evaluated at 6-week intervals until healing has occurred. Implants do not need to be removed unless they cause a problem.

COMPLICATIONS

Screw loosening, the most common implant-related complication, occasionally results in loss of fracture alignment with compromise of the pelvic canal.

PROGNOSIS

The prognosis is excellent for return to normal function with most iliac fractures after surgery. Factors that warrant a more guarded prognosis include ipsilateral acetabular fractures, with potential development of osteoarthrosis. The prognosis for isolated pubic and ischial fractures is excellent for return to normal function. If other pelvic fractures are present, the prognosis depends on how these fractures heal rather than on the pubic or ischial fracture outcome.

ACETABULAR FRACTURE

DEFINITION

Acetabular fractures occur through the articular surface and medial fossa of the acetabulum.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Acetabular fractures occur after blunt trauma, most often vehicular accidents, and are usually concomitant with other pelvic pathology. Approximately 12% of pelvic fractures in dogs and 7% in cats are acetabular fractures. Additionally, approximately one-fourth of pelvic fractures involve bilateral iliac body fractures, bilateral acetabular fractures, or acetabular fractures coupled with a contralateral iliac body fracture. The hip joint transfers weight from the rear limb through the pelvis to the spine. Sequelae to conservatively treated acetabular fractures include degenerative joint disease and loss of function. Occasionally the sciatic nerve may be injured in association with acetabular fractures.

Fractures of the acetabulum are classified as cranial, central, or caudal according to the location of involvement of the articular surface. The cranial and central portions of the acetabulum are the weight-bearing areas, and fractures of these areas should be anatomically reduced and rigidly fixed for optimal function. Caudal fractures are less demanding but may result in degenerative joint disease with conservative treatment.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, breed, or sex of dog or cat may be affected.

History

Acetabular fractures usually result from motor vehicle accidents but may be associated with other forms of blunt trauma or falls.

Physical Examination Findings

Affected animals are generally brought in for evaluation of non–weight-bearing lameness. Some patients bear weight on the affected limb if fracture displacement is minimal. Pain can generally be elicited on manipulation of the hip joint, but crepitation may or may not be present.

Diagnostic Imaging

Most of these animals are in pain and require sedation or general anesthesia for proper positioning to obtain quality radiographs (see Tables 31.2 and 31.3). Ventrodorsal and lateral radiographs should be taken to assess the pelvic fractures. Oblique radiographs taken by positioning the animal in lateral recumbency and abducting the unaffected leg to expose the affected acetabulum often help delineate acetabular fractures. CT can be used to better delineate fractures of the pelvis, particularly if these fractures involve the acetabulum. Reconstructed two-dimensional and three-dimensional CT images allow a better appreciation of the fracture appearance and facilitate fracture planning.

Laboratory Findings

Consistent laboratory abnormalities are not present. Traumatized animals undergoing surgery should have sufficient bloodwork done to determine the optimal anesthetic regimen.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include capital physeal fractures, coxofemoral luxations, proximal femoral fractures, and ipsilateral iliac or ischial fractures.

MEDICAL MANAGEMENT

Medical treatment of animals with acetabular fractures includes analgesics for posttraumatic pain (see Chapter 13). To maintain joint congruity and the normal distribution of stress over the articular surface, reduction and stabilization of acetabular fractures are recommended in all cases. If the client cannot afford surgery, conservative treatment should be considered; however, it may result in less than optimal limb function. Initial conservative treatment, followed by a delayed femoral head and neck excision, may be indicated in cases when surgery and anesthesia are initially contraindicated due to medical reasons. Conservative treatment for acetabular fractures is similar to that for sacroiliac luxations (see p. 1092).

SURGICAL TREATMENT

Anatomic reconstruction of the articular surface and rigid stabilization are mandatory to restore acetabular joint function and minimize degenerative joint disease. Plate and screw fixation is the preferred approach for acetabular reconstruction and stabilization, regardless of the fracture assessment score. However, accurate plate contouring is essential for maintaining anatomic reduction. As screws are tightened, the bone shifts to match the plate, often disrupting the reduction. Techniques such as plate

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luting and the use of screws and orthopedic wire reinforced with polymethylmethacrylate have been described to improve the accuracy of reduction. Dogs with irreparable acetabular fractures may be candidates for femoral head and neck ostectomy (see p. 1216), but the acetabulum should still be bridged and stabilized with a bone plate to decrease pain and to encourage early motion, which is paramount to a successful outcome.

Preoperative Management

Urination and bowel movements should be monitored before surgery. Respirations should be monitored, and chest radiographs are indicated. An electrocardiogram is useful to determine cardiac rhythm, particularly if peripheral pulses appear weak or irregular. Analgesics should be provided to posttraumatic animals (see Chapter 13).

Anesthesia

Refer to Tables 32.1 and 32.2 for anesthetic management of patients with fractures.

Surgical Anatomy

The hip joint is a ball-and-socket joint constituting the femoral head and acetabulum. The normal conformation, surrounding musculature, suction-like effect of the synovial fluid, and ligament of the femoral head act to stabilize the joint. The articular surface is on the dorsolateral face of the acetabulum, and the medial face is occupied by the round ligament. The fibrous joint capsule originates from the lateral acetabular rim and inserts onto the femoral neck. Stabilizing musculature surrounding the hip includes the gluteal muscles, internal and external rotators, and the iliopsoas muscle medially.

The sciatic nerve courses dorsomedial to the acetabulum. Fractures cause the caudal acetabular segment to become displaced medial and cranial to the cranial segment. The sciatic nerve may be positioned directly dorsal or dorsolateral to the caudal acetabular segment. Caution must be exercised when exposing and reducing the caudal segment to avoid damaging this nerve. Soft tissue bruising and swelling often distort the normal anatomy. Dissection adjacent to the greater trochanter facilitates correct tissue identification and reflection. The insertion of the deep gluteal muscle at the greater trochanter can be used as a landmark for dissection.

Positioning

The animal is positioned in lateral recumbency with the dorsum raised 30 degrees from the operating table. The hip should be prepared from the dorsal midline to the tarsus. A hanging-leg preparation facilitates manipulation of the limb during surgery.

SURGICAL TECHNIQUE

Approach to the Acetabulum via Trochanteric Osteotomy

Make a skin incision centered over the cranial border of the greater trochanter. Begin the incision 3 to 4 cm proximal to the dorsal ridge of the greater trochanter, and curve it distally 3 to 4 cm, following the cranial border of the femur. Incise the superficial leaf of the fascia lata at the cranial border of the biceps femoris muscle, and retract the muscle caudally (Fig. 33.80A). Incise the deep leaf of the fascia lata, and carry the incision proximally through the insertion of the tensor fasciae latae muscle at the greater trochanter and along the cranial border of the superficial gluteal muscle. Incise through the insertion of the superficial gluteal muscle at the third trochanter. Reflect the superficial gluteal muscle proximally and the biceps femoris caudally to find and visualize the course of the sciatic nerve (Fig. 33.80B). Perform an osteotomy of the greater trochanter with an osteotome and mallet or Gigli wire (Fig. 33.80C). Position the osteotome just proximal to the insertion of the superficial gluteal muscle at the third trochanter. Angle the osteotome 45 degrees to the long axis of the femur to remove the trochanter with the insertions of the deep gluteal and middle gluteal muscles (Fig. 33.80D). Reflect the gluteal muscles and the greater trochanter from the joint capsule with a periosteal elevator. Visualize the insertions of the gemellus muscle and tendon of the internal obturator, and preplace a suture through the two insertions near the trochanteric fossa. Incise both structures together at the trochanteric fossa, and elevate the gemellus muscle from the caudolateral surface of the acetabulum with a periosteal elevator (see Fig. 33.80E). Use the suture to retract the muscle proximally and caudally. For additional exposure of the ilium, combine the lateral approach to the ilium (see p. 1097) with the approach to the acetabulum.

Application of Bone Plates and Screws

Adequate exposure and manipulation of the caudal acetabular fragment are important components of anatomic reduction and plate application. Accurate plate contouring is essential for retaining anatomic reconstruction of the articular surface. A specially designed curved canine acetabular plate is easier to contour to the dorsal rim of the acetabulum than standard plates and is recommended for transverse or short oblique acetabular fractures. This plate does not require precontouring. Alternatively, reconstruction plates that can be contoured with specially designed bending pliers in the direction of its long and short (width) axes can be used. It is helpful to precontour this plate to a similarly sized bone specimen before surgery. Smaller dogs and cats may require 2.7 or 2.0 T or L plates.

Acetabular fractures combined with iliac body fractures may be treated with two plates: a dynamic compression plate applied to the iliac body fracture and an acetabular plate applied to the acetabular fracture. Alternatively, a reconstruction plate can span the hemipelvis (Fig. 33.81). Comminuted acetabular fractures must be reconstructed with lag screws and supported with a plate. *For caudal fragment control, expose the tuber ischium and place a bone-holding forceps on it (Fig. 33.82). When reducing the fracture, pay particular attention to the alignment of the articular surface, which is facilitated by incising the joint capsule (see Fig. 33.80E). Place plates on the dorsal rim of the acetabulum. Attempt to place at least two plate screws in the caudal fragment and three plate screws in the cranial fragment (see Fig. 33.82). After reduction and stabilization, suture the gemellus and the internal obturator tendon to their points of insertion. Suture the joint capsule. Reduce and stabilize the greater trochanter with two K-wires and a tension band wire (see p. 1015). Place interrupted sutures in the insertion of the superficial gluteal muscle and a continuous suture in the insertion of the tensor fasciae latae muscle and deep leaf of the fascia lata. Use a continuous suture in the superficial leaf of the fascia lata and the subcutaneous tissue. Suture the skin with an interrupted suture pattern.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Equipment is needed for application of an acetabular bone plate or reconstruction plate. An osteotome and mallet or Gigli wire

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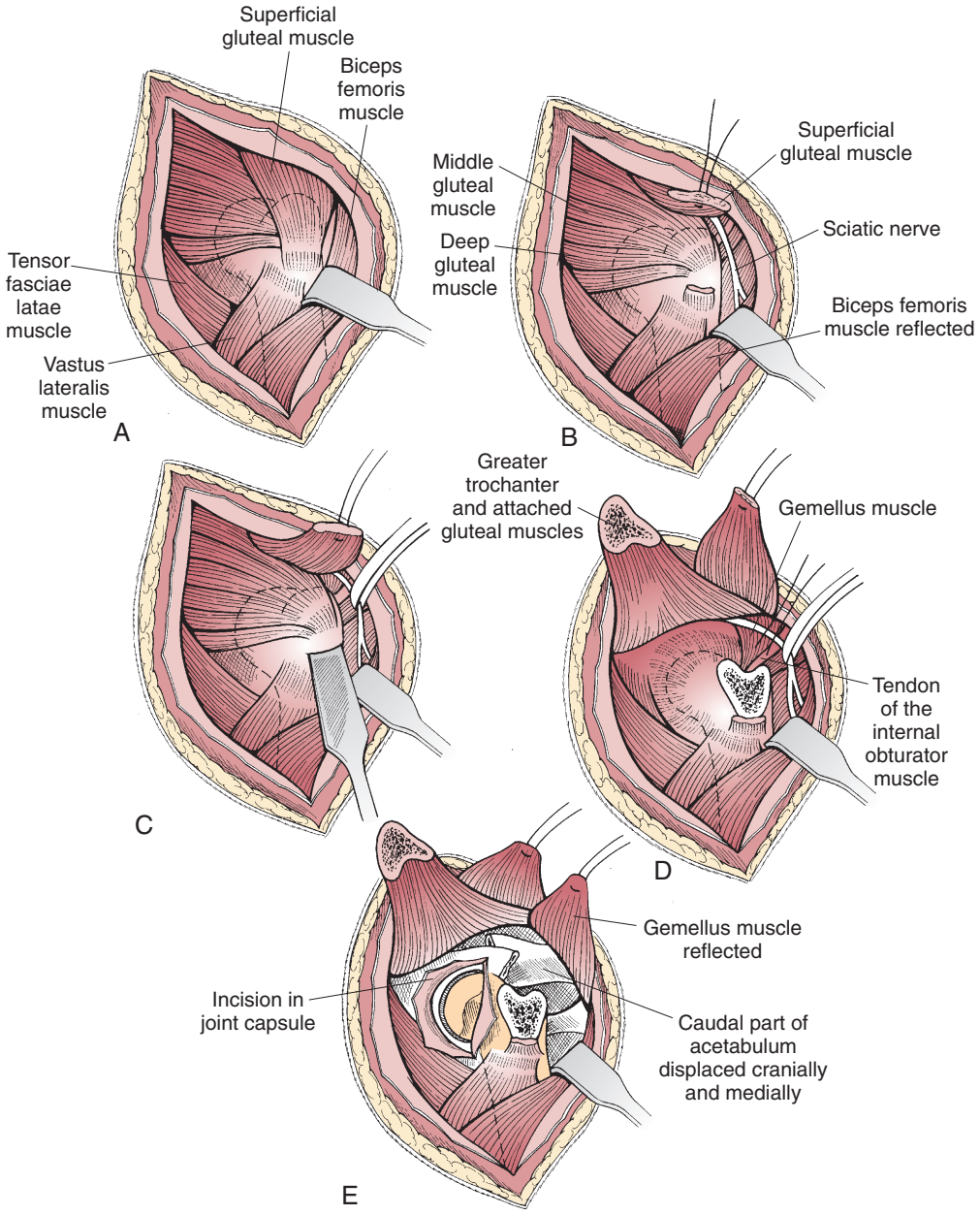


FIG. 33.80 (A) To expose the hip joint through an osteotomy of the greater trochanter, retract the biceps femoris muscle caudally. (B) Reflect the superficial gluteal muscle to expose the third trochanter. (C–D) Osteotomize the greater trochanter, and reflect the attached musculature dorsally. (E) Incise and reflect the gemellus and internal obturator muscles. Incise the joint capsule. Note that the caudal fragment of the fracture is displaced craniomedially.

is necessary to perform a trochanteric osteotomy. Small pins and wire are used to stabilize the trochanteric osteotomy with a tension band.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiographs should be taken to assess alignment, apparatus, and apposition. Postoperative pain management may be indicated (see Boxes 32.1 and 32.2 and Table 32.4). Activity should be restricted to leash walking and physical rehabilitation until the fracture has healed. Physical rehabilitation (see Table 33.3 and Chapter 11) encourages controlled limb use and optimal limb function after fracture healing. Care must be taken to develop customized protocols for each patient depending on location of the fracture, stability and type of fracture fixation, potential for healing, abilities and attitudes of the patient, and willingness or ability of the client to participate in the animal's care. Radiographs should be evaluated at 6-week intervals until healing has occurred. Healing generally occurs between 6 and 12 weeks, depending on the biological fracture assessment. Implants are not routinely removed. Occasionally the tension band wire and K-wires need to be removed because of soft tissue irritation.

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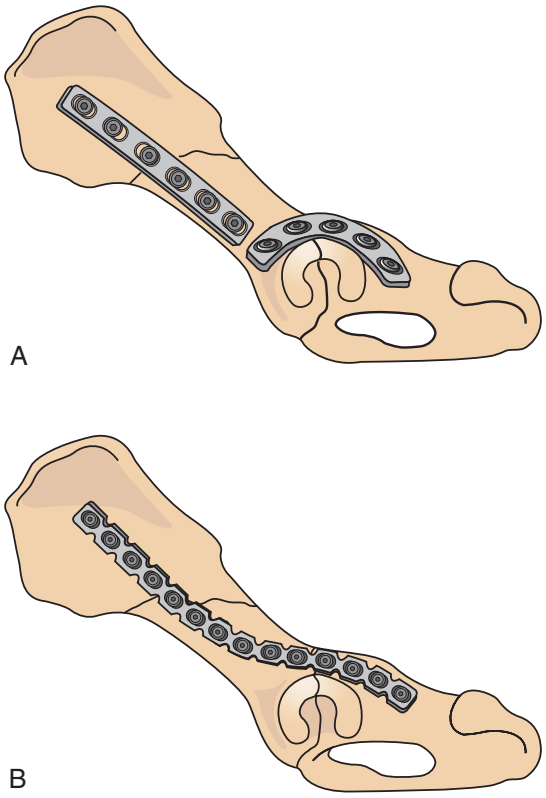


FIG. 33.81 Plate application for combined acetabular and iliac body fractures. (A) Application of two plates: a dynamic compression plate applied to the iliac body fracture and an acetabular plate applied to the acetabular fracture. (B) Alternatively, a reconstruction plate can span the hemipelvis. (Modified from Johnson AL, Houlton JEF, Vannini R, eds. *AO Principles of Fracture Management in the Dog and Cat*. New York: Thieme; 2005.)

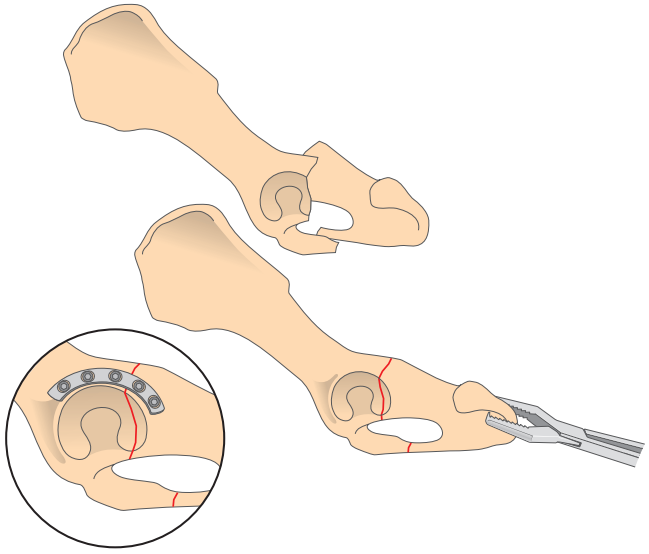


FIG. 33.82 Reduce the acetabular fracture by manipulating the caudal fragment with a bone-holding forceps secured to the ischium. Hold the fracture in reduction with bone-holding forceps placed across the lesser ischiatic notch, and stabilize it with a bone plate and screws *(inset)*.

COMPLICATIONS

Acetabular fractures may result in postoperative degenerative joint disease, although this is minimized with careful reduction and rigid fixation. Other complications include sciatic nerve injury, delayed union, nonunion, osteomyelitis, and fixation failure. Poor decision making regarding implant choice relative to the fracture assessment is the most common reason for complications (see p. 1031 for treatment of complications).

PROGNOSIS

The prognosis is good to excellent if the acetabular fracture is anatomically reduced and appropriately stabilized.

FEMORAL FRACTURES

FEMORAL DIAPHYSEAL AND SUPRACONDYLAR FRACTURES

DEFINITIONS

Femoral diaphyseal fractures result in disruption of the continuity of the diaphyseal cortical bone. Supracondylar fractures are fractures of the distal diaphysis.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Femoral fractures are usually caused by trauma. Occasionally a patient will present with an acute femur fracture without discernible or historic trauma; fractures in these patients may occur secondary to a preexisting bone pathologic condition. Primary or metastatic bone tumors are the most common cause of pathologic fractures. When preexisting disease is present, radiographs made at the time of injury show cortical lysis and new bone formation in the area of the fracture.

High-velocity injuries are the most common type of trauma that causes femoral fractures in veterinary patients. Most injuries result from automobile accidents, but gunshot injuries and blunt trauma are also common. A thorough physical examination is necessary to rule out concurrent injury (e.g., thoracic trauma, coxofemoral luxations, pelvic girdle injuries). Careful thoracic auscultation and percussion help detect cardiac or airway abnormalities. Abnormal heart rhythm and pulse deficits suggest traumatic myocarditis, whereas lack of normal air movement on auscultation may indicate pulmonary contusion, pneumothorax, or diaphragmatic hernia. Thoracic radiographs and a lead II electrocardiogram are useful and should be done routinely as part of the anesthetic preoperative database for patients sustaining high-velocity injuries.

Coxofemoral luxations (see p. 1220) may occur concurrently with femoral fractures. Diagnosis is often made when radiographs are taken to evaluate the femur because swelling in the limb often precludes palpation of bony landmarks used for assessing femoral head position relative to the hip joint. Concurrent fractures or luxations must be considered when the appropriate implant is chosen for fracture stabilization. Observation of pelvic girdle symmetry and gentle rectal palpation help determine the presence of pelvic fracture. Additional radiographs centered on the pelvis are indicated if abnormalities are found. If fractures

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of the pelvis are found, careful assessment of urinary tract integrity is recommended.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, breed, or sex of dog or cat may be affected, but young male dogs are most likely to have trauma-induced femoral fractures.

History

Trauma may or may not have been observed. Motor vehicle accidents cause most cases of femoral diaphyseal fracture. Other modes of injury include gunshots and falls.

Physical Examination Findings

Patients with femoral diaphyseal fractures are usually non–weight-bearing and have varying degrees of limb swelling. Pain and crepitus can often be elicited with limb manipulation. Proprioception may appear abnormal because the animal may not lift its paw when it is placed on its dorsum. The animal's reluctance to move the limb may be caused by pain.

Diagnostic Imaging

Both craniocaudal and lateral radiographs of the femur are necessary to assess the extent of bone and soft tissue injury. Most of these animals are in pain and require sedation or general anesthesia for proper positioning to obtain quality radiographs (see Tables 31.2 and 31.3). Alternatively, radiographs can be taken under anesthesia just before surgery; however, this reduces the amount of time available for planning surgical repair. Radiographs of the contralateral limb are useful in assessing normal bone length and shape. These radiographs can be used to contour a bone plate more precisely before surgery, reducing operative time. Radiographs are also used as a reference to select appropriately sized implants.

Laboratory Findings

Consistent laboratory abnormalities are not present. Animals sustaining fractures secondary to trauma should have sufficient bloodwork done to determine appropriate anesthetic regimens and concurrent diseases.

DIFFERENTIAL DIAGNOSIS

Femoral fractures should be differentiated from muscle contusion, coxofemoral luxation, fractures of the pelvic girdle, and ligamentous injury to the stifle.

MEDICAL MANAGMENT

Medical treatment of animals with femoral fractures includes analgesics for posttraumatic pain (see Chapter 13) and antibiotics to treat open fractures. Casts or splints are not recommended for femoral fractures because adequate stabilization of the femur is difficult using these methods.

SURGICAL TREATMENT

IM pins, interlocking nails, IM pins plus ESF, external skeletal fixators alone, and bone plates may be used to repair femoral diaphyseal fractures. The implant system chosen should reflect the patient's fracture assessment (Box 33.14).

BOX 33.14 Implant Use for Fractures of the Femoral Diaphysis According to Fracture Assessment Score (FAS)

FAS 0 to 3

Bone plate and intramedullary (IM) pin
Bone plate and screws
Interlocking nail

FAS 4 to 7

External skeletal fixation with IM pin tie-in
Bone plate and screws with or without IM pin
Interlocking nail

FAS 8 to 10

IM pin with cerclage wires or external fixator
Flexible bone plate and screws

Preoperative Management

Because these fractures result from trauma, all affected animals should be examined for concurrent injury and stabilized if necessary before surgery. Fractures of the femur are not usually immobilized preoperatively because of the difficulty in applying coaptation splints. Patients should be confined to a small area until surgery. Analgesics should be provided to posttraumatic animals (see Chapter 13).

Anesthesia

Refer to Tables 32.1 and 32.2 for anesthetic management of patients with fractures.

An epidural anesthetic may make fracture reduction easier.

Surgical Anatomy

The diameter of the femoral marrow cavity varies along its length and is narrower proximally than distally. The narrowest area of the marrow cavity is called the *isthmus.* In the femur, the isthmus is located within the proximal third of the bone, just distal to the third trochanter. The canine femur is normally curved in a cranial-to-caudal direction; the curvature is most accentuated in the distal third of the femur. The amount of curvature varies between breeds. The cross-sectional diameter of the feline femur is uniform from proximal to distal.

NOTE The anatomy of the canine and feline femur differs. The cat femur is straighter, with little or no cranial-to-caudal bend.

The normal anatomy of the femur and surrounding tissue may be less apparent when fractures are present. Soft tissue swelling and bruising vary depending on the velocity of the injury. The vastus lateralis muscle often appears swollen and bruised when the fascia lata is incised. Cranial retraction of the vastus lateralis may be aided by releasing the muscle from the distal femoral caudolateral surface. Hematomas and serum are frequently encountered, which may make fractured bones difficult to identify. Proximal and distal fracture segments can be identified using a combination of gentle retraction and probing. It is often

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useful to begin dissection proximal or distal to the fracture site in an area of more normal anatomy. The dissection is then carried into the fracture zone.

Fracture of the femur frequently results in external rotation of the proximal segment due to the pull of the internal and external obturator muscles. Restoration of proper alignment is critical to avoid lameness due to hip subluxation and patella subluxation or luxation. Rotational alignment is achieved in nonreducible comminuted fractures by (1) aligning the roughened caudal surface of the femur of the attachment of the adductor magnus muscle, (2) achieving the normal spatial relationship (generally 90 degrees) between the patella and greater trochanter and/or femoral neck, and (3) achieving approximately equal range of motion in internal and external rotation of the patella when the fracture has been reduced and stabilized.

Positioning

The patient is positioned in lateral recumbency. The leg is prepped from the dorsal midline to the tarsal joint. It is advantageous to use a hanging-leg preparation to allow maximum manipulation of the limb during surgery. A donor site for cancellous bone graft harvest is prepared at the ipsilateral proximal humerus. Alternatively, the ipsilateral iliac wing or proximal tibia is used.

SURGICAL TECHNIQUE

Surgical Approach to the Femoral Diaphysis

Make an incision along the craniolateral border of the thigh (Fig. 33.83A). Ensure that the incision is made slightly more cranial than lateral because the exposure plane will be at the cranial border of the biceps. The length of the incision depends on the type of implant used for stabilization and fracture configuration. In general, bone plate insertion and comminuted fracture patterns require a longer incision. The approach to distal diaphyseal fractures may include a lateral approach to the stifle joint (see p. 1234). *Incise the superficial leaf of the fascia lata along the cranial border of the biceps femoris muscle for the length of the incision (Fig. 33.83B). Retract the biceps femoris caudally to expose the vastus lateralis muscle (Fig. 33.83C). Incise the fascial septum of the vastus lateralis as it inserts at the caudal lateral border of the femur. Reflect the vastus lateralis from the surface of the femur to expose the femoral diaphysis (Fig. 33.83D). Carefully manipulate soft tissue and fracture hematoma to allow fracture reduction and application of a fixation system.*

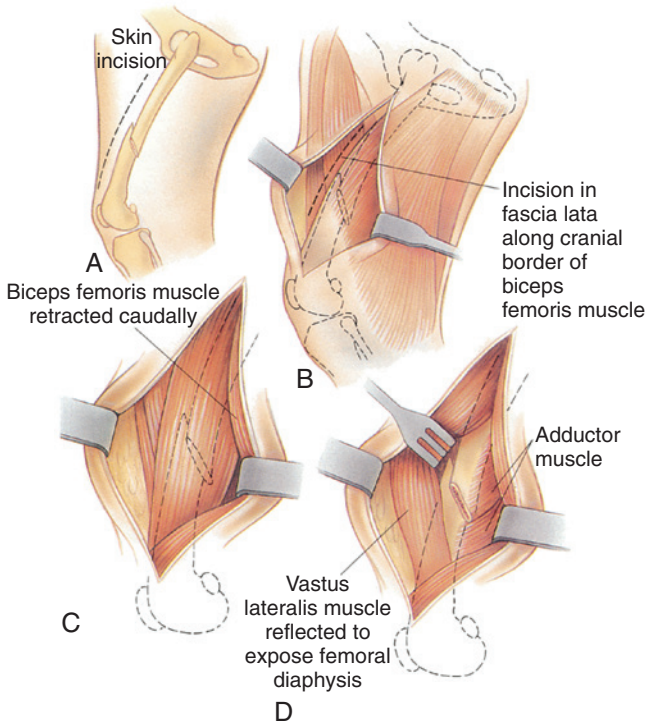


FIG. 33.83 (A) To expose the femoral diaphysis, make an incision along the craniolateral border of the thigh. (B) Incise the superficial leaf of the fascia lata along the cranial border of the biceps femoris muscle for the length of the incision. (C) Retract the biceps femoris caudally to expose the vastus lateralis muscle. (D) Reflect the vastus lateralis from the surface of the femur to expose the femoral diaphysis.

Implant Application Techniques Specific to the Femur

Application of Intramedullary Pins

An IM pin can be used to stabilize femoral mid-diaphyseal fractures, providing excellent resistance to bending but not to rotational forces or axial loading. Additional implants must be used to provide rotational and axial support for most fractures (Fig. 33.84) (for general principles of IM pin application, see p. 1008). Generally, an IM pin should equal 70% to 80% of the diameter of the marrow cavity. When an appropriate IM pin is chosen, the diameter of the marrow cavity at the isthmus must be considered and can be estimated from preoperative radiographs. The curvature of the femur also governs pin size: the greater the curvature and the more distal the fracture, the smaller the pin must be for proper seating within the femoral condyles. An IM pin can be normograded or retrograded for placement in the femur. The advantage of normograde placement is that the pin can be placed laterally, adjacent to the greater trochanter. This positions the pin so that it passes through less soft tissue than when placed in a retrograde fashion and ensures that the pin is positioned lateral to the sciatic nerve. The disadvantage of normograde placement is that it is difficult to identify the correct entry point into the bone because insertion of the pin into the trochanteric fossa is generally done blindly. *To normograde an IM pin, make a small skin incision at the point of pin entry over the bony prominence of the greater trochanter.* Palpation of the greater trochanter may be difficult if the limb is swollen secondary to trauma of the soft tissue. Limited surgical exposure may be necessary to locate the greater trochanter prominence. *Push the point of the pin through soft tissue until it contacts the most proximal trochanteric ridge. Walk the pinpoint off the medial edge of the greater trochanter until it falls into the trochanteric fossa. Secure the proximal segment with Kern bone-holding forceps, and drive the pin through the proximal metaphyseal cancellous bone in a slightly caudomedial direction. As the pinpoint emerges from the marrow cavity at the fracture site, reduce the fracture and drive the pin into the distal segment. Use a second pin equal in length to that placed in the marrow cavity for a point of reference to estimate appropriate pin penetration into the distal bone segment. Remove the proximal excessive pin by pushing the skin down and cutting the pin below the level of the skin. Suture the skin over the pin.* After cutting the pin, it may be impacted deeper with a pin set or Jacobs chuck and a mallet. *Ensure that there is adequate bone stock in the distal femur so it does not penetrate the bone within the stifle joint.* The advantage of retrograde pin placement

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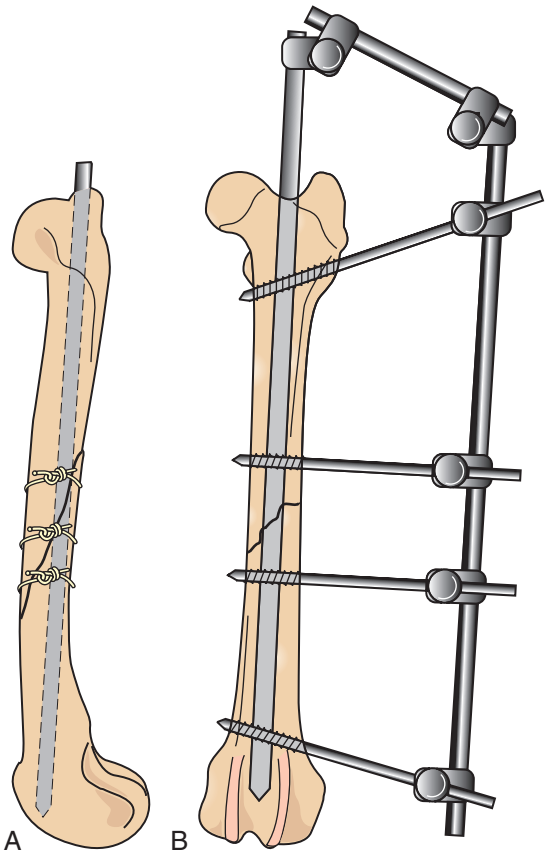


FIG. 33.84 Placement of an intramedullary (IM) pin in the femur. Depending on fracture configuration, the IM pin may be supported with (A) cerclage wires or (B) an external fixator, which may be tied into the IM pin. (Modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat.* St. Louis: Elsevier; 2005.)

in the femur is the ability to visualize the pin's site of insertion at the fracture. The disadvantage of retrograde placement is the difficulty in controlling the pin's site of exit at the trochanteric fossa because the pin may exit too far medially, causing soft tissue irritation, increasing the risk of sciatic palsy, and interfering with femoral head development in immature animals. *To retrograde a pin, expose the fracture and insert the pin into the medullary canal of the proximal segment. Secure the proximal bone segment with a Kern bone-holding forceps, and drive the pin proximally, forcing the shaft of the pin against the caudomedial cortex to help ensure that the pin exits laterally in the fossa. Hold the limb in a hip extended and adducted position while the pin exits the trochanteric fossa to prevent penetration of the sciatic nerve. Pull the pin proximally until the point is within the canal. Reduce the fracture, and drive the pin distally.* After cutting the pin, it may be impacted deeper with a pin set or Jacobs chuck and a mallet. *Ensure that there is adequate bone stock in the distal femur so it does not penetrate the bone within the stifle joint.*

NOTE Warning: Cutting the pin at the level of the trochanter may allow the pin end to injure the sciatic nerve. After cutting the pin, it may be impacted deeper with a pin set or Jacobs chuck and a mallet.

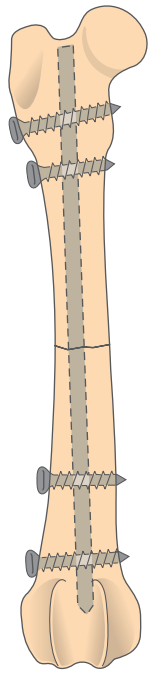


FIG. 33.85 Placement of interlocking nail in the femur.

NOTE Normograde placement of the pin is advantageous because it allows the pin to be inserted more laterally at the proximal aspect of the bone than can be done with retrograde placement. To help identify the correct entry point for the pin, consider a limited surgical exposure of the greater trochanter.

NOTE Warning: Failure to hold the femur adducted and the hip in extension when driving an IM pin retrograde through the trochanteric fossa may injure the sciatic nerve.

NOTE Always have a reference pin available that is the same length as the IM pin being inserted.

Application of Interlocking Nails

Interlocking nails can be used to stabilize both single and comminuted mid-diaphyseal femoral fractures. The interlocking nail provides resistance to bending, rotational, and axial loading forces and can offer effective IM fixation to bridge a nonreducible fracture (for general principles of interlocking nail application, see p. 1011). An open approach is used to reconstruct reducible fractures. An "open but don't touch" approach is used when major segment alignment is the goal. The size of the nail selected should correspond to the width of the medullary canal at the isthmus of the bone. *Ream the medullary canal using a normograde or retrograde technique. Insert the interlocking nail in a normograde manner starting at the trochanteric fossa. Make a limited approach to the trochanter to expose the point of insertion. Slightly overreduce the distal femur to insert the nail an appropriate distance into the distal segment (Fig. 33.85).*

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NOTE Overreduction is positioning the distal fragment with the femoral condyle further cranially resulting in a gap at the caudal aspect of the fracture line. This decreases the effect of the normal femoral curvature, enabling IM pins, bone plates, or interlocking nails to be placed further distally on or in the femur.

Application of External Skeletal Fixation

The surrounding muscle mass and the proximity of the abdomen and motion of the stifle make external fixator application to the femur challenging. Generally, an IM pin combined with a type Ia fixator with the pin tied into the fixator is used to supply bending, axial, and rotational support to the fracture. The external fixator pins in the central third of the diaphysis often pass through muscles and cause pain during motion of the leg. The number and type of fixation pin vary with the rigidity of fixation desired and the length of time the fixator must remain in place (for general principles of ESF application, see p. 998). *Insert an IM pin that occupies 50% to 60% of the medullary canal in a normograde or retrograde manner (see earlier, IM pin application). Apply a type Ia fixator to the lateral surface of the femur. Insert the proximal fixation pin laterally at the level of the third trochanter. Use a small K-wire as a "feeler" pin to identify a proper angle of insertion for the fixation pin that avoids intersecting the IM pin. Use the same technique to insert the distal fixation pin laterally 2 to 3 cm proximal to the femoral condyles. Insert the remaining pins in the femoral diaphysis. Connect the IM pin to the external fixator frame to increase the stiffness of the fixation (see Fig. 33.84B).*

To construct a modified type Ib external skeletal fixator, place an additional fixation pin in a cranial lateral–to–caudal medial direction in the proximal half of the femur. Connect the cranial pin with the distal-most lateral fixation pin with a connecting bar. Tie the IM pin into the frame. A third frame may be added by using a transfixation pin as the distalmost pin in the lateral frame and connecting the medial aspect of that pin to the proximal cranial pin.

Application of Bone Plates and Screws

Bone plates are ideally suited for complex or stable fractures of the femur. Plate size depends on patient size and plate function. The plate may serve as a compression plate, neutralization plate, or bridging plate with or without an IM pin (see p. 1016 for general principles of plate application) (Fig. 33.86). *Place an appropriately sized and contoured plate on the lateral surface of the femur. Combine the plate with an IM pin sized 40% to 50% of the medullary canal width for additional stability for comminuted fractures.* A locking plate applied as an internal fixator to stabilize comminuted femoral fractures does not need to anatomically conform to the shape of the bone as long as the bone is aligned when the plate is applied; however, with comminuted fractures, it is helpful to contour the plate to the normal shape of the femur and secure the bone segments to the plate with reduction forceps before inserting the locking screws (Fig. 33.87).

Fixation Techniques for Specific Fractures

Stabilization of Midshaft Transverse or Short Oblique Fractures

Stabilization of a transverse or short oblique fracture requires rotational and bending support; short oblique fractures also require axial support. This can be achieved with bone plates, an interlocking nail, or an IM pin with an external fixator.

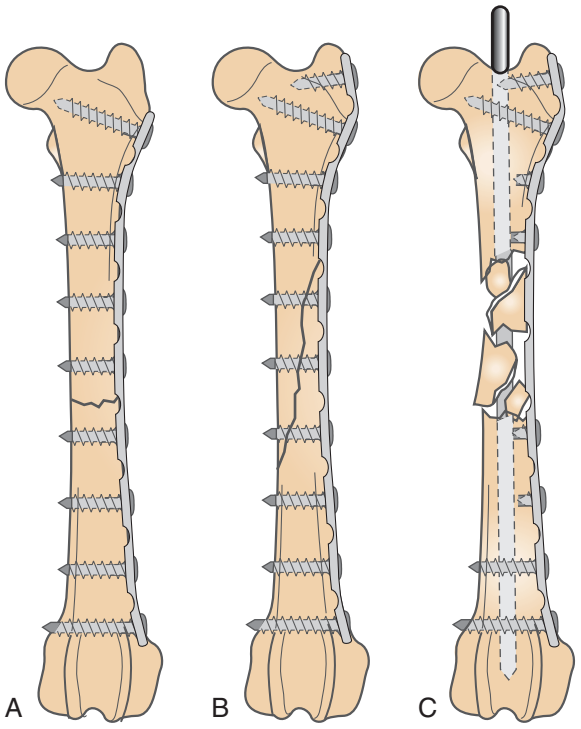


FIG. 33.86 A plate placed on the lateral surface of the femur may function as (A) a compression plate for transverse fractures, (B) a neutralization plate to support long oblique fractures reconstructed with lag screws, or (C) a bridging plate combined with an IM pin to span a nonreducible fracture. (Modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat*. St. Louis: Elsevier; 2005.)

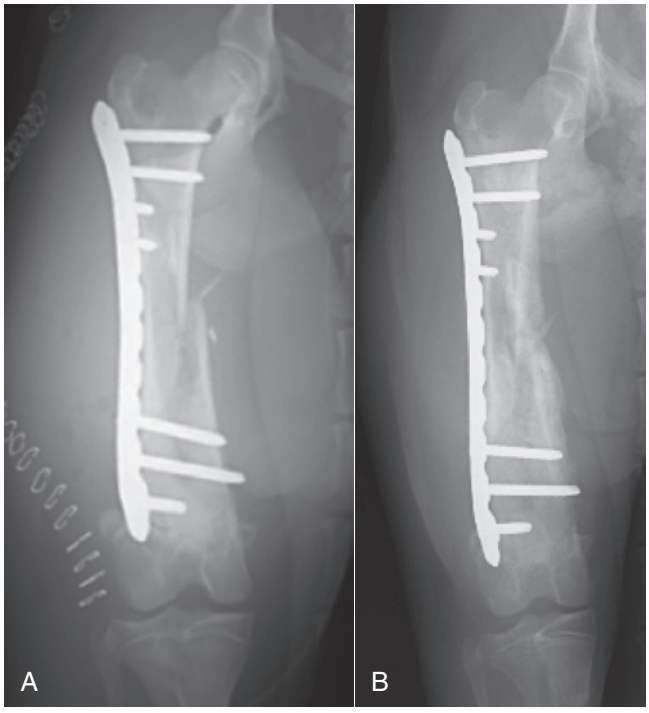


FIG. 33.87 Postoperative radiographs. (A) Comminuted femoral fracture in a dog score stabilized with a locking plate bridging the fracture. (B) Healing fracture 5 weeks later.

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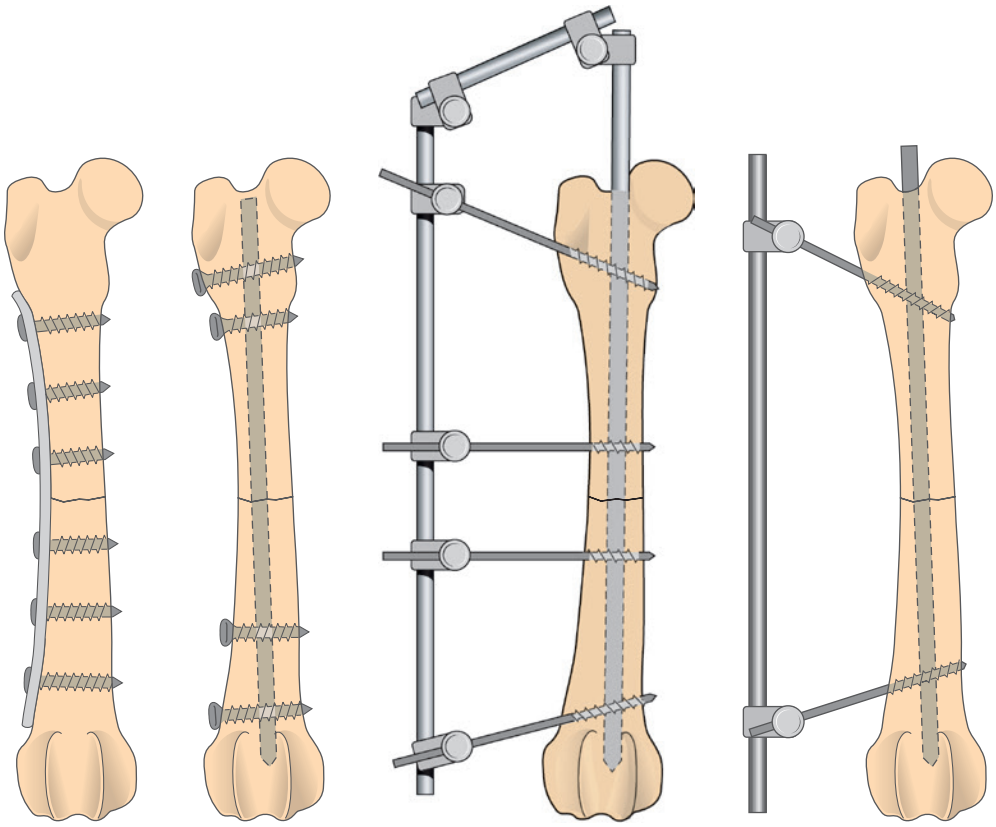


FIG. 33.88 Recommended methods for stabilizing transverse or short oblique femoral fractures based on fracture assessment score. If fracture assessment score is 0 to 3, a compression plate or interlocking nail may be used. If fracture assessment score is 4 to 7, a compression plate, an interlocking nail, or a four-pin external skeletal fixator plus intramedullary (IM) pin (tie-in configuration) may be used. With fracture assessment scores of 8 to 10, an IM pin plus a two-pin external skeletal fixator provides necessary stability. (Third figure modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat.* St. Louis; Elsevier; 2005.)

Fixation systems that are useful with a low fracture assessment score include a bone plate and screws inserted to function as a compression plate or an interlocking nail. With a moderate fracture assessment score, a compression plate, an interlocking nail, or an IM pin tied in with a type Ia external skeletal fixator would be functional. Transverse or short oblique fractures are common in puppies and kittens because their owners often step on them. These patients have a high fracture assessment score and can be stabilized with an IM pin with a two-pin type Ia external skeletal fixator (Fig. 33.88). Alternatively, flexible plating with a veterinary cuttable plate is an option in immature dogs (see p. 1016).

Stabilization of Midshaft Long Oblique Fractures or Comminuted Fractures With One or Two Large Butterfly Fragments

These fractures can be anatomically reduced and interfragmentary compression applied with cerclage wire or lag screws. Axial, rotational, and bending forces generated by weight bearing are neutralized with bone plates, interlocking nails, or IM pins and external fixators.

Fixation systems that are useful with a low fracture assessment score are lag screws combined with neutralization plates. With a moderate fracture assessment score, lag screws or cerclage wire combined with a neutralization plate, or cerclage wire combined with an interlocking nail or an IM pin tied into an external fixator, may be used. With a high fracture assessment score, an IM pin combined with cerclage wire for interfragmentary compression is a useful method of stabilizing the fracture (Fig. 33.89).

Stabilization of Midshaft Comminuted Fractures With Multiple Fragments

These fractures are realigned using indirect reduction techniques. Because no load sharing occurs between the implant and the bone until biological callus forms to provide support, these fractures need rigid axial, rotational, and bending support. Very high stresses will be imposed on the implant and its connection to the bone. If the biological assessment is favorable, the imposed stresses will be of short duration, reducing the likelihood of implant failure. If the biological assessment is not favorable, however, imposed stresses will act on the implant for an extended period, making implant failure more likely. Enhancing the biological response by applying the concept of bridging osteosynthesis (see p. 986) and inserting an autogenous cancellous bone graft (see p. 991) is recommended.

Fixation systems useful in managing patients with a low fracture assessment score include a bone plate-pin combination. Patients with a moderate fracture assessment score may be treated with a bone plate functioning as a bridging plate with an IM pin or an interlocking nail. An external skeletal fixator, with a tie-in to the IM pin, may also be used. A patient with this type of fracture would have a high fracture assessment score only when the biological assessment is extremely favorable (e.g., 4- to 5-month-old animal with a closed, single-limb injury). These animals may be treated with bridging osteosynthesis with an IM

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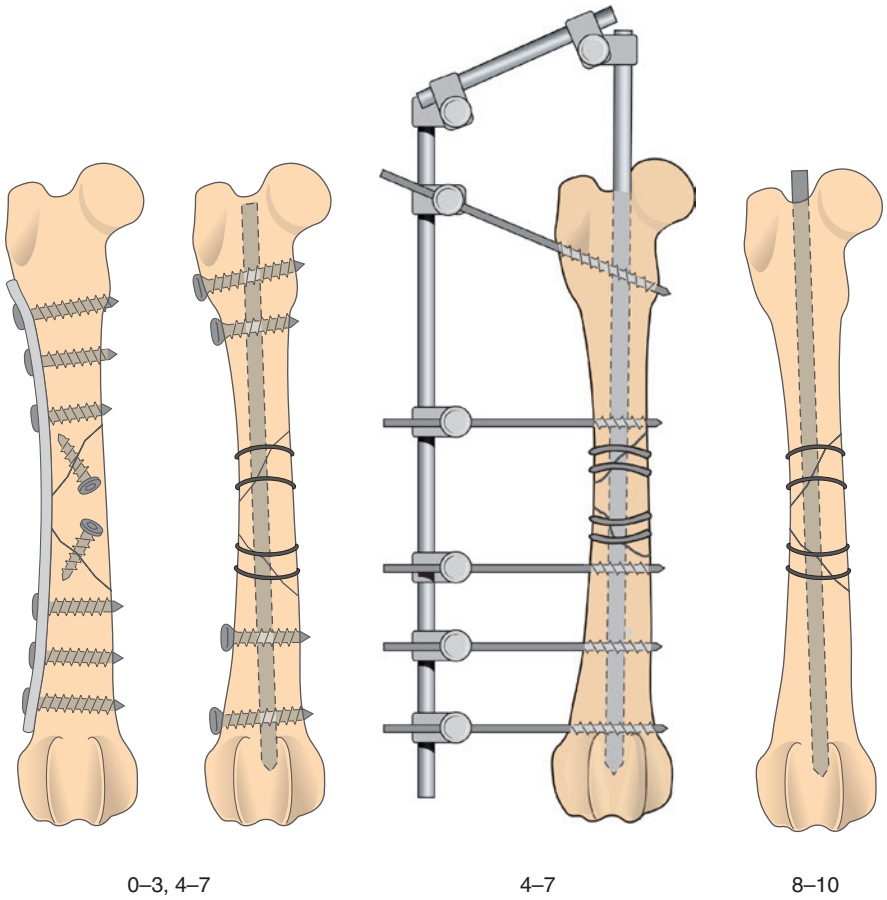


FIG. 33.89 Recommended methods for stabilizing long oblique fractures or reducible comminuted femoral fractures (single large fragment) based on fracture assessment score. If fracture assessment score is 0 to 3, a neutralization plate with lag screws or an interlocking nail with cerclage wire may be used. If fracture assessment score is 4 to 7, a neutralization plate with lag screws, an interlocking nail with cerclage wire, or an external skeletal fixator plus intramedullary (IM) pin (tie-in configuration) and cerclage wire may be used. With fracture assessment scores of 8 to 10, an IM pin plus cerclage wire provides necessary stability. (Third figure modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat.* St. Louis: Elsevier; 2005.)

pin, with a tie-in to a type Ia external skeletal fixator (Fig. 33.90). Immature dogs may be treated with elastic plating techniques using a veterinary cuttable plate and screws (see p. 1018).

Stabilization of Supracondylar Fractures

Supracondylar fractures are usually transverse or short oblique fractures. Occasionally, a comminuted fracture with multiple small fragments will be seen. When comminution is present, however, the length of bone involved is usually limited to a small area. Stabilization of supracondylar fractures depends on the fracture configuration and the patient's fracture assessment. Transverse or short oblique fractures require rotational and bending support, whereas comminuted fractures require axial, rotational, and bending support. The caudal curve of the femoral condyles and the likelihood of comminution contribute to the difficulty in stabilizing these fractures. In patients with low and moderate fracture assessment scores, a compression plate is preferred for transverse fractures, and a plate combined with an IM pin is preferred for comminuted fractures. Locking plate technology is useful for treating these fractures (see p. 1018). Hybrid external fixators have been used successfully in dogs and cats with distal diaphyseal or metaphyseal fractures.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Equipment necessary for pin and wire placement includes retractors, Kern bone-holding forceps, reduction forceps, a Jacobs pin chuck, IM pins, K-wires, orthopedic wire, wire cutters, and pin cutters. Additional equipment needed for external fixation includes a drill bit, a drill guide, a low-RPM power drill, and external fixation clamps and bars. The interlocking nail equipment is needed for nail application. Plating equipment and a high-speed drill are necessary for application of plates and screws.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiographs are taken to evaluate alignment, apparatus, and apposition. Postoperative pain management may be indicated (see Boxes 32.1 and 32.2 and Table 32.4). Activity should be restricted to leash walking and physical rehabilitation until the fracture has healed. Physical rehabilitation (see Tables 33.2 and 33.4 and Chapter 11) encourages controlled limb use and optimal limb function after fracture healing. Care must be taken to develop customized protocols for each patient depending on location of the fracture, stability and type of fracture fixation, potential for healing, abilities and attitudes of the patient, and willingness or ability of the client to provide for the animal's care. External fixator management includes daily pin care and pin packing as needed. Radiographs should be taken and evaluated in 6 weeks. External fixators may be destabilized at this time if healing is progressing satisfactorily. Radiographs should be repeated at 6-week intervals until fracture bridging is observed. Time to bone union depends on the patient's fracture assessment. IM pins and ESFs should be removed when healing occurs; cerclage wire, lag screws, interlocking nails, and bone plates generally are not removed unless a problem is associated with their presence.

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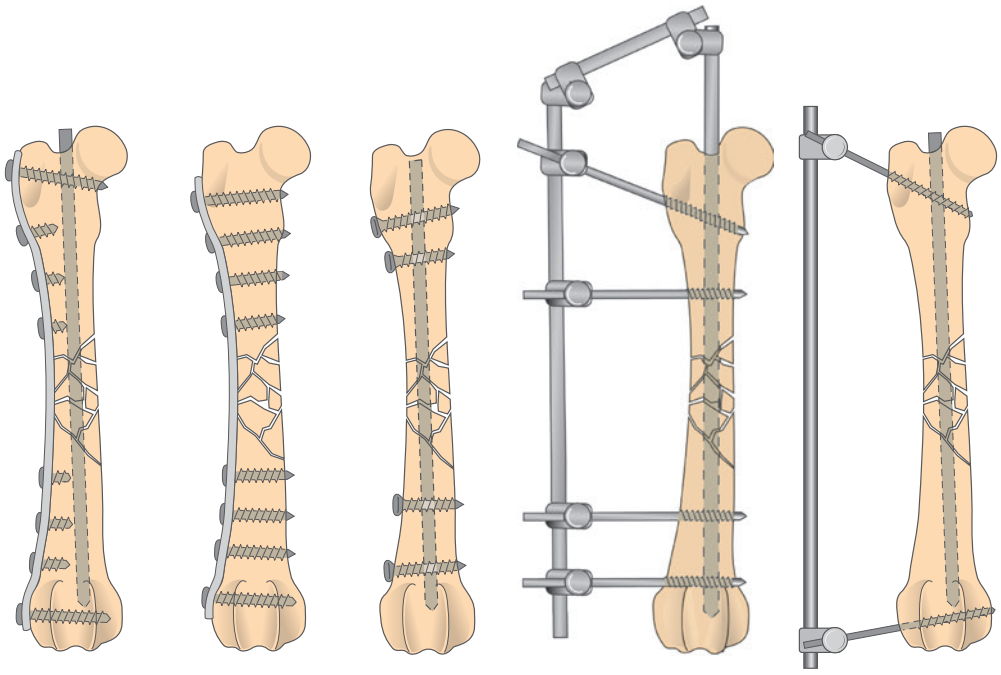


FIG. 33.90 Recommended methods for stabilizing nonreducible comminuted femoral fractures (multiple fragments) based on fracture assessment score. If fracture assessment score is 0 to 3, a plate–intramedullary (IM) pin combination should be used. If fracture assessment score is 4 to 7, a plate–IM pin combination, a bridging plate, an interlocking nail, or an external skeletal fixator plus IM pin (tie-in configuration) may be used. With fracture assessment scores of 8 to 10, an external skeletal fixator plus IM pin provides necessary stability.

COMPLICATIONS

Complications include delayed union, nonunion, malunion, osteomyelitis, pin tract infection, and fixation failure (see p. 1031 for treatment of complications). Sciatic nerve injury occurs with improperly placed IM pins. Premature loosening and migration of IM pins, external skeletal fixator fixation pins, and cerclage wire often results from poor implant choice relative to the fracture assessment (Box 33.15). When inappropriate or improper implants or techniques are chosen, the implant and its bony connection are subjected to excessive stress, which promotes micromotion at the implant-bone interface. Alternatively, the stress may be moderate, but it may occur over time, during which the implant may reasonably be expected to remain stable. In either case, bone resorption and eventual implant loosening result. Breakage of implants can occur through fatigue, most often with bone plates when reduction and stabilization of a zone of comminution with cerclage wire or lag screws are unsuccessful, causing devascularized bone fragments and small fracture gaps. The small gaps are unfavorable for healing and concentrate stress over a small section of the bone plate.

PROGNOSIS

The prognosis is generally excellent if proper fracture management procedures are followed.

FEMORAL METAPHYSICAL AND ARTICULAR FRACTURES

DEFINITIONS

Femoral neck fractures occur at the base of the neck where it joins the metaphysis of the proximal femur. **Articular fractures** involve the joint surface. **Epiphyseal fractures** and **metaphyseal fractures** occur in the trabecular bone at the proximal or distal end of the femur.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Femoral neck fractures are generally single basilar fractures, but comminution of the femoral neck can occur. Mechanically, these are highly unstable fractures because (1) the length of the moment arm acting at the fracture is extensive (entire femoral neck) and (2) the plane of the fracture is along lines of maximal shear stress. Compression of the fracture surface is necessary to resist the high shear stress. The fracture plane is extracapsular, preserving blood flow to the fracture zone after injury. Femoral neck fractures may accompany comminuted proximal femoral fractures.

Articular fractures may occur through the femoral head or the femoral trochlea. Anatomic reconstruction and rigid internal fixation are essential to minimize degenerative joint disease and

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TABLE 33.4 Sample Rehabilitation Protocol for Patient With a Distal Femoral Physeal Fracture

Treatments/ Modalities	Day 1–7	Day 7–21	3–8 Wk	8–12 Wk and Beyond
Pain medications	As directed	As directed	PRN	PRN
Cryotherapy	10–15 min 3 times daily before walks or exercises First session immediately after surger	Use after exercise for 15 min	PRN after exercises	PRN after exercises
Heat therapy		Apply heat to the quadriceps muscles 10 min before PROM or exercise	As before 10 min twice daily	PRN
Massage	Twice daily for edema from toes toward heart	Continue twice daily	Massage quadriceps before active exercise	Massage quadriceps before active exercise
PROM	Gentle stifle flexion/ extension 10 reps, 3–4 times daily Elicit flexor reflex; stifle flexion is very important	Bicycling, stifle flexion/ extension mild resistance 10–15 reps 3 times daily	Continue PROM and bicycling as previously described up to 4 wk	
Laser therapy	Daily or every other day for first week	Every other day for first week then twice weekly	PRN	
Walks	At 5 d start slow controlled leash walks for 5 min twice daily to encourage active stifle ROM	Increase each walk by 2–3 min each week	Add small incline walking and gradually increase to 10–15 min twice daily	20- to 30-min walks twice daily including 10 min of incline work
Balancing	Gentle balancing exercises on foam pad to encourage weight bearing and active stifle ROM 5 min twice daily	5 min 3 times daily After 10 d increase the instability of the surface to challenge the muscles	10 min 2–3 times daily Consider balancing on a ball or inflatable disc with assisted support	Incorporate challenging surfaces to increase stifle flexion: sand, snow, tall grass
Underwater treadmill[a]		10–15 min once daily after day 10—incision healed	15–20 min daily	20–30 min 3 times weekly
Sit to stand[a]			Likely 6+ wk after surgery, 10-20 reps twice daily	10–20 reps twice daily
Stairs[a]			Likely 6+ wk after surgery Start with one flight and add one flight per week	Work up to 5 flights twice daily
Cavalettis[a]			5–10 reps over 5 rails once daily	10–15 reps over 5 rails twice daily
Swimming[a]			10–15 min with breaks daily	20–30 min once to twice daily

For first 2 to 4 days after surgery, dog's leg may be bandaged in a 90-degree/90-degree stifle tarsal flexion sling or bandage between rehabilitation exercise sessions. This is to prevent stifle extension and quadriceps contracture.
[a]Should only be done after radiographic evidence of healing has occurred and implant integrity is verified by surgeon.

BOX 33.15 Common Errors in Femoral Diaphyseal Fracture Fixation

- Failure to provide adequate rotational stability may lead to delayed union and nonunion, even in young animals.
- Using a single IM pin to stabilize a femoral diaphyseal fracture results in fracture instability and implant migration.
- Failure to appropriately reduce rotational malalignment may lead to hip or stifle subluxation, osteoarthritis, and gait abnormality.
- Attempting to reconstruct nonreducible fractures destroys the biological environment and delays healing, contributing to implant failure.

maximize function. Physical rehabilitation is also essential for restoring joint function.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, breed, or sex of dog or cat may be affected. Femoral head and neck fractures occur more often in mature patients after the femoral capital physeal growth plate has closed. Trochlear fractures are uncommon.

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History

Most injuries result from motor vehicle accidents, but some are caused by falls.

Physical Examination Findings

Most affected animals are brought in for evaluation of non–weight-bearing lameness. Pain and crepitation are evident on manipulation of the hip joint or stifle. The stifle is swollen and unstable when a trochlear fracture is present.

Diagnostic Imaging

Femoral head and neck fractures do not require special radiographic views for detection. If only a lateral view of the hip joint is taken, however, the diagnosis may be missed. Femoral trochlear fractures are diagnosed with standard craniocaudal or caudocranial and lateral views of the femur. Because of the manipulation necessary for diagnostic radiographs, some animals may require sedation (see Tables 31.2 and 31.3).

Laboratory Findings

Complete blood count and serum chemistry evaluation should be done to assess anesthetic risk in an animal sustaining trauma. Consistent laboratory abnormalities are not present.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include coxofemoral luxation, acetabular fracture, proximal femoral fracture, and capital physeal fracture in young patients. Differential diagnosis for trochlear fractures includes deranged stifle and tumor.

MEDICAL MANAGEMENT

Medical or conservative management is not a treatment option. Surgical intervention is required for optimal outcome.

SURGICAL TREATMENT

Femoral head and neck fractures with a single fracture plane are best treated with a lag screw and K-wires. If irreparable comminution is present, total hip replacement and femoral head and neck ostectomy are treatment options. If financial restraints preclude fracture repair, a femoral head and neck ostectomy may be performed (see p. 1216). Trochlear fractures are reconstructed with lag screws. Comminuted fractures are supported with a buttress plate.

Preoperative Management

Because femoral head, neck, and trochlear fractures occur secondary to trauma, all affected animals should be examined for concurrent injury and stabilized if necessary before surgery. Analgesics should be provided to posttraumatic animals (see Chapter 13).

Anesthesia

Refer to Tables 32.1 and 32.2 for anesthetic management of patients with fractures.

Surgical Anatomy

The femoral neck–femoral shaft junction in the frontal plane is known as the *angle of inclination*. This angle is normally 135 degrees and should be approximated when surgical reduction is performed. The normal angle of anteversion is 15 to 20 degrees. This angle must be taken into consideration when screws or pins are inserted into the femoral neck.

Normal anatomy of the hip joint is described on p. 1214. A craniolateral approach is performed for exposure of these fractures (Fig. 33.91). Care must be taken to ensure adequate ventral reflection of the vastus lateralis muscle to visualize the fracture surface. The femoral shaft is located craniodorsal to the femoral head and neck, which remain in the acetabulum. Adequate longitudinal incision of the joint capsule is necessary for accurate reduction. A greater trochanteric osteotomy may be required (see Fig. 33.80) if visualization is not adequate for anatomic reduction and placement of implants.

Surgical anatomy of the distal femur is described on p. 1103. A craniolateral parapatellar approach is frequently used to expose the distal femur (Fig. 33.92). Fractures of the medial condyle should be approached medially, and a tibial tuberosity osteotomy may be indicated for additional exposure of comminuted trochlear fractures.

Positioning

The animal is usually positioned in lateral recumbency with the affected leg up. A hanging-leg preparation will facilitate manipulation of the limb during surgery. Positioning the animal in dorsal recumbency facilitates exposure of the medial portion of the stifle.

SURGICAL TECHNIQUE

Femoral Head and Neck Fractures

A craniolateral approach to the hip joint (as for capital physeal fractures) is most often used (see Fig. 33.91). If alignment of the fracture is difficult, a trochanteric osteotomy can be performed to improve accessibility (see Fig. 33.80). Unless the biological assessment is extremely favorable (allowing K-wires to be used), femoral head and neck fractures are best stabilized with lag screws.

Stabilization of an Avulsion Fracture of the Femoral Head With a Lag Screw

Visualize the fracture fragment, and determine whether it is large enough to repair. Reduce the fragment and maintain its position with pointed reduction forceps. Drill a glide hole through the fragment. Drill a thread hole into the femoral head. Use the countersink to seat the screw head below the level of the articular cartilage. Measure, tap the thread hole, and insert the screw. If the fragment cannot be repaired, consider a femoral head and neck ostectomy.

Stabilization of a Femoral Neck Fracture With a Partially Threaded Cancellous Lag Screw

Place two K-wires so that they lie at the most proximal and distal level of the fracture surface (Fig. 33.93A). Drive the wires from medial to lateral, beginning at the fracture surface, or from the lateral surface medially to exit at the fracture surface. Reduce the fracture, and drive the K-wires into the femoral head. Take care to avoid penetrating the articular surface. Drill a thread hole through the femoral epiphysis with a drill bit of appropriate size parallel to and centered between the K-wires. Measure the length of screw needed, and tap the thread hole. Insert a partially threaded cancellous screw 2 mm shorter than the length measured so that all threads cross the fracture plane and are seated into the femoral head. Leave

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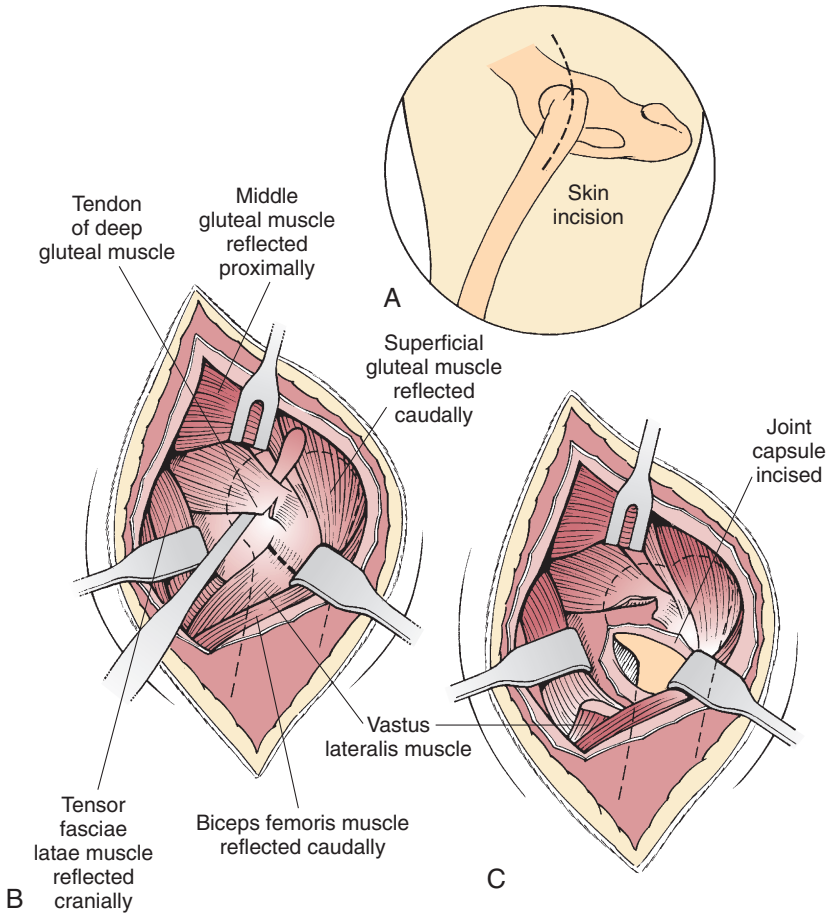


FIG. 33.91 (A) To perform a craniolateral exposure of the hip joint, incise skin 5 cm proximal to the greater trochanter. Curve the incision distally adjacent to the cranial ridge of the trochanter, and extend it distally for 5 cm over the proximal femur. (B) Reflect the tensor fasciae latae muscle cranially and the superficial gluteal and biceps femoris muscles caudally. Incise the deep gluteal tendon for one-third to one-half of its width at its point of insertion into the greater trochanter. (C) Incise the joint capsule and the origin of the vastus lateralis muscle to expose the hip joint.

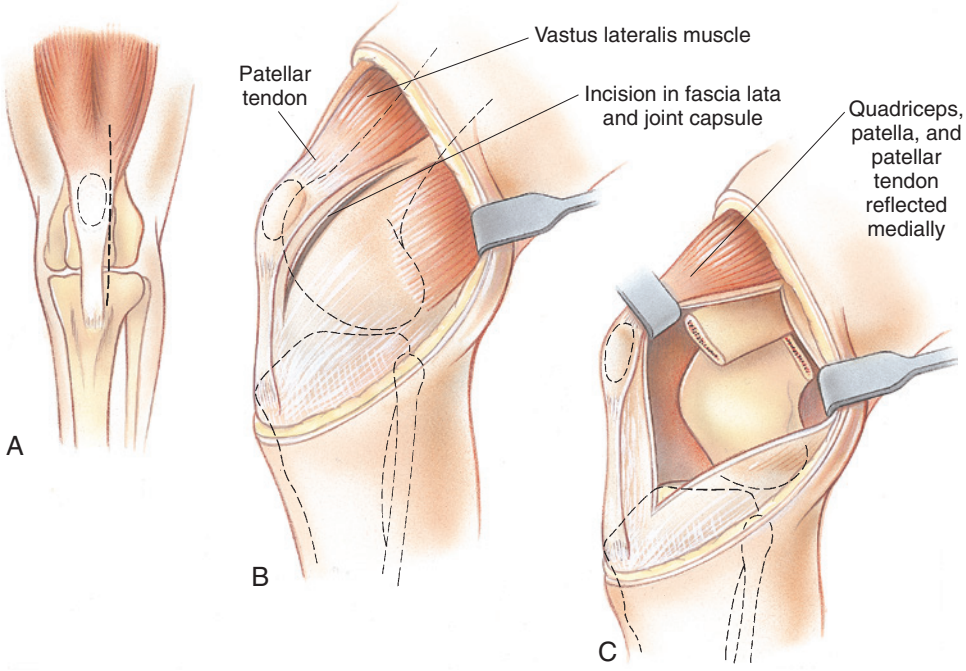


FIG. 33.92 (A) To expose the distal femur, make an incision on the craniolateral surface of the stifle joint centered over the palpable femoral shaft. (B) Create a parapatellar arthrotomy through the distal fascia lata and joint capsule. (C) Reflect the quadriceps muscles, patella, and patellar tendon medially to expose the articular surface of the femoral condyles.

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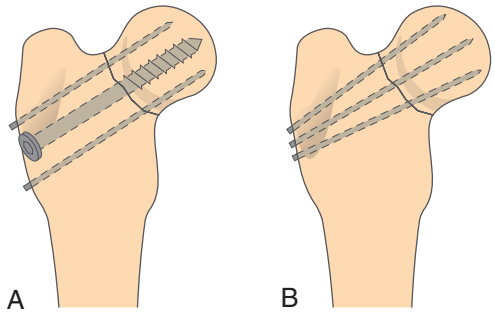


FIG. 33.93 Femoral neck fracture repair. (A) A lag screw is centered between two K-wires. (B) Triangulated K-wires.

one or both wires in place to serve as antirotational devices. To stabilize a femoral neck fracture with a cortex bone screw used as a lag screw, drill the glide hole through the femoral neck with a drill bit equal to the diameter of the threads of the screw.

Stabilization of a Femoral Neck Fracture With Triangulated K-wires

Insert three K-wires from the fracture surface with the wires parallel to one another to form a triangle (Fig. 33.93B). Retrograde the wires to exit the bone near the third trochanter. Alternatively, normograde the wires so that they enter the bone at the third trochanter and exit at the fracture site. Reduce the fracture, and drive the wires into the femoral epiphysis. Take care not to penetrate the articular surface.

Stabilization of Intertrochanteric Fractures

Intertrochanteric fractures are often complex and involve the femoral neck, greater trochanter, and proximal femoral metaphysis. The method of fixation depends on the fracture assessment score (see p. 985) but includes lag screw stabilization of the femoral neck with some method of fixation applied to the proximal femur. The exception is the immature patient with a favorable biological assessment, in whom multiple small pins can be used in the femoral neck and femoral metaphysis. In adult patients, IM pins are not used because the screw traverses the proximal metaphysis and does not allow placement of an IM pin. The preferred method of stabilization in adult patients is a bone plate. If the metaphyseal fracture is a transverse or short oblique fracture, the plate functions as a compression plate. If the area of comminution is reconstructed with cerclage wire and lag screws, the reconstruction can be protected with a neutralization plate. Alternatively, the area of comminution can be bridged with a buttress plate without manipulating the fracture fragments. The lag screw used for stabilization of the femoral neck fracture can be placed through a plate hole or offset caudally to the plate.

Stabilization of a Unicondylar Fracture With a Lag Screw

Reduce the fracture and temporarily stabilize it with pointed reduction forceps and a K-wire. Prepare for and insert a lag screw starting just proximal to the trochlear ridge on the same or opposite side of the femur depending on the fracture line orientation (Fig. 33.94A). Orient the screw perpendicular to the fracture line for optimal compression and to prevent the fragment from shifting as the screw is tightened. The K-wire may be removed or left in place for rotational stability.

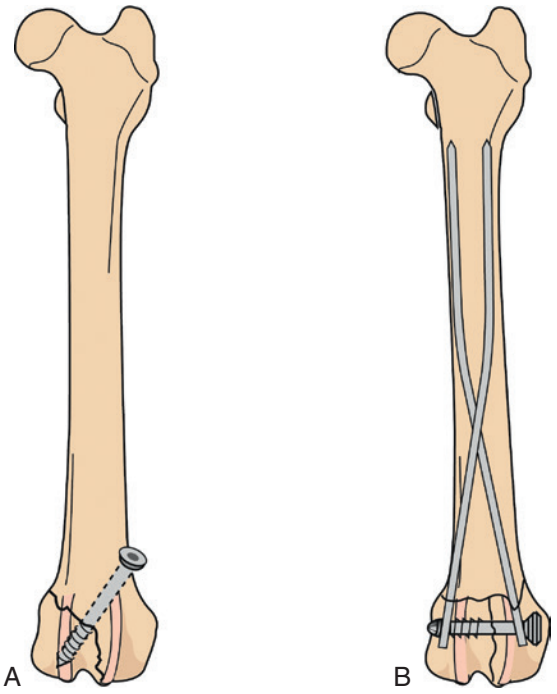


FIG. 33.94 (A) Stabilization of a femoral unicondylar fracture may be done with a lag screw. (B) Repairing a bicondylar fracture requires stabilization of the condyles with a lag screw. The condyles are then attached to the shaft of the femur with Steinmann pins or a bone plate. (Modified from Johnson AL, Houlton JEF, Vannini R, eds. *AO Principles of Fracture Management in the Dog and Cat.* New York: Thieme; 2005.)

Stabilization of a Bicondylar Fracture With a Lag Screw, and With Steinmann Pins or a Bone Plate

Reduce the femoral condyles, and apply compression with a lag screw placed across the fracture. After condylar reduction and stabilization, insert two small Steinmann pins as described for stabilization of Salter I and II fractures (Fig. 33.94B; see also p. 984). If the animal is approaching maturity, stabilize with a reconstruction plate on the lateral surface.

Stabilization of Comminuted Distal Femoral Fractures

Comminuted distal femoral fractures often involve the trochlea and the articular surface of the femoral condyles. Visualization of the fracture planes is necessary to achieve the anatomic reduction and rigid fixation required with articular fractures. Surgical exposure is best accomplished through a combination of the standard approach for osteotomy of the tibial crest and a medial arthrotomy. Proximal reflection of the patellar tendon, patella, and quadriceps muscle group allows excellent visualization. *Reconstruct the articular surface using a combination of lag screws and K-wires. Once the articular fractures have been reconstructed, reduce the femoral condyles and stabilize them with a reconstruction plate. Contour the reconstruction plate to the lateral surface of the distal femur and femoral condyles.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Appropriate surgical instrumentation for application of K-wires or lag screws and plates is needed. Helpful instruments include

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a battery-powered or air-driven drill with a wire-driving attachment, self-retaining retractors, pointed reduction forceps, and wire cutters.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiographs are taken to evaluate alignment, apparatus, and apposition. Postoperative pain management may be indicated (see Boxes 32.1 and 32.2 and Table 32.4). Activity should be restricted to leash walking and physical rehabilitation until the fracture has healed. Physical rehabilitation (see Table 33.4 and Chapter 11) encourages controlled limb use and optimal limb function after fracture healing and is especially important after fractures affecting the stifle. Care must be taken to develop customized protocols for each patient depending on location of the fracture, stability and type of fracture fixation, potential for healing, abilities and attitudes of the patient, and willingness or ability of the client to provide for the animal's care. Radiographs should be repeated at 6-week intervals until fracture bridging is observed. Femoral metaphyseal and articular fractures may take 6 to 12 weeks to heal depending on the biological fracture assessment. Implants are generally not removed unless they cause a problem.

COMPLICATIONS

Inappropriate reduction and poor implant choice are the most common problems reported with femoral neck fractures. Significant bending and shear stress across the fracture plane place extreme bending loads on implants. The most common implant error is the use of K-wires or small pins when the fracture assessment indicates prolonged healing. Micromotion at the pin-bone interface arising from high physiologic stress may cause pins to loosen early. This problem can be avoided or treated by using a lag screw and an antirotational pin except when the biological assessment indicates rapid healing. The animal with a femoral neck fracture that fails to heal is usually treated with a femoral head and neck ostectomy. Intraarticular fractures may result in postoperative degenerative joint disease, although this is minimized with careful reduction and rigid fixation.

PROGNOSIS

The prognosis is generally good if proper fracture management procedures are followed. Femoral condylar fractures generally heal rapidly because they are located in cancellous bone and generally occur in young animals.

FEMORAL PHYSEAL FRACTURE

DEFINITIONS

Articular fractures involve the joint surface. **Physeal fractures** involve the growth plates in immature animals. The **capital physis** is the proximal femoral physis of the femoral head.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Because femoral physeal fracture occurs through cartilage of the growth plate, capital physeal injuries may occur without significant trauma. This is particularly true in young heavy male cats that have been neutered before 6 months of age. Delayed physeal closure and cartilage abnormalities may increase the susceptibility of these cats to physeal fracture (Fig. 33.95). The femoral neck is usually externally rotated and displaced craniodorsally such that it lies adjacent to the iliac wing. The physis of the greater trochanter may also be fractured, causing the femoral shaft to be displaced more dorsally than expected. Proximal physeal fractures are generally Salter I or Salter II fractures (see p. 984). Distal femoral physeal fractures are generally Salter II physeal fractures.



FIG. 33.95 Ventrodorsal radiograph of young cat with bilateral capital physeal fractures.

The capital physis functions to provide femoral neck length until the animal is approximately 8 months of age. The distal physis functions to provide most of the femoral length. In most fractures, the growth cartilage is damaged by the traumatic incident, in the posttraumatic period, or during surgery. Physeal fractures heal rapidly, but most of the time, the physis does not continue to function. The younger the animal, the more dramatic are the effects of premature closure of the injured physis.

DIAGNOSIS

Clinical Presentation

Signalment

Most affected animals are younger than 10 months. Young male dogs are more likely to sustain trauma resulting in femoral physeal fracture, probably because of their tendency to roam. Young heavy male cats that have been neutered before 6 months of age are also at risk.

History

Most animals are presented for evaluation of acute non–weight-bearing lameness. The trauma may or may not be witnessed by

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the owner. Femoral physeal fractures are often caused by motor vehicular accidents. However, minor trauma such as a fall may be sufficient to separate the growth plate.

Physical Examination Findings

Animals with proximal physeal fractures usually exhibit non–weight-bearing lameness with pain and crepitation on manipulation of the hip joint. Some animals are weight-bearing and do not have detectable crepitus referable to the hip joint. These animals usually have minimal displacement of the femoral head. Animals with distal femoral physeal fractures present with swelling, pain, and crepitus on manipulation of the stifle region.

Diagnostic Imaging

Standard ventrodorsal and mediolateral projections of the femur are required to confirm the diagnosis. Some animals may require sedation (see Tables 31.2 and 31.3). A fractured capital physis with minimal displacement may be difficult to detect using an extended limb ventrodorsal radiographic projection. A ventrodorsal view with the limbs in "frog position" may help confirm the diagnosis in such cases. If a greater trochanteric physeal fracture is also present, the cap of the greater trochanter will be superimposed over the shaft of the femur and will appear as a half-moon radiodense object on a ventrodorsal radiograph. On a lateral projection, the cap of the trochanter will appear as a radiodense fragment caudal to the femur. With distal physeal fractures, the femoral shaft is displaced cranially and distally and may be superimposed over the femoral condyles. If the displacement is slight, the fracture may be missed when a single craniocaudal radiograph is taken. Additional oblique and skyline projections are useful in evaluating the articular surface of the trochlea and femoral condyles if fissures or fractures of these structures are suspected.

NOTE Superimposition may be such that the fracture could be missed with a single radiograph. Always take two orthogonal views.

Laboratory Findings

Complete blood count and serum chemistry evaluation should be done to evaluate the status of the animal for anesthesia and to determine whether concurrent injury or damage to the renal or hepatobiliary systems exists. Consistent laboratory abnormalities are not present.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for proximal physeal fractures include coxofemoral joint luxation, femoral neck fracture, and acetabular fracture. For distal physeal fractures, the differential diagnosis includes diaphyseal fractures and ligamentous injury of the stifle.

MEDICAL MANAGEMENT

Surgical intervention is required to prevent severe degenerative joint disease and lameness.

SURGICAL TREATMENT

Surgical treatment of physeal fractures consists of anatomic reduction and stabilization with K-wires or small pins that are smooth so as to not interfere with any residual physeal function. These fractures heal rapidly, and smooth implants are generally sufficient. In animals that are close to maturity, threaded implants may be used to increase the stability of the fixation. Anatomic reduction is critical for optimal outcome with capital physeal fractures. Mechanically, prevention of movement of the reduced capital physeal and distal physeal fractures is assisted by the shape of the fractured physeal surfaces. If separated, the physis of the greater trochanter must also be anatomically reduced and stabilized with a tension band to counteract the distractive forces of the gluteal muscles.

Preoperative Management

Because these fractures occur secondary to trauma, all affected animals should be examined for concurrent injury and stabilized if necessary before surgery. Analgesics should be provided to posttraumatic animals (see Chapter 13).

Anesthesia

Refer to Tables 32.1 and 32.2 for anesthetic management of patients with fractures.

Surgical Anatomy

The proximal capital physis lies between the femoral epiphysis and the femoral neck and acts as a barrier for passage of blood vessels from the femoral neck to the femoral epiphysis. The blood supply to the femoral epiphysis involves a series of cervical ascending vessels lying outside the femoral neck that cross the physis and then penetrate the epiphysis. See p. 2014 for surgical anatomy of the hip.

The distal femoral growth plate is W-shaped and lies at the joint capsule reflection. The configuration of the growth plate and the cancellous bone surface provide a degree of inherent stability for the fracture. The position of the growth plate necessitates an arthrotomy incision to facilitate exposure (see p. 1233 for surgical anatomy of the stifle).

Positioning

The animal is positioned in lateral recumbency with the affected limb up. The limb should be clipped circumferentially from dorsal midline to distal tibia (distal physeal fractures) and draped from a hanging position to allow maximal manipulation during surgery.

SURGICAL TECHNIQUE

Surgical Approach to the Proximal Femur and Hip Joint

Incise the skin 5 cm proximal to the greater trochanter. Curve the incision distally adjacent to the cranial ridge of the trochanter, and extend it distally for 5 cm over the proximal femur (see Fig. 33.91A). Incise subcutaneous tissue and the juncture at the superficial leaf of the fascia lata and the cranial border of the biceps femoris muscle. Incise the deep leaf of the tensor fascia lata between the tensor fasciae latae muscle and the deep border of the biceps femoris muscle and the superficial gluteal muscle. Reflect the tensor fasciae latae muscles cranially and the superficial gluteal and biceps femoris muscles caudally (see Fig. 33.91B). Visualize the tendinous insertion of the deep gluteal muscle by retracting the middle gluteal muscle proximally. Place a periosteal elevator beneath the deep gluteal muscle near its insertion, and separate the deep gluteal muscle from the joint capsule using a sweeping motion with the elevator. Incise the deep gluteal tendon for one-third to one-half of its width

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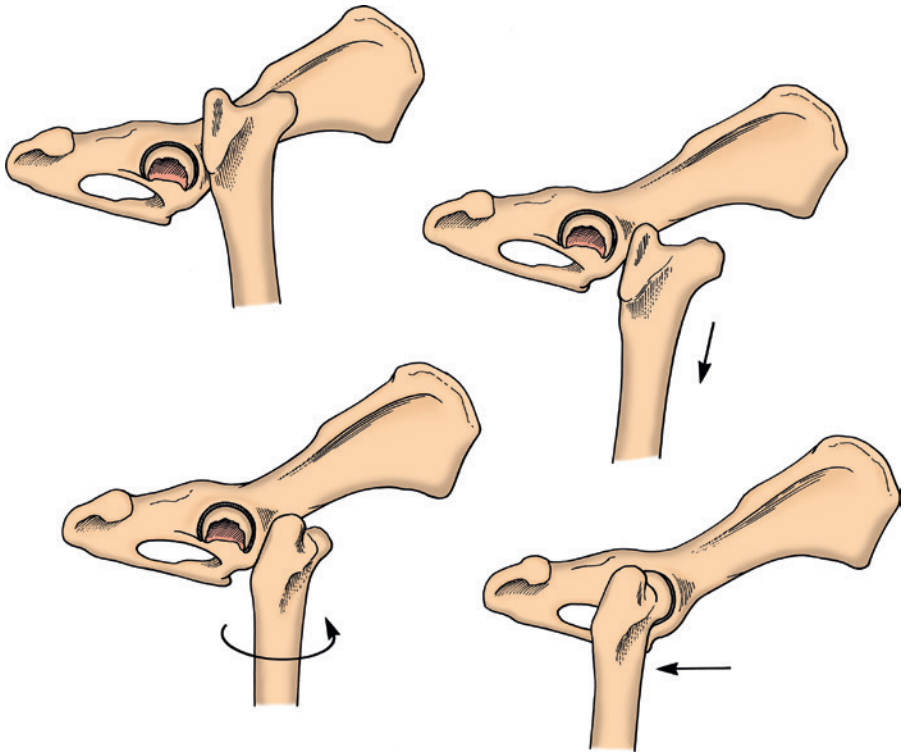


FIG. 33.96 To reduce a capital physeal fracture, bring the femoral neck ventral until it is level with the hip joint. Then derotate the femur and slide it caudally.

at its point of insertion onto the greater trochanter (see Fig. 33.91C). Leave 1 to 2 mm of tendon on the trochanter for closure, but make the incision through the tendon close to the bone. If the joint capsule is lacerated, exposing the fracture surface of the femoral neck, enlarge the opening in the joint capsule with an incision from the rim of the acetabulum laterally through the point of origin of the vastus lateralis muscle. If the joint capsule is intact, incise it parallel to the long axis of the femoral neck near its proximal ridge. Continue the joint capsule incision laterally through the point of origin of the vastus lateralis muscle on the cranial face of the proximal femur. It is important to keep this cut at the proximal point of origin just under the cut edge of the deep gluteal tendon. *Reflect the vastus lateralis distally to expose the hip joint. To reduce the proximal femoral physeal fracture, (1) bring the femoral neck distally so it lies cranial and level with the acetabulum, (2) derotate the femur to correct for the abnormal anteversion, and (3) slide the fracture surface of the femoral neck caudally into the matching surface of the femoral epiphysis (Fig. 33.96).*

Surgical Approach to the Distal Femur

The most recognizable structure is often the palpable distal end of the femoral shaft, which serves as the center of the incision. *Make an incision on the craniolateral surface of the stifle joint centered over the palpable femoral shaft. Begin the incision 4 to 5 cm proximal to the center point and extend it 4 to 5 cm distally (see Fig. 33.92A). Incise the subcutaneous tissue along the line, and identify the fascia lata and patellar tendon. Create a parapatellar arthrotomy through the distal fascia lata and joint capsule (see Fig. 33.92B). Make the incision along the caudal border of the vastus lateralis muscle through the intermuscular septum of the fascia lata.* Most often the distal femoral metaphysis lies cranial and lateral to the femoral condyles and is exposed while incising through the joint capsule and fascia lata (see Fig. 33.92C). *Reflect the quadriceps muscles, patella, and patellar tendon medially to expose the articular surface of the femoral condyles. To reduce the distal femoral fracture, lever the condyles cranially and distally with a "spoon" Hohmann retractor placed between the fracture fragments.*

Stabilization of Proximal Femoral Physeal Fracture With Triangulated K-wires

Place three K-wires parallel to one another and positioned in the femoral neck so that they lie in a triangle. Two wires are sufficient in cats. *Insert the wires from the lateral aspect of the femur paralleling the angle of the femoral neck; the points of the wires should be just visible at the fracture surface (Fig. 33.97A). Reduce the fracture by securing the proximal femur with a reduction forceps and maneuvering it into position. Hold the reduction by pressing against the fragment in the acetabulum, and drive the wires into the femoral epiphysis (Fig. 33.97B).* A common problem with this technique is that the pins penetrating the articular cartilage are not visible to the surgeon at surgery. The exception is the feline femoral head, which may be distracted enough to view the articular cartilage. The femoral head is a domed structure, so the length of pin placed in the periphery will be different from the length of pin placed in the center of the dome. *To determine the proper length of wires, place the first wire in the periphery of the femoral epiphysis so that it penetrates the articular cartilage where it is visible. Estimate the length of the wire that would not penetrate the articular cartilage, and use it as a gauge for the remaining wires. Alternatively, estimate the distance into the epiphysis and mark one wire with a hemostat to provide a guide. Secure an adjacent wire in the pin driver at the estimated epiphyseal distance from the hemostat. Drive the pin until the pin driver is adjacent to the hemostat. Drive the remaining wires into the femoral epiphysis using a similar guiding process. Drive the remaining wires into the femoral epiphysis, and place the joint through a normal range of motion after each wire to ensure that*

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it has not penetrated the articular surface. Bend the wires at the lateral surface and cut off the excess. Close the incision routinely.

Stabilization of Proximal Femoral Physeal Fracture With Lag Screw

Initially, place two K-wires in the femoral neck in a lateral-to-medial direction so that they lie parallel to one another. Position them so that one wire lies in the dorsal section of the femoral neck and one wire lies in the ventral section of the femoral neck. Drill a glide hole between the two wires to emerge at the center of the fracture surface (Fig. 33.98A). Angle the K-wires and glide hole to correct for normal anteversion of the femoral neck. Reduce the fracture, and drive the K-wires into the femoral epiphysis. Place a drill insert into the glide hole for accurate drilling of the thread hole into the femoral epiphysis (Fig. 33.98B). Countersink the glide hole, and determine the appropriate screw length by measuring the distance from the lateral surface of the greater trochanter to the femoral epiphyseal articular surface. Choose a screw that is 2 mm shorter than that measured with the depth gauge. Tap the thread hole and insert the screw (Fig. 33.98C). The wires may be left to provide additional rotational stability. Close the incision routinely.

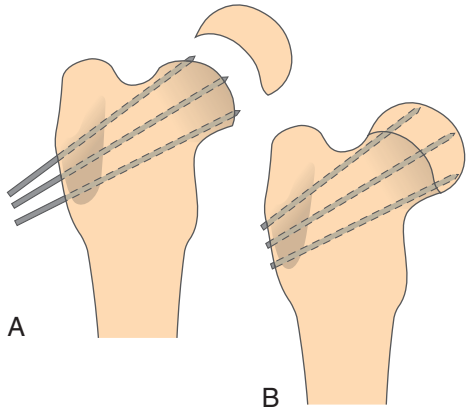


FIG. 33.97 (A) To stabilize a femoral capital physeal fracture with K-wires, place three wires through the femoral neck to the fracture surface. Triangulate the K-wires. (B) Reduce the fracture, and advance the wires into the epiphysis.

Stabilization of Trochanteric Physeal Fracture With Tension Band Wire

Reduce the trochanter, and start two K-wires in the fragment. Drive the wires across the physis to lodge in the proximal femur, and check the repair to see whether stabilization is sufficient to prevent avulsion of the fracture. If not, use a tension band wire, even though it may prevent physeal growth. To place a tension band wire, (1) drill a transverse hole in the major bone segment, (2) pass a figure-eight wire through the hole and around the K-wires, and (3) tighten the wire (see p. 1015 for tension band wire techniques).

Stabilization of Salter I or II Distal Femoral Physeal Fractures With Steinmann Pins

With Salter I or Salter II fractures, Steinmann pins may be used alone or in combination with small K-wires. The pins may be placed in a Rush pin manner, as an IM pin, or as a crossed pin (Fig. 33.99). *Reduce the fracture by levering the condyles cranially and distally with a blunt Hohmann retractor placed between the fracture fragments. Maintain reduction during pin placement by placing a Kern bone-holding forceps on the lateral aspect of the distal femoral metaphysis. Place a pointed reduction forceps from the trochlear surface to the jaws of the Kern on the cranial surface of the femur.*

When using a Steinmann pin as an IM pin, insert the pin through the articular cartilage cranial to the origin of the caudal cruciate ligament. Direct the pin in a normograde fashion across the fracture, proximally into the femur, to exit at the trochanteric fossa. Cut the distal part of the pin and countersink it below the level of the articular cartilage; cut any excessive pin off below the skin above the trochanteric fossa. Use a small-diameter pin so it can bend with the curvature of the femoral canal as it passes proximally. Consider adding a small crossed pin at the fracture to establish rotational stability, as necessary (Fig. 33.100).

When using Steinmann pins as crossed pins, position them so that they enter the epiphysis at a point cranial to the medial and lateral epicondyles, and drive them proximally to a point where they are just visible at the fracture surface. Overreduce the fracture, and drive the pins into the femoral metaphysis and through the cortices. The distal ends of the pins can be countersunk deeper after cutting with a nail set or closed Jacobs chuck and a mallet.

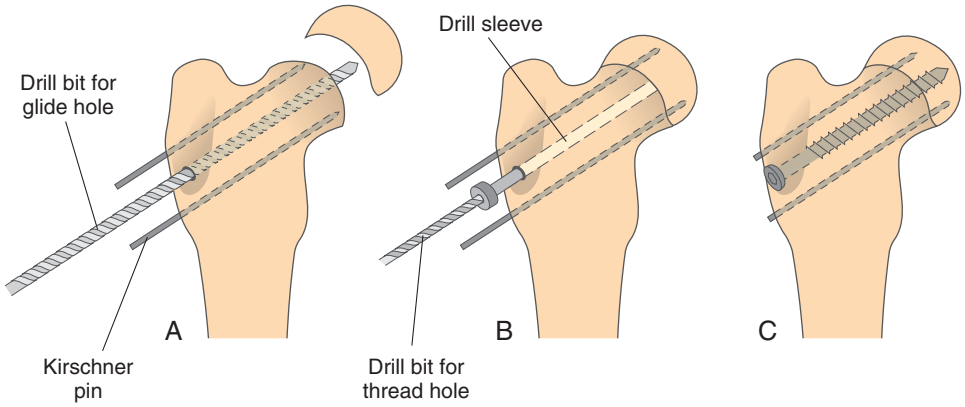


FIG. 33.98 (A) To stabilize a femoral capital physeal fracture with a cortex bone screw used as a lag screw, place two K-wires (one superior and one inferior) in the femoral neck, perpendicular to the fracture surface. Drill a glide hole between the K-wires. (B) Reduce the fracture, and advance the K-wires into the femoral epiphysis. Drill the thread hole in the epiphysis, measure to determine screw length, and tap threads in the epiphysis. (C) Insert the lag screw.

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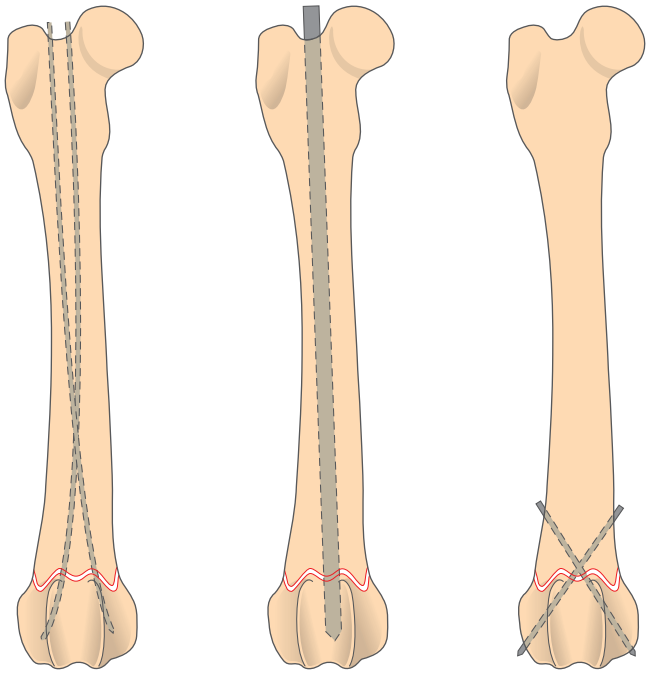


FIG. 33.99 Stabilization of distal femoral physeal fractures may be done with multiple pins placed in Rush fashion, a single intramedullary pin, or crossed pins.

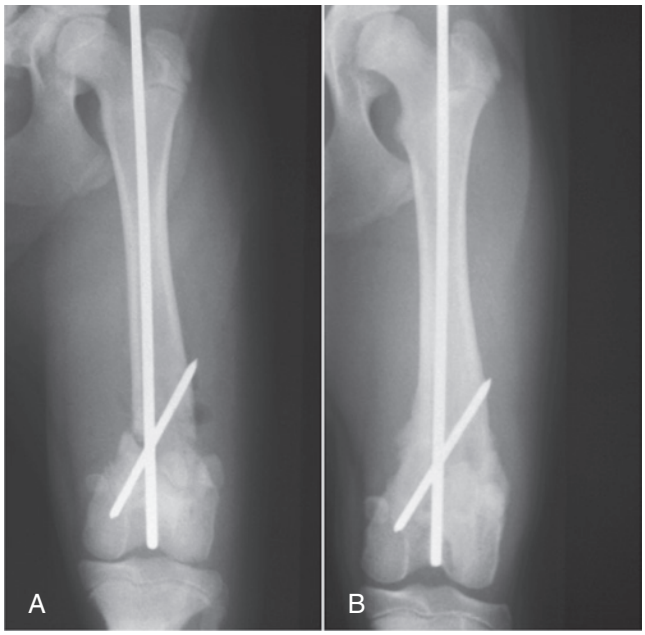


FIG. 33.100 Postoperative radiographs. (A) Salter II fracture through the distal femoral physis stabilized with intramedullary pin and crossed pin. (B) Healed fracture 6 weeks later.

Suture the joint capsule using an interrupted suture pattern. Close subcutaneous tissue and skin routinely.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

A battery-operated or air-driven drill with a wire driver attachment is helpful for inserting K-wires in proximal femoral physeal fractures. Instruments that facilitate fracture reduction include Hohmann retractors, Kern bone-holding forceps, and pointed reduction forceps. Small IM Steinmann pins and an assortment of K-wires are needed.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiographs are taken to evaluate alignment, apparatus, and apposition. Postoperative pain management may be indicated (see Boxes 32.1 and 32.2 and Table 32.4). Activity should be restricted to leash walking and physical rehabilitation until the fracture has healed. Physical rehabilitation (see Table 33.4 and Chapter 11) encourages controlled limb use and optimal limb function after fracture healing and is especially important after fractures affecting the stifle. Care must be taken to develop customized protocols for each patient depending on location of the fracture, stability and type of fracture fixation, potential for healing, abilities and attitudes of the patient, and willingness or ability of the client to provide for the animal's care. Radiographs should be repeated at 4 to 6 weeks (see Fig. 33.100). K-wires and crossed pins are generally not removed unless they cause a problem. IM pins should be removed after the fracture has healed.

COMPLICATIONS

If a proximal physeal fracture is not appropriately reduced, or if implants penetrate the articular cartilage, significant degenerative changes may develop, necessitating additional surgical treatment.

PROGNOSIS

The prognosis for long-term, pain-free function after proximal physeal fracture is good if the fracture is adequately reduced and appropriately stabilized (Fig. 33.101). In animals younger than 5 months, however, a shortened femoral neck may result from closure of the capital physis, which may cause hip joint subluxation and may increase the risk of degenerative joint disease.

For distal physeal fractures, bone union occurs within 4 to 6 weeks. Although growth retardation or premature closure of the distal femoral growth plate is likely, the prognosis for normal limb use is excellent with Salter I or II fractures. The exception is seen when injury occurs at 3 to 5 months of age in a large or giant breed dog, when considerable growth potential is still present.

PATELLAR FRACTURES

DEFINITION

Patellar fractures result from loss of bony and articular continuity between the superior and inferior poles of the patella.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Fractures of the patella may result from direct or indirect trauma but are uncommon. Direct trauma results from an external blow to the cranial surface of the patella. The resulting fracture may consist of a transverse separation midway between the proximal and distal poles, fragmentation of the proximal or distal pole, or comminution of the patellar body. Indirect trauma is caused

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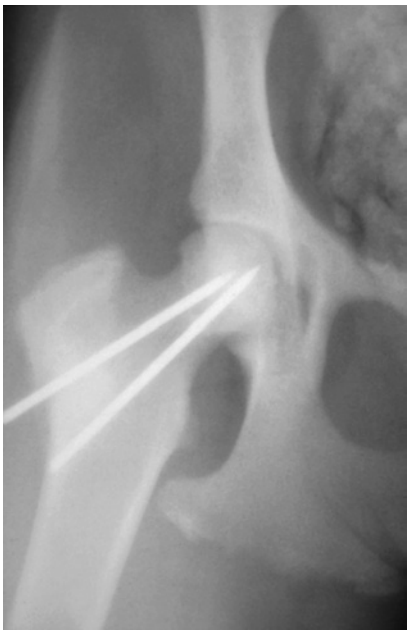


FIG. 33.101 Postoperative radiographs of a proximal femoral physeal fracture stabilized with K-wires 6 weeks after surgery. Note the loss of bone density circumferentially on the femoral neck. This "apple coring" is a frequent postoperative observation but rarely has clinical significance.

by forceful contraction of the quadriceps muscle group, which causes excessive tensile forces to be applied across the body of the patella. These forces result in a transverse fracture midway between the superior and inferior poles. Tearing of the parapatellar fibrocartilage, quadriceps tendon, and straight patellar ligament may also occur. Transverse or comminuted fractures through the patellar body are disabling injuries because they cause loss of quadriceps function and resultant inability to bear weight on the affected limb. Without treatment, the proximal and distal fragments of the patella separate owing to countering forces of the quadriceps muscle and patellar tendon. A fibrous union develops between the fragments and affords some stability, but the support is insufficient to allow normal function. Additionally, loss of articular surface congruity results in degenerative arthritis of the patellar-femoral articulation. Small fragments at the proximal or distal poles of the patella may not be disabling if the integrity of the insertion of the quadriceps muscle group is maintained.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, breed, or sex of dog may be affected. Breeds with congenital myotonia and sporting breeds are most prone to transverse fractures caused by forceful contraction of the quadriceps. Young adult cats may develop transverse stress fractures with minimal trauma; the fractures are often bilateral.

History

Animals with patellar fractures resulting from trauma are usually evaluated because of non–weight-bearing lameness. Owners seldom witness the trauma itself. Indirect trauma usually occurs when the animal is undergoing strenuous activity and acutely becomes non–weight-bearing. Some cats have an insidious onset of lameness.

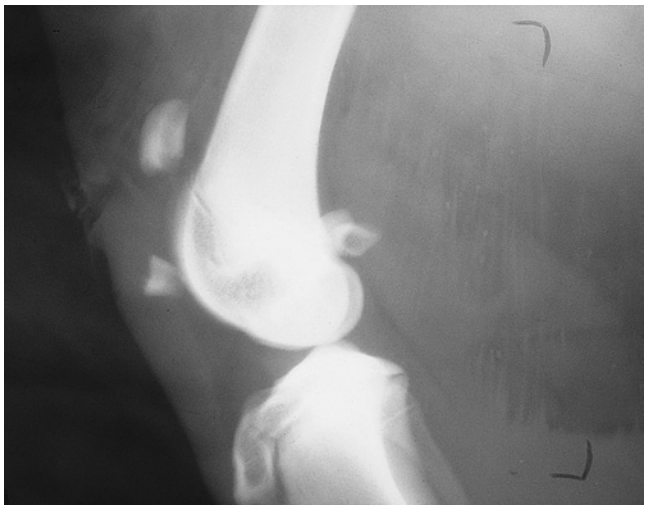


FIG. 33.102 Lateral radiograph of the stifle of a dog with transverse patellar fracture.

Physical Examination Findings

Animals are usually non–weight-bearing on the affected limb. Palpation shows pain and swelling over the cranial surface of the stifle. Crepitation is not usually noted because the fragments separate. Palpation may detect a void in the quadriceps muscle-patellar tendon mechanism.

Diagnostic Imaging

Standard craniocaudal or caudocranial and mediolateral radiographs should be obtained (Fig. 33.102). Many animals require sedation to obtain quality radiographs (see Tables 31.2 and 31.3). Radiographs of the contralateral stifle in cats may reveal sclerosis of the patella, which possibly precedes fracture.

Laboratory Findings

Consistent laboratory abnormalities are not present.

DIFFERENTIAL DIAGNOSIS

Patellar fractures should be differentiated from lacerations or rupture of the patellar tendon resulting in quadriceps insufficiency. This differentiation is made based on the radiographic appearance of the patella.

MEDICAL MANAGEMENT

Conservative management is indicated in cats if minimal displacement of the patellar segments occurs. Conservative management (i.e., rest or passive physical therapy) is not indicated in dogs if a transverse or comminuted fracture has separated the proximal and distal poles of the patella. Fibrous union may occur, but the stability afforded is insufficient to allow normal activity. Small fragments near the proximal or distal pole may be managed conservatively if they do not interfere with motion of the patellofemoral joint.

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SURGICAL TREATMENT

Preoperative Management

The animal should be confined to a cage until surgery is performed. A soft-padded bandage with a lateral splint will help support the stifle. Analgesics should be provided to posttraumatic animals (see Chapter 13).

Anesthesia

Refer to Tables 32.1 and 32.2 for anesthetic management of patients with fractures.

Surgical Anatomy

The patella, the largest sesamoid in the body, is embedded within the tendons of the quadriceps muscle group. The undersurface tracks within the trochlear groove of the femur and is formed of hyaline articular cartilage. The function of the patella is twofold: (1) to maintain straight-line stability during contraction of the quadriceps muscle group and (2) to provide mechanical efficiency for the quadriceps muscle group. During surgery, care must be taken to preserve the insertions of the quadriceps tendon and patellar ligament.

Positioning

The patient is positioned in dorsal recumbency. The limb should be clipped and surgically prepared from the greater trochanter proximally to the tarsus distally. A hanging-leg preparation facilitates manipulation of the limb during surgery.

SURGICAL TECHNIQUE

Make a craniolateral skin incision 1 cm lateral to the patella. Begin the incision 5 cm proximal to the patella and extend it distally to the tibial crest. Incise subcutaneous tissue overlying the patella and the patellar tendon to expose the fragment ends. Place the limb in extension to reduce the fracture, then stabilize the fragments with a tension band placed over the cranial surface of the patella (Fig. 33.103). Because the patella is composed of very dense bone, predrill the holes to facilitate K-wire placement. In small dogs and cats, it may be possible to place only one K-wire. *Place orthopedic wire around the K-wire tips, and tighten it. Visualize the articular surface of the patella to ensure anatomic reduction after tightening the wire.* Circumferential orthopedic wire placed through the patellar tendon and ligament to hold the fragments in apposition may be indicated in cats with stress fractures; the bone may be brittle and subject to fracture when a K-wire is placed.

Comminuted fractures are repaired with a tension band if two fragments are large enough to stabilize. Small fragments are removed. The primary concern is restoring the integrity of the quadriceps mechanism.

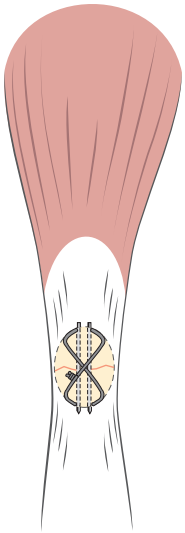


FIG. 33.103 Stabilization of transverse patellar fracture with a tension band wire.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Self-retaining retractors are useful to reflect adjacent soft tissue. Orthopedic wire, small pins, a drill bit, and an air-driven or battery-operated drill are necessary for placement of the tension band.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiographs are taken to evaluate fracture alignment, apparatus and apposition. Postoperative pain management may be indicated (see Boxes 32.1 and 32.2 and Table 32.4). Activity should be restricted to leash walking and physical rehabilitation until the fracture has healed. Strict adherence to limited activity is mandatory because forceful contraction of the quadriceps will disrupt the repair. Physical rehabilitation (see Tables 33.3 and 33.4) encourages controlled limb use and optimal limb function after fracture healing and is especially important after fractures affecting the stifle. Care must be taken to develop customized protocols for each patient depending on location of the fracture, stability and type of fracture fixation, potential for healing, abilities and attitudes of the patient, and willingness or ability of the client to provide for the animal's care (see Chapter 11). Radiographs should be repeated at 6-week intervals until fracture healing is observed. Implants are generally not removed unless they cause a problem.

COMPLICATIONS

Feline patellar fractures treated with pin and tension band fixation have a high complication rate, with greater than 85% of cases resulting in additional fracture and fragment displacement. Transverse proximal tibial fractures may occur in cats with chronic nonunion of transverse patellar fractures.

PROGNOSIS

Return to athletic function in dogs depends on adequate healing and reduction of the articular surface. Radiographic evidence of degenerative joint disease will usually develop. Functional prognosis is good to excellent if postoperative instructions are followed closely and if the integrity of the patellofemoral joint is maintained. Most cats with patellar fractures progress to a fibrous or functional nonunion, with many cats exhibiting intermittent lameness. Osteoarthritis and peripatellar ossification are commonly seen in cases given long-term follow-up.

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TIBIAL AND FIBULAR FRACTURES

TIBIAL AND FIBULAR DIAPHYSEAL FRACTURES

DEFINITIONS

Tibial diaphyseal fractures and **fibular diaphyseal fractures** occur as a result of trauma to the rear limb and are a disruption of the continuity of the diaphyseal cortical bone. **Open fractures** (wounds through the skin over the bone) may occur because of sparse soft tissue coverage.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Fractures of the tibia in dogs and cats are primarily the result of trauma, including motor vehicle trauma, gunshots, fights with other animals, and falls. Although the fibula is usually fractured as well, it is seldom stabilized unless the stability of the stifle or hock is threatened. Underlying pathologic conditions (e.g., skeletal tumors) may predispose to fracture. The tibia is subject to several mechanical forces, and fractures can be avulsion fractures or transverse, oblique, spiral, comminuted, or severely comminuted. The paucity of soft tissue increases the possibility of open fractures and potentially decreases the extraosseous blood supply, both of which can delay bone healing, but is advantageous for placing an external fixator. Minimal soft tissue coverage of bone plates rarely results in tissue irritation and cold hypersensitivity.

Because tibial fractures are most often caused by trauma, the entire animal must be evaluated to detect concurrent injuries, such as pulmonary contusions, pneumothorax, rib fractures, and traumatic myocarditis. Concurrent injuries to the limb may include extensive soft tissue damage or loss.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, breed, or sex of dog or cat may be affected. Young animals more often sustain vehicular trauma.

History

Affected animals usually present with non–weight-bearing lameness after trauma. Owners may be unaware that the trauma has occurred.

Physical Examination Findings

Affected animals are usually non–weight-bearing on the affected limb and have palpable swelling, crepitation, and pain at the fracture site. The fracture may be open, with or without soft tissue loss. Affected animals often appear to have abnormal proprioceptive responses because they are reluctant to move the limb.

Diagnostic Imaging

The extent of bone and soft tissue damage should be assessed on craniocaudal and lateral radiographs that include the stifle and hock adjacent to the affected tibia. Fractious animals and those in pain may require sedation (see Tables 31.2 and 31.3) or general anesthetic for radiography after it has been determined that no contraindications (e.g., shock, hypotension, or severe dyspnea) to administration of sedatives or anesthetics exist. Thoracic radiography should be performed to evaluate for thoracic trauma.

Laboratory Findings

Complete blood count and serum chemistry evaluation should be done to evaluate the status of the animal for anesthesia and to determine whether concurrent injury or damage to the renal or hepatobiliary system exists.

DIFFERENTIAL DIAGNOSIS

Diagnosis of tibial fractures is based on physical and radiographic examination. Animals with tibial and fibular fractures should be evaluated to determine whether fractures are the result of trauma or an underlying pathologic condition (neoplasia or metabolic disease).

MEDICAL MANAGEMENT

Medical treatment of animals with tibial and fibular fractures may include analgesics (see Chapter 13) and antibiotics for open fractures. Conservative management of tibial and fibular diaphyseal fractures consists of splints and casts and is reserved for closed, nondisplaced, or greenstick fractures in immature animals. Cast fixation is appropriate for these fractures because the joint above and the joint below the fractured bone (stifle and hock) can be immobilized, and the fracture should heal rapidly.

> **NOTE** When choosing cast fixation, consider whether the animal will be able to bear weight on the other three limbs.

SURGICAL TREATMENT

The decision to perform an open or a closed reduction of tibial diaphyseal fractures is made on the basis of fracture configuration, fracture assessment score, and implant selection (Box 33.16) (see section on fracture reduction, p. 988). If open reduction of the fractured tibia is performed, consideration should be given to harvesting autogenous cancellous bone to enhance bone healing. The most accessible site for cancellous bone harvest is the ipsilateral distal femur (see p. 991). Fixation systems applicable to the tibial diaphysis include casts; IM pins with cerclage wire

BOX 33.16 Decision Making for Open or Closed Reduction for Tibial Diaphyseal Fractures

Open Reduction
- Displaced reducible fractures with internal fixation

Limited Open Reduction
- Displaced reducible fractures with external skeletal fixation
- Comminuted fractures necessitating cancellous bone graft

Closed Reduction
- Nondisplaced fractures with external coaptation or external skeletal fixation
- Comminuted nonreducible fractures with external skeletal fixation

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BOX 33.17 Implant Use for Fractures of the Tibial Diaphysis According to Fracture Assessment Score (FAS)

FAS 0 to 3
Bone plate and screws
Bone plate and intramedullary (IM) pin combination
Type II external skeletal fixation
Interlocking nail

FAS 4 to 7
Type Ib or type II external skeletal fixation
Bone plate and screws
Interlocking nail

FAS 8 to 10
Type Ia external skeletal fixation
IM pin and cerclage wire or external skeletal fixation
Cast

or external fixator support; interlocking nails; linear, circular, or hybrid external fixators; and bone plates (Box 33.17). The implant system chosen should reflect the fracture assessment score (see p. 985).

Preoperative Management

Open wounds should be managed initially by carefully clipping surrounding hair, cleaning the wound, and obtaining a swab for bacterial culture and susceptibility testing. Cultures of open wounds should be obtained before antibiotics are administered. The limb should be temporarily stabilized with a Robert Jones bandage or soft bandage and splint to immobilize the fragments, decrease or prevent soft tissue swelling, protect or prevent open wounds, and enhance patient comfort until surgery can be performed. Analgesics should be provided to posttraumatic animals (see Chapter 13). Concurrent injuries should be managed before the animal is anesthetized for fracture fixation. Prophylactic antibiotics are indicated when open reduction is performed (see Chapter 13).

Anesthesia

Refer to Tables 32.1 and 32.2 for anesthetic management of patients with fractures.

Surgical Anatomy

The tibial diaphysis is round in cross-section and resembles an S-shaped curve when viewed from the cranial aspect. The craniomedial surface of the tibia is not covered by muscle and can be easily palpated to serve as a landmark for location of the incision. Extensor muscles on the lateral surface of the tibia and flexor muscles caudal to the tibia can be retracted to expose the bone. The medial saphenous vein crosses the medial portion of the distal tibia.

Positioning

The leg should be prepared from the hip to below the hock. If cancellous bone graft harvest is anticipated, a donor site should also be prepared. For closed reduction or limited open reduction and ESF, the animal should be positioned with the affected leg suspended from the ceiling to improve visualization of correct joint alignment (see p. 990). Open reduction and plate application

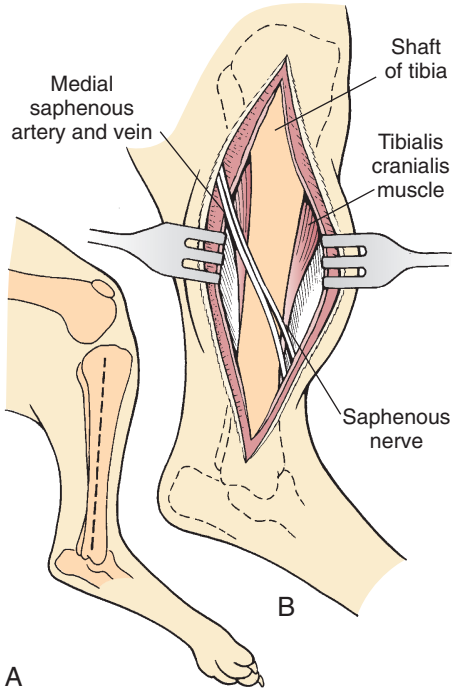


FIG. 33.104 (A) To approach the craniomedial aspect of the tibia, make a craniomedial skin incision. Extend the incision the length of the tibia if a plate is being applied. (B) Dissect through fascia, avoiding the medial saphenous vein and nerve, crossing the middle to distal third of the tibial diaphysis.

can also be performed with the leg suspended. If IM pin or interlocking nail application is anticipated, the animal should be positioned in dorsal recumbency and the limb draped out and released to expose the medial surface.

SURGICAL TECHNIQUE

Craniomedial Approach to the Tibia

Make a craniomedial skin incision parallel to the tibial crest, and extend it the entire length of the tibia (Fig. 33.104A). Continue dissection through the fascia, avoiding the medial saphenous vein and nerve and crossing the middle to distal third of the tibial diaphysis (Fig. 33.104B).

Implant Application Techniques Specific to the Tibia

Application of Casts

Casts can be applied as the sole method of fixation for stable fractures in young dogs or cats when the fracture will maintain adequate reduction and heal quickly (for general principles of cast application, see p. 995). A bivalve cast, which is applied over a padded bandage cut on the lateral and medial sides and taped together, is occasionally used to support internal fixation. *Position the dog in lateral recumbency with the fractured leg uppermost. Have the assistant hold the limb in slight extension with varus angulation. Apply stockinette, cast padding, and cast material (Fig. 33.105). To construct a bivalve cast, use extra cast padding while applying the cast. When the cast is hard, cut it longitudinally along the medial and lateral surfaces, and tape it in position.* Bivalve casts do not offer as rigid a fixation as cylinder casts but provide additional support with pin or plate fixation and are easily changed to allow wound treatment.

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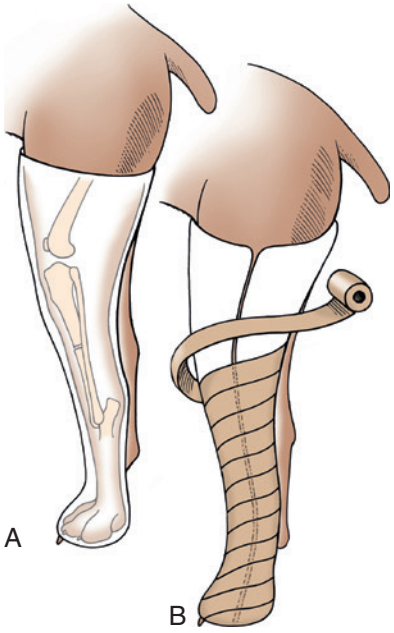


FIG. 33.105 Casts are used to stabilize closed, nondisplaced tibial-fibular fractures in patients with fracture assessment scores of 8 to 10. (A) Full cylinder cast, which immobilizes the stifle and hock, is placed with the limb in slight extension and varus angulation. (B) Cast can be bivalved by placing cast material over multiple layers of padding, cutting it on the lateral and medial aspects, and securing the two halves around the limb with elastic tape.

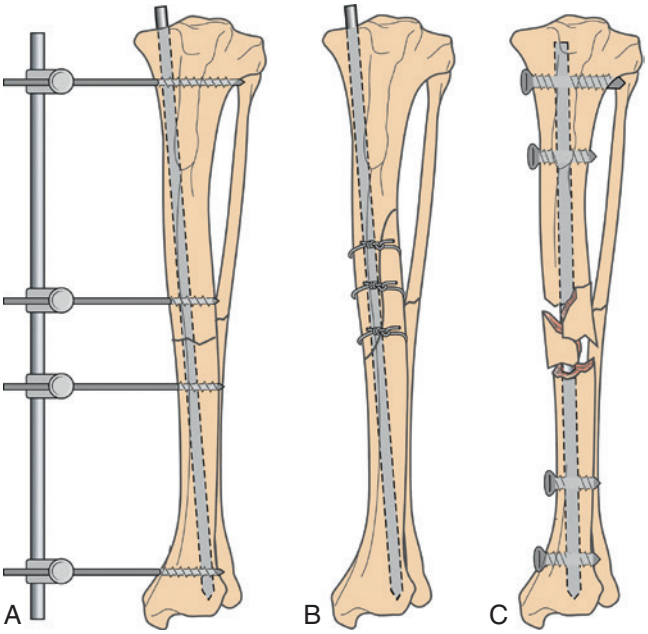


FIG. 33.106 (A) Transverse or short oblique fractures may be stabilized with an intramedullary (IM) pin and a unilateral external fixator. (B) Spiral or oblique fractures may be treated with an IM pin and multiple cerclage wires. (C) An interlocking nail may be used to support nonreducible fractures. (Modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat*. St. Louis: Elsevier; 2005.)

Application of Intramedullary Pins

An IM pin can be used to stabilize tibial diaphyseal fractures, providing excellent resistance to bending but no resistance to rotational forces or axial loading. Additional implants must be used to provide rotational and axial support for most fractures (for general principles of IM pin application, see p. 1008). Transverse or short oblique fractures treated with an IM pin require a concurrent unilateral external fixation splint to control rotation (Fig. 33.106A). Spiral or oblique fractures, in which the length of the fracture line is two to three times the diaphyseal diameter, can be treated with an IM pin and multiple cerclage wires (see Fig. 33.106B). Comminuted nonreducible fractures require an interlocking nail (see Fig. 33.106C). Correct placement of the IM pin is important to avoid interfering with the stifle joint. Retrograde pinning techniques risk damage to intraarticular structures in dogs and to the patellar tendon in cats. *To normograde the pin, insert it on the medial aspect of the proximal end of the tibia so that it penetrates the bone at a point midway between the tibial tubercle and the medial tibial condyle on the medial ridge of the tibial plateau (Fig. 33.107), and drive it in line with the medullary canal to exit at the fracture. Reduce the fracture and drive the pin distally. Seat the pin into the distal bone segment, stopping before penetrating the articular surface.* To estimate the appropriate pin penetration into the distal bone segment, a second pin equal in length to that placed in the marrow cavity can be used for a point of reference. If an external skeletal fixator is used with the IM pin, the pin should be small enough that fixation pins can be placed through the tibial diaphysis.

NOTE Estimate pin size from the preoperative radiograph, but a smaller pin may be necessary to allow for the curve of the tibia.

NOTE Pins must be placed in a normograde manner starting at the proximal tibia. Always manipulate the hock to ensure that the pin does not interfere with the joint.

Application of Interlocking Nails

Interlocking nails are used to stabilize both single and comminuted tibial diaphyseal fractures. The interlocking nail provides resistance to bending, rotational, and axial loading forces and can effectively bridge a nonreducible fracture (for general principles of interlocking nail application, see p. 1011). The size of nail selected should correspond to the width of the medullary canal at the isthmus of the bone. *Insert the interlocking nail in a normograde manner, starting at the craniomedial aspect of the tibial plateau. Make a medial parapatellar approach (see p. 1235) to expose the point of insertion. Flex the stifle 90 degrees to facilitate nail insertion.*

Application of External Skeletal Fixation

ESF is particularly useful for treating a wide variety of tibial diaphyseal fractures. The stiffness of a fixator can be increased in animals with low fracture assessment scores by adding fixation pins and using bilateral and biplanar frames. Because tibial fractures are frequently open, the use of external fixation is desirable to avoid invading the fracture site with metal implants. Implant removal is desirable because of the lack of soft tissue

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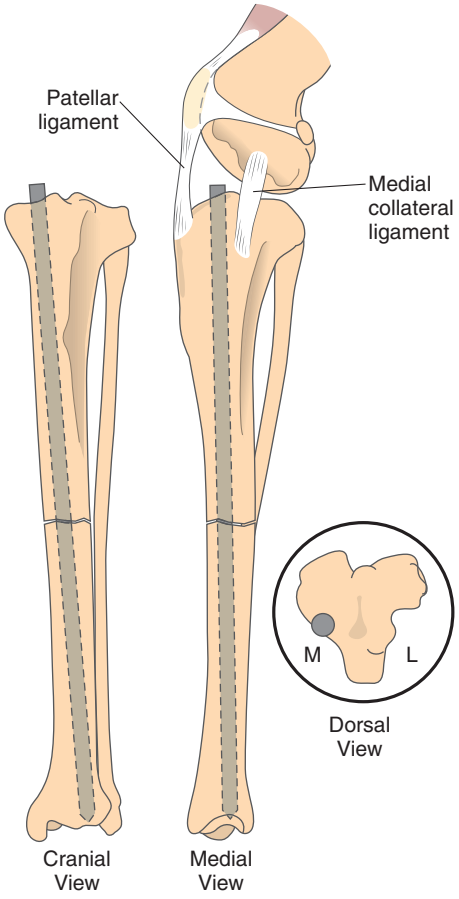


FIG. 33.107 Correct normograde placement of intramedullary pin in the tibia. A pin is inserted through the skin on the medial aspect of the proximal tibia so that it penetrates bone at a point midway between the tibial tubercle and the medial tibial condyle on the medial ridge of the tibial plateau.

coverage and the frequency of open fractures and is easily accomplished with external fixators. *Position the dog in dorsal recumbency and drape the leg while it is hanging from the ceiling. Use limited open reduction for reducible fractures and closed reduction for nonreducible fractures.*

Apply a type Ia frame to the cranial medial surface of the tibia (Fig. 33.108A) (see p. 998 for general principles of external fixator application). This location avoids penetration of the major muscle masses with fixation pins and thus decreases morbidity. *Apply type Ib frames to the cranial lateral and cranial medial surfaces of the tibia (Fig. 33.108B).*

Type II Frames

Penetration of major muscle masses is unavoidable with type II external fixators, but they are often favored because of their increased stiffness, and because proximal and distal transfixation pins can be used as guide pins for limb alignment. *To apply a type II frame, insert transfixation pins into the proximal and distal metaphyses of the tibia. Alternatively, place the proximal transfixation pin in the proximal diaphysis to decrease pin morbidity. Center the transfixation pins in the bone in the frontal plane and parallel to the respective joint surfaces. Reduce the fracture and secure the pins with medial and lateral connecting bars. Fill out the frame by placing at least two pins (preferably three) proximal and distal to the fracture (Fig. 33.108C).* For the additional stiffness of a maximal type II frame, transfixation pins that connect to both the medial and lateral bars may be placed using a guiding system (see p. 998). Postoperative radiographs should be taken to ensure that appropriate fracture reduction, pin placement, and joint alignment were achieved. *Correct valgus or varus malalignment by loosening the clamps and distracting the appropriate side of the limb until the joint surfaces are parallel (Fig. 33.109). Correct mild rotation by reversing the position of the clamps on the appropriate side of the pins in the distal fragment. Make every effort to correctly align the limb before the procedure is completed.* Circular external fixators may be used effectively for tibial diaphyseal fractures, especially comminuted nonreducible fractures treated with closed reduction. The fixation frame may be manipulated postoperatively to correct angular deformities in both craniocaudal and mediolateral planes. Circular fixators allow controlled axial micromotion of stabilized bone segments, promoting rapid bone union. However, circular fixators require considerable preoperative planning and frame construction to shorten surgical time, as well as optimal aftercare to minimize implant-related complications (see p. 1003 for general principles of circular fixator application). Combining a distal ring and wires with a linear connecting rod and fixation pins placed proximally, a hybrid construct, is useful in stabilizing complex radial fractures with short distal bone segments (see p. 1008 for general principles of hybrid frame application).

Application of Bone Plates and Screws

Bone plates are an excellent method of stabilizing tibial diaphyseal fractures, neutralizing axial, rotational, and bending forces. The function of the plate depends on the fracture (Fig. 33.110) (see p. 1016 for general principles of plate and screw application). *Incise the skin cranial to the anticipated position of the plate to avoid implant irritation of healing tissues. Contour the plate to match the curved medial surface of the tibia. When treating a nonreducible fracture, contour the plate to match the craniocaudal radiographic view of the contralateral tibia. Apply the plate to the medial surface of the tibia.*

NOTE Careful contouring of the plate to match the normal configuration of the tibia is essential. Failure to reproduce the normal curve of the tibia will result in valgus angulation of the limb.

Fixation Techniques for Specific Fractures

Stabilization of Midshaft Transverse or Short Oblique Fractures

Stabilization of a transverse or short oblique fracture requires rotational and bending support; short oblique fractures also require axial support. Useful fixation systems in animals with a low fracture assessment score include a bone plate and screws inserted to function as a compression plate or a type II external skeletal fixator. In animals with a moderate fracture assessment score, a compression plate, an interlocking nail, a minimal type II external skeletal fixator, or a type Ib external skeletal fixator may be used. Transverse or short oblique fractures in patients with a high fracture assessment score may be stabilized with a six-pin type Ia external skeletal fixator, an IM pin with a type Ia external fixator, or a cast if the fracture is greenstick or nondisplaced (Fig. 33.111).

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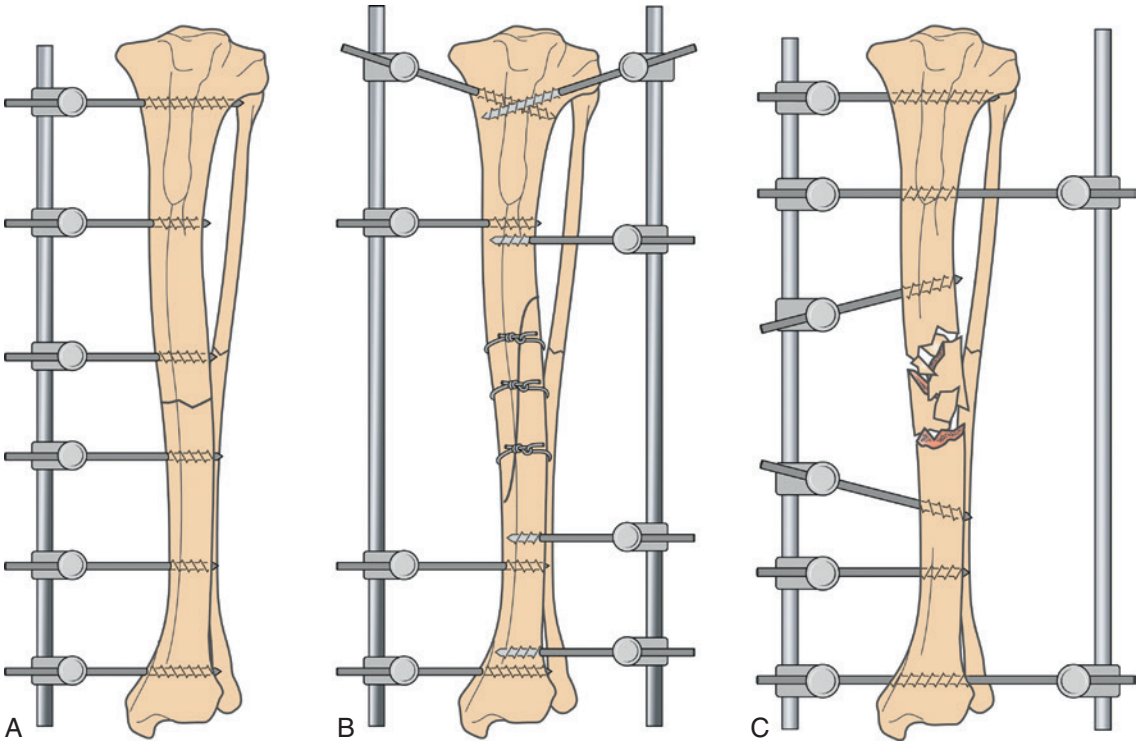


FIG. 33.108 Type Ia and Ib external fixators are used to treat tibial fractures in patients with high and moderate fracture assessment scores. (A) A type I external fixator is placed on the craniomedial surface of the tibia to stabilize a transverse fracture. (B) Anatomic reconstruction of long oblique fracture with cerclage wire restores the bony column and allows load sharing with a type Ib external fixator. (C) Type II external fixators are used to treat patients with low fracture assessment scores. A minimal type II frame is constructed using unilateral fixation pins to fill out the frame. (Modified from Johnson AL, Dunning D. *Atlas of Orthopedic Surgical Procedures of the Dog and Cat.* St. Louis: Elsevier; 2005.)

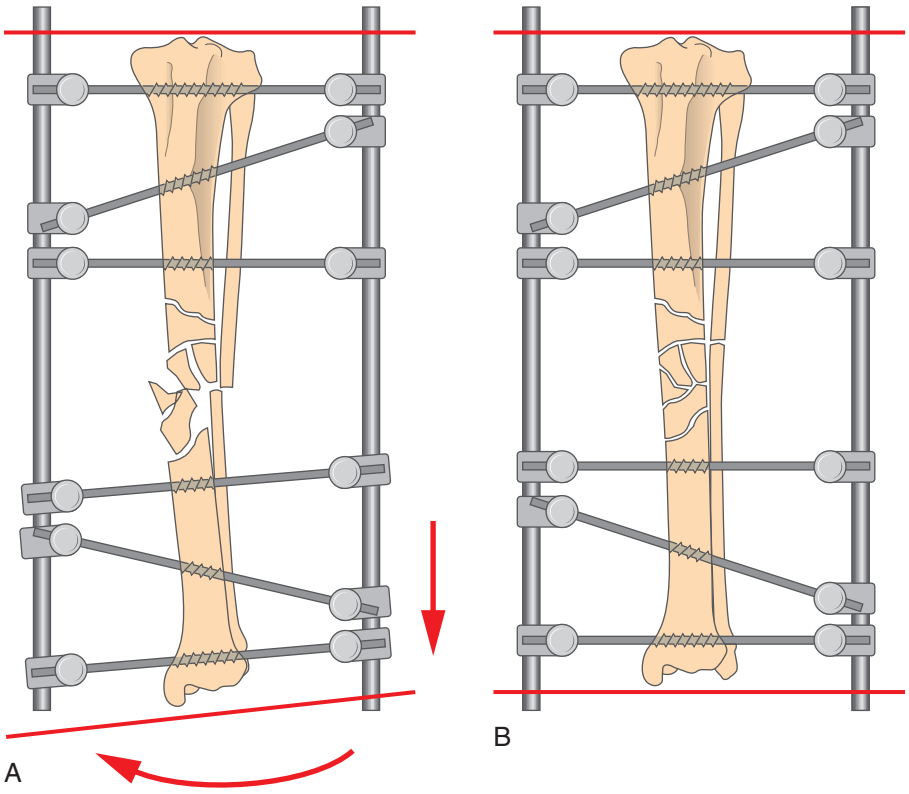


FIG. 33.109 (A) To correct a valgus angulation of the tibia, loosen the clamps distal to the fracture. Reposition the distal segment of bone by moving the clamps distally on the lateral bar and proximally on the medial bar until the joints are parallel. Reverse the procedure to correct a varus angulation. (B) When the joints are aligned, tighten the clamps.

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Stabilization of Midshaft Long Oblique Fractures or Comminuted Fractures With a Large Butterfly Fragment

These fractures can be anatomically reduced and interfragmentary compression applied with cerclage wire or lag screws. Axial, rotational, and bending forces generated by weight bearing are neutralized with bone plates, interlocking nails, IM pins with the cerclage wire, or external fixators.

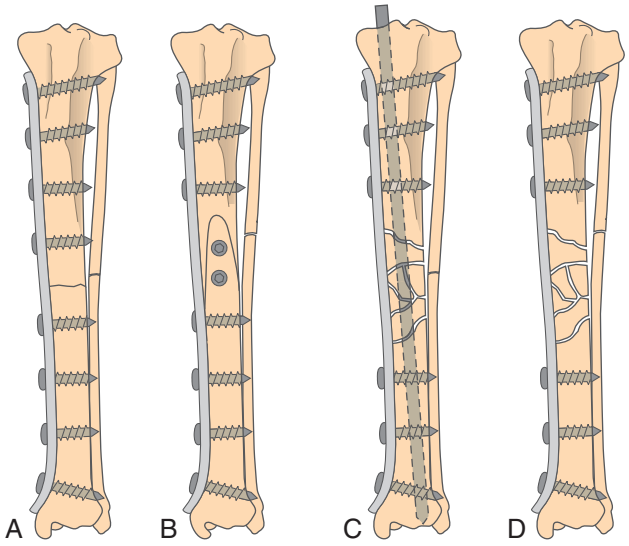


FIG. 33.110 A plate placed on the medial surface of the tibia may function as (A) a compression plate for transverse fractures, (B) a neutralization plate to support long oblique fractures reconstructed with lag screws, or (C–D) a bridging plate with or without an intramedullary pin to span a nonreducible fracture.

Useful fixation systems in long oblique fractures in patients with low fracture assessment scores include using the combination of interfragmentary compression with cerclage wire or lag screws to reconstruct the cylinder of the bone and then bridging the area with a bone plate, interlocking nail, or type II external fixator. With a moderate fracture assessment score, a neutralization plate, an interlocking nail, or a type II or type Ib external fixator combined with lag screws or cerclage wire for interfragmentary compression may be used. Long oblique fractures in patients with a high fracture assessment score may be stabilized with an IM pin and cerclage wires or cerclage wires and a type Ia external fixator (Fig. 33.112).

Stabilization of Midshaft Comminuted Fractures With Multiple Fragments

These fractures are realigned using indirect reduction techniques (see p. 990). Because no load sharing occurs between the implant and the bone until biological callus forms to provide support, these fractures need rigid axial, rotational, and bending support. Very high stresses will be imposed on the implant and its connection to the bone. If the biological assessment is favorable, the imposed stresses will be of short duration, reducing the likelihood of implant failure. If the biological assessment is not favorable, however, imposed stresses will act on the implant for an extended period, making implant failure more likely.

Stabilization may be achieved in patients with low fracture assessment scores with a bone plate used in a bridging fashion, with or without an IM pin, an interlocking nail, or a maximal type II external skeletal fixator. Patients with a moderate fracture assessment score may be managed with a bone plate functioning as a bridging plate, an interlocking nail, or a minimal type II external skeletal fixator. A patient with this type of fracture would have a high fracture assessment score only when the biological assessment is extremely favorable (e.g., 4- to 5-month-old animal with a low-velocity, closed, single-limb injury) and could be treated with a type Ib external skeletal fixator (Fig. 33.113).

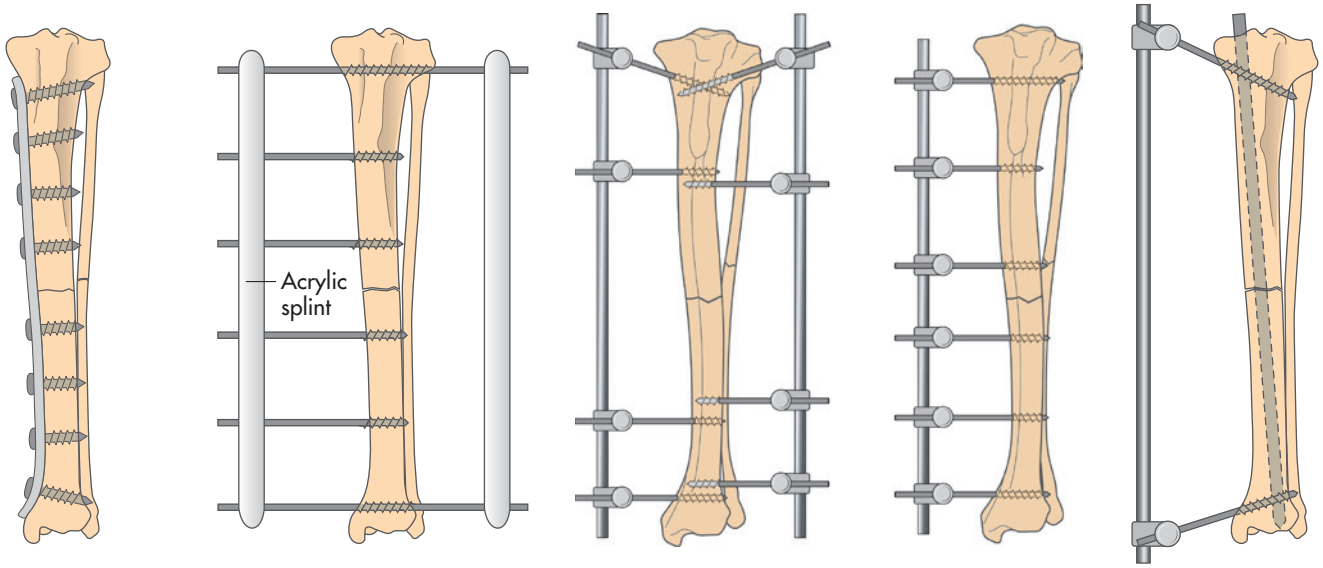


FIG. 33.111 Suggested methods for stabilizing transverse or short oblique tibial fractures based on fracture assessment score.

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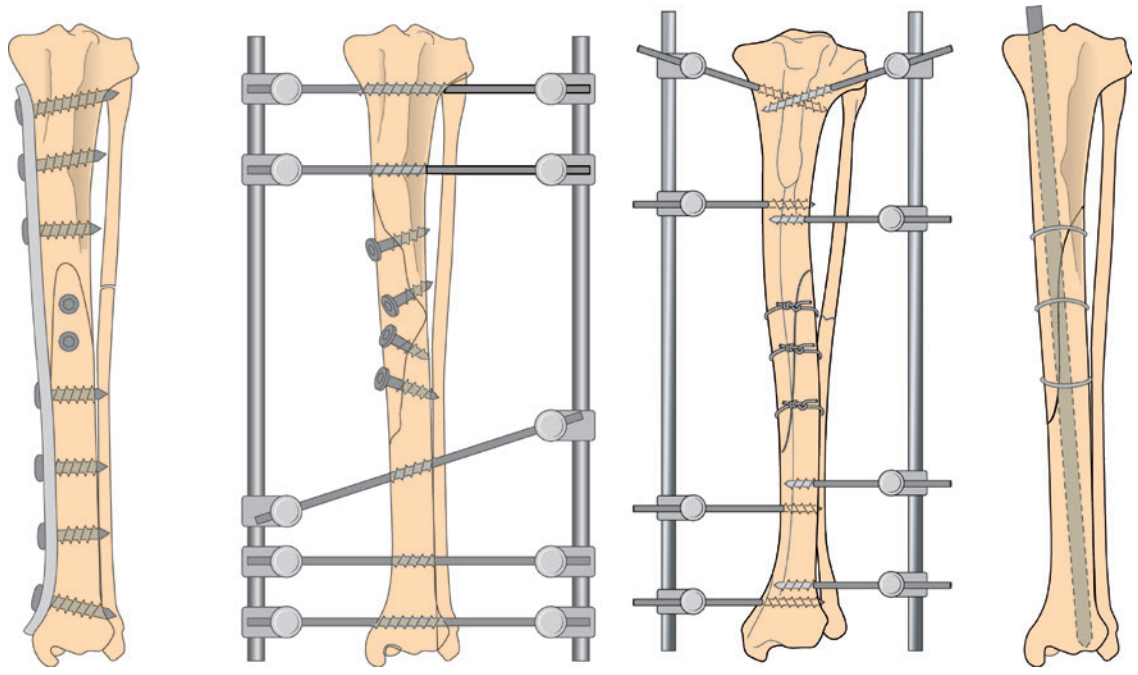


FIG. 33.112 Suggested methods for stabilizing long oblique or reducible comminuted tibial fractures based on fracture assessment score.

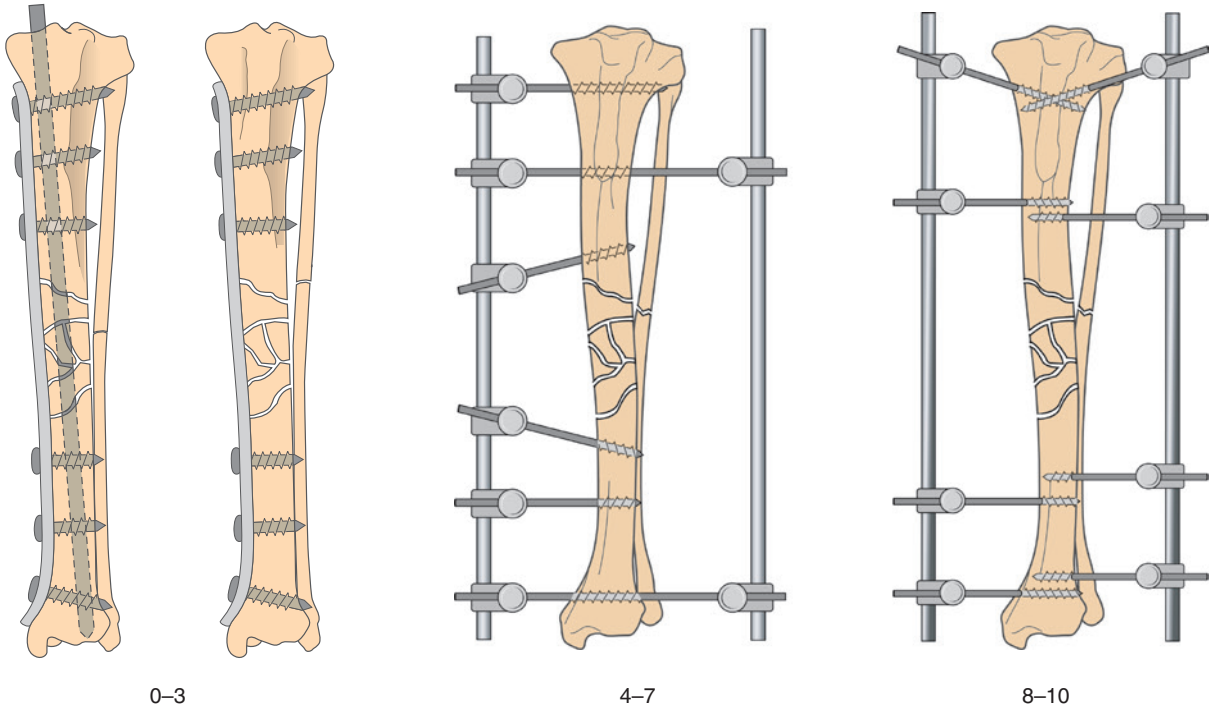


FIG. 33.113 Recommended methods for stabilizing nonreducible comminuted tibial fractures based on fracture assessment score.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Equipment necessary for pin and wire placement includes retractors, bone-holding forceps, reduction forceps, a Jacobs pin chuck, IM pins, K-wires, orthopedic wire, wire twisters, and wire and pin cutters. Additional equipment needed for external fixation includes a low-RPM power drill and an external fixator system. Interlocking nail equipment is necessary for nail application. Plating equipment and a high-speed drill are required for application of plates and screws.

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POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiographs should be taken to document alignment, apparatus and apposition. Postoperative pain management may be indicated (see Boxes 32.1 and 32.2 and Table 32.4). Activity should be restricted to leash walking and physical rehabilitation until the fracture has healed. Physical rehabilitation (see Table 33.2 and Chapter 11) encourages controlled limb use and optimal limb function after fracture healing. Care must be taken to develop customized protocols for each patient depending on location of the fracture, stability and type of fracture fixation, potential for healing, abilities and attitudes of the patient, and willingness or ability of the client to provide for the animal's care.

Casts require intensive management by owners and frequent evaluations by the veterinarian. Abrasions and wounds are caused by pressure from the cast. Management involves removal of the cast or replacement with a bivalve cast and wound treatment. Early destabilization of the fracture to treat wounds can delay healing.

After open reduction with ESF, the incision should be covered. Gauze sponges are opened and stuffed to fill the space between the fixation bar and the skin around the pins, then are secured in place with a bandage around the fixator. Care should be taken to bandage the paw as well during the immediate postoperative period to prevent swelling. Open wounds should be treated daily with wet-to-dry dressings until a granulation bed has formed. Wounds are then covered with a nonadhesive pad and the bandage changed as necessary. Daily hydrotherapy aids in cleaning open wounds, reduces postoperative swelling, and cleans pin sites. The animal should be released to the owner with instructions to limit exercise and prevent fixator snares. If the external fixator bandage is maintained, it should be changed weekly. If a bandage is not used, daily hydrotherapy can be administered with a handheld shower massage. Checkups should be scheduled at 2 weeks for suture removal and fixator evaluation, and every 6 weeks for radiographic evaluation.

Although a rigidly stabilized fracture site is optimal for initial bone formation, subsequent bone remodeling is enhanced by increasing the load through the fractured bone. Removal of one-half of a type Ib external fixator (converting to a type Ia frame) or removal of selected transfixation pins from a type I or type II frame decreases rigidity of the fixator, allowing increased load on the remodeling bone while still protecting the healing fracture. Destabilization is usually done at 6 weeks after surgery, depending on the fracture assessment. Early bone formation and bridging of the fracture site should be radiographically evident before destabilization is done. The external fixation splint can be removed completely when radiographic evidence shows complete bone bridging of the fracture lines. A support bandage or a splint may be used to protect the healing bone for a few weeks after fixator removal.

After placement of a plate on a diaphyseal fracture, the limb should be supported for a few days with a soft-padded bandage to reduce swelling. The animal will usually be fully weight bearing within 2 to 3 weeks. Confinement is recommended until radiographic signs of bone union are noted.

Plate removal may rarely be necessary after bone union because tissue irritation and cold sensitivity have been associated with minimal soft tissue coverage over the plate. IM pins may be removed after the fracture has healed, but interlocking nails are usually left in place unless implant-related complications occur.

COMPLICATIONS

Complications with tibial fractures include osteomyelitis, implant migration resulting in soft tissue irritation, malunion, delayed union, and nonunion. Poor decision making regarding implant choice relative to the fracture assessment is the most common reason for complications. Cats treated with rigid external fixators may be at risk for delayed healing or nonunion. Complications encountered with external fixation of the tibia include pin loosening and pin tract drainage.

PROGNOSIS

The prognosis is generally good after tibial fracture repair if proper fracture management procedures are followed.

TIBIAL AND FIBULAR METAPHYSEAL AND ARTICULAR FRACTURES

DEFINITIONS

Metaphyseal fractures and **epiphyseal fractures** occur in trabecular bone. Articular fractures disrupt the joint surface.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Fractures of the proximal tibial metaphysis and epiphysis occur infrequently in mature dogs and cats. These fractures are usually transverse or short oblique in nature but may be comminuted as a result of severe trauma, such as a gunshot. Fractures of the distal tibia in mature animals usually involve the malleoli, either as fractures of the malleoli or as erosion injuries that remove the malleoli. Loss of malleolar stability results in loss of collateral ligament function and talocrural instability. Accurate alignment of the articular surface of malleolar fractures and rigid fixation of the fragment are necessary to achieve joint stability and minimize subsequent development of degenerative joint disease.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, breed, or sex of dog or cat may be affected. Young animals more frequently sustain vehicular trauma.

History

Affected animals usually have non–weight-bearing lameness after trauma.

Physical Examination Findings

Because of the traumatic nature of these tibial and fibular fractures, the entire animal must be assessed to detect abnormalities of other body systems. Palpation of the limb may reveal swelling, pain, crepitation, and instability of the adjacent joint. A shearing injury of the tarsus (see p. 1269) may be associated with fracture of the distal tibia. Although no major nerves are in the immediate area, dogs often appear to have abnormal proprioceptive responses because they are reluctant to move the limb.

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Diagnostic Imaging

The extent of bone and of soft tissue damage should be assessed on craniocaudal and lateral radiographs that include joints proximal and distal to the affected tibia. Stress radiographs may demonstrate abnormal laxity of the tarsal joints or motion of the proximal tibial epiphysis associated with the fracture. Fractious animals and those in extreme pain may require sedation (see Tables 31.2 and 31.3) or general anesthesia after it has been determined that no contraindications (e.g., shock, hypotension, severe dyspnea) to administration of sedatives or anesthetics exist. Thoracic radiography should be performed to evaluate for thoracic trauma.

Laboratory Findings

Complete blood count and serum chemistry evaluation should be done to evaluate the status of the animal for anesthesia and to determine whether concurrent injury to the renal or hepatobiliary systems exists.

DIFFERENTIAL DIAGNOSIS

Animals brought in with proximal or distal tibial fractures should be evaluated to determine whether the fractures are caused by trauma or by an underlying pathologic condition (neoplasia, metabolic disease). Joint luxations can be differentiated from fractures radiographically.

MEDICAL MANAGEMENT

Medical treatment of animals with tibial and fibular metaphyseal and epiphyseal fractures includes analgesics (see Chapter 13) and administration of antibiotics to treat open fractures (see Chapter 9). Nondisplaced proximal tibial fractures may be treated with a cast.

SURGICAL TREATMENT

Simple proximal tibial metaphyseal fractures may be treated with IM pins and Steinmann pins or K-wires placed as crossed pins. Comminuted tibial plateau fractures are best treated with buttress plate application. Cancellous bone autograft is used to augment the repair and speed healing. Articular fractures require anatomic reduction and stabilization with a lag screw or plate. Malleolar fractures require tension band wire techniques to resist the pull of the collateral ligaments. Animals with unstable shearing injuries to the hock are treated by reestablishing the stability to the hock with an external fixator or synthetic ligament and bivalve cast coupled with appropriate wound management (see p. 1269).

Preoperative Management

Extensive damage and loss of tissue may be noted in the area of the fracture. Open wounds should be managed initially by carefully clipping surrounding hair, cleaning the wound, and taking a culture from deep tissue for microbial sensitivity testing. The tibia should be temporarily stabilized with a Robert Jones bandage (see p. 982) to immobilize fragments, decrease or prevent soft tissue swelling, protect or prevent open wounds, and enhance patient comfort until surgery can be performed. Concurrent injuries should be managed before anesthetic induction for fracture fixation. Perioperative pain management should be instituted (see Chapter 13).

Anesthesia

Refer to Tables 32.1 and 32.2 for anesthetic management of patients with fractures.

Surgical Anatomy

The medial aspect of the proximal tibia is covered only with skin and subcutaneous tissue and can easily be palpated and approached. The saphenous nerve lies caudal to the medial surface of the proximal tibia. The lateral aspect of the tibia and fibula is covered by the cranial tibial muscle, and the tendon of the long digital extensor muscle is visible. The lateral collateral ligament attaches from the lateral femoral condyle to the fibular head and stabilizes the stifle. The peroneal nerve and the popliteal artery lie caudal to the fibular head.

The medial malleolus of the distal tibia and the lateral malleolus of the fibula extend distal to the articulating surfaces of the distal tibia and talus. The long and short parts of the medial collateral ligaments arise from the medial malleolus. The long and short parts of the lateral collateral ligaments arise from the lateral malleolus. These ligaments are essential for hock stability. Tendons of the cranial tibial and long digital extensor muscles cross the cranial surface of the distal tibia. The medial saphenous vein crosses the medial surface of the distal tibia.

Positioning

The limb should be prepared from the hip to below the hock. If cancellous bone graft harvest is anticipated, a donor site should be prepared (ipsilateral proximal tibia or proximal humerus). The animal should be positioned in lateral or dorsal recumbency and the limb draped out for proximal tibial and fibular fractures. The animal is positioned in dorsal recumbency for treatment of distal tibial and fibular fractures.

SURGICAL TECHNIQUE

Surgical Approach to the Proximal Tibia

The craniomedial approach to the tibia (see p. 1122) is extended proximally to include the metaphysis. The lateral approach to the stifle (see p. 1234) is used to expose the fibular head for stabilization.

Surgical Approach to the Distal Tibia

Extend the craniomedial approach to the tibia (see p. 1122) distally to expose the medial malleolus. Make a lateral skin incision over the malleolus and use blunt and sharp dissection to expose the bone.

Stabilization of Proximal Tibial Fractures

Fractures of the proximal tibial metaphysis may be stabilized with a cast if the fracture can be reduced closed, is stable, and will heal rapidly (fracture assessment score of 8 to 10).

Refer to p. 995 for general casting techniques and p. 1121 for application of a cast to the tibia. If open reduction is necessary, stabilize transverse or short oblique proximal tibial fractures with IM pins as described for diaphyseal fractures of the tibia (see p. 1123). If the fracture is not rotationally stable, angle a K-wire across the fracture line. Place a figure-eight orthopedic wire around both ends of the K-wire to provide compression to the fracture line

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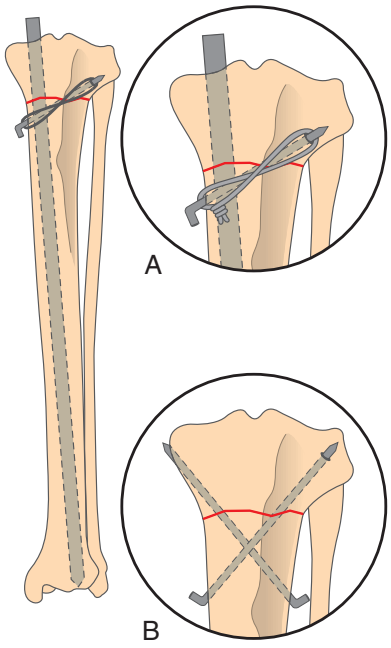


FIG. 33.114 Simple single fractures of the proximal tibial metaphysis in dogs with high fracture assessment scores can be stabilized with an intramedullary pin. (A) K-wire and figure-eight orthopedic wire may be added for rotational stability. (B) Crossed K-wires may also be used.

(Fig. 33.114A). These fractures can also be stabilized with crossed pins. Reduce the fracture and drive a K-wire from the lateral surface of the tibial epiphysis across the fracture and into the tibial metaphysis, exiting the medial cortex. Drive a second wire from the medial tibial epiphysis across the fracture and into the metaphysis, exiting the lateral cortex. Take care to avoid penetrating the articular surface. Alternatively, the K-wires may be driven from the epiphysis to the metaphysis (Fig. 33.114B). Additional external support may be required for dogs with moderate fracture assessment scores.

Comminuted tibial plateau fractures must be supported to prevent collapse of the tibial plateau. *Apply a bone plate functioning as a buttress plate to the medial side of the proximal tibia. If bone loss is noted on the lateral side of the proximal tibia, consider adding a type Ia external fixator to support the tibial plateau. Add cancellous bone autograft to any defects. Support the repair as necessary with a splint or a transarticular external fixator.*

Stabilization of Malleolar Fractures

Fractures of the malleoli are usually treated with open reduction and internal fixation with a tension band wiring technique; however, a bone screw functioning as a lag screw can also be used if the fracture assessment score is low and the fragment is large enough (Fig. 33.115A–B). Additional external fixation or coaptation is used as necessary to support the internal fixation device.

Application of Tension Band

Reduce the fracture (medial malleolus of tibia or lateral malleolus of fibula), and start two K-wires in the fragment (one wire may be all that fits in the fibular fragment). Drive the wires across the fracture line to lodge in the major bone segment. Place a transverse

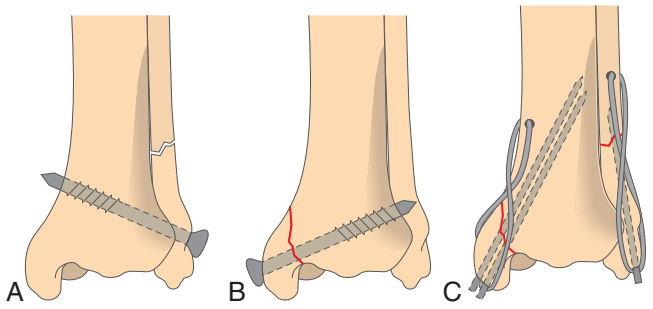


FIG. 33.115 (A–B) Fractures of the tibial and fibular malleoli can be repaired with a lag screw. (C) Tension band wires can be used in patients with high fracture assessment scores.

drill hole in the major bone segment, pass a figure-eight wire through the hole and around the K-wire, and tighten it (Fig. 33.115C).

Application of Lag Screws

For the medial malleolus, reduce the fracture and drill a gliding hole (equal to the diameter of the threads on the screw) into the malleolar fragment. Place an insert drill sleeve into the gliding hole, and drill a smaller hole (equal to the core diameter of the screw) across the tibia. Measure, tap, select, and place a screw of appropriate length. Compression of the fracture should occur (see Fig. 33.115A–B and p. 1019 for lag screw techniques). When the proximal or distal fibula is stabilized to the tibia, the fibula is treated as the fragment by drilling the gliding hole through the fibula and the tapped hole in the tibia.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Instruments needed include a high-speed power drill, equipment for lag screws, K-wires, IM pins, orthopedic wire, wire twisters, wire cutters, and reduction forceps. Bone-plating equipment is needed for buttress plate application.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiographs should be taken to document alignment, apparatus, and apposition. Postoperative pain management may be indicated (see Boxes 32.1 and 32.2 and Table 32.4). After internal fixation, a soft-padded bandage should be applied for a few days to control swelling and to support soft tissue. Active dogs may require a splint for 3 to 6 weeks to protect distal tibial implants. Physical rehabilitation (see Table 33.3 and Chapter 11) encourages controlled limb use and optimal tarsal function after fracture healing. Care must be taken to develop customized protocols for each patient depending on location of the fracture, stability and type of fracture fixation, potential for healing, abilities and attitudes of the patient, and willingness and ability of the client to provide for the animal's care. Physical rehabilitation for patients with fractures supported by splints for 6 weeks generally begins after the splint has been removed. Open wounds are treated daily with wet-to-dry dressings until a granulation bed has formed. The wounds are then covered with a nonadhesive pad, and the bandage is changed as needed. Daily hydrotherapy aids in cleaning open wounds and reducing postoperative swelling. Rechecks should be scheduled at 2 weeks for suture removal, and every 6 weeks for radiographic evaluation. Implant removal may be necessary after bone healing if the implants interfere with or irritate soft tissue.

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COMPLICATIONS

Fractures that involve the articular surface predispose the joint to development of degenerative joint disease. Malalignment of articular fractures routinely causes degenerative joint disease.

PROGNOSIS

Metaphyseal fractures usually heal quickly because of the large amounts of cancellous bone surrounding the fracture. In general, trabecular bone heals with minimal formation of callus.

TIBIAL AND FIBULAR PHYSEAL FRACTURES

DEFINITIONS

Physeal fractures may occur through the cartilaginous growth plate of the proximal or distal tibia or the tibial tuberosity in immature animals. Physeal fractures are also called *epiphyseal plate fractures* or *slipped physes*.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The cartilaginous physis is weaker than surrounding bone and ligaments, making it more susceptible to injury (see p. 984). The Salter-Harris classification is used to categorize physeal fractures on the basis of radiographic and histologic appearance (see p. 984). Tibial physeal fractures may or may not be displaced. Proximal tibial physeal fractures are usually Salter I or II fractures, although rarely Salter III or IV fractures occur. With Salter I or II fractures and concurrent fracture of the fibula, the epiphysis may be displaced caudolateral to the tibial diaphysis, and additional injury to collateral ligaments may occur. Open reduction and internal fixation are usually required to restore normal anatomy. Distal tibial physeal fractures are also usually Salter I or II fractures.

DIAGNOSIS

Clinical Presentation

Signalment

These fractures occur in immature dogs or cats with open physes.

History

Affected animals usually have non–weight-bearing lameness after trauma. Owners may be unaware that trauma has occurred.

Physical Examination Findings

Because of the traumatic nature of physeal fractures, the entire animal must be assessed to detect abnormalities of other body systems. Palpation of the limb may reveal swelling, pain, crepitation, and instability of the adjacent joint. Dogs often appear to have abnormal proprioceptive responses because they are reluctant to move the limb.

Diagnostic Imaging

Craniocaudal and lateral radiographs of the affected tibia and fibula (which include the proximal and distal joints) are required to diagnose Salter I to Salter IV fractures. With minimally displaced fractures, it may be difficult to determine whether the normally radiolucent physis is really fractured. Comparison radiographs of the opposite limb are often beneficial, particularly with tibial tuberosity avulsions. Fractious animals and those in extreme pain may require sedation (see Tables 31.2 and 31.3) or general anesthetic for radiography after it has been determined that no contraindications (e.g., shock, hypotension, severe dyspnea) to administration of sedatives or anesthetics exist. Thoracic radiography should be performed to evaluate for thoracic trauma.

> **NOTE** Radiographs taken at the time of injury do not provide information about crushing injuries to the physis or damage to the physeal blood supply. Therefore it is difficult to give an accurate prognosis for growth with these fractures at the time of injury.

Laboratory Findings

Complete blood count and serum chemistry evaluation should be done to evaluate the status of the animal for anesthesia and to determine whether concurrent injury or damage to the renal or hepatobiliary system exists.

DIFFERENTIAL DIAGNOSIS

Physeal fractures can be differentiated from joint luxations or soft tissue trauma radiographically.

MEDICAL MANAGEMENT

Medical treatment of animals with tibial physeal fractures may include analgesics (see Chapter 13) and antibiotics to treat open fractures (see Chapter 9). Nondisplaced or minimally displaced tibial physeal fractures may be adequately stabilized with a cast.

SURGICAL TREATMENT

Most physeal fractures are classified as having assessment scores of 8 to 10 because affected animals are young and physeal fractures heal quickly. Therefore the implant system chosen need not function for a long time. Surgical treatment of displaced physeal fractures consists of anatomic reduction and stabilization with K-wires or small pins that are smooth so as not to interfere with physeal function. Pins placed perpendicular to the physis allow growth more readily than pins placed obliquely and anchored in cortical bone (cross-pinning technique). In animals that are close to maturity, threaded implants may be used to compress the fractured physis.

> **NOTE** Use smooth implants when crossing the physis in animals with potential growth.

Preoperative Management

The rear limb should be temporarily stabilized with a Robert Jones bandage (see p. 981) or soft padded bandage and splint to immobilize fragments, decrease or prevent soft tissue swelling, protect or prevent open wounds, and enhance patient comfort until surgery can be performed. Concurrent injuries should be managed before anesthetic induction for fracture fixation. Perioperative pain management should also be instituted (see Chapter 13).

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Anesthesia

Refer to Tables 32.1 and 32.2 for anesthetic management of patients with fractures.

Surgical Anatomy

Surgical anatomy of the tibia and fibula is provided on p. 1122.

Positioning

The limb should be prepared from the hip to below the hock. The animal is positioned in dorsal recumbency and the limb draped out for physeal fractures of the tibia.

SURGICAL TECHNIQUE

Surgical Approach to the Craniomedial Tibia

Use a proximal extension of the craniomedial approach to the tibia (see p. 1122) for proximal tibial physeal fractures and tibial tuberosity avulsions.

Surgical Approach to the Distal Physis

Approach distal physeal fractures with a distal extension of the craniomedial approach to the tibia or with a cranial skin incision and retraction of the extensor tendons.

Stabilization of Nondisplaced Fractures

Treat nondisplaced physeal fractures with closed reduction and a cast that immobilizes the stifle and the hock (see Fig. 33.105). Casts applied to the tibia should reach from the toes to above the stifle. The stifle should be extended to reduce proximal tibial physeal fractures during cast application.

Stabilization of Displaced Fractures

Displaced fractures require a surgical approach and gentle reduction to restore physeal alignment. Crossed K-wires or Steinmann pins may be used to stabilize tibial physeal fractures. *To stabilize proximal tibial physeal fractures, drive one K-wire from the lateral surface of the tibial epiphysis across the physis, into the tibial metaphysis, and through the medial cortex. Drive the second wire from the medial tibial epiphysis across the physis, into the metaphysis, and through the lateral cortex (Fig. 33.116).* A third wire may be driven from the tibial tuberosity into the metaphysis for additional stabilization.

To stabilize distal tibial physeal fractures, drive a K-wire from the medial malleolus across the physis and into the lateral metaphysis. Drive the second wire from the lateral epiphysis or the lateral malleolus across the physis and into the metaphysis. Seat both wires in the cortex of the metaphysis (see Fig. 33.116).

Stabilization of Avulsion Fractures of the Tibial Tuberosity

Fractures through the physis of the tibial tuberosity result in proximal displacement of the tuberosity and must be reduced and stabilized to restore quadriceps muscle function and stifle extension. Occasionally, closed reduction through sufficient extension of the stifle realigns the tubercle. The limb can then be cast in extension for 2 to 3 weeks. If open reduction is necessary to reduce the tuberosity, then the fracture should be stabilized with K-wires with or without a tension band wire. *Reduce the tibial tuberosity and drive two K-wires across the physis to lodge in the proximal tibia. Check the repair to determine whether stabilization is sufficient to prevent avulsion of the fracture; if not, place a tension band wire, even though it may prevent physeal growth. To place a tension band wire, drill a transverse hole in the major bone segment, pass a figure-eight wire through the hole and around the K-wire, and tighten the wire (Fig. 33.117) (see p. 1015 for tension band application).*

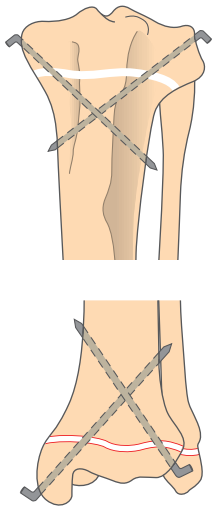


FIG. 33.116 Displaced tibial physeal fractures can be treated with open reduction and placement of crossed K-wires.

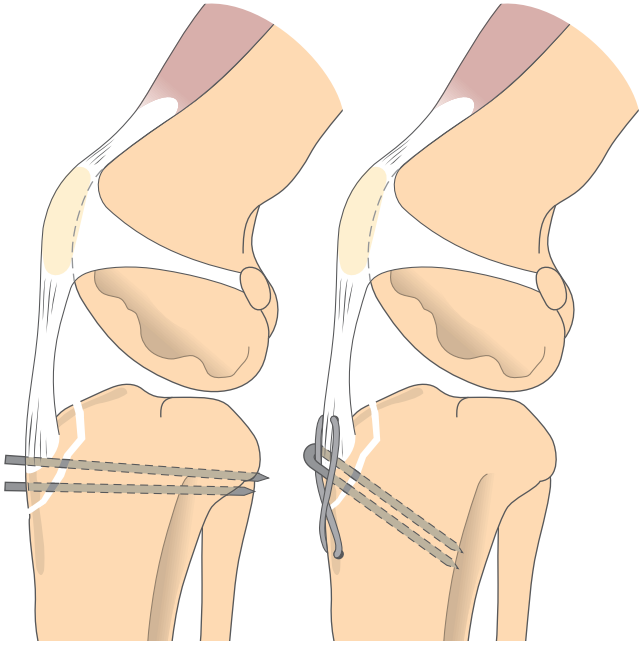


FIG. 33.117 Avulsions of the tibial tuberosity can be reduced and stabilized with two K-wires. If displacement of the fragment occurs when the stifle is flexed, a figure-eight orthopedic wire may be added.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

K-wires, Steinmann pins, orthopedic wire, wire twisters, wire cutters, and reduction forceps are needed.

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POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiographs should be taken to document limb alignment, apparatus, and fracture apposition. After internal fixation, a soft-padded bandage should be applied for a few days to control swelling and to support soft tissue. Postoperative pain management may be indicated (see Boxes 32.1 and 32.2 and Table 32.4). Activity should be restricted to leash walking and physical rehabilitation until the fracture has healed. Physical rehabilitation (see Chapter 11) encourages controlled limb use and optimal joint function after fracture healing. Care must be taken to develop customized protocols for each patient depending on location of the fracture, stability and type of fracture fixation, potential for healing, abilities and attitudes of the patient, and willingness or ability of the client to provide for the animal's care. Rechecks should be scheduled at 2 weeks for suture removal and at 4 to 6 weeks for radiographic evaluation of fracture healing. Radiographs of the injured bone and the contralateral bone can be made and compared for length as early as 2 to 3 weeks after the injury to determine physeal function. Cartilage physes that heal in a manner allowing continued function appear radiographically as a lucent line. Additionally, increased bone length should be apparent. If the physeal line appears as a bone density, endochondral ossification has occurred and continued physeal function is unlikely. Implant removal is indicated after physeal healing (4 weeks) to allow bone growth (if the physis is functional after the trauma). Three to four weeks postoperatively, the tension band wires used to stabilize the tibial tuberosity are removed to encourage physeal function and to prevent deformity.

PROGNOSIS

Although the prognosis for healing of a physeal fracture is excellent, the prognosis for continued function or growth of the physis depends on the amount of damage sustained by the zone of proliferating cells. The prognosis is good for future physeal growth after a fracture that separates the cartilaginous physis at the zone of hypertrophied cells. The prognosis is poor for future physeal growth after trauma that crushes the physis. Unfortunately, most trauma-induced physeal fractures sustain damage to the growing cells and have a guarded prognosis for growth. Although the tibia and the fibula constitute a paired bone system, premature closure of the proximal or distal tibial physis usually results in a short but straight limb, for which the animal compensates by extending the stifle. Caudal malalignment of the proximal tibial epiphysis may result in an increased tibial plateau angle. Premature closure of the tibial tuberosity physis can alter the conformation of the proximal tibia, resulting in impaired function and degenerative joint disease of the stifle.

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34

Diseases of the Joints



GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

The term **arthrosis** denotes an articulation or joint between two bones; this term is also used to signify a degenerative disease of a joint. **Polyarthritis** is inflammation that simultaneously affects several joints. **Osteoarthritis** or **osteoarthrosis** is a primarily noninflammatory degenerative joint disease (DJD) characterized by articular cartilage degeneration, marginal bone hypertrophy (**osteophytosis**), and synovial membrane changes. **Ankylosis** is the result of DJD or inflammatory diseases, in which the joint is fused after new bone production.

Synovial joints (e.g., shoulder joint, hip, elbow, or stifle) are lined with **synovial membrane** and allow for relatively free movement. Components of **fibrous** (e.g., skull and tooth sockets) and **cartilaginous** (e.g., mandibular symphysis and growth plates) **joints** are connected with fibrous tissue or cartilage, respectively; therefore these joints allow for little or no movement. **Arthroscopy** is the use of an endoscope to examine and treat joints. **Arthrotomy** is the surgical exposure of a joint. **Arthroplasty** is revision of a joint structure. **Arthrodesis** is surgical treatment leading to joint fusion. **Dysplasia** is the abnormal development of tissues, organs, or cells and is frequently diagnosed in dogs as hip or elbow dysplasia.

GENERAL CONSIDERATIONS

A joint should be considered an organ composed of cartilage, subchondral bone, joint fluid, synovium, and associated ligaments. Its function and health are directly connected with the health of numerous other structures and organs, including bones, muscles, and tendons. Diagnosis and treatment of joint disorders are important aspects of veterinary orthopedic practice. Many joint diseases are managed medically rather than surgically, and a basic knowledge of nonsurgical joint diseases is necessary to differentiate surgical and nonsurgical joint disease and prescribe appropriate therapy. Knowledge of normal joint structure and function, the joint's response to injury, and treatment of joint diseases is also essential to selection of appropriate treatment regimens and accurate prognostication.

The common arthropathies of dogs and cats are generally categorized as inflammatory or noninflammatory (Box 34.1). Inflammatory arthropathies are further classified as infectious or noninfectious. Noninfectious arthropathies may be erosive or nonerosive. The common noninflammatory arthropathies in dogs and cats are DJD (which is often due to dysplasia of the hip or elbow or is due to cranial cruciate ligament [CCL] rupture) and those resulting from trauma or neoplasia. Numerous etiologic agents have been associated with infectious arthropathies in dogs and cats, including bacteria, spirochetes (e.g., *Borrelia burgdorferi*), rickettsiae (*Anaplasma phagocytophilum, Ehrlichia ewingii, Neorickettsia risticii,* and *Rickettsia rickettsii*), mycoplasmas, fungi, caliciviruses (cats), bacterial L-forms (cats), and protozoa. Nonerosive, noninfectious arthropathies include idiopathic immune-mediated nonerosive polyarthritis, chronic inflammatory-induced polyarthritis, plasmacytic-lymphocytic synovitis, and arthritis associated with systemic disease (systemic lupus erythematosus [SLE]). Erosive or deforming arthropathies include rheumatoid arthritis (rare in dogs and cats), feline chronic progressive polyarthritis, erosive polyarthritis of greyhounds, and periosteal proliferative arthropathy. The pathophysiologies of the most commonly diagnosed arthropathies are discussed individually in this chapter (see the later discussion). Further information about arthropathies may also be found in most medical texts.

DIAGNOSIS OF JOINT DISEASES

Clinical Presentation

The history and clinical presentation of joint disease vary depending on the arthropathy (see the sections on various arthropathies in this chapter). Dogs are typically presented with either acute or chronic histories of varying degrees of lameness. Joint disease affects dogs of all breeds, age, and size. Osteoarthritis, the most frequently diagnosed arthropathy, is estimated to affect 20% of dogs over 1 year of age. Cats are either affected less often or joint disease often goes unrecognized in this species. Importantly, there is growing evidence that feline osteoarthritis is more common than previously reported.

Physical Examination Findings

Dogs show varying degrees of lameness associated with arthropathies. Muscular asymmetry (between limbs) and joint enlargement may be palpable. Joint enlargement may be due to increased joint effusion, periarticular fibrosis, or osteophytosis. Abnormalities in range of motion, instability, pain, and crepitation during joint manipulations may be noted. For a more complete discussion of physical examination findings, see the sections on individual joints in this chapter.

Diagnostic Imaging

Survey radiography is an important and common method of screening affected joints; however, changes with many diseases may be similar, often making radiography nonspecific. Radiographic findings in affected joints include proliferative or erosive

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ABSTRACT

Joint disease is the leading cause of lameness in small animals. A majority of joint disease is due to dysplastic diseases such as hip dysplasia or elbow dysplasia or degenerative diseases such as cruciate ruptures. Veterinarians should develop an efficient algorithm for the diagnosis or joint diseases and understand the application of different diagnostic tools, including imaging and joint fluid analysis. A thorough understanding of treatment options is important for practitioners whether or not they are performing the procedure themselves or referring.

KEYWORDS

Arthritis
Arthrosis
Dysplasia
Luxation
Subluxation
Suppurative
Degenerative
Cartilage
Synovium

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BOX 34.1 Classification of Arthropathies in Dogs and Cats

Inflammatory

Infectious

Bacteria
Viruses
Rickettsiae
Spirochetes
Fungi
Mycoplasmas
Protozoa

Noninfectious

Erosive

Rheumatoid arthritis
Feline chronic progressive polyarthritis
Erosive polyarthritis of greyhounds
Periosteal proliferative arthropathy

Nonerosive

Idiopathic immune-mediated polyarthritis
Chronic inflammatory-induced polyarthritis
Plasmacytic-lymphocytic synovitis
Systemic lupus erythematosus

Noninflammatory

Dysplasia
Degenerative joint disease
Trauma
Neoplasia

TABLE 34.1 Radiographic Findings of Arthropathies in Dogs and Cats

Categorization[a]	Radiographic Changes
Inflammatory	
Infectious	Subchondral bone may be sclerotic or lytic ± Periarticular bone formation ± Joint space narrowing ± Joint capsule distention and adjacent soft tissue swelling
Noninfectious	**Nonerosive** Soft tissue swelling and joint capsule distention without bony changes; multiple joints affected **Erosive** Joint space collapse; subchondral destruction; periosteal new bone formation along with soft tissue swelling; multiple joints affected
Noninflammatory	
Degenerative joint disease	Soft tissue swelling and intracapsular distention; diminished joint space, periarticular osteophytosis; subchondral bone plate, usually intact but may be sclerotic
Trauma	Depends on the trauma (e.g., fracture, luxation); ultimately, may lead to degenerative joint disease
Neoplasia	Soft tissue swelling and intracapsular distention; destruction of the subchondral bone plate (often on both sides of the joint) with aggressive bone proliferation

[a]Detection of these signs depends on stage of the disease, type of disease, and joint(s) affected.

bone lesions, increased joint fluid, and adjacent soft tissue changes, including muscle atrophy. Radiographic findings help guide clinicians to a definitive diagnosis (Table 34.1); however, absence of radiographic changes does not ensure that the joint is normal. Radiography is particularly insensitive for mild to moderate disease affecting articular cartilage.

Cross-sectional imaging (computed tomography [CT] and magnetic resonance imaging [MRI]) is being more commonly used in evaluation of joint diseases. Both CT and MRI are superior to radiography because they enable visualization of joint structures without superimposition of overlying structures. CT is particularly useful for evaluating bony changes and is often helpful in identifying joint incongruities and fragmentation in osteoarthritic joints. MRI is widely used in the diagnosis of human joint disease and is increasingly being investigated for use in small animal joint diseases, particularly those of the shoulder. The primary indication for MRI is evaluation of menisci or soft tissue structures surrounding diseased joints. Cartilage of small animals is thin, which limits the usefulness of most MRI magnets for diagnosis of cartilage disease. Ultrasonography may also be used to evaluate intraarticular and extraarticular soft tissue structures, particularly in the canine shoulder as well as the meniscus. Bone scintigraphy (bone scan) has been used extensively in small animal orthopedics for localization of joint disease and screening for bone tumors. Although this technique is extremely sensitive for detecting bone abnormalities, it is not specific and not readily available to most practitioners.

Laboratory Findings

Synovial fluid is often evaluated to help differentiate between arthropathies. Cytologic findings range from normal to the presence of phagocytic mononuclear cells, nondegenerative neutrophils, or degenerative neutrophils plus organisms. Cytologic findings may help make a definitive diagnosis (Table 34.2) or may localize disease, but they are typically nonspecific.

TABLE 34.2 Cytologic Findings With Arthropathies in Dogs and Cats

Synovial Fluid Findings	Arthropathies
Phagocytic mononuclear cells	Degenerative joint disease
Nondegenerative neutrophils	Systemic lupus erythematosus Feline chronic progressive polyarthropathy Plasmacytic-lymphocytic synovitis Idiopathic immune-mediated nonerosive polyarthritis Chronic inflammatory-induced polyarthritis Rheumatoid arthritis Infectious arthritis (including bacteria)
Degenerative neutrophils	Bacterial arthritis Rickettsial or spirochetal polyarthritis

Synovial Fluid Collection

Joint taps to obtain synovial fluid are essential for characterizing and differentiating arthropathies. Sedation or general anesthesia is recommended, especially if the animal is fractious (see pp. 961–962). Equipment needed includes sterile gloves, needles

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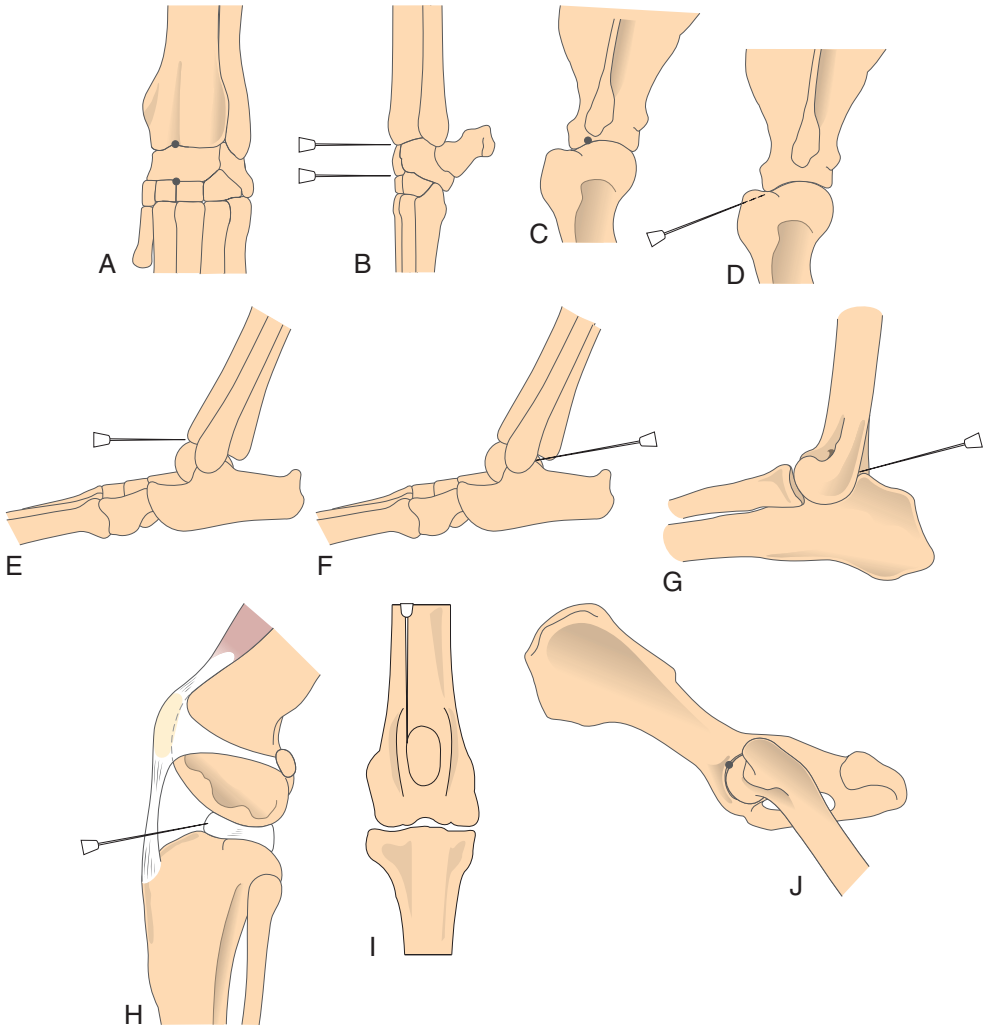


FIG. 34.1 Synovial fluid collection; recommended sites for arthrocentesis in dogs and cats. (A–B), *Carpus:* partly flex the joint. Palpate and enter the craniomedial aspect of the middle carpal or radiocarpal space. (C) *Shoulder (lateral approach):* insert the needle just distal to the acromion process. (D) *Shoulder (cranial approach):* insert the needle just medial to the greater tubercle and ventral to the supraglenoid tubercle of the scapula. (E) *Hock (anterior approach):* palpate the space between the tibia and tibiotarsal bone on the craniolateral surface of the hock; insert the needle in the shallow palpable space. (F) *Hock (lateral approach):* partly flex the joint and insert the needle under the lateral malleolus of the fibula. (G) *Elbow:* insert the needle just medial to the lateral epicondylar ridge, proximal to the olecranon process. Advance parallel to the olecranon process into the olecranon fossa. (H) *Stifle (lateral approach):* insert the needle just lateral to the straight patellar ligament and distal to the patella. (I) *Stifle (dorsal approach):* insert the needle just dorsal to the patella so it passes between the patella and trochlear groove of the femur. (J) *Coxofemoral joint:* abduct and medially rotate the limb. Insert the needle dorsal to the greater trochanter and angle ventrally and caudally.

(25-gauge needles for small joints, 22-gauge 1.5-inch [40 mm] needles for shoulder, elbow, and stifle joints in larger dogs, or 22-gauge 3-inch [80 mm] needles for the hip joint), and 3-mL syringes. Only a small amount of fluid is needed to determine viscosity, estimated cell count, and differential cell count, and for culture.

Synovial fluid aspirates commonly show no bacterial growth when cultured directly on blood agar plates. More reliable results are obtained if the synovial fluid sample is incubated for 24 hours in a blood culture medium or specific enrichment broth before culturing onto blood agar plates (Videos 34.1 and 34.2).

Select the joint or joints that are swollen for initial taps. Clip the appropriate area over the joint and prepare the site for an aseptic procedure (Fig. 34.1A). Use a gloved hand to palpate landmarks. Insert a needle attached to a syringe into the joint. Apply gentle suction to the syringe. After the fluid has been collected, release the negative pressure on the syringe and withdraw the needle. If blood appears in the syringe, withdraw the syringe immediately; contamination with blood can alter cell counts. If only a few drops of fluid are obtained (common in small dogs and cats), spray the material directly onto a slide and examine it cytologically. Estimate viscosity as the fluid drops from the needle to the slide. Normal

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joint fluid is viscous and forms a long string. *Place a drop of fluid on a slide and make a smear for an estimated complete cell count and a differential cell count. Culture fluid for bacterial and mycoplasmal growth: with the same needle and syringe, aseptically aspirate an appropriate culture broth (e.g., trypticase soy broth from a blood culture vial). Rinse the barrel of the syringe and hub of the needle with the culture medium and then inject this culture medium into a blood culture vial with the same medium.*

NOTE Not all joints with arthritis have large volumes of joint fluid. If you can only retrieve a small amount of fluid, it is still important to do both cytology and culture. Spray the joint fluid onto a slide for cytologic evaluation and then rinse the syringe with bacterial culture medium, and place the fluid in a blood culture vial for culture as described previously.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include all of the noninflammatory and inflammatory arthropathies (see Box 34.1). The definitive diagnosis is made by evaluating the animal's history, clinical signs, radiographic findings (see Table 34.1), results of other imaging modalities, and joint cytology (see Table 34.2).

MEDICAL MANAGEMENT

Medical management of specific arthropathies is provided in the later discussion of specific diseases. Specific therapies may include antibiotics based on culture and susceptibility testing and immunosuppressive medications for immune-mediated joint disease. Regardless of cause, for virtually all joint diseases, there are five basic principles of medical management (Box 34.2).

Principle 1: Weight Management

Patients with joint disease can benefit significantly from proper body weight management. Excessive body weight places increased loads on joints, exacerbating concurrent joint disease. Excessive body weight accelerates degeneration of joints with DJD and has been demonstrated to exacerbate clinical signs of osteoarthritis. Excessive body weight also exacerbates signs of dysplastic diseases (e.g., hip dysplasia). Evidence suggests that proper body weight management can lessen and delay clinical signs of osteoarthritis and decrease the need for antiinflammatory medication and surgery.[1]

Recommended body condition scoring (BCS) for dogs is 4.5 on a 1 to 9 scale (Fig. 34.2). Each BCS level above the target level of the 1 to 9 scale represents 10% excess body weight. Weight reduction programs should aim to decrease body weight by 1% to 2% per week until the target BCS is attained. Simplified diet plans recommend feeding 80% to 100% of the caloric resting energy requirements (RER = 70 × [kg of body weight]0.75) for the targeted body weight. When calculating the daily caloric intake, it is critical that all food, including treats, be included.

NOTE Although numerous diet programs are available for the reduction of body weight in cats and dogs, studies suggest that the most important element is repeated body weight evaluation and follow-up with a veterinarian. The importance of counseling and guiding owners in managing their pet's body weight cannot be overemphasized.

Principle 2: Nutritional Supplementation

Omega-3 Fatty Acids

Omega-3 fatty acids are nutritional supplements added to canine diets specifically for management of joint disease. They may be added to commercial foods, or the owner may supplement them. They are antiinflammatory when given at the correct dosage and work by replacing arachidonic acid in cell walls with eicosapentaenoic acid, which decreases pain and inflammation associated with joint injury or osteoarthritis. Omega-3 fatty acids may also help ease inflammation by blocking some of the genes that cause inflammation in osteoarthritic joints.

Studies in dogs suggest that omega-3 fatty acids can ease the pain of osteoarthritis. Recent studies have demonstrated that the use of omega-3 fatty acids can improve weight bearing and decrease the need for nonsteroidal antiinflammatory drugs (NSAIDs) in dogs with osteoarthritis.[2,3] Fatty-acid supplements rarely cause gastrointestinal problems, although sometimes dog owners claim that the supplement has given their dog "fishy-smelling breath." Omega-3 fatty acids appear to be safe.

Slow-Acting, Disease-Modifying Osteoarthritis Agents

Alternative therapies for managing osteoarthritis focus on administering chondroprotective agents to slow cartilage degradation and promote cartilage matrix synthesis. Oral chondroprotective supplements are reported to provide supraphysiologic amounts of glucosamine and chondroitin sulfate to the joints to act as precursors for synthesis of hyaline cartilage matrix. These compounds appear to be safe; they cross the gastrointestinal-blood barrier intact when administered orally and may modify pain associated with osteoarthritis. Meta-analysis demonstrates a moderate level of comfort for the efficacy of a combination of glucosamine, chondroitin, and manganese in the treatment of osteoarthritis in dogs.[1]

Principle 3: Exercise Moderation

Exercise moderation is critical to proper management of joint disease. The type and degree of exercise moderation depend on the stage of the disease, time relative to surgery (if any), and function of the pet. Specific recommendations for exercise moderation for each disease are provided later in the discussion.

Principle 4: Physical Rehabilitation Therapy

Rehabilitation therapy can provide tremendous benefits in the management of small animal joint disease, particularly in dogs (see Chapter 11). The primary targets of physical therapy are strengthening, endurance, and range of motion. Specific recommendations for physical therapy are described in the section on the specific joint diseases.

BOX 34.2 Five Principles of Medical Management of Osteoarthritis

Body weight management
Nutritional supplementation
Exercise moderation
Physical therapy
Antiinflammatory medication

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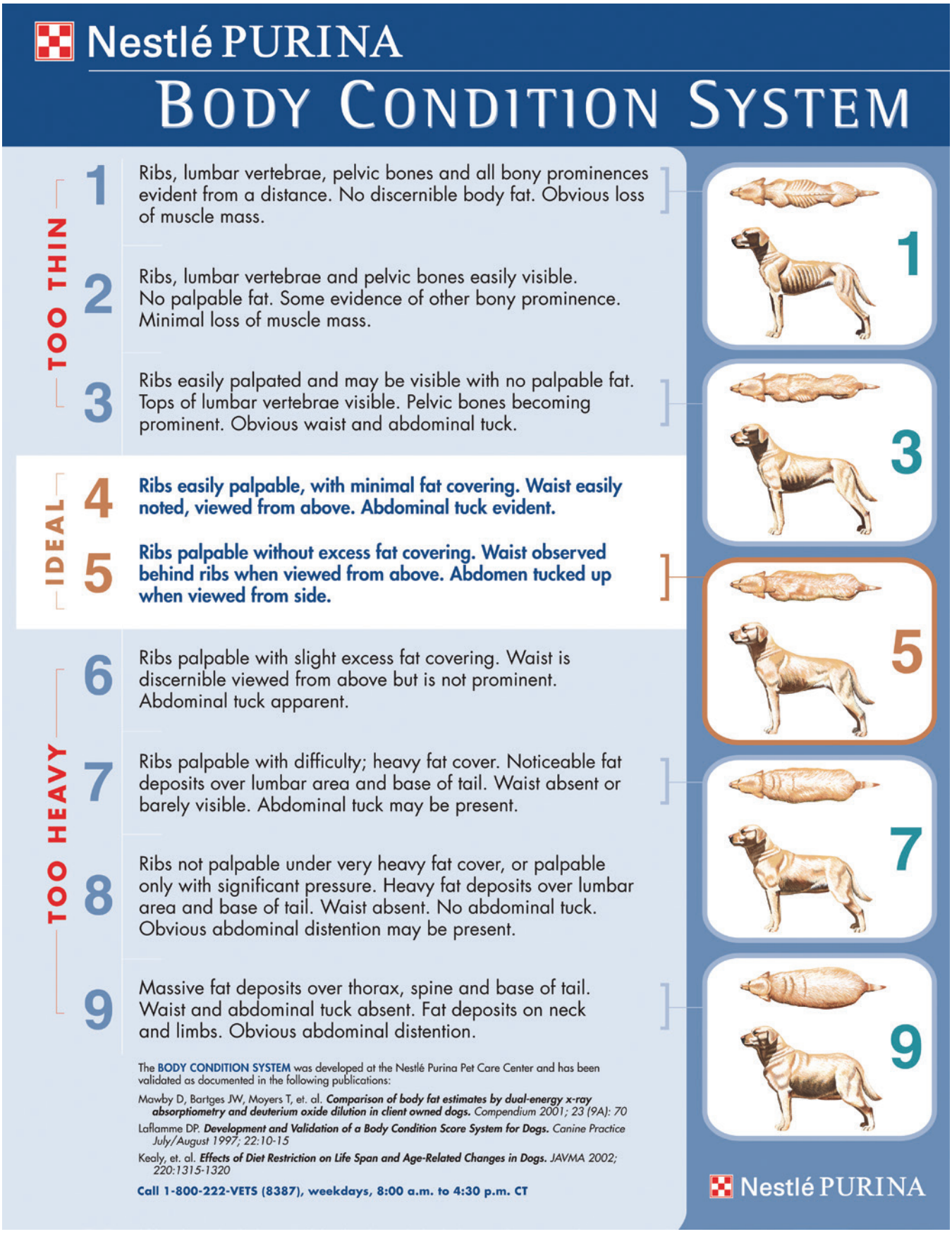


FIG. 34.2 Body condition score table using 1 through 9 categorization.

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NOTE The science of exercise moderation and rehabilitation therapy is expanding rapidly. Veterinarians are encouraged to consult with experts in the field and their publications for the optimum therapy and rehabilitation of patients with joint disease.

Principle 5: Nonsteroidal Antiinflammatory Drug Therapy and Other Medical Therapies

Nonsteroidal Antiinflammatory Drugs

Medical management of DJD often includes therapy with NSAIDs. NSAIDs reduce proinflammatory mediators (e.g., thromboxanes, prostaglandins, prostacyclin, and oxygen radicals) by inhibiting cyclooxygenase-1 and -2 (COX-1 and COX-2) pathways. Inhibiting COX-1 inhibits normal physiologic responses in the gastrointestinal and renal systems. Uses of NSAIDs that significantly inhibit COX-1 (e.g., aspirin, ibuprofen, and phenylbutazone) have greater potential to cause gastrointestinal ulceration and/or nephrotoxicity (Fig. 34.3). Most contemporary veterinary NSAIDs (Table 34.3) are touted to preferentially inhibit COX-2. It was initially believed that these drugs would provide clinical benefit without the risk of the older NSAIDs; however, the mechanisms of action and adverse effects of these drugs are not as clear as was initially postulated. Numerous studies have reported conflicting results regarding the COX-1:COX-2 ratios of these drugs. The method by which an NSAID is determined to affect COX-2 versus COX-1 involves in vitro tests that may or may not accurately reflect what happens in vivo. Presently, it is probably best to say that some drugs appear to be COX-1 sparing instead of COX-2 selective, and it may be more accurate to state that these drugs are "safer" as opposed to saying that they are "safe." There is strong evidence for the efficacy of NSAIDs in lessening the clinical signs of osteoarthritis in dogs.

Side effects of the NSAIDs vary depending on the specific drug and means of reporting adverse events. Owners should be cautioned that antiinflammatory medication may cause gastrointestinal, liver, or kidney disease (see Table 34.3). In dogs, gastric ulceration/erosion tends to be the predominant side effect, whereas in cats, nephrotoxicity seems to be the predominant side effect. Other potential side effects include altered platelet function, extended clotting times, and keratoconjunctivitis sicca. Concurrent administration of drugs to prevent gastric ulceration/erosion is probably not needed in most cases. However, if protracted NSAID therapy is required, or if other factors make gastric ulceration a greater risk (e.g., poor blood perfusion, other ulcerogenic drugs), or if the patient has had repeated clinical signs consistent with gastric ulceration/erosion when treated with NSAIDs, then proton pump inhibitors appear to provide as good a protection against NSAID-induced gastric lesions (Box 34.3) as misoprostol.

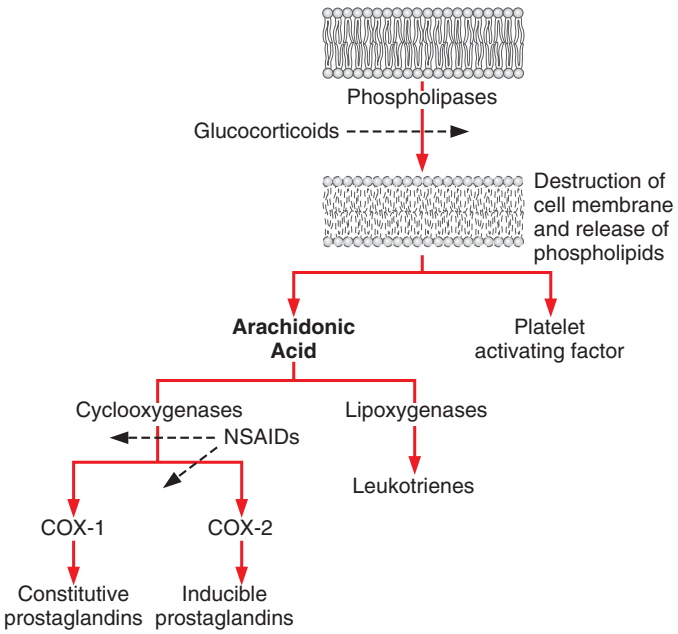


FIG. 34.3 Method of action of glucocorticoids and nonsteroidal antiinflammatory drugs in inhibiting inflammatory pathways. *COX,* Cyclooxygenase; *NSAIDs,* nonsteroidal antiinflammatory drugs.

NOTE Any NSAID can be harmful to dogs, but some (e.g., naproxen, indomethacin, ibuprofen) are especially dangerous and should not be used. The concept of a "wash out" period when switching from one NSAID to another has been widely publicized. There are currently no good data to support this concept; however, it is generally accepted that one should not use two NSAIDs concurrently.

Other Medical Therapies

Compounds such as polysulfated glycosaminoglycans and hyaluronic acid (HA) enhance macromolecular synthesis by chondrocytes and hyaluron synthesis by synoviocytes, inhibit degradative enzymes or inflammatory mediators, and remove or prevent the formation of fibrin, thrombi, or plaques in synovia or subchondral blood vessels. Although there are anecdotal reports of positive results with both polysulfated glycosaminoglycans and HA, no controlled studies have been performed in dogs to establish their efficacy or safety.

Antibiotics

Antibiotics should be administered for infectious arthritis and prophylactically for specific surgical procedures (see Chapter 9). In general, antibiotics should be selected for treatment of bacterial septic arthritis on the basis of identification of the organism and susceptibility testing. Broad-spectrum bactericidal antibiotics should be administered until culture and susceptibility testing results are obtained and then adjusted as necessary (Table 34.4). Gram-positive bacteria are most commonly cultured from septic joints; therefore initial treatment with first generation cephalosporins is usually indicated before culture and bacterial susceptibility reports are received. If bacterial L-forms are suspected (primarily in cats), doxycycline is the antibiotic of choice. Chloramphenicol and erythromycin might also be effective. Antibiotics for these infections should typically be administered for 4 to 6 weeks and

BOX 34.3 Protectants Against Nonsteroidal Antiinflammatory Drug–Induced Gastrointestinal Erosion

Omeprazole (Prilosec)
Dogs: 1–2 mg/kg PO q12h
Cats: 1 mg/kg PO q12h

Misoprostol (Cytotec)
Dogs: 2–5 µg/kg PO q8–12h
Do not give to pregnant animals

IV, Intravenous; *PO,* orally.

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TABLE 34.3 Antiinflammatory Drugs for Treatment of Degenerative Joint Disease in Dogs and Cats

Analgesic	Use/Indication	Route of Administration	Dose
Amantadine	Chronic pain	PO	*Dogs:* 3–5 mg/kg q24h, *or* 2–10 mg/kg q8–12h *Cats:* 3–5 mg/kg q12–24h
Carprofen	Antiinflammatory Mild to moderate pain	SC, PO SC	*Dogs:* 2–4 mg/kg once; 4.4 mg/kg q24h, *or* 2.2 mg/kg q12–24h *Cats:* 4 mg/kg, once
Deracoxib	Chronic pain Acute postoperative pain	PO PO	*Dogs:* 1–2 mg/kg q24h *Dogs:* 3–4 mg/kg q24h, do not give for >7 days
Etodolac	Antiinflammatory	PO	*Dogs:* 5–15 mg/kg q24h
Firocoxib	Antiinflammatory	PO	*Dogs:* 5 mg/kg q24h
Gabapentin	Treat neuropathic pain	PO	The dose is variable depending on the level of pain and may need to be increased over time to be effective. Starting dose: 5–15 mg/kg q12h May increase to: *Dogs:* 40 mg/kg q8–12h (see text) *Cats:* 8–10 mg/kg q8h, or 3 mg/kg q6h (see text)
Grapiprant	Pain and inflammation associated with osteoarthritis	PO	*Dogs:* 2 mg/kg, q24h
Meloxicam	Antiinflammatory	IV, SC, PO SC, PO	*Dogs:* 0.2 mg/kg once then 0.1 mg/kg SC, PO q24h *Cats:* 0.05 mg/kg PO q24h with reduction in dose if long-term treatment is pursued. Long-term treatment may be reduced to 0.05 mg/kg PO q48h and as low as q72h. Single dose of 0.15 mg/kg SC but approved for doses up to 0.3 mg/kg SC.
Pregabalin	Neuropathic pain	PO	*Dogs:* start with 4 mg/kg PO q12h and increase dose gradually if necessary *Cats:* start with 2 mg/kg PO q12h and increase to 4 mg/kg PO q12h
Robenacoxib	Antiinflammatory	SC between the shoulder blades PO	*Cats:* 2 mg/kg *Dogs:* 1.0–2.0 mg/kg q24h *Cats:* ≤3 days (based on weight of cat): 2.5–6 kg; 6 mg/cat q24h 6.1–12 kg; 12 mg/cat q24h *Cats:* 3–11 days: 1 mg/kg q24h

IV, Intravenous; *PO,* orally, *SC,* subcutaneous.

TABLE 34.4 Antibiotics for Septic Arthritis Before Culture Results

Suspected Type	Antibiotic
Gram positive	Cephalexin 22 mg/kg PO, IV q8–12h Amoxicillin-clavulanate 12.5–25 mg/kg PO q12h
Borrelia, rickettsiae, *Mycoplasma,* bacterial L-forms	Doxycycline 5 mg/kg PO q12h, or 10 mg/kg PO q24h
Gram negative	Baytril 5–20 mg/kg PO, IV[a] q24h
Anaerobes	Clindamycin 11–33 mg/kg q12h PO, or 11 mg/kg IV (dilute and give slowly when administering IV) Metronidazole 10 mg/kg PO, IV[a] q12h

[a]Dilute and give slowly over 20 minutes.
IV, Intravenous; *PO,* orally.

at least 2 weeks after the cessation of clinical signs. If *Borrelia* spp., *E. ewingii, N. risticii, A. phagocytophilum,* or *R. rickettsii* is suspected, doxycycline is the antibiotic of choice and should generally be used for 28 days.

Antibiotics may be administered initially when differentiation between immune-mediated and rickettsial infection has not been made; doxycycline is the antibiotic of choice (see Table 34.4). Doxycycline may be administered until tick titer results are available or as a trial to observe for resolution of clinical signs before initiating steroid therapy for presumptive nonerosive immune-mediated joint disease.

Antibiotics should be administered prophylactically to prevent surgical infections, especially in joint replacement procedures, but they are not a substitute for aseptic technique (see Chapters 1, 2, 5, and 6). A broad-spectrum, bactericidal antibiotic, such as cefazolin (22 mg/kg intravenous [IV]), should be administered after an IV line has been placed and anesthesia induced. This dose can be repeated every 90 minutes to 3 hours during surgery. Antibiotics can be discontinued after surgery, or they may be continued until culture results are obtained. If culture results are negative, antibiotics should be discontinued; if results are positive, antibiotics may be continued or changed according to the results of susceptibility testing.

Glucocorticoids

Glucocorticoids effectively reduce synovial inflammation by inhibiting activity of phospholipase A (see Fig. 34.3), reducing production of both cyclooxygenases and lipoxygenases. Glucocorticoids may also protect cartilage matrix by reducing metalloproteinase activity;

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however, they depress chondrocyte metabolism and alter the matrix composition by diminishing proteoglycan and collagen synthesis. Because of the adverse systemic effects of long-term corticosteroid administration and their deleterious effects on cartilage, they are seldom indicated for treatment of cartilage injury or DJD. Glucocorticoids are often used to treat inflammatory noninfectious arthropathies. When used long term, dosages should be titrated to the smallest amount that achieves an effect, and short-acting steroids (e.g., prednisolone) should be administered every other day if possible to prevent suppression of the hypothalamic-pituitary-adrenal axis; however, inadequate dosage may increase resistance to effective treatment. Efficacy of glucocorticoids in the management of immune-mediated joint disease must be based on repeat joint taps and not on clinical evaluation of lameness. Prednisolone is preferred to prednisone, and it must be recognized that dexamethasone is much more ulcerogenic than prednisolone. Routine prophylaxis to prevent gastric ulceration/erosion in dogs treated with glucocorticoids by using proton pump inhibitors is currently of uncertain value. Attempted prophylaxis would seem reasonable in patients that have had gastrointestinal signs consistent with gastric ulceration/erosion while receiving steroids, and patients on long-term and/or high-dose glucocorticoid therapy. It is thought that raising the gastric pH may help lessen bleeding even if it does not prevent ulceration/erosion.

SURGICAL TREATMENT

See individual joints for specific surgical procedures.

SURGICAL ANATOMY

Synovial joints permit motion while providing stability for load transfer between bones (Box 34.4). Synovial joint cavities are surrounded by joint capsules made up of an outer layer of fibrous connective tissue lined with a synovial membrane. Nerves, blood vessels, and lymphatic vessels are located between the synovial membranes and the fibrous capsules. Synovial fluid is formed as a dialysate of plasma from the rich vascular supply of synovial membranes. This fluid filters through the vascular endothelium and synovial interstitium to lubricate the joint and provide nutrition for the articular cartilage. The synovial membrane is composed of synovial A and B cells and dendritic cells. Muco-proteins, such as HA, are added to the fluid by synovial B cells; synovial A cells function as phagocytes and secrete interleukin-1 and prostaglandin E. The articulating joint surfaces are covered with 1 to 5 mm of a dense, white connective tissue, usually hyaline cartilage. This articular cartilage facilitates the gliding motion of the joint, distributes mechanical loads, and prevents or minimizes injury to underlying subchondral bone. Some synovial joints (e.g., the stifle joint) also have intraarticular ligaments, menisci, and fat pads that further facilitate joint function and stress reduction during weight bearing (Fig. 34.4). External joint support is provided by surrounding ligaments and tendons.

Loss of joint stability occurs after ligament rupture (i.e., ruptured cranial cruciate ligament) or secondary to developmental or anatomic abnormalities (i.e., canine hip dysplasia). Abnormal joint motion places abnormal physiologic loads on portions of the articular cartilage. These loads can cause the cartilage matrix to fracture or fissure, disrupting the collagen fibril network and causing cell death. The tissue response to cartilage loss is similar to that which occurs during healing of cartilage defects (see p. 1142). Subchondral bone sclerosis, osteophyte formation, periarticular soft tissue fibrosis, and synovial membrane inflammation are all definitive signs of DJD, and all are physiologic responses to joint instability.

Hyaline cartilage is a dense, white connective tissue made up of chondrocytes (10%) dispersed within an extracellular matrix (90%). Seventy percent of the weight of articular cartilage is fluid. Because articular cartilage is avascular and devoid of nerve endings, it relies on synovial fluid for nutrition. Chondrocytes produce matrix and are most numerous and active during cartilage formation; their numbers and metabolic activity decline with age. The noncellular matrix is composed of collagen, proteoglycans, and noncollagenous proteins. Collagen fibrils embedded in the matrix form a supporting scaffold for cartilage. Proteoglycans are primarily glycosaminoglycan chains (e.g., chondroitin sulfate, keratin sulfate, and HA) that repel each other and help to give form to the cartilage. Interaction between chondrocytes and a matrix is facilitated by the presence of noncollagenous proteins. This macromolecular conglomerate organizes and holds water in the extracellular matrix of articular cartilage.

Adult articular cartilage is categorized into four distinct zones based on cellular morphology and spatial arrangement (Fig. 34.5). The *superficial zone* has a thin, cell-free matrix that provides the gliding surface of articular cartilage. Deep to this

BOX 34.4 Joint Classification

Fibrous Joints

Syndesmosis (e.g., temporohyoid joints)
Sutures (e.g., skull)
Gomphosis (e.g., tooth sockets)

Cartilaginous Joints

Hyaline cartilage or synchondrosis (growth plates)
Fibrocartilage or amphiarthrosis (e.g., mandibular symphysis)

Synovial Joints

Articular hyaline cartilage (e.g., shoulder joint)

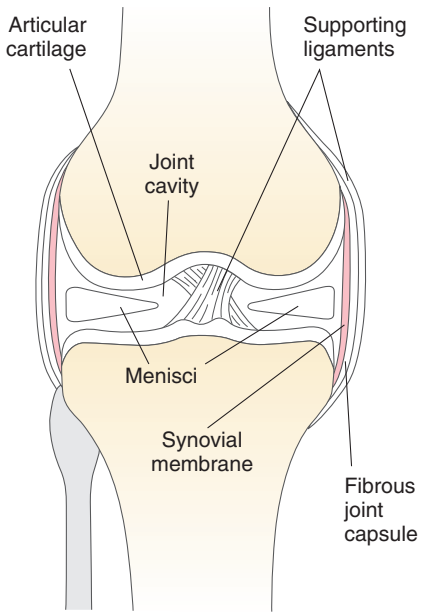


FIG. 34.4 Structure of a synovial joint.

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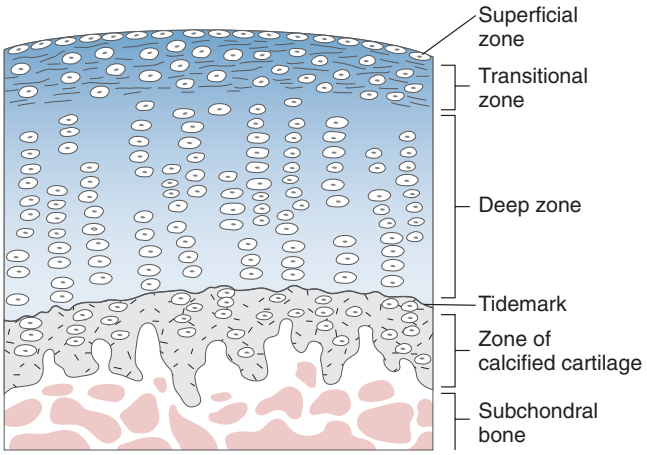


FIG. 34.5 Histologic structure of adult articular cartilage.

layer are thin, elongated chondrocytes oriented parallel to the articular surface. This *transitional zone* is wider than the superficial zone and is made of spherical chondrocytes and matrix with large collagen fibrils. The *deep zone* is the largest zone and contains small chondrocytes that are arranged in short columns perpendicular to the joint surface. The deep zone has the highest proteoglycan content and the least water of the cartilage zones. The *zone of calcified cartilage* is separated from the preceding zones by the "tidemark," which is visible when articular cartilage is stained with hematoxylin and eosin. The zone of calcified cartilage anchors cartilage to subchondral bone (see Fig. 34.5).

Articular cartilage functions as a gliding surface to facilitate joint movement and as a shock absorber to buffer forces applied to long bones during locomotion. The dynamic fluid mechanics of cartilage provide these capabilities. Fluid movement through cartilage plays a fundamental role in (1) augmenting transport of nutrients into and waste products out of cartilage, (2) controlling cartilage deformation, and (3) lubricating joint surfaces during exudation and imbibition of fluid associated with cartilage deformation during weight bearing. Interstitial fluid containing water, metabolites, and small proteins is filtered from synovial fluid and absorbed into the cartilage matrix. This fluid nourishes cartilage cells and adds bulk and resilience to the matrix. As the cartilage deforms with weight bearing, some of this fluid is extruded into the joint, carrying waste products and lubricating joint surfaces. When the load is removed and the cartilage expands, interstitial fluid is resorbed by the matrix.

SURGICAL TECHNIQUE

Arthrotomy

An arthrotomy is an open surgical approach to a joint using traditional surgical instrumentation. Principles of arthrotomy are listed in Box 34.5. Arthrotomies should be based on a thorough knowledge of the local anatomy, particularly the local musculature and neurovascular structures. Standard approaches to the joints are typically recommended. *Make the skin incision in a direction that permits easy retraction of the superficial musculature. When possible, reflect muscles by incising the adjacent fascia versus making an intramuscular incision. If you must incise through the muscles, make your incision parallel to the muscle fibers. Regardless of the technique, be sure that you have adequate exposure to provide proper visualization of the joint. Incise the joint capsule in such a way that it provides exposure while allowing it to be easily closed. At completion of the arthrotomy, close the joint capsule, except in diseases in which release of the joint capsule and adjacent fascia may be necessary for proper alignment (i.e., patellar luxation). The joint capsule is not a holding layer; do not close it with tension.* Closure of the joint capsule does not need to result in a watertight seal because the joint capsule incision will seal with synovial tissue within days of the surgery. Similarly, if the joint capsule cannot be completely closed, a new capsule will form around the joint rapidly. The fascia surrounding the joint capsule is the holding layer and for most joints plays a role in the stability of the joint. Careful tissue layer apposition is important to joint stability and limb function. *Reappose muscles in individual layers to permit proper function, and minimize dead space and seroma formation. Close the remainder of the wound routinely.*

BOX 34.5 Principles of Articular Surgery

- Surgical approaches to joints should be implemented so as to minimize damage to the supportive structures of the joints.
- Damage to the articular cartilage should always be avoided during surgery.
- Although usually attempted, complete closure of joint capsule is not necessary as a synovial layer will rapidly reform, reestablishing the joint capsule.
- In suturing of the joint capsule, it is appropriate to prevent having a nonabsorbable suture within the joint where it may cause chronic irritation, contributing to osteoarthritis.
- Debridement of osteophytes has little value in alleviation of clinical signs of osteoarthritis.

TABLE 34.5 Reasons for Closed Reduction of Traumatic Joint Luxations

Rule	Reason
Closed reduction is generally preferred over open reduction of traumatic luxations of otherwise normal joints	Closed reduction minimizes contamination Closed reduction minimizes anesthetic time Closed reduction lessens iatrogenic soft tissue trauma

Endoscopically Assisted Joint Surgery

General principles of arthroscopy are discussed in Chapter 14.

Closed Versus Open Joint Reduction

Closed reduction of luxated joints is generally preferred to open reduction whenever possible because it minimizes contamination, decreases soft tissue damage, and promotes rapid healing (Table 34.5). When determining if closed reduction and stabilization are appropriate, it is critical to determine if there are underlying diseases (e.g., hip dysplasia, congenital elbow luxation) that significantly decrease the chances of success with closed reduction. Other contraindications to closed reduction include fractures and significant soft tissue interposition.

HEALING OF CARTILAGE DEFECTS AND RESPONSE OF CARTILAGE TO TREATMENT

Loss of proteoglycans from matrix occurs with infection, inflammation, and joint immobilization; when cartilage is exposed

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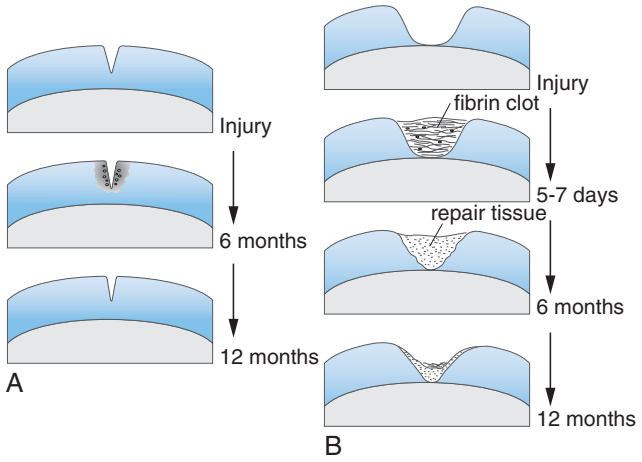


FIG. 34.6 (A) Healing of superficial and (B) deep lacerations of articular cartilage.

during surgery; or as a result of traumatic disruption of synovial membranes. With reversible damage, chondrocytes may replace the lost matrix components after the insult is removed. However, irreversible damage may occur. Lacerations or abrasions of the cartilage surface destroy chondrocytes and disrupt the matrix. A standard inflammatory response does not occur with superficial lacerations (those that do not penetrate to subchondral bone) because inflammatory cells from marrow and blood vessels cannot access the joint (Fig. 34.6A). Chondrocytes near the injury respond by proliferating and synthesizing new matrix; however, this response is usually inadequate for healing. Although superficial lacerations do not heal, they seldom progress. When a full-thickness cartilage defect occurs, marrow cells capable of participating in an inflammatory response gain access to the defect (see Fig. 34.6B). The size of the defect affects healing; small defects (1 mm in diameter) heal more completely than large defects. The defect is initially filled with a fibrin clot, which is replaced within 5 days by fibroblast-like cells and collagen fibers. Metaplasia of these fibroblast-like cells into chondrocytes occurs after 2 weeks. These chondrocytes do not function normally, as is indicated by lower concentrations of proteoglycans in the reparative tissue at 6 months after injury. The reparative tissue (fibrocartilage) is also thinner than articular cartilage and is prone to fibrillation and erosive changes.

Joint Stabilization

Joint stabilization that recreates normal anatomic relationships and permits normal joint function promotes cartilage repair in animals with acute joint instability (i.e., traumatic joint luxations and acute cruciate ligament injuries), if irreversible damage is not present and the cartilage was normal before injury. Dogs with CCL injuries are often presented for treatment after initial changes leading to DJD have occurred. In addition, most surgical techniques for treatment of ruptured CCL injury do not restore normal anatomic relationships or allow normal joint function. Therefore many animals develop progressive DJD despite surgical treatment.

Joint Immobilization

Prolonged immobilization of synovial joints causes progressive proteoglycan loss and depression of proteoglycan synthesis, which leads to softening of the cartilage. When limited activity is allowed, cartilage is restored; however, forced activity after immobility may further damage softened cartilage. Rigid joint immobilization (i.e., that obtained with a transarticular external fixator) results in more severe cartilage degeneration than does less rigid immobilization (i.e., with a cast).

Continuous Passive Motion

Experimentally, early motion and weight bearing are beneficial for repair of full-thickness cartilage defects. Continuous passive motion also appears to accelerate repair of full-thickness cartilage defects, which results in repair tissue that more closely approximates hyaline cartilage. Although continuous passive motion is impractical in small animals, physical rehabilitation provides techniques that may have similar benefits. See Chapter 11 for additional information on physical rehabilitation.

POSTOPERATIVE CARE AND ASSESSMENT

Both medical and surgical management of animals with joint diseases must incorporate nutritional management, physical therapy, and rehabilitation.

Nutritional Management

Nutritional management is important both in modifying developmental skeletal diseases (i.e., hip dysplasia and osteochondrosis) and in managing dogs with osteoarthritis. Avoiding excessive calories and obesity in growing and adult dogs with osteoarthritis will aid in managing their joint disease. Nutrient excesses are risk factors in development of skeletal abnormalities in fast-growing, large-breed dogs. Weight control can help reduce abnormal forces placed on joints and can prevent or delay the onset of symptomatic disease in predisposed patients or help alleviate symptoms in affected patients.

Physical Rehabilitation

Physical rehabilitation is an important component of the treatment of joint diseases. Physical rehabilitation increases muscle strength, endurance, and joint range of motion; reduces muscle spasm and pain; and improves performance. Passive physical manipulation, heat, and cold are important considerations in rehabilitation of joint injuries. For additional information on general principles of physical rehabilitation, see Chapter 11. Supervised, regular short exercise periods of walking or swimming, when the animal is no longer showing signs of inflammation or after surgery to stabilize the joint, help strengthen supporting muscles. Exercise should be avoided when the animal shows signs of acute inflammation and should be limited to prevent discomfort. Effects of a physical rehabilitation program can be measured in terms of increases in muscle circumference, joint range of motion documented with a goniometer, and exercise endurance.

SELECTED NONSURGICAL DISEASES OF JOINTS

DEGENERATIVE JOINT DISEASE

DEFINITION

DJD, or *osteoarthritis*, is a noninflammatory, noninfectious degeneration of articular cartilage accompanied by bone formation at the synovial margins and by fibrosis of periarticular soft tissue. Although classified as noninflammatory, a low-grade (mononuclear), ongoing inflammatory process is associated with this condition.

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GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

DJD may be classified as primary or secondary, depending on the cause. *Primary osteoarthritis* is a disorder of aging in which cartilage degeneration occurs for unknown reasons. *Secondary osteoarthritis* occurs in response to abnormalities that cause joint instability (e.g., CCL rupture) or abnormal loading of articular cartilage (i.e., developmental or anatomic abnormalities, such as hip or elbow dysplasia, or trauma leading to articular malunion), or in response to other recognizable joint disease (e.g., infection and immune-mediated inflammation). Secondary osteoarthritis is more common than primary osteoarthritis in dogs and cats (see Box 34.1). Abnormal joint motion increases the physiologic loads placed on some portions of normal articular cartilage, initiating molecular changes that lead to osteoarthritis. Normal stress on abnormal cartilage (i.e., injured by genetic or metabolic cartilage disorders, inflammation, or immune responses) initiates identical changes. Initially, fibrillation of the superficial cartilage layer results in roughening of the articular surface, with fissures eventually extending to subchondral bone. Free cartilage fragments can initiate an inflammatory response from synovium with production of inflammatory mediators (i.e., cytokines and prostaglandins). Cartilage degradation results from altered chondrocytes, depletion of matrix proteoglycans, and damage to the collagen fibril network. Collagen breakdown is induced by cytokines (i.e., interleukin-1 and tumor necrosis factor) and by upregulation of the release of destructive enzymes (i.e., matrix metalloproteinases and aggrecanase) from chondrocytes, synoviocytes, and inflammatory cells. Affected cartilage is more susceptible to breakdown from the loads of weight bearing. The result is a vicious cycle of inflammation and cartilage destruction. Thus articular fibrillation, cartilage loss, subchondral bone sclerosis, osteophyte formation, periarticular soft tissue fibrosis, and synovial membrane inflammation cause pain and loss of function in osteoarthritis.

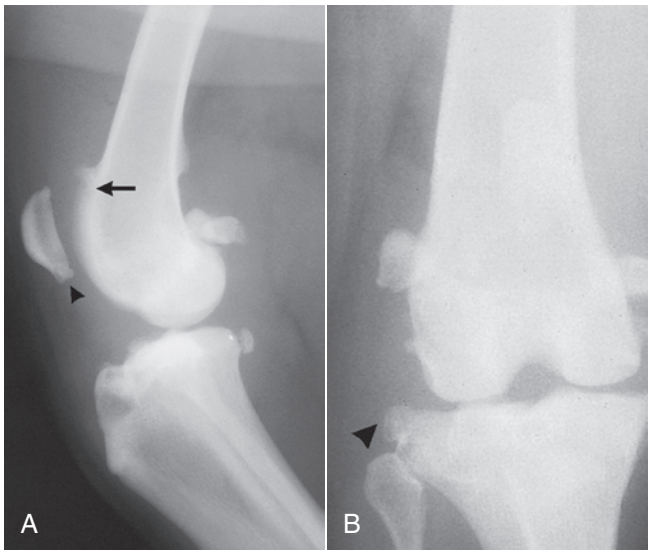


FIG. 34.7 (A) Lateral radiograph of a canine stifle with degenerative joint disease. Note the periarticular osteophytes along the trochlear ridges *(arrow)* and patella *(arrowhead)*. Joint effusion is causing cranial displacement of the infrapatellar fat pad and caudal distention of the joint capsule. (B) On a craniocaudal view, periarticular osteophytes *(arrowhead)* are evident off the lateral aspect of the tibial plateau and distal to the medial fabella. Increased prominence of periarticular soft tissue can be seen medially and laterally.

DIAGNOSIS

Clinical Presentation

Signalment

Osteoarthritis may affect any age or breed of dog or cat. Dysplastic diseases leading to osteoarthritis are often breed specific; however, osteoarthritis caused by trauma is not specific for age or breed.

History

The most common clinical sign of osteoarthritis is lameness, which may be acute or chronic and persistent or intermittent. Most animals have a history of exercise intolerance, particularly when multiple joints are affected (e.g., hip dysplasia). There may be a previous history of joint fractures, osteochondritis dissecans (OCD), congenital or chronic joint luxations, inflammatory joint disease, septic arthritis, and/or neuropathies. Other causes in the forelimbs include fragmented coronoid processes (FCPs) (see p. 1174), ununited anconeal processes (UAPs) (see p. 1188), incomplete ossification of the humeral condyle (IOHC) (see p. 1192), and angular limb deformity (see p. 1079). In the rear limbs, hip dysplasia, aseptic necrosis of the femoral head, patellar luxation, and cruciate ligament rupture may also cause osteoarthritis.

Physical Examination Findings

Unilateral lameness is usually evident when affected animals stand or ambulate. Bilateral conditions (e.g., hip dysplasia) often appear as unilateral lameness if one joint is more severely affected than another. Acutely affected joints may be swollen because of joint effusion, but swelling is more commonly caused by periarticular fibrosis in chronic disease. Reduced range of motion, palpable crepitus during motion, and joint instability are common. Joint palpation may elicit pain.

Diagnostic Imaging

The severity of radiographic change depends on chronicity. Radiographic signs observed include (Fig. 34.7A) subchondral bone sclerosis, articular and periarticular osteophyte formation, joint effusion, greater prominence of periarticular soft tissue (see Fig. 34.7B), and muscle atrophy. Because most radiographic changes are associated with chronic joint changes, osteoarthritis and significant cartilage damage may exist long before radiographic changes are apparent.

Arthroscopy

Arthroscopy typically allows early detection of osteoarthritis. Arthroscopic findings in osteoarthritis include varying grades of cartilage damage and synovial proliferation (Fig. 34.8).

Laboratory Findings

Laboratory abnormalities associated with systemic disease are seldom present with osteoarthritis. Rheumatoid factor, SLE preparations, and antinuclear antibody test results are typically normal. Synovial fluid analysis may reveal decreased synovial fluid viscosity, increased synovial fluid volumes, and increased numbers of mononuclear phagocytic cells (6000–9000 white blood cells [WBCs] per microliter).

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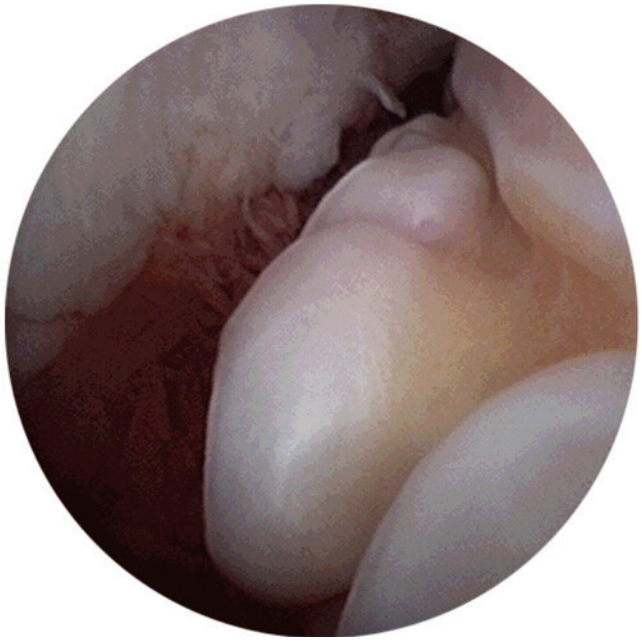


FIG. 34.8 Arthroscopic view of osteoarthritis. Note the osteophytes and synovial proliferation.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for osteoarthritis include septic arthritis, immune-mediated polyarthritis, neoplasia, and joint trauma.

MEDICAL MANAGEMENT

Because osteoarthritis in small animals usually develops secondary to other orthopedic problems, the underlying problem should be corrected if possible. Surgical therapy plus medical treatment is often necessary to reduce or eliminate clinical signs. However, correction of the inciting problem, although necessary to reestablish function, does not usually eliminate or prevent progression of degenerative changes. Correction of the primary problem is not possible or indicated in some animals. Many animals with radiographic signs of osteoarthritis are asymptomatic and may or may not develop lameness as they age. Owners should be apprised of the animal's condition; however, they should be reassured that the animal may function normally with minimal or no treatment.

Inflammatory synovitis is often responsible for clinical signs of pain and lameness associated with osteoarthritis. Physical rehabilitation can play a key role in strengthening the periarticular structures, which will lead to improved comfort, stronger musculature to support compromised joints, and increased functional range of motion. These lifestyle changes can significantly improve comfort and quality of life for these patients (see also Chapter 11). Because excessive or uncontrolled exercise exacerbates and prolongs inflammation, rest for 2 to 3 days plus judicious use of antiinflammatory drugs may be necessary to break the cycle and return the animal to controlled levels of activity.

NOTE Do not treat asymptomatic dogs just because they have radiographic signs of osteoarthritis.

Weight Management and Exercise/Physical Therapy

Many dogs showing clinical signs of osteoarthritis are obese. Obesity may be a causative or inciting factor in the development of osteoarthritis, or it may be a response to chronic pain. Dogs that are reluctant to exercise may gain weight if food intake is not regulated. Weight loss or weight control for a dog with osteoarthritis should be stressed to the owners. Weight loss can be associated with diminished pain and increased function in animals with chronic osteoarthritis. Once the inflammatory phase of disease is under control, moderate regular exercise (not severe enough to cause lameness) is important to maintain joint mobility, muscle strength, and joint support. Swimming appears to maintain muscle strength without increasing joint loads.

Medication

Veterinary NSAIDs are effective for relieving pain associated with osteoarthritis and generally have few clinically obvious gastrointestinal side effects (see Table 34.3); however, gastric ulceration/erosion, hepatocellular necrosis, and renal dysfunction may occur with any NSAID. Cats tend to have more difficulty metabolizing these drugs through glucuronidation, and the result is that they often have a longer half-life than in dogs. Using NSAIDs designed for humans may be especially dangerous. In dogs, gastrointestinal ulceration/erosion tends to be the predominate side effect, and it may be asymptomatic or manifested by anorexia, vomiting, diarrhea, anemia, dyspnea, and/or melena. It is noteworthy that many dogs with substantial gastric ulceration/erosion have anorexia as the sole clinical signs. If any of these signs are noted, NSAID use should be discontinued to prevent worsening of the disease. In cats, renal dysfunction tends to be the more serious side effect of NSAIDs.

Glucocorticoids have antiinflammatory effects on joint tissues; however, they depress chondrocyte metabolism and alter cartilage matrix, which causes decreased proteoglycan and collagen synthesis (see p. 1140). Because of this and the adverse systemic effects of long-term glucocorticoid administration, they should be used in osteoarthritic animals only as a last resort. If they are used, they should be administered infrequently, and the animal should be restricted from exercise for several weeks after treatment to protect the cartilage.

NOTE Always counsel owners about side effects of NSAIDs and steroids and especially the combination of the two. These drugs should not be given together unless there are specific reasons necessitating such an approach. The owners need to be counseled about the risks of such combination therapy.

SURGICAL TREATMENT

Surgical treatment of the underlying disease may be indicated to control signs of osteoarthritis (e.g., stabilization of stifles with CCL ruptures). Uncontrollable pain and loss of limb function associated with osteoarthritis warrant surgical intervention. Several surgical procedures are available to salvage limb function. Arthroplasty techniques, such as femoral head and neck ostectomy (FHO; see p. 1213), have been described in veterinary patients. After resection of bone, a fibrous tissue joint, or pseudoarthrosis, is allowed to form. Function varies after these procedures. Although the joint mechanics are not normal, in many cases limb function is satisfactory. Replacement of an arthritic joint

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with prosthesis improves the chances for normal limb function and superior pain relief. Prosthetic joint replacements are available for the hip, elbow, and knee in dogs, although the success rates vary tremendously with the joint being replaced. The biomechanical nature of the elbow joint makes successful design and placement of a prosthetic joint significantly more complicated than a total hip replacement (THR). Knee and elbow replacements are readily available, although no long-term outcome information is available. Hip replacement has been described in the cat. Arthrodesis (surgical fusion of the joint) can relieve pain and allow good function of the limb when used to treat carpal or tarsal instability. Arthrodesis of the elbow, knee, or shoulder results in fair to poor function. Amputation is a salvage procedure that should be considered only in selected patients—those that are non–weight-bearing and have unilateral disease causing unrelenting pain that has been unresponsive to other therapies. Joint allografts have been performed in a limited number of patients with fair results.

Preoperative Management

See previous Medical Management section. Specific preoperative treatment depends on the underlying disease.

Anesthesia

Anesthetic management of animals with orthopedic disease is provided in Tables 32.1 and 32.2.

Surgical Anatomy

Surgical anatomy of the joints is provided under the specific procedure.

Positioning

Positioning depends on the procedure being performed (see the later discussion).

SURGICAL TECHNIQUE

For arthroplasty techniques (i.e., FHO and glenoid cavity resection), see pp. 1164 and 1213.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Suture materials and special instruments required for joint surgery are listed under the specific procedure.

POSTOPERATIVE CARE AND ASSESSMENT

For postoperative care after joint surgery, see the specific procedure.

PROGNOSIS

Prognosis for patients with osteoarthritis varies tremendously depending on severity of the disease, number of joints affected, and medical condition of the patient. In many cases of mild to moderate osteoarthritis, proper medical management can return the patient to near normal function. In more severe cases, particularly with multiple joint involvement, the prognosis for return to normal activity is guarded. However, in most cases, with the exception of severe end-stage osteoarthritis, function as a house pet can be restored. Important considerations for management and prognosis of animals with osteoarthritis are listed in Box 34.6.

BOX 34.6 Important Considerations in Degenerative Joint Disease

- In most instances of degenerative joint disease (osteoarthritis) in dogs, a primary joint problem (e.g., hip dysplasia, fragmented coronoid process, or ruptured cruciate ligament) was the inciting cause. The underlying disease must be diagnosed and treated appropriately.
- Osteoarthritis is usually progressive, regardless of treatment.
- Diagnosis of osteoarthritis is based on radiographic evaluation, but treatment is based on clinical signs.
- Medical management of osteoarthritis includes weight control, exercise, or physical therapy to maintain joint mobility and muscle mass, rest during acute clinical signs, and medication.

SEPTIC (BACTERIAL) ARTHRITIS

DEFINITION

Septic arthritis *(infective arthritis, suppurative arthritis)* is joint infection caused by bacterial organisms.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Septic arthritis may be due to hematogenous spread of infection from respiratory, digestive, urinary, umbilical, or valvular infections, but more often it is caused by direct bacterial inoculation of joints from penetrating trauma (e.g., cat bite), surgical procedures, or intraarticular injections. Common organisms include staphylococci, streptococci, *Corynebacterium* spp., and coliforms.[4] Bacterial contamination of synovium causes inflammation and promotes extravasation of fibrin, clotting factors, polymorphonuclear leukocytes, and proteinaceous serous fluid into the joint. Fibrin deposition on articular cartilage surfaces inhibits synovial fluid penetration. Leukocytes phagocytize bacteria and release lysosomal enzymes that break down cartilage matrix and expose collagen fibrils to further enzymatic destruction. Enzymatic destruction of matrix and collagen fibrils, loss of normal synovial fluid nutrition, and mechanical trauma on diseased cartilage cause loss of the cartilaginous joint surface. Eventually the infective process invades subchondral bone, causing bacterial osteomyelitis.

Severity of the pathologic changes in affected joints depends on the chronicity of infection. Early changes include hyperemia, edema, synovial inflammation, and a purulent-appearing joint fluid. Both hyperplasia and hypertrophy of synovial cells occur. Cartilage fibrillation and granulation tissue production within the joint soon ensue with loss of articular cartilage. The articular surface fills with granulation and fibrous tissue; eventually these tissues calcify, causing ankylosis. Septic arthritis may be suspected based on radiographic findings (see Table 34.1). Diagnosis of septic arthritis is based on synovial fluid analysis (see Table 34.2) and positive results on bacterial culture.

Osteoarthritis is known to increase the risk of septic osteoarthritis in people and likely increases the risk in dogs.[5] Diseases such as canine hip dysplasia may increase the risk of septic arthritis, and this diagnosis should be considered when examining patients with hip pain and severe osteoarthritis.

DIAGNOSIS

Clinical Presentation

Signalment

Septic arthritis can be found in any aged dog, but males of larger breeds are more commonly affected.

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BOX 34.7 Clinical Signs of Septic Arthritis

Acute
- Single-limb lameness
- Severe lameness, may carry the limb
- Joint swollen, painful, and warm

Chronic
- Single-limb lameness or multiple-limb lameness with subacute endocarditis
- Weight-bearing lameness
- Joint swelling

History

Animals with septic arthritis after joint inoculation usually show a notable unilateral lameness (Box 34.7). Onset of clinical signs may be acute or gradual. Penetrating wounds, surgical intervention, or joint injection are often historical findings. Dogs with septic arthritis from septicemia (e.g., bacterial endocarditis) usually have multiple limb involvement and no history of joint intervention.

Physical Examination Findings

Animals with acute onset of signs are often severely lame or non–weight-bearing on the affected limb. The affected joint may be swollen, painful, warm, and crepitate; drain purulent material; and have reduced range of motion. Systemic signs (pyrexia, lethargy, and anorexia) occur in a small percentage of animals with septic arthritis. The signs may be subtle (i.e., only lameness and joint swelling) with low-grade, chronic infections. Dogs with septic arthritis caused by bacterial endocarditis typically have multiple joint involvement, lameness, pyrexia, lethargy, anorexia, and/or cardiac murmurs.

Diagnostic Imaging

Early radiographic signs of septic arthritis are joint effusion and soft tissue swelling. Later changes include bone lysis, periosteal new bone formation, joint surface irregularities, subchondral bone sclerosis, and joint subluxation (Fig. 34.9). Echocardiography may demonstrate valvular lesions of bacterial endocarditis in dogs without heart murmurs; however, it is important to note that bacterial endocarditis can be associated with septic or nonseptic arthritis/polyarthritis.

Laboratory Findings

Complete blood cell counts (CBCs) may indicate an inflammatory response in acute stages of septic arthritis. Chronic disease is essentially walled-off within the joint and is seldom associated with significant changes in hemograms or serum biochemistry profiles. Joint fluid obtained by means of arthrocentesis is the most sensitive/specific way to achieve a definitive diagnosis. Finding bacteria cytologically or via culture is definitive of septic arthritis. Culture of bacteria in particular allows susceptibility testing and optimal antibiotic selection. Results of bacterial culture of synovial fluid from septic joints vary, but positive results can be facilitated by securing a sample before antibiotics are administered and by using enrichment broth to facilitate bacterial growth from synovial fluid. Culture of surgically retrieved synovial samples may be necessary to grow bacteria. Blood and urine cultures sometimes grow the organism(s) when joint fluid does not. Purulent joint fluid with an increased number of polymorphonuclear leukocytes (40,000 cells/μL to more than 100,000 cells/μL), especially if they are degenerative, is very suggestive; however, septic joints do not always contain degenerative neutrophils.



FIG. 34.9 Craniocaudal radiograph of a stifle joint with septic arthritis. There is extensive osteolysis of the femur and tibia. The subchondral margins of the joint surfaces are irregular and difficult to identify.

NOTE Bacterial endocarditis may occur in dogs that do not have heart murmurs. Be sure to perform echocardiograms in animals with septic arthritis (especially if multiple joints are affected).

DIFFERENTIAL DIAGNOSIS

Other infectious agents causing arthropathies (e.g., rickettsiae, fungi, mycoplasmas, and protozoa) must be differentiated from bacterial causes. Noninfectious causes of arthropathy, including joint neoplasia, immune-mediated polyarthritis, and osteoarthritis, also must be differentiated from infectious causes. Samples of joint fluid and synovium for bacterial culture and susceptibility testing should be obtained to identify causative organisms and determine appropriate antibiotic therapy.

MEDICAL MANAGEMENT

Treatment options for management of septic arthritis include administration of antimicrobials only, administration of antimicrobials plus surgical lavage, administration of antimicrobials plus arthroscopic lavage, administration of antimicrobials plus surgical or arthroscopic lavage and drain placement, or administration of antimicrobials and open joint management. Antibiotics should be administered immediately after samples for culture are obtained, and antibiotic therapy should be adjusted after microbial susceptibility tests are obtained. Because of the high

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BOX 34.8 Treatment of Rickettsial/Anaplasmal Polyarthritis and Bacterial L-Forms

Doxycycline
10 mg/kg PO q24h[a,b]

Chloramphenicol
50 mg/kg PO q8h (can rarely cause fatal aplastic anemia in people; therefore do not eat or drink while handling this product; do not touch eyes; wear gloves; wash hands after use; warn owners of same)

Enrofloxacin[c]
3 mg/kg PO or IV q24h

[a]Must not be given with dairy products.
[b]Must wash down with water or food because retention of medication in esophagus may cause esophagitis +/– benign esophageal stricture.
[c]Specifically for L-forms.
IV, Intravenous; *PO,* orally.

incidence of gram-positive infections, first-generation cephalosporins are often recommended until susceptibility results are available (see Table 34.4). If bacterial L-forms are considered likely, doxycycline is the drug of choice (Box 34.8). Antibiotics are administered for 4 to 6 weeks and for at least 2 weeks after clinical signs have resolved. It is important to remember that tetracycline drugs have some antiinflammatory activity, so care must be taken when ascribing cause and effect to an arthritic patient that improves after receiving doxycycline or tetracycline. NSAIDs are often necessary to alleviate pain in affected joints.

SURGICAL TREATMENT

Goals of surgical treatment are to control infections rapidly and remove purulent material and fibrin from joints to minimize cartilage destruction. Immediate joint exploration, including debridement of fibrin and granulation tissue and sterile saline lavage, is indicated for postoperative joint infections, septic joints for which treatment was delayed for 72 hours or longer, joints that have not responded to 72 hours of appropriate medical therapy, and penetrating joint wounds. Surgery is not indicated in dogs with inflammatory joint disease that has occurred secondary to bacterial endocarditis.

Preoperative Management

The overall health of the animal should be determined before surgery. Most patients with septic arthritis are otherwise healthy; however, some patients may have secondary systemic effects, including dehydration and systemic inflammation. Antibiotics should not be administered until fluid has been obtained for culture and susceptibility testing.

Anesthesia

General anesthesia is required for exploration of the joint. Anesthetic protocols should be based on signalment, physical examination findings, and laboratory analysis. Patients who are brought in without preoperative findings suggestive of major organ dysfunction or systemic effects of joint infection can be managed using a variety of anesthetic techniques (see Table 32.1). Anesthetic protocols for management of traumatized animals with orthopedic disease are provided in Table 32.2.

Surgical Anatomy

Surgical anatomy of specific joints is provided elsewhere in this chapter.

Positioning

Positioning for arthroscopy or arthrotomy depends on the joint being evaluated (see sections on specific joints).

SURGICAL TECHNIQUE

Exploration of the joint may be performed either with a traditional arthrotomy or by arthroscopy (see sections on specific joints). *Aggressively lavage the joint and remove fibrin deposits. In severe cases, perform synovectomy.* When performing arthroscopic debridement, adequate lavage is provided by routine arthroscopic fluid lavage. *Perform debridement with a power shaver. After arthrotomy and joint lavage, install a system of ingress and egress drains, if necessary, so that joint lavage can be performed two or three times daily.* In most cases, only egress drains are required. Joint drains routinely clog with synovium and fibrin debris. Synovectomy may aid in preventing drain clogging. Use of a proper drain—such as a flat, multifenestrated, closed suction system—also aids in maintaining drainage. *Avoid use of Penrose drains because they may encourage retrograde contamination. In severe cases, leave the joint incision open for daily lavage; heavy sedation or general anesthesia is generally needed. Replace sterile bandages once or twice a day, as needed. Close the joint after 2 to 4 days of open drainage.* This type of management is extremely time-intensive, expensive, and rarely necessary. *When performing ingress-egress lavage, pay strict attention to sterile technique. For lavage fluid, use saline or a balanced electrolyte solution, either of which has minimal effects on joint tissue.* Povidone-iodine at a concentration of 0.1% has minimal adverse effects on synovium. However, stronger concentrations of povidone-iodine and chlorhexidine cause a chemical synovitis. *Perform ingress-egress flushing for 1 or 2 days. Maintain the egress drain until drainage is limited to that caused only by the presence of the drain itself, which usually amounts to a few milliliters per day.*

NOTE Acute septic arthritis is a surgical emergency.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Absorbable sutures are recommended for closure of the joint capsule. Closed suction drains are valuable for drainage of septic joints.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative care for animals with septic arthritis consists of oral antibiotics, analgesics (see Chapter 13), daily wound management until joint drainage is no longer purulent, and passive range of motion. Oral antibiotics should be continued for 4 to 6 weeks. Physical rehabilitation by a trained practitioner is strongly recommended due to the highly destructive nature of infection on the joint. Joint flushing and range of motion exercises are usually quite painful in septic osteoarthritis patients; therefore adequate pain management with opioids or NSAIDs is indicated.

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PROGNOSIS

Prognosis for normal joint function varies and depends on the amount of cartilage that has been destroyed; however, most dogs with bacterial septic arthritis respond well to treatment and are free of clinical signs of the disease at follow-up. Salvage procedures for chronically painful joints include arthrodesis or amputation. Joint replacement may be possible following resolution of the infection, although the risk of a repeat infection is significantly increased. The prognosis for animals with bacterial endocarditis is poor if there is impending cardiac failure secondary to valvular dysfunction, especially aortic valvular insufficiency.

RICKETTSIAL AND ANAPLASMAL POLYARTHRITIS

DEFINITION

Polyarthritis may be caused by obligate intracellular microorganisms of the families Rickettsiales (i.e., *Rickettisia rickettsii)* and Anaplasmataceae (i.e., *Ehrlichia* spp., *N. risticii*, *A. phagocytophilum*).

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Polyarthritis in dogs may be caused by *A. phagocytophilum, N. risticii, E. ewingii*, or *R. rickettsii*. *R. rickettsii* (the cause of Rocky Mountain spotted fever [RMSF]) is transmitted by ticks.[6] These agents have been observed in much of the United States, but especially in the southeastern portions. The reader is referred to a medical text for a more complete discussion.

DIAGNOSIS

Clinical Presentation

Signalment

Any breed dog of any age can be affected. Dogs that are exposed to ticks (e.g., hunting dogs) may be more likely to become infected.

History

RMSF is most commonly diagnosed from March through October in endemic areas. It typically causes an acute onset of symptoms, including multiple-limb lameness, joint pain, fever, petechial hemorrhages, lymphadenopathy, neurologic signs, facial edema, and edema of the extremities. Arthritis is typically just one aspect of a much larger picture of a dog with a serious systemic illness.

Ehrlichiosis and anaplasmosis have a great breadth of clinical findings, ranging from acute to subclinical to chronic with signs varying from relatively mild (e.g., anorexia, depression, fever, or weight loss) to dramatic (e.g., epistaxis) to severe (e.g., seizures, hyperesthesia, and stupor).

Physical Examination Findings

Clinical signs associated with *E. ewingii* are usually acute and can include fever, depression, lethargy, neurologic abnormalities, and/or arthritis. *A. phagocytophilum* usually causes acute disease as seen by fever, lethargy, anorexia, depression, lymphadenopathy, splenomegaly, hepatomegaly, and/or arthritis. Clinical findings in RMSF classically include fever, cutaneous edema and/or hyperemia, scrotal edema, stiff gait, petechiation, and/or central nervous system aberrations.

Diagnostic Imaging

Radiographic changes of rickettsial polyarthritis include joint effusion and periarticular soft tissue swelling. Surfaces of bones and cartilage usually appear normal. Occasionally a pulmonary interstitial pattern is noted. Splenomegaly and/or hepatomegaly can be seen.

Laboratory Findings

Thrombocytopenia is typically present in infections by all of these agents. Joint fluid analysis usually reveals a neutrophilic synovitis and/or arthritis. Diagnosis of these polyarthritides is usually based on serologic or DNA testing (rarely on finding morula) plus other evidence of the disease during historical, physical, and clinicopathologic evaluation. Polymerase chain reaction is the most definitive test for *E. ewingii* and *A. phagocytophilum*. However, all of these agents typically respond well to doxycycline therapy, making the need for specific, definitive diagnosis unimportant in most cases. The reader is referred to a medical text for a more complete discussion of serology and molecular diagnosis of these various tick-borne agents affecting dogs.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for rickettsial/anaplasmal joint infections include joint neoplasia, bacterial infection, and immune-mediated polyarthritis.

MEDICAL MANAGEMENT

Doxycycline for 28 days (see Box 34.8) is typically the preferred treatment of tick-borne polyarthritis, regardless of the agent responsible. Chloramphenicol is also effective and is especially desirable when treating puppies less than 6 to 12 months of age (to avoid discoloring the teeth). Sometimes it is helpful to also administer short-term glucocorticoids to patients with severe ehrlichiosis because there may be an immune-mediated component to the disease in these animals. Limiting exposure to ticks by removing ticks daily from dogs is important in preventing future infections.

SURGICAL TREATMENT

Surgical therapy is not indicated for these tick-borne polyarthritis.

PROGNOSIS

Dogs treated for *Ehrlichia* and *Anaplasma* infections generally respond well to appropriate medical therapy. Prognosis for dogs with RMSF varies, depending on clinical signs and promptness of the diagnosis. RMSF is a potential zoonosis, so use appropriate precautions.

LYME ARTHROPATHY

DEFINITION

Lyme arthropathy is a form of polyarthritis caused by *B. burgdorferi,* a gram-negative bacteria of the spirochete class.

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GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

B. burgdorferi infection should be suspected in dogs with transient or recurrent arthritis if they have been in an area endemic for borreliosis. Endemic foci exist in northeastern United States, northern California, and the Midwest.[7] The disease can be transmitted by larval, nymphal, and adult *Ixodes* ticks. A feeding time of 24 to 50 hours is necessary for the tick to transmit the spirochete. Septicemia then occurs, with spread to target organs. Any dog that has the potential for contact with vectors of the disease may be infected; however, joint signs may occur months after exposure. Although most humans with exposure to *B. burgdorferi* will demonstrate clinical signs, 95% of exposed dogs will remain asymptomatic. There is currently little evidence that cats develop disease from these agents. Clinical signs may be intermittent, with animals appearing normal between acute exacerbations; episodes may include multiple-limb lameness, fever, lymphadenopathy, and anorexia.

The efficacy of vaccination against Lyme disease is not absolute. In a consensus statement by the American College of Veterinary Internal Medicine, all respondents agreed that the vaccine is unnecessary in nonendemic locations and the majority of respondents did not recommend the vaccine in endemic areas either. Tick control and avoidance is important in minimizing the risk of Lyme disease and other tick-borne illnesses. The reader is referred to a medical text for a more complete discussion of vaccination/prevention.

DIAGNOSIS

Clinical Presentation

Signalment

Dogs of any age or breed may be affected. Dogs that are in endemic areas and are exposed to ticks are most likely to be infected.

History

Dogs with Lyme disease can have acute fever, shifting leg lameness, anorexia, and/or depression. A history of being exposed to ticks in an endemic area is important.

Physical Examination Findings

Although clinical disease may be transient, the pathologic joint condition may be progressive. Clinical signs may be intermittent, with animals appearing normal between acute exacerbations; episodes may include multiple-limb lameness, fever, lymphadenomegaly, and anorexia.

Diagnostic Imaging

Radiographs of affected joints may show effusion during the acute phase of the disease.

Laboratory Findings

Diagnosis is very difficult because most dogs develop a subclinical infection and then have antibody titers that persist for years. Detection of *B. burgdorferi* by culture or polymerase chain reaction has good sensitivity and specificity, but it is often difficult, time-consuming, and/or expensive. There are no hematologic or serum biochemistry panel findings that are particularly useful in diagnosing borreliosis (mild to moderate thrombocytopenia is common, but nonspecific; Lyme nephritis sometimes develops and causes renal failure). Synovial fluid may be normal, or it may be less viscous and contain 5000 to 10,000 nondegenerate neutrophils/μL. Synovial biopsy typically shows invasion of the synovial lining with plasmacytes and lymphocytes.

Serology for borreliosis is potentially confusing because there are numerous tests, each with its own nuances. As a result, borreliosis is probably overdiagnosed. It is important to note that serology alone is not sufficient for diagnosis because clinically normal animals may have positive serologic tests. The reader is referred to a medical text for a more complete discussion of serology and diagnosis of borreliosis.

NOTE Positive serologic findings reflect exposure to the organism, not necessarily active infection.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include other inflammatory, nonerosive, and erosive arthropathies (e.g., immune-mediated polyarthritis, septic arthritis, rheumatoid arthritis, osteoarthritis) and joint neoplasia.

MEDICAL MANAGEMENT

Doxycycline (28 days) is generally effective for treating Lyme disease (Box 34.9). Amoxicillin is often used in young animals in which doxycycline can stain the teeth. Azithromycin, ceftriaxone, and cefotaxime also have been used (see Box 34.9). NSAIDs can be used to control joint pain, if necessary. Patients with acute disease generally respond clinically within 2 days of beginning therapy.

SURGICAL TREATMENT

Surgery is not indicated for this disease.

PROGNOSIS

Dogs treated promptly for acute infections generally have an excellent prognosis. The prognosis for dogs with chronic infections is uncertain, but patients with chronic renal disease (i.e., Lyme nephritis) caused by *B. burgdorferi* have a poor prognosis. The

BOX 34.9 Treatment of Lyme Disease in Dogs

Doxycycline (Vibramycin)
10 mg/kg PO q24h for a minimum of 1 month

Amoxicillin
22 mg/kg PO q12h

Azithromycin (Zithromax)
10 mg/kg PO q24h for 5–7 days and then decrease to q48h

Ceftriaxone (Rocephin)[a]
50 mg/kg, IM or IV q12h

[a]In human beings, this drug is typically reserved for central nervous system disease.
IM, Intramuscular; *IV,* intravenous; *PO,* orally.

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spirochete may survive antibiotic treatment, and the disease can possibly be reactivated in immunosuppressed animals.

NONEROSIVE IDIOPATHIC IMMUNE-MEDIATED POLYARTHRITIS

DEFINITION

Nonerosive idiopathic immune-mediated polyarthritis is a noninfectious joint disease of unknown etiology. The term *canine idiopathic polyarthritis* is sometimes used for this condition.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Idiopathic nonerosive inflammatory arthropathies have no identifiable cause and are diagnosed by ruling out all other causes of polyarthritis, septic arthritis, rickettsial arthritis, rheumatoid arthritis, other inflammatory nonerosive polyarthropathies, and osteoarthritis. The cause is unknown but presumed to be rooted in immune complex formation.[8] The synovium is thickened, congested, and edematous and may contain fibrin deposits. Cartilage and bone are relatively unaffected; however, superficial fibrillation of the articular cartilage is an occasional finding.

Idiopathic immune-mediated nonerosive polyarthritis is diagnosed by synovial fluid analysis and negative culture, by joint radiographs that do not show erosive or proliferative bone lesions, and by eliminating other known causes. A therapeutic trial of antibiotics is sometimes used to help eliminate occult infectious causes, but it must be remembered that tetracyclines such as doxycycline (which are commonly used) have antiinflammatory properties that can make it difficult to accurately decipher cause-and-effect in such cases. The reader is referred to a medical text for a more complete discussion of this disease.

DIAGNOSIS

Clinical Presentation

Signalment

All breeds and ages of dogs are susceptible to immune-mediated polyarthritis. Some studies have suggested breed predilections in German shepherds, Doberman pinschers, collies, spaniels, retrievers, terriers, and poodles. Females are more commonly affected.

History

Stiffness, difficulty rising, pyrexia, anorexia, and/or lethargy may be present. Although more than one joint is usually involved, single-limb lameness is common. Affected animals may or may not have difficulty rising and walking. Some animals have a chronic fever of unknown origin.

Physical Examination Findings

Joint palpation may reveal pain, effusion, or loss of range of motion. It is important to note that joint effusion may be absent. Cervical pain and vertebral hypersensitivity may reflect intervertebral involvement. Other systemic abnormalities (dermatitis, glomerulonephritis, uveitis) may be found.

Diagnostic Imaging

Radiographs of affected joints usually reveal either no abnormalities or synovial fluid effusion and periarticular soft tissue swelling.

Laboratory Findings

Hematology often reveals a nonspecific neutrophilic leukocytosis, but it is not invariable. C-reactive protein was elevated in all dogs with polyarthritis in one study.[9] Synovial fluid is thin and turbid, and the mucin clot test is usually normal. Nucleated cell counts in joint fluid are notably elevated, with predominantly nondegenerative neutrophils. Most dogs test negative for or have insignificant titers of antinuclear antibody and rheumatoid factor. Synovial biopsy usually reveals hypertrophy of the synovial lining plus polymorphonuclear or mononuclear cell infiltration. Microbial organisms are not observed and bacterial cultures are negative. Serology and molecular testing for tick-borne agents are negative.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for nonerosive idiopathic immune-mediated polyarthritis include all other arthropathies (e.g., joint neoplasia, infectious arthritis, rheumatoid arthritis, osteoarthritis). Nonerosive inflammatory polyarthritis may occur secondary to any chronic inflammatory disorder or to persistent antigenic stimulus. Another disease (e.g., chronic infection, bacterial endocarditis, gastroenteritis, ulcerative colitis, or neoplasia) or drug therapy incites formation of immune complexes, which mediate the arthritis. SLE may also induce polyarthritis.

MEDICAL MANAGEMENT

Glucocorticoids are the initial treatment of choice (Box 34.10).[10] The dosage should be titrated to the lowest amount that prevents clinical signs; however, many animals require lifelong therapy, which can be associated with side effects (e.g., polyuria, urinary tract infections, iatrogenic hyperadrenocorticism). Cyclophosphamide can be used in difficult-to-control patients; however,

BOX 34.10 Treatment of Idiopathic Immune-Mediated Polyarthritis

Prednisolone

2.2–4.4 mg/kg/d for 2 wk; then if clinical response is seen, can decrease to 1.1–2.2 mg/kg/d for 2 wk; if animal is clinically normal at this time and if synovial inflammation has subsided, reduce dosage to 1.1 mg/kg q48h for 4 wk.

Azathioprine (Imuran)

Dogs: 2 mg/kg/d given orally for 5 days, then 2 mg/kg q48h if clinical signs resolve; giving 2 mg/kg q48h from the start will require longer to achieve remission but may lessen the chance of hepatitis or pancreatitis.

Cyclosporine

3–5 mg/kg PO q12h with food; must do therapeutic drug monitoring to check trough levels and then adjust dose.

Leflunomide

Dogs: 3–4 mg/kg/d (usually divided q12h) given orally; duration varies with dog and with response to therapy; hematologic variables, serum biochemical analysis results, and clinical signs of immune-mediated polyarthritis should be monitored for evidence of adverse effects to treatment with leflunomide.

PO, Orally.

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it is not recommended due to potentially serious side effects. Azathioprine can be administered in cases resistant to prednisolone therapy (see Box 34.10). Azathioprine can cause myelosuppression (principally when used daily as opposed to every other day), and a CBC and platelet count should initially be monitored periodically or whenever the patient seems unwell. Azathioprine may also cause hepatitis or pancreatitis. Cyclosporine is an immunosuppressive drug that is often effective and generally has few serious side effects; however, therapeutic drug monitoring is often needed because absorption varies greatly among different products and different patients. Chronic cyclosporine therapy may be associated with opportunistic fungal infections, particularly in warmer climates. Leflunomide has been reported as a safe and effective alternative to glucocorticoids (see Box 34.10).[11]

SURGICAL TREATMENT

Surgery is not indicated.

PROGNOSIS

The prognosis for nonerosive immune-mediated polyarthritis varies. Remission or cure can be achieved in most cases, but owners should be warned of the possibility of recurrence. A positive response to therapy should be based on arthrocentesis and cytology, not on clinical signs alone, because medical therapy may result in clinical improvement but incomplete cessation of the inflammatory activity.

NOTE A positive response to therapy should be based on arthrocentesis and cytology and not on clinical signs alone because medical therapy may result in clinical improvement but incomplete cessation of the inflammatory activity.

RHEUMATOID ARTHRITIS

DEFINITION

Rheumatoid arthritis is an erosive, noninfectious, inflammatory joint disease characterized by chronic, bilaterally symmetric, erosive destruction of the joints.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The etiology of rheumatoid arthritis is unknown, but it is considered an immune-mediated arthropathy.[12] The antigens are altered host immunoglobulins (IgG and IgM), which are known as *rheumatoid factors*. Resultant immune complexes are deposited in the synovium, initiating an inflammatory response followed by synovial cell proliferation, villous hypertrophy, pannus formation over the cartilage surface, cartilage and subchondral bone destruction, joint swelling, and rupture of the collateral ligaments. The outcome is a nonfunctional joint. The synovial membrane is generally discolored, edematous, congested, and thickened and may contain fibrin deposits. Pannus of granulation tissue originates at the periphery of the joint and covers portions of the articular cartilage, which may fibrillate and ulcerate.

Diagnosis of classic rheumatoid arthritis requires the presence of destructive lesions seen radiographically, a positive rheumatoid factor, characteristic histopathologic changes in the synovial membrane, and four additional criteria listed in Box 34.11. Rheumatoid arthritis is usually diagnosed when 7 of the 11 criteria are present and joint signs have been present for at least 6 weeks. Subcutaneous nodules are rare in dogs.

! BOX 34.11 Characteristics of Rheumatoid Arthritis

- Stiffness after rest
- Pain in at least one joint
- Swelling in at least one joint
- Swelling of at least one other joint within 3 months
- Symmetric joint swelling
- Subcutaneous nodules over bony prominences or extensor surfaces, or in juxtaarticular regions
- Destructive radiographic lesions
- Positive rheumatoid factor
- Poor mucin precipitate from synovial fluid
- Characteristic histopathologic changes in the synovial membrane
- Characteristic histopathologic changes in subcutaneous nodules

DIAGNOSIS

Clinical Presentation

Signalment

Rheumatoid arthritis can affect any breed of dog, but it is primarily found in adults, usually greater than 5 years old.

History

Most affected dogs have a history of stiffness after rest, limping, or difficulty walking.

Physical Examination Findings

In chronic cases, joints are generally enlarged with periarticular soft tissue swelling and joint effusion. The distal joints (i.e., carpi and tarsi) may be unstable, with obvious deformity and angulation.

Diagnostic Imaging

Radiographs of the joints show a generalized loss of mineralization, radiolucent foci, and irregular joint margins. Bone proliferation may also be present. Soft tissue swelling and joint effusion may be evident (Fig. 34.10).

Laboratory Findings

Hematology may reveal anemia of chronic inflammation and a nonspecific leukocytosis. The synovial fluid is often yellow, turbid, and increased in volume. The mucin clot may be poor and friable. Joint fluid nucleated cell counts are greatly elevated with predominantly nondegenerative neutrophils. However, dogs with no obvious evidence of rheumatoid arthritis may have positive titers; therefore care is warranted in interpreting tests for rheumatoid factor.

Synovial biopsy generally shows villous hypertrophy, proliferation of synovial cells, and lymphocyte and plasma cell infiltration. Immunofluorescent studies demonstrate complexes of IgG or IgM in synovial lining cells, blood vessel walls, and extracellular tissue. Microbial organisms are not observed on histologic samples, and bacterial cultures are negative.

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FIG. 34.10 Dorsopalmar radiograph of the canine carpus with advanced abnormalities associated with rheumatoid arthritis. Note the erosion of subchondral bone plates and the collapse of the joint space, suggesting erosion of the articular cartilage.

BOX 34.12 Therapy for Rheumatoid Arthritis

Prednisolone
2.2–4.4 mg/kg/d PO for 2 wk; then 1.1–2.2 mg/kg/d PO for 2 wk; then 1.1 mg/kg q48h

Azathioprine (Imuran)
Dogs: 2 mg/kg/d PO for 5 d; then 2 mg/kg q48h if clinical signs resolve; giving 2 mg/kg q48h from the start will require longer to achieve remission but may lessen the chance of hepatitis or pancreatitis

Cyclosporine
3–5 mg/kg PO q12h with food; must do therapeutic drug monitoring to check trough levels and then adjust dose

Leflunomide
Dogs: 3–4 mg/kg/d given orally for 6 wk (usually divided q12h); duration varies with dog and with response to therapy; hematologic variables, serum biochemical analysis results, and clinical signs of immune-mediated polyarthritis should be monitored for evidence of adverse effects to treatment with leflunomide

PO, Orally.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include joint neoplasia, infectious arthritis, inflammatory nonerosive polyarthropathies, and osteoarthritis.

MEDICAL MANAGEMENT

A combination of immunosuppressive drugs (i.e., prednisolone, azathioprine, cyclosporine, mycophenolate, leflunomide) is typically necessary to achieve remission of clinical signs (Box 34.12).[12] Therapy is usually initiated with prednisolone, and then additional immunosuppressive drugs (e.g., azathioprine) are added. Cyclophosphamide should be avoided because it poses significant risks to the dog and the person administering the drug. The dog should be reevaluated and synovial fluid analyzed monthly. The dose of glucocorticoid is gradually reduced with a goal of ultimately only needing 1 mg/kg every other day (plus azathioprine, mycophenolate, and/or cyclosporine). Joints that have lost all collateral ligament support may benefit from therapeutic wrap braces.

SURGICAL TREATMENT

Surgical treatment is not indicated.

PROGNOSIS

Rheumatoid arthritis is progressive, and dogs rarely, if ever, make a full recovery. Lameness and stiffness usually persist despite treatment.

FELINE CHRONIC PROGRESSIVE POLYARTHRITIS

DEFINITION

Feline chronic progressive polyarthritis is an immune-mediated disease most commonly affecting male cats, which can exist as a progressive periosteal-proliferative form and a deforming, erosive form. It has been described in one case report in a female cat.[13]

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The cause may involve exposure to the feline syncytium-forming virus and feline leukemia virus (FeLV); however, these viruses cannot be used to experimentally induce the disease. The periosteal-proliferative form of the disease results in osteoporosis and periosteal new bone formation around the joint. Periarticular erosions and collapse of the joint space with fibrous ankylosis occur with time. The erosive form of the disease causes joint changes similar to those seen in canine rheumatoid arthritis. The synovium is infiltrated with lymphocytes and plasma cells.

DIAGNOSIS

Clinical Presentation

Signalment

This condition affects mostly male cats. Affected cats are generally between the ages of 1.5 and 4.5 years. Any breed of cat may be affected.

History

Most cats have a history of lameness, reluctance to move, depression, anorexia, weight loss, and occasionally deformity of affected joints.

Physical Examination Findings

Pyrexia, depression, lymphadenopathy, and multiple-joint involvement are common findings. Joint palpation causes pain. The joints are sometimes swollen and may be deformed in animals with the erosive form of the disease.

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FIG. 34.11 Radiograph of the tarsus of a cat with the periosteal-proliferative form of feline progressive arthritis. There is proliferative new bone formation on the periphery of the joints, generalized loss of opacity of subchondral bone, and loss of the joint space. Radiolucent foci are visible throughout the tarsal bones. (From Olmstead ML. *Small Animal Orthopedics*. St. Louis: Mosby; 1995.)

Diagnostic Imaging

Changes noted on radiographs of affected joints include proliferative new bone formation on the periphery of the joints, generalized loss of opacity of the subchondral bone, and loss of the joint space. Radiolucent foci in the subchondral bone and irregular joint margins may also occur. Periarticular soft tissue swelling and joint effusion may be noted (Fig. 34.11).

Laboratory Findings

Feline syncytium-forming virus is identified in all cats with feline chronic progressive polyarthritis, and FeLV is identified in 60% of cats with the disease. Synovial fluid analysis demonstrates elevated neutrophil counts. Routine blood work may demonstrate leukocytosis.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include neoplasia, septic arthritis, DJD, hypervitaminosis A, and Scottish fold arthropathy/osteochondrodysplasia.

MEDICAL MANAGEMENT

Treatment involves immunosuppressive drugs and usually begins with prednisolone (Box 34.13). The cat often requires lifelong therapy. Chlorambucil may help control the signs. Cyclophosphamide is myelosuppressive in cats and should be avoided unless absolutely necessary. Never use azathioprine in a cat.

BOX 34.13 Treatment of Feline Chronic Progressive Polyarthritis

Prednisolone
4.4–6.6 mg/kg/d PO; if cat improves after 2 wk, reduce dosage to 2.2 mg/kg/d; then place on maintenance therapy (1.1–2.2 mg/kg q48h)

Chlorambucil (Leukeran)[a]
1 mg/cat PO twice weekly in cats <3.5 kg; 2 mg/cat PO twice weekly in cats >3.5 kg, *or*
2 mg/cat PO every second or third day, *or*
15 mg/M^2 daily for 4 days, then cycle repeated every 3 wk

Cyclophosphamide (Cytoxan)
6.25–12.5 mg/cat PO given up to 4 consecutive days of each week for up to 4 mo; this drug can have substantial and life-threatening side effects; use this drug only if all other drugs fail

[a]There are various protocols for the use of this drug in cats.
PO, Orally.

NOTE If you lower the dose of medications too quickly in these patients, clinical signs often recur and are more resistant to therapy.

SURGICAL TREATMENT

Surgical therapy is not indicated.

PROGNOSIS

Prognosis is good for remission of signs but guarded for complete control of the disease. Other FeLV-related disorders may occur in cats that test positive.

Management of Specific Joint Diseases

SHOULDER JOINT

OSTEOCHONDRITIS DISSECANS OF THE PROXIMAL HUMERUS

DEFINITIONS

OCD, in which a flap of cartilage is lifted from the articular surface, is a manifestation of a general syndrome called *osteochondrosis*. *Osteochondrosis* is a disturbance in endochondral ossification that leads to cartilage retention. Detached pieces of articular cartilage are often referred to as **joint mice.**

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

OCD commonly occurs in the shoulders, elbows, stifles, and hocks of immature large- and giant-breed dogs; it is rarely

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reported in small-breed dogs. Despite unilateral lameness, this condition is often bilateral. In the shoulder, it is usually evidenced as a cartilage flap found on the midline or lateral aspect of the dorsocaudal humeral head. In some cases, the subchondral bone defect occupies half the area of the humeral head. The abnormal cartilage may fissure and cause protrusion of a loose flap of cartilage into the joint, or the cartilage may completely detach from the underlying bone and become lodged in the caudoventral joint pouch or bicipital bursa.

NOTE OCD is often bilateral; radiograph and evaluate both shoulders even if the animal exhibits unilateral lameness.

OCD begins with a failure of endochondral ossification in either the physis or the articular epiphyseal complex that is responsible for long bone epiphyseal formation. The cause of OCD is unknown but is considered to be multifactorial, with management, genetic, and nutritional interactions occurring in young growing dogs. Risk factors for OCD include age, gender, breed (genetic), rapid growth, and nutrient excesses (primarily calcium). Failure of endochondral ossification leads to cartilage thickening (osteochondrosis). Because developing cartilage is nourished initially by synovial fluid and later by vascularization through subchondral bone, increased cartilage thickness may result in malnourished, necrotic chondrocytes. Loss of chondrocytes deep in the cartilage layer leads to formation of a cleft at the junction of calcified and noncalcified tissue. Subsequently, normal activity may lead to development of vertical fissures in the cartilage that eventually communicate with the joint, forming a cartilage flap (Fig. 34.12). This communication allows cartilage fragments and inflammatory mediators to reach the synovial fluid, induce joint inflammation, and initiate the cycle of DJD (see p. 1143). OCD does not apparently cause clinical signs until a cartilage flap forms. Free cartilage flaps can lodge in joints and may increase in size with calcification until they become radiographically visible joint mice. In some cases, the cartilage flaps are gradually resorbed. DJD is often the final outcome.

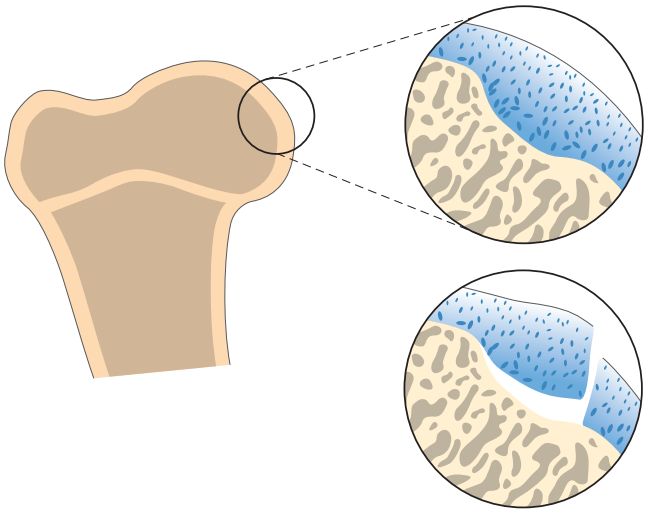


FIG. 34.12 Failure of endochondral ossification leads to cartilage thickening. Loss of chondrocytes deep in the cartilage layer produces a cleft and causes development of vertical fissures in the cartilage. These fissures eventually communicate with the joint, forming a cartilage flap.

NOTE OCD has a hereditary component; counsel owners against breeding affected dogs.

DIAGNOSIS

Clinical Presentation

Signalment

Large- and giant-breed dogs are commonly affected; this disease is rarely diagnosed in cats or small dogs. Males are more commonly affected than females. Clinical signs often develop between 4 and 8 months of age; however, some dogs may not be presented for veterinary evaluation until they are mature or middle-aged.

History

Affected animals are usually seen with unilateral forelimb lameness. Owners typically report a gradual onset of lameness that improves after rest and worsens after exercise. Owners may occasionally associate the onset of lameness with trauma.

Physical Examination Findings

The shoulder should be palpated and moved through a complete range of motion. Crepitation or palpable swelling of the joint is seldom evident, but affected animals usually have muscle atrophy and show pain when the shoulder is moved into extreme extension (i.e., moving the humerus forward with one hand while the other hand is positioned as a fulcrum on the cranial aspect of the shoulder; see p. 963). Extreme flexion of the shoulder may also cause pain.

NOTE Be careful not to confuse elbow pain with shoulder pain when hyperextending the elbow to extend the shoulder.

Diagnostic Imaging

Diagnosis of OCD is based on radiographic findings evident on lateral projections of the scapulohumeral joints; craniocaudal projections do not contribute to the diagnosis, but they may help identify the location of a joint mouse.[14] Both shoulders should be imaged because this condition is often bilateral despite unilateral lameness. The dog should be positioned in lateral recumbency with the shoulder of interest dependent and the head elevated to prevent superimposition of the humeral head over the cervical spine (Fig. 34.13). The uppermost forelimb is retracted caudally, and the dependent forelimb is extended cranially. Slight external rotation of the humerus may help silhouette the affected portion of the humeral head. Sedation may be required for quality radiographs (see Table 31.2). Multiple views made while the humerus is rotated (pronated and supinated) may be necessary to locate the lesion.

The earliest radiographic sign of OCD is flattening of the subchondral bone of the caudal humeral head. As the disease progresses, a saucer-shaped radiolucent area in the caudal humeral head may be visualized (Fig. 34.14). Calcification of the flap may allow visualization of the flap in situ or in the joint if it has detached from the underlying bone. In chronic cases, large calcified joint mice are often observed in the caudoventral joint pouch or along bicipital bursa.

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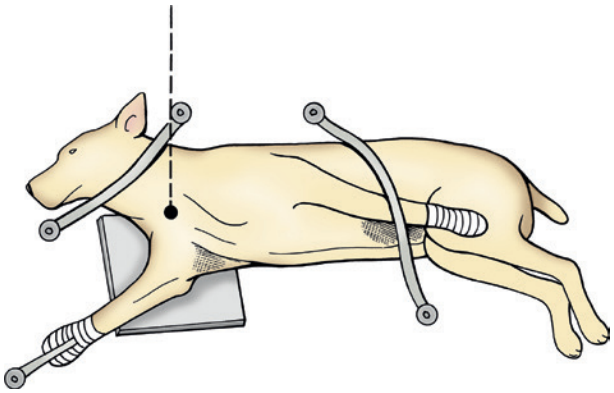


FIG. 34.13 Proper positioning of a dog to obtain a medial-lateral view of the scapulohumeral joint.

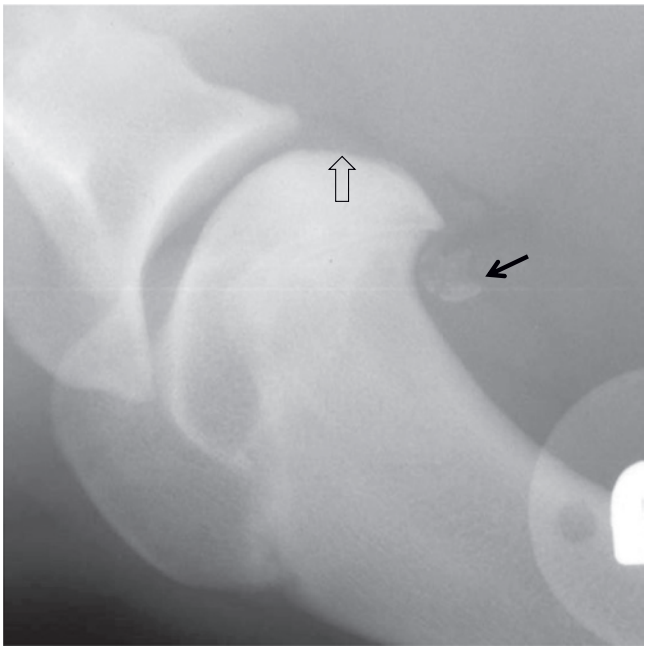


FIG. 34.14 Radiograph of a shoulder with osteochondritis dissecans (OCD) of the caudal humeral head. Note the flattening and irregularity of the subchondral bone of the caudal aspect of the humeral head *(open arrow)* and free OCD flap in the caudoventral joint pouch *(solid arrow)*.

Ultrasound has also been shown to be effective in the diagnosis of OCD of the shoulder.

Laboratory Findings

Analysis of synovial fluid in animals with OCD reflects underlying inflammation and the development of DJD. Other specific laboratory abnormalities are not present.

DIFFERENTIAL DIAGNOSIS

Forelimb lameness in large immature dogs can be caused by many diseases: OCD, UAP, or FCP of the elbow; panosteitis; premature closure of the physes; retained cartilage cores; and hypertrophic osteodystrophy. Differentials for lameness attributable to the shoulder joint include injuries to the biceps, supraspinatus, subscapularis or infraspinatus tendon, and injuries to the medial or lateral glenohumeral ligaments.

MEDICAL MANAGEMENT

Conservative therapy may provide short-term benefit to some dogs with OCD of the shoulder; however, long-term resolution of lameness requires arthroscopy or arthrotomy. If shoulder OCD has been definitively diagnosed, risks of medical or conservative management include prolonged lameness and muscle atrophy, migration of the flap and associated complications, and severe osteoarthritis/synovitis/biceps tendon disease. Body weight management in overweight dogs aids in decreasing loads on the joint and thus may reduce the signs of associated osteoarthritis.

SURGICAL TREATMENT

Preoperative Management

The overall health of the animal should be determined before surgery. A complete physical examination should be performed to determine if other joints are similarly affected.

Anesthesia

These animals are usually young and healthy, and a variety of anesthetic regimens can be used (see Table 32.1).

Arthroscopic Treatment of Shoulder Osteochondritis Dissecans

Indications

Indications for shoulder arthroscopy include OCD, bicipital tenosynovitis, shoulder instability (i.e., joint capsule or ligament tears or both), and diagnostic examinations (e.g., biopsy of bone, cartilage, or the soft tissue envelope) (Video 34.3). Management of OCD is the most common indication. Arthroscopy provides outstanding exposure for visualization and treatment of OCD lesions and may be performed bilaterally with minimal morbidity (Video 34.4).

Instrumentation

A 30-degree foreoblique arthroscope is commonly used in the shoulder joint. In most dogs, a 2.7-mm arthroscope can be easily inserted into the joint space. In small breeds of dogs, a 2.4-mm arthroscope is preferable to prevent iatrogenic cartilage damage during insertion or surgical manipulation. Use of a blunt obturator is always recommended over a sharp trocar.

> **NOTE** Whichever size arthroscope is chosen, remember to consider the outside diameter of the arthroscope cannula because it too must enter the joint.

Shoulder arthroscopy requires an assortment of hand instruments (see Chapter 14). These include instruments to assist in the inspection of intraarticular structures (probes), grasping forceps for removal of free bodies, biopsy forceps, and instruments for surface abrasion. Instruments commonly used for abrasion arthroplasty are handheld burrs, curettes, or a motorized shaver. Instruments can be inserted into the joint through an open instrument port, instrument cannulas, or a combination of the two. If the surgeon chooses to work through an instrument cannula, different size cannulas and "switching sticks" are necessary.

Portal Sites and Technique

Clip and prepare the area as if an open shoulder arthrotomy were to be performed, so that the arthroscopy procedure can be aborted

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and an open arthrotomy performed if necessary. This occurs most often when the surgeon is beginning to learn arthroscopy. *Use one of two methods for limb preparation, depending on the limb maneuverability desired. For maximum maneuverability of the limb during surgery, use a hanging-limb preparation.* As you become more experienced and adept at shoulder arthroscopy, less maneuverability is needed, and you may wish to perform a lateral limb preparation for each limb. *However, perform the lateral limb preparation such that an open procedure can be performed if needed. Use two or three portal sites for shoulder arthroscopy, depending on the purpose of arthroscopic intervention. If visual exploration of the shoulder joint is all that is required, use only an egress portal and an arthroscope portal. If tissue biopsy or treatment of a pathologic joint condition is undertaken, use an additional instrument portal. Alternatively, use the instrument portal as an egress portal as well.*

Position the dog in lateral recumbency with the leg to be operated on uppermost. Remember to support the limb in neutral position to prevent excessive adduction that closes the joint line between the glenoid and humeral head. Infuse and distend the joint first at the site of the arthroscope portal. This aids in determining the proper location for this portal. *Use a hypodermic needle (18-gauge, 1.5-inch [40 mm]; a spinal needle may be required in giant-breed dogs). Insert the needle just in front of and slightly distal to the acromion process of the scapula. Insert the needle perpendicular to the skin surface and maintain this orientation through the soft tissue as the needle enters the joint.*

In most cases, when the needle has been properly placed, synovial fluid is easily aspirated. *If synovial fluid is not aspirated and if you believe that the joint has been entered, instill fluid (lactated Ringer's solution) into the joint.* If the needle is located in the joint, fluid is easily instilled. In addition, as one begins to fill the joint cavity with fluid, reverse pressure is felt on the syringe plunger from the instilled fluid. *Remove the needle to maintain joint distention.*

Establish the arthroscope portal next. Insert the arthroscope cannula with the blunt obturator first. A pointed blunt (conical) obturator is preferred. *Use a No. 11 Bard-Parker blade to make a small entry wound through the skin and superficial soft tissue at the site of the previous needle insertion. Do not enter the joint with the scalpel blade because extravasation of fluid outside the joint cavity is more likely when this occurs. Insert the arthroscope cannula with the attached pointed blunt obturator. Once in the joint, remove the obturator from the cannula.* Fluid will flow freely from the cannula, confirming correct placement. *Attach the fluid ingress line to the cannula and insert the arthroscope, and establish egress portal. Inspect the medial compartment for evidence of inflammation and then the cranial compartment. Thoroughly inspect the region of the biceps tendon for inflammation or free cartilage pieces (Fig. 34.15).*

Direct the arthroscope caudally, and position the light post to inspect the caudal articular surface and joint capsule. Once the OCD lesion has been identified, pass a guide needle to position the instrument portal (Fig. 34.16). Use an 18-gauge, 1.5-inch (40-mm) needle, or in larger dogs a spinal needle or catheter stylet. Insert the needle into the joint from a point approximately 2 cm caudal to the arthroscope portal. Insert the needle so that it can be visualized at the site of the lesion and direct it to the site of the OCD lesion. The most common error is overangling the needle and crossing the needle over the arthroscope. *Once the position for the instrument portal has been established, decide if you are going to work through an open instrument portal, an instrument cannula, or a combination of the two. If you choose to work through an open portal site, use a No. 11 Bard-Parker scalpel blade to make a 0.5- to 1-cm soft tissue tunnel adjacent to the guide needle. If the cartilage flap is still attached (generally cranial and/or medial), insert a probe or elevator to free the edge of the flap. Do not free the cartilage flap completely; leave it attached at one or more sites. Insert grasping forceps and hold the cartilage flap with the forceps and remove it (Fig. 34.17). To facilitate removal of the flap, twist the forceps to fold the flap longitudinally, and ease passage through the joint capsule. Remove the cartilage flap as a single large fragment or, as is often the case, in two or three smaller pieces.*

If working through an instrument cannula, insert the small instrument cannula with a sharp trocar into the joint adjacent to the guide needle. Place larger cannulas with the use of switching sticks, but use the smallest instrument cannula through which the lesion can be treated. Pass a hand curette, hand burr, or motorized shaver through the cannula, and use it to break the cartilage flap into small pieces. The pieces generally are small enough to flow out the instrument cannula. *If a cartilage piece is too large to pass freely through the cannula, insert small grasping forceps and capture the fragment. Pull the fragment next to the instrument cannula, and remove the cannula and forceps at the same time. Reestablish*

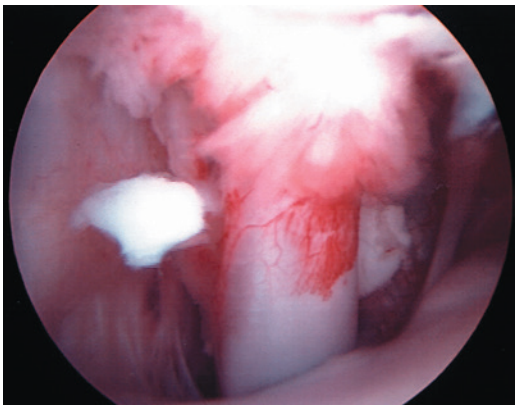


FIG. 34.15 View of the biceps tendon; note the free cartilage fragments adjacent to the tendon.

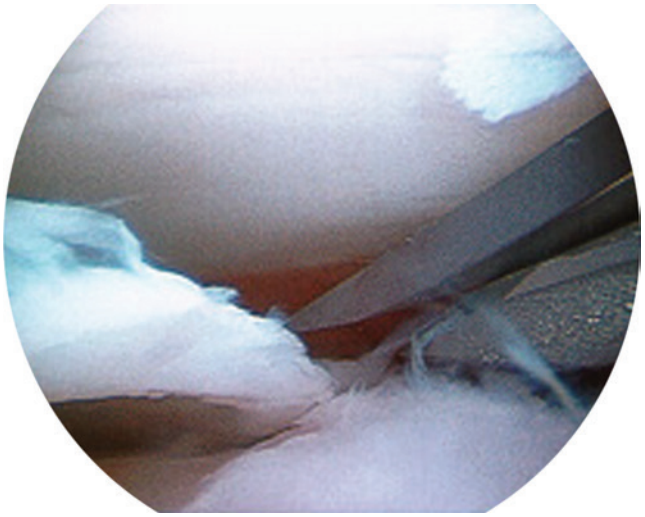


FIG. 34.16 Arthroscopic triangulation within the shoulder joint for removal of an osteochondritis dissecans fragment.

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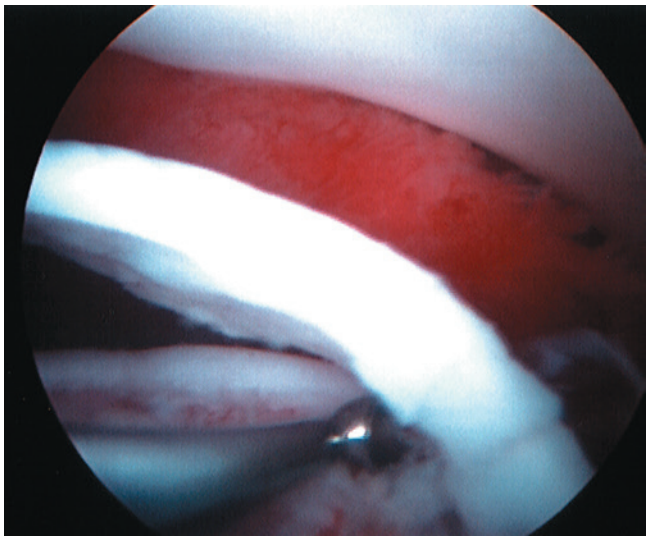


FIG. 34.17 Osteochondritis dissecans cartilage fragment raised from the underlying bone surface.

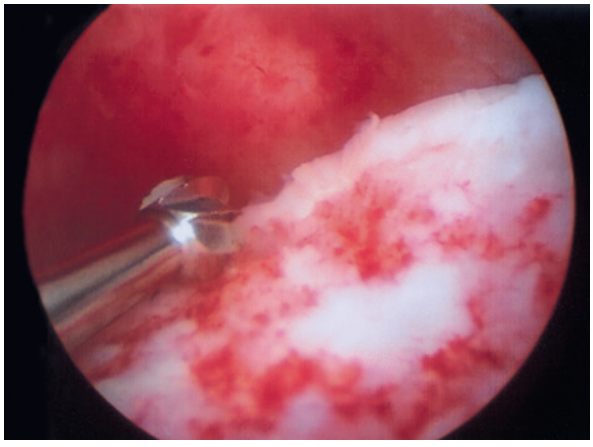


FIG. 34.18 Abrasion arthroplasty of the surface of an osteochondritis dissecans lesion on the humeral head using a hand burr.

the instrument port by placing a switching stick into the joint followed by the cannula. Continue to break the cartilage flap into small pieces until the cartilage lining the periphery of the lesion bed is firmly attached to the underlying subchondral bone.

Next, treat the lesion bed by surface abrasion or microfracture (most surgeons use surface abrasion) (Fig. 34.18). Whether using an open portal or instrument cannula to facilitate removal of the cartilage flap, treatment of the lesion bed is best accomplished through an instrument cannula. *If an open portal was used to remove the cartilage flap, insert an instrument cannula over a switching stick. Treat the surface of the lesion with aggressive abrasion using a handheld curette, handheld burr, or motorized shaver. Continue to abrade the surface until the underlying bone bleeds freely. Stop ingress fluid as needed to observe the extent of bleeding bone. On completion of surface abrasion or microfracture, flush small bone or cartilage fragments from the joint by increasing the ingress flow and allowing egress through a large instrument cannula. Inspect the joint for remaining bone or cartilage fragments, and then remove the arthroscope and instrument cannula. Suture the portals with nonreactive, nonabsorbable suture.*

Postoperative Care and Assessment

The animal can usually be released to the owner the day of surgery or the following day. The client should be instructed to limit the animal's activity for 1 month to allow soft tissue to heal and to provide time for formation of fibrocartilage in the OCD lesion. The dog can then be returned gradually to full activity. The portal sites should be observed for drainage, which usually resolves without therapy.

Open Surgical Techniques for Osteochondritis Dissecans

Surgical treatment involves exploratory arthrotomy and removal of the cartilage flap. The goals of surgery are to remove the cartilage flap from the humeral head and curette the edges of the bony defect to ensure removal of all affected cartilage. Subchondral bone that appears pale and sclerotic should also be curetted. The joint should be carefully explored and flushed extensively to identify and remove any pieces of dislodged cartilage.

One of several different approaches may be used to expose the scapulohumeral joint surgically, depending on the location of the defect and any detached cartilage flaps. Infraspinatus tenotomy affords excellent exposure of the humeral head and access to both the cranial and caudal joint compartments. However, because the infraspinatus tendon is cut and the joint is subluxated during the procedure, this approach is more traumatic than approaches that do not involve tenotomy. Longer postoperative recovery periods should be expected than with other techniques. A caudal approach to the joint affords good exposure of the humeral head (with adequate retraction) and excellent access to the caudal ventral joint compartment without tenotomy; however, it does not allow the cranial aspect of the joint to be explored.

NOTE In animals with unilateral lameness that have bilateral radiographic lesions, surgery should be performed on the lame leg. The other leg may require surgery if lameness subsequently develops in that limb.

Surgical Anatomy

Important anatomic landmarks for identifying the location of the scapulohumeral joint are the acromion process of the scapular spine, the greater tubercle, and the acromial head of the deltoid muscle. The omobrachial vein is located superficially over the acromial head of the deltoid muscle. The caudal circumflex humeral artery and vein and axillary nerve are encountered and must be protected during the caudal approach to the shoulder.

Positioning

The dog is positioned in lateral recumbency with the affected limb up. The limb should be prepared from the dorsal midline to below the elbow.

SURGICAL TECHNIQUE

Infraspinatus Tenotomy for Exposure of the Scapulohumeral Joint

Make an incision in the skin and subcutaneous tissue from just proximal to the acromion process to the proximal humerus

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(Fig. 34.19A). Curve the incision over the joint along the palpable cranial margin of the deltoid muscle's acromial head. Incise the deep fascia along the cranial margin of the acromial portion of the deltoid muscle, and retract the muscle caudally (Fig. 34.19B). Isolate the infraspinatus tendon, and place a stay suture in its proximal portion. Incise the tendon 5 mm from its insertion on the humerus and retract it caudally (Fig. 34.19C). Incise the joint capsule midway between the glenoid rim and the humeral head (Fig. 34.19D). Internally rotate the humerus until the head subluxates, exposing the caudal surface of the humeral head (Fig. 34.19E). Remove the cartilage flap from the humeral head and curette the edges of the bony defect to ensure removal of all affected cartilage (Fig. 34.19F). Flush all parts of the joint thoroughly to remove any cartilage debris or joint mice. Close the joint capsule with 3-0 absorbable suture in a simple interrupted pattern. Reappose the infraspinatus tendon with an absorbable suture in a Bunnell or locking-loop pattern (see p. 1283). Close the muscular fascia, subcutaneous tissue, and skin separately.

Caudal Approach to the Scapulohumeral Joint

Make an incision in the skin, subcutaneous tissue, and deep fascia that extends from the midscapular spine to the midhumeral diaphysis (Fig. 34.20A). Incise the intermuscular septum between the caudal border of the scapular portion of the deltoid muscle and the long head of the triceps muscle and separate the muscles (Fig. 34.20B). Use blunt dissection to free the deltoid muscle and expose the caudal circumflex artery and vein, the muscular branch of the axillary nerve, and the teres minor muscle (Fig. 32.20C). Elevate and retract the teres minor muscle cranially, exposing the axillary nerve and joint capsule. Place a Penrose drain around the nerve, and gently retract it caudally (Fig. 34.20D). Incise the joint capsule 5 mm from and parallel to the glenoid rim to expose the humeral head (Fig. 34.20E). To expose OCD lesions of the humeral head, internally rotate the humerus and flex the shoulder. Explore the joint and remove the cartilage as described previously. Close the joint capsule in an interrupted pattern with 3-0 absorbable suture. Then, suture the intermuscular septum, deep fascia, subcutaneous tissue, and skin as separate layers.

POSTOPERATIVE CARE AND ASSESSMENT

The dog can usually be released to the owner within 1 or 2 days after surgery. The client should be instructed to limit the animal's activity for 1 month to allow soft tissue to heal. The dog then can be returned gradually to full activity. The incision site should be observed for seroma formation, which usually resolves without therapy. Healing of shoulder OCD lesions following flap removal benefits from rehabilitation therapy. The most commonly applied treatments are passive and active range of motion and controlled therapeutic exercises (see Chapter 11).

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Army-Navy retractors or Gelpi retractors are used to retract soft tissue and improve visualization of the humeral head. A bone curette is used to remove damaged articular cartilage from the humeral head.

PROGNOSIS

Prognosis for normal limb function with OCD of the shoulder is good. After surgery, most dogs become sound within 7 to 60 days. Approximately 75% of dogs have excellent function after fragment removal on long-term evaluation. Despite the absence of lameness in these dogs, DJD may develop; owners should be forewarned of this possibility.

SCAPULOHUMERAL JOINT LUXATION

DEFINITION

Scapulohumeral joint luxation occurs when loss or damage of supporting structures of the joint is sufficient to cause separation of the humerus from the scapula. Synonyms include a *dislocated shoulder* or a *shoulder luxation*.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Scapulohumeral luxations may be caused by trauma or may be congenital in origin. The scapulohumeral joint is supported by the joint capsule, glenohumeral ligaments, and surrounding tendons (supraspinatus, infraspinatus, teres minor, and subscapularis). Significant structures include the biceps tendon and medial and lateral glenohumeral ligaments. When these structures are torn or deficient, humeral head luxation may occur. Scapulohumeral luxations are named for the direction in which the humeral head deviates. Medial or lateral deviations are most common; cranial and caudal luxations are rare.

Traumatic luxations are usually the result of shoulder injury. Traumatic lateral humeral luxations occur after lateral glenohumeral ligament and infraspinatus tendon rupture, whereas traumatic medial humeral luxations are associated with tearing of the medial glenohumeral ligament and subscapularis tendon. Concurrent thoracic trauma is common (i.e., pneumothorax, hemothorax, pulmonary contusions, or fractured ribs).

Congenital or developmental laxity of the capsule and ligaments may result in medial displacement and medial luxation of the humeral head. The glenoid cavity may be sufficiently deformed or hypoplastic (glenoid dysplasia) to prevent reduction of the humeral head. This condition often occurs bilaterally in affected animals.

Subluxation or shoulder instability (without complete luxation) associated with incomplete tearing of the medial glenohumeral ligament, biceps tendon, or glenoid labrum; distention of the joint capsule; synovitis; and varying degrees of DJD may cause chronic shoulder pain and lameness in dogs, and are treated as distinct conditions from scapulohumeral luxation (discussed later in this chapter).

> **NOTE** Be sure to differentiate traumatic from congenital luxations because the treatment and prognosis are different.

DIAGNOSIS

Clinical Presentation

Signalment

Traumatic luxation may occur in any age or breed of dog; it is rare in cats. Congenital, medial luxation usually occurs in small and miniature dog breeds, such as toy poodles and Shetland sheepdogs; lameness usually appears when the animal is young.

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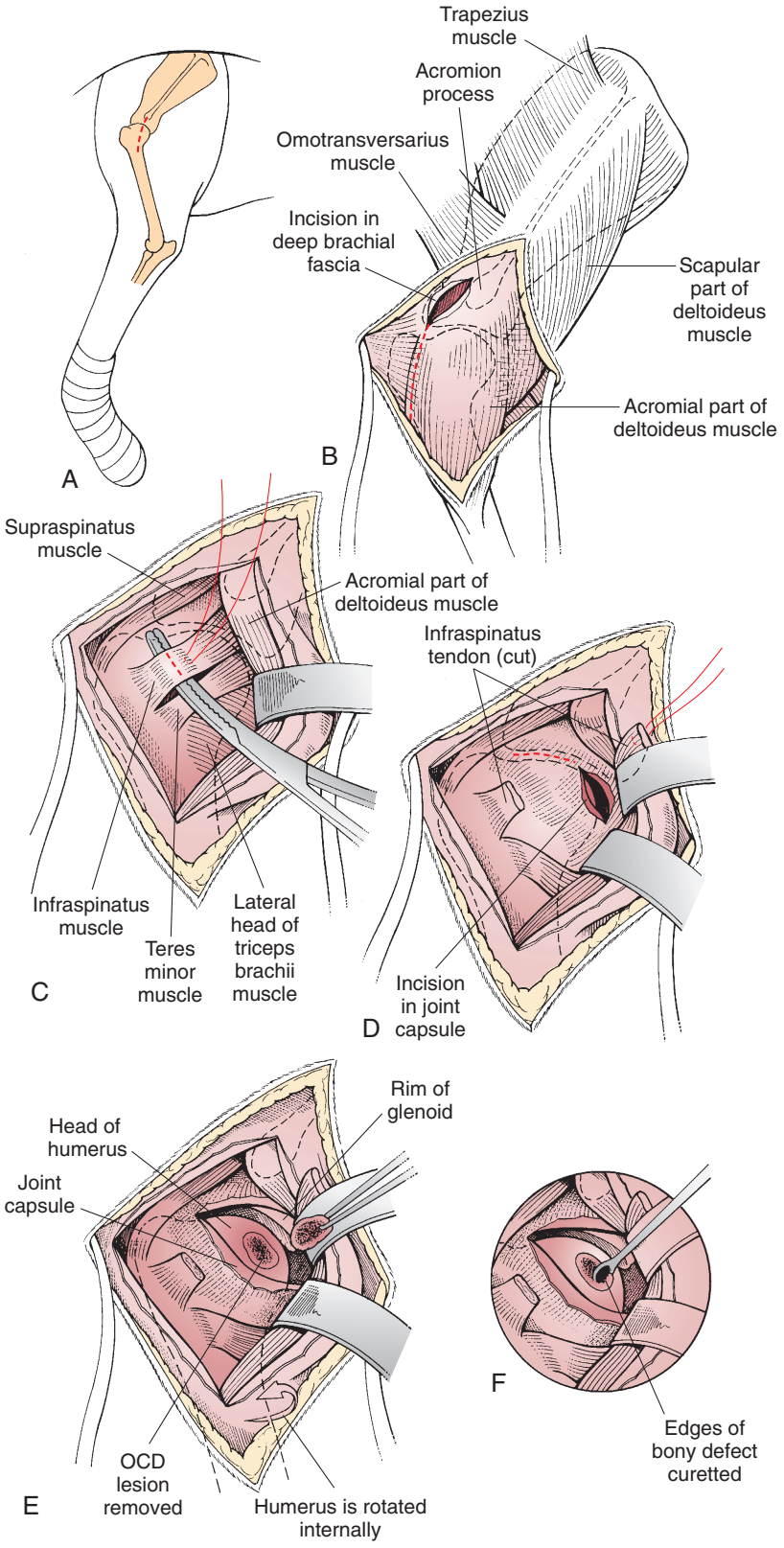


FIG. 34.19 Craniolateral approach to the shoulder. (A) Make an incision in the skin and subcutaneous tissue from just proximal to the acromion process to the proximal humerus. (B) Incise the deep fascia along the cranial margin of the acromial portion of the deltoideus muscle and retract the muscle caudally. (C) Isolate the infraspinatus tendon, place a stay suture in its proximal portion, and incise the tendon. (D) Incise the joint capsule midway between the glenoid rim and the humeral head. (E) Internally rotate the humerus until the head subluxates and remove the cartilage flap. (F) Curette the edges of the bony defect to ensure removal of all affected cartilage.

History

Dogs with traumatic luxation usually have a history or evidence of trauma. Chronic forelimb lameness that first becomes evident at a young age and without a history of trauma suggests congenital luxation.

Physical Examination

With traumatic luxation, affected animals may be non–weight-bearing, and the limb is often carried in a flexed position. With lateral luxation, the foot is rotated internally, and the greater tubercle is palpable lateral to its normal position. With medial

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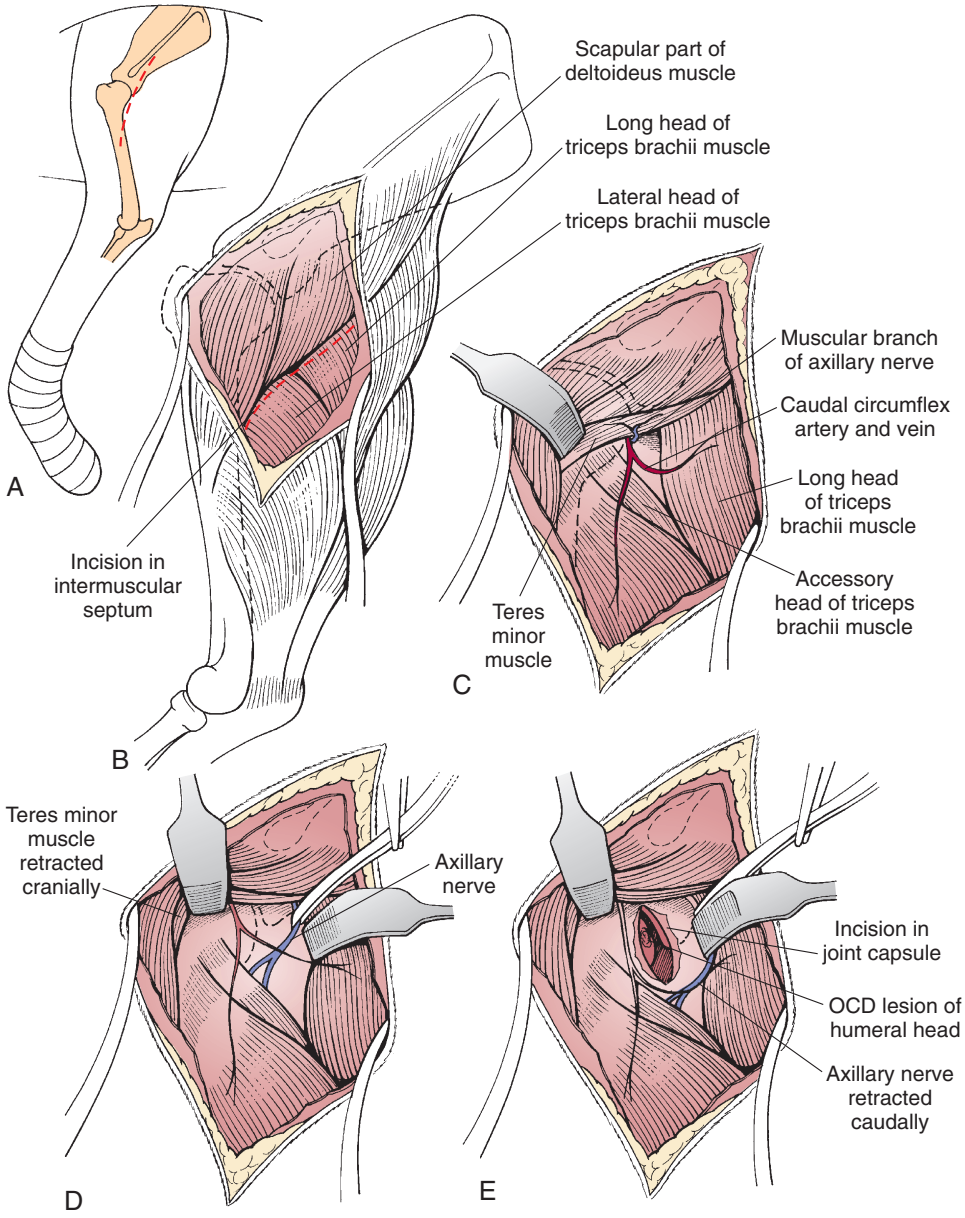


FIG. 34.20 Caudal approach to the shoulder. (A) Make an incision in the skin, subcutaneous tissue, and deep fascia that extends from the midscapular spine to the midhumeral diaphysis. (B) Incise the intermuscular septum between the caudal border of the scapular portion of the deltoideus muscle and the long head of the triceps muscle. (C) Elevate and retract the teres minor muscle cranially, exposing the axillary nerve and joint capsule. (D) Place a Penrose drain around the nerve and gently retract it caudally. (E) Incise the joint capsule 5 mm from and parallel to the glenoid rim to expose the humeral head. *OCD,* Osteochondritis dissecans.

luxation, the foot is rotated externally and the greater tubercle is palpated medial to its normal location. Pain and crepitation are evident with shoulder manipulation.

Dogs with chronic, congenital medial luxation are often lame. The joint is easily luxated and reduced, but manipulation does not usually cause pain. If the glenoid cavity is deformed, reduction of the humeral head may be impossible. Some small dogs with chronic medial luxation show only mild intermittent lameness and minimal DJD.

Diagnostic Imaging

Lateral and ventrodorsal radiographs of the shoulder are evaluated to confirm the diagnosis (Fig. 34.21). With traumatic luxation, special attention should be given to identifying concurrent scapular fractures or thoracic injuries.

Laboratory Findings

Specific laboratory abnormalities are not seen with traumatic or congenital luxation. Traumatized animals undergoing surgery

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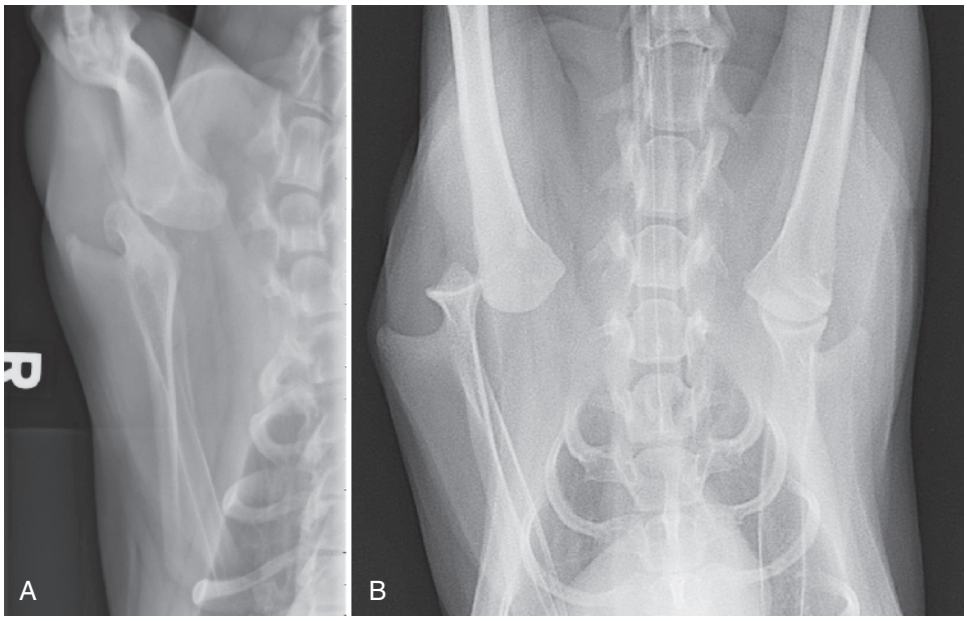


FIG. 34.21 Ventrodorsal radiographs showing (A) congenital and (B) traumatic medial luxation of the humeral head.

should have sufficient measurement of blood values to determine the optimum anesthetic regimen. Analysis of synovial fluid from affected animals may reflect associated inflammation and the development of DJD.

DIFFERENTIAL DIAGNOSIS

DJD, humeral osteosarcoma, and contracture of the infraspinatus or supraspinatus tendons must be differentiated from luxation on the basis of physical findings and the results of radiography.

MEDICAL MANAGEMENT

Dogs with chronic medial luxation, only mild intermittent lameness, and minimal DJD may be managed with exercise restriction and administration of antiinflammatory medications during acute exacerbations. Closed reduction can be attempted for a traumatic luxation presented for treatment soon after injury, provided the condition is not associated with humeral or scapular fractures. General anesthesia is required for closed reduction. A lateral luxation is reduced with the leg held in extension. Medial pressure is applied to the humeral head, and lateral pressure is applied to the medial surface of the scapula (Fig. 34.22). The humeral head should remain in place when it is gently moved through a normal range of motion. If the luxation appears stable, a lateral spica splint should be applied for 10 to 14 days (see p. 1166). A medial luxation caused by trauma is reduced in the opposite manner and immobilized by placing the limb in a Velpeau sling (see p. 1166). Temporary stabilization with transarticular cross pins or external fixators in a closed fashion has been described for treatment of a traumatic shoulder luxation.

Body weight management in overweight dogs aids in decreasing loads on the joint and may reduce clinical signs associated with osteoarthritis.

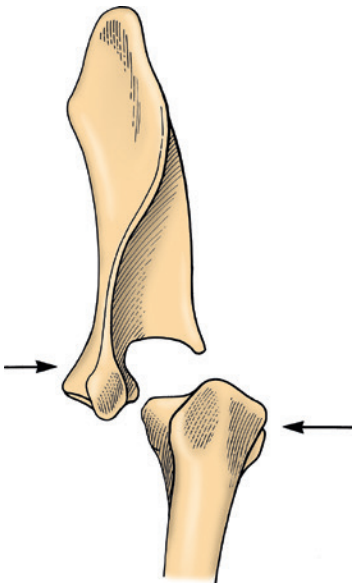


FIG. 34.22 For closed reduction of a lateral luxation, apply medial pressure to the humeral head and lateral pressure to the medial surface of the scapula.

SURGICAL TREATMENT

If a traumatic luxation is unstable enough after closed reduction that reluxation occurs or if the luxation is chronic, open reduction and stabilization with capsulorrhaphy or biceps tendon transposition are required. Temporary stabilization with a transarticular locking bone plate has also been described for treatment of a traumatic shoulder luxation.

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Surgery is warranted in animals with congenital luxations that cause severe or persistent lameness. In cases of severe joint dysplasia or DJD, open reduction and stabilization are unsuccessful, and salvage procedures (arthrodesis or glenoid excision) must be performed.

Surgical arthrodesis of the shoulder is reserved for animals with chronic intractable luxation, comminuted fractures of the humeral head or glenoid, or severe DJD that precludes primary fixation.[15] This is considered a salvage procedure and should be done only as a last resort and only when other joints are normal. Because scapular mobility compensates for loss of motion in the scapulohumeral joint, most animals have good limb function after shoulder arthrodesis.

Excision arthroplasty is a salvage procedure that causes a pseudoarthrosis to form between the scapula and the humerus, allowing limited scapulohumeral joint motion. This procedure does not require implantation of orthopedic hardware (plates, screws), and most dogs bear weight on the limb at a walk or run. However, gait abnormalities and mild or moderate atrophy of shoulder muscles are usually noted after surgery.

Preoperative Management

The animal's overall health should be determined before surgery. Thoracic radiography and electrocardiographic analysis are warranted in animals with traumatic luxation.

Anesthesia

General anesthesia is required for closed or open reduction. A variety of anesthetic regimens can be used in young, healthy animals (see Table 32.1). Special care should be exercised in anesthetizing older animals or those with concurrent injuries.

Surgical Anatomy

Important landmarks used to identify the location of the scapulohumeral joint are the acromion process of the scapular spine, the greater tubercle, and the acromial head of the deltoid muscle. Anatomic landmarks for positioning the skin incision include the acromion of the scapula, the greater tubercle of the humerus, and the pectoral muscles. The suprascapular nerve courses over the craniolateral surface of the scapula, and the caudal circumflex humeral artery and axillary nerve pass on the caudolateral aspect of the shoulder and should be avoided.

Positioning

For either medial or lateral luxation, the animal is positioned in dorsal recumbency with the affected leg draped. The prepared area should extend from the dorsal and ventral midlines to below the elbow.

SURGICAL TECHNIQUE

Surgical Stabilization of a Medial Luxation

A craniomedial approach to the shoulder joint is used to expose the luxated joint. It may be helpful to reduce the joint before the approach is made to reestablish normal anatomic relationships. *Beginning at the medial aspect of the acromion, incise the skin and subcutaneous tissue over the greater tubercle, and continue the incision medially to the midhumeral diaphysis (Fig. 34.23A). Incise the fascia along the lateral border of the brachiocephalicus muscle, and retract the muscle medially (Fig. 34.23B). Incise the insertions of the superficial and deep pectoral muscles from the humerus, and retract them medially. Carefully incise the fascial attachment between the deep pectoral muscle and supraspinatus muscle to prevent damaging the suprascapular nerve (Fig. 34.23C). Retract the supraspinatus muscle laterally. Transect the tendon of the coracobrachialis muscle to expose the subscapularis muscle tendon (Fig. 34.23D). If the joint capsule is not torn, incise it to inspect the joint and assess the condition of the humeral head and medial labrum of the glenoid (Fig. 34.23E).* If the labrum is torn, the prognosis for successful stabilization of the shoulder is poor. The tendon of the coracobrachialis muscle may be torn and retracted with traumatic luxation. *Reduce the joint, and imbricate the capsule and subscapularis tendon with nonabsorbable mattress sutures.* If this does not sufficiently stabilize the joint, prosthetic capsulorrhaphy/ligament augmentation or biceps tendon transposition are required. *For shoulder joint stabilization, place two bone anchors in the distal scapula at the sites of origin of the medial glenohumeral ligament and a single bone anchor in the humerus at the point of insertion (Fig. 34.24). Connect the humeral and scapular bone anchors with heavy gauge suture. Tighten the sutures to minimize abduction of the joint but not so tightly as to limit joint motion. Close the surgical site routinely.* Alternatively, biceps tendon transposition may be performed by freeing and moving the tendon medially, and securing it to the humerus with a bone screw and spiked washer (Fig. 34.25). *For closure, suture the joint capsule, and then suture the pectoral muscles to the deltoid fascia. Suture the subcutaneous tissue and skin separately.*

Surgical Stabilization of a Lateral Luxation

A lateral approach to the shoulder joint is used to expose the luxated joint (see Fig. 34.19). *Make a lateral skin incision from the midportion of the scapular spine distally to the lateral surface of the humerus. Retract the omotransversarius and trapezius muscles craniodorsally and the acromial part of deltoideus muscle caudally to expose the acromion process and greater tubercle of the humerus. Reduce the joint and imbricate the capsule with nonabsorbable mattress sutures.* If sufficient stabilization is not achieved, prosthetic capsulorrhaphy/ligament augmentation or biceps tendon transposition are required. Prosthetic capsulorrhaphy/ligament augmentation with heavy suture can be achieved with bone anchors, bone tunnels, or a combination of these. *Close as described previously.*

Shoulder Arthrodesis

Perform a combined craniolateral and cranial approach to the shoulder with osteotomy of the acromion process (see p. 1049) and greater tubercle (Fig. 34.26). Incise the skin and subcutaneous tissue and deepen the incision through the pectoral muscles as described previously for a medial luxation (Fig. 34.26A). Perform an osteotomy of the greater tubercle that includes the supraspinatus muscular insertion (Fig. 34.26B). Incise the joint capsule to inspect the joint (Fig. 34.26C). Detach the biceps tendon from the supraglenoid tubercle. Using an oscillating saw, perform ostectomies of the glenoid process and humeral head, which parallel each other when the humerus is held at an angle of approximately 105 degrees to the scapula (the exact angle should be customized to the individual animal) (Fig. 34.27A). Take care to preserve the suprascapular nerve and caudal circumflex humeral artery. Appose the flat surfaces and temporarily stabilize the bones with a K-wire driven through the cranial aspect of the humerus and into the glenoid. Contour an 8- or 10-hole plate to fit from the dorsocranial junction of the spine and scapula over the cranial aspect of the humerus (Fig. 34.27B). Use of a locking plate may significantly increase the stiffness of the construct and decrease the risk of nonunion.

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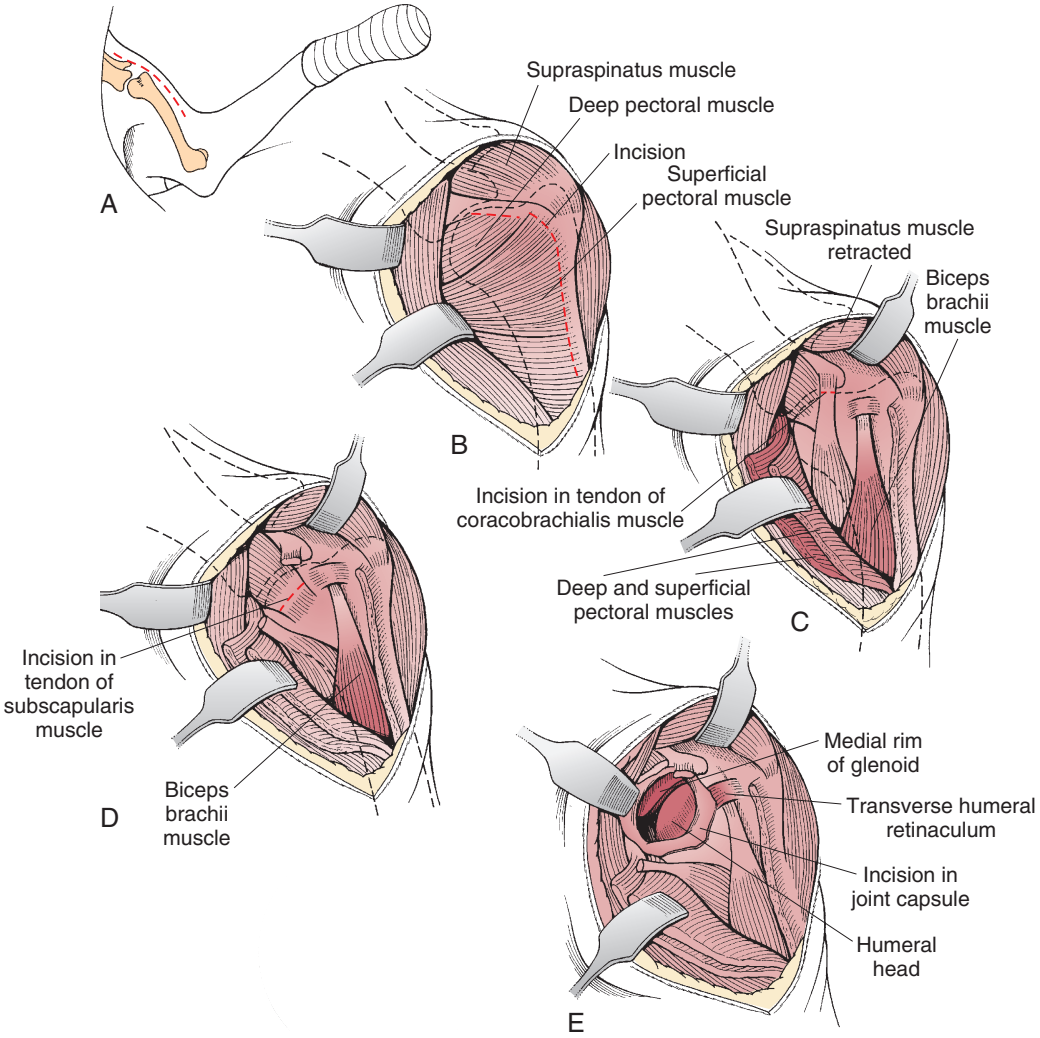


FIG. 34.23 Surgical stabilization of a medial shoulder luxation. (A) Perform a craniomedial approach with biceps tendon transposition. Incise the skin and subcutaneous tissue over the greater tubercle and continue the incision medially to the midhumeral diaphysis. (B) Incise the fascia along the lateral border of the brachiocephalicus muscle. (C) Incise the insertions of the superficial and deep pectoral muscles from the humerus and retract them medially. Retract the supraspinatus muscle laterally. (D) Transect the tendon of the coracobrachialis muscle to expose the subscapularis muscle tendon. Incise the tendon of the suprascapularis muscle. (E) Incise the joint capsule to inspect the joint.

Be sure that the plate does not impinge on the suprascapular nerve. If necessary, contour the cranial humerus with rongeurs to obtain a better fit for the plate. Place a cancellous bone autograft between and around the ostectomies to aid in bone healing. Insert one of the screws across the apposed ostectomy lines as a lag screw. Remove the K-wire and attach the biceps tendon to the surrounding fascia, or attach it to the humerus with a bone screw and spiked washer. Secure the greater tubercle to the humerus lateral to the plate with K-wires or bone screws. Suture the soft tissue and close the skin routinely.

Excision Arthroplasty

Perform a craniolateral approach to the shoulder with osteotomy of the acromion process (see Fig. 33.16). Detach the biceps tendon from the supraglenoid tubercle and incise the joint capsule. Using an oscillating saw or osteotome, perform an ostectomy of the glenoid (Fig. 34.28). Take care to preserve the suprascapular nerve and caudal circumflex humeral artery when performing the excision. Bevel the glenoid so that the lateral edge is longer than the medial edge. Part of the humeral head can be ostectomized to create a vascular surface to promote pseudoarthrosis. *Pull the teres minor muscle over the glenoid ostectomy site, and suture it to the medial joint capsule and biceps tendon to provide soft tissue interposition between bone surfaces. Pull the acromion process proximally until the deltoid muscle is taut. Wire the process to the scapular spine (see p. 1049), then close the soft tissue.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Stabilization techniques for scapulohumeral luxations require retractors, a periosteal elevator, osteotome and mallet, K-wires, orthopedic wire, wire driver, wire twister, bone screw and spiked

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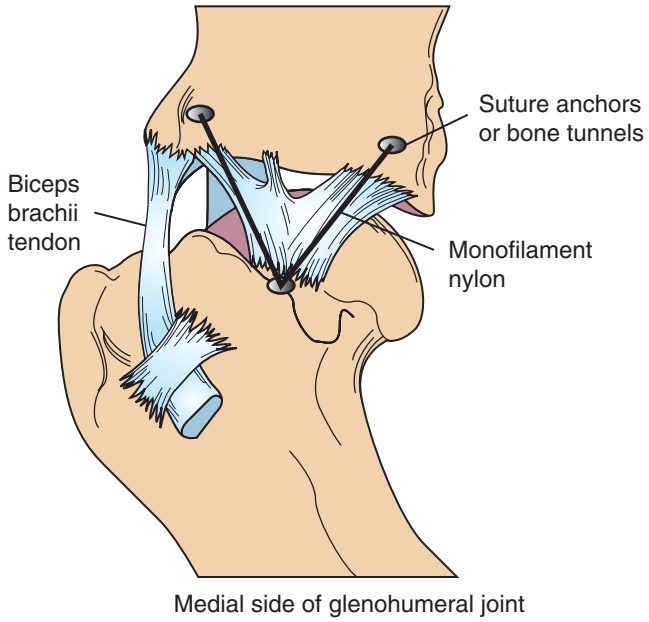


FIG. 34.24 Positioning of bone anchors for stabilization of medial glenohumeral luxation.

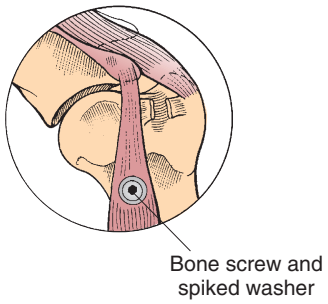


FIG. 34.25 To transpose the biceps tendon, incise the transverse humeral ligament. Make a small incision in the joint capsule under the biceps tendon to free it and move the tendon medially. Secure it to the humerus with a bone screw and spiked washer.

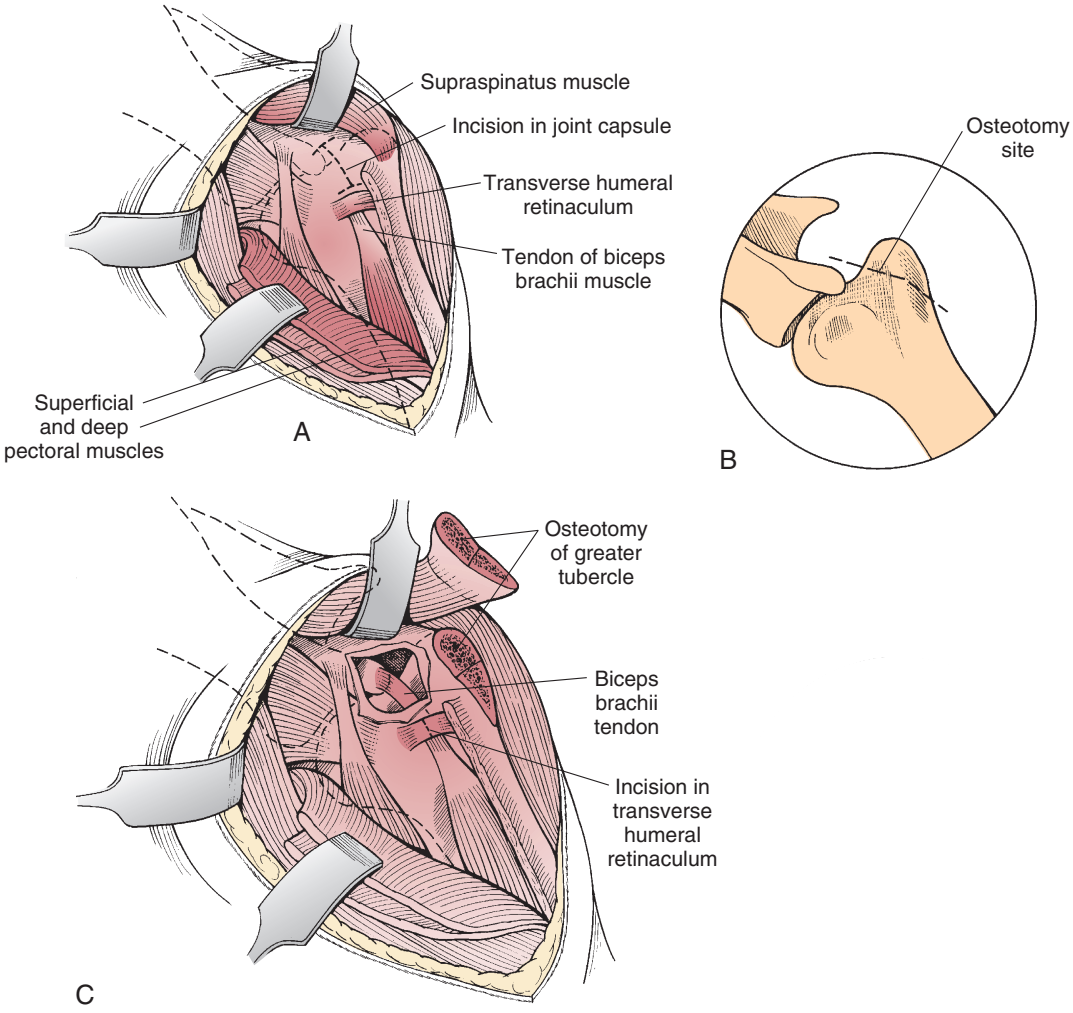


FIG. 34.26 A combined craniolateral and cranial approach to the shoulder is performed for shoulder arthrodesis. (A) Expose the joint. (B) Perform an osteotomy of the greater tubercle, including the supraspinatus muscle insertion. (C) Incise the joint capsule and the transverse humeral ligament over the biceps tendon.

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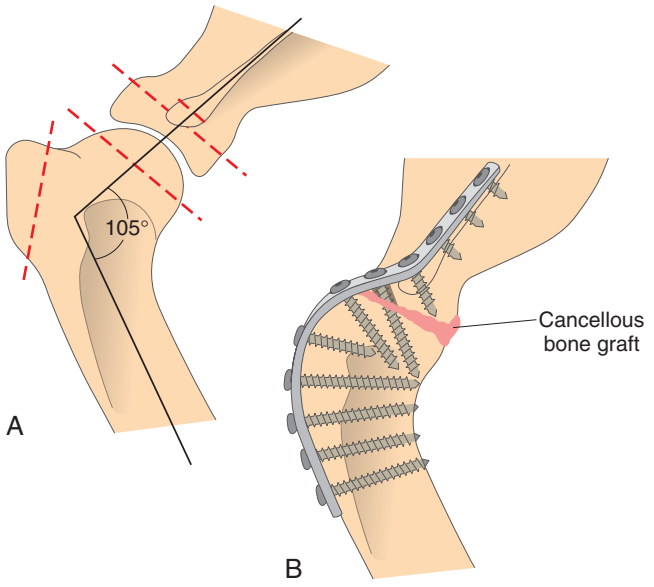


FIG. 34.27 Shoulder arthrodesis. (A) Osteotomize the greater tubercle and acromion process. Make the ostectomy lines for the glenoid and humeral head parallel to each other when the humerus is held at 105 degrees to the scapula. (B) Compress the bone surfaces with a contoured compression plate. Place a lag screw across the fracture line. A cancellous bone autograft may be added.

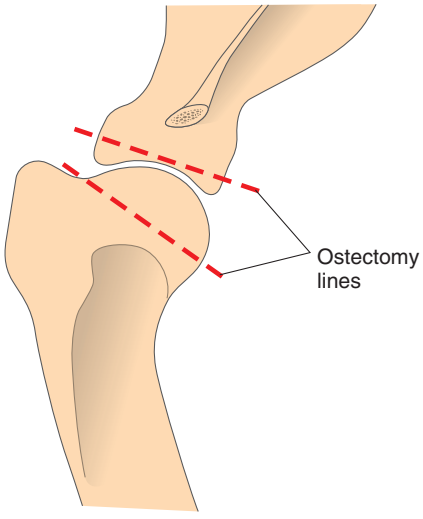


FIG. 34.28 Ostectomy lines for glenoid excision.

washer, drill, drill bit, tap, depth gauge, and screwdriver. Plating equipment is required for arthrodesis. Excision arthroplasty requires instruments similar to those for luxations.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiography is performed to document the position of the humeral head and any implants used. After lateral luxation, the limb should be supported in a spica splint (Fig. 34.29) for 10 to 14 days; after medial luxation, it should be supported in a Velpeau sling (Fig. 34.30) for a similar period. Owners should be cautioned to limit activity and protect the bandage. After removal of the bandage, rehabilitation exercises can begin; however, uncontrolled activity should be limited for an additional 6 to 12 weeks (see Chapter 11).



FIG. 34.29 Spica splint applied to forelimb to temporarily immobilize the shoulder joint and scapula.



FIG. 34.30 Velpeau sling applied to forelimb to prevent weight bearing and provide some stability to the proximal forelimb and shoulder joint.

Spica Splints

Spica splints envelop the torso and affected limb and are often used as temporary splints to immobilize shoulder joint. *Place adhesive tape stirrups on the cranial and caudal surfaces of the limb, and apply cotton cast padding to the limb and torso. Begin the padding at the paw and wrap it proximally in a spiral fashion, overlapping it 50%. When the axillary region is reached, wrap the cotton padding around the animal's torso several times, alternating cranially and caudally to the affected limb. Then wrap elastic gauze over the cast padding (50% overlap). Wrap the gauze firmly around the limb for mild compression of the soft tissue. Reinforce the spica bandage with fiberglass casting material to provide additional stabilization of the shoulder joint. Fold the casting tape onto itself to provide a lateral splint that is four to six layers thick, extending from toes to dorsal midline. Use*

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Vetrap or elastic adhesive tape to hold the fiberglass cast to the limb and provide an outer covering for the splint (see Fig. 34.29).

Velpeau Slings

Velpeau slings prevent weight bearing and provide some stability to the proximal forelimb (see Fig. 34.30). They are most often used to help maintain closed or open reduction of medial shoulder luxations. *With the shoulder and elbow flexed and the limb adducted against the body wall, begin by placing two or three layers of padding around the torso and limb. Wrap layers cranial and caudal to the opposite limb to prevent slippage of the incorporated limb. Place an additional layer in a similar manner with gauze (e.g., Kling) to add mild compression. Place an outer layer of elastic tape or Vetrap to provide support.* Velpeau slings should not be used for more than 2 weeks. Long-term limb immobilization in a flexed position makes rehabilitation difficult.

After shoulder arthrodesis, postoperative radiographs should be made to evaluate the implant's position and to serve as a comparison for subsequent evaluations. A spica splint is applied until radiographic signs of bone union are seen, usually in 6 to 12 weeks. Once bone union is evident, the splint can be removed and the animal gradually returned to full activity.

After excision arthroplasty, postoperative radiographs are assessed to document alignment of the scapula and humeral head. Early postoperative use of the limb should be encouraged. The animal should be walked on a leash and physical therapy (range of motion exercises) should be performed beginning 1 or 2 days after surgery. Once the sutures have been removed (10–14 days after surgery), activity should be encouraged to promote rapid formation of a pseudoarthrosis. Lameness is often evident for 4 to 8 weeks after surgery. Physical therapy may be of particular benefit following excision arthroplasty. Programs usually consist of passive range of motion and cryotherapy postoperatively followed by swimming and walking beginning at 3 weeks.

COMPLICATIONS

The major complication associated with surgical repair of scapulohumeral joint luxations is reluxation of the humeral head. With traumatic luxation, most dogs regain normal limb use; however, some DJD may occur and require periodic treatment with analgesics.

PROGNOSIS

After closed reduction, the prognosis is good for maintenance of reduction and return of limb function if the joints are stable during forelimb manipulation. However, the prognosis is guarded if the joints appear unstable; open reduction and joint stabilization should be considered in such cases. The prognosis is guarded for dogs with congenital or developmental medial luxation. When it is not possible to maintain reduction of scapulohumeral joints, a salvage procedure (e.g., arthrodesis) may be indicated.

SHOULDER INSTABILITY

DEFINITION

Shoulder instability is characterized by a pathologic increase of the range of motion of the shoulder joint, most commonly in the mediolateral plane. Synonyms include *shoulder subluxation* and *glenohumeral instability*.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Shoulder instability is due to tearing or stretching of the medial or lateral supportive structures of the shoulder joint. The injury may be due to low-grade chronic trauma or acute blunt trauma, resulting in instability and subluxation of the joint without complete luxation. Damaged structures may include the medial or lateral glenohumeral ligaments and subscapularis tendon. Damage to the biceps tendon may also be apparent. Dogs with shoulder instability may have varying degrees of osteoarthritis.

DIAGNOSIS

Clinical Presentation

Signalment

Dogs of any size may be affected.

History

This disease occurs most commonly in active dogs, but it may be diagnosed in older animals after years of exercise and activity. The animal typically has a history of moderate chronic lameness. Less commonly, there may be an acute onset of lameness and a history of mild trauma.

Physical Examination Findings

A moderate weight-bearing lameness is usually observed on gait analysis. Orthopedic examination may reveal moderate muscle atrophy of the affected limb. Palpation of the limb may demonstrate increased angles of abduction in cases of medial shoulder instability (Fig. 34.31). Most dogs have pain on manipulation of the shoulder joint.

Diagnostic Imaging

Radiographs are usually normal in shoulder instability, although some cases may have changes compatible with osteoarthritis. Dogs with concurrent bicipital tenosynovitis may have osteophytosis and sclerosis of the bicipital groove on skyline views.

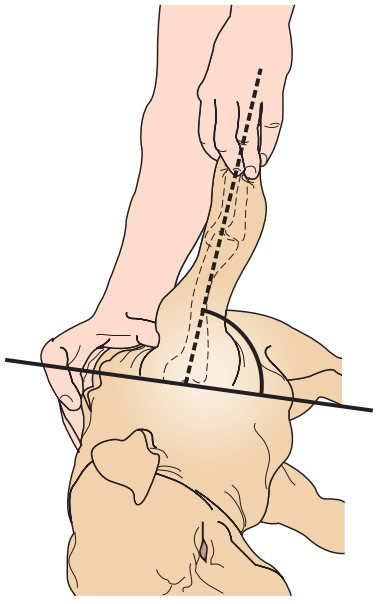


FIG. 34.31 Abduction palpation of the shoulder joint to evaluate for medial instability.

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Arthroscopy

Definitive diagnosis of shoulder instability is made by arthroscopy. Arthroscopic findings may include tearing or laxity of the medial glenohumeral ligament, lateral glenohumeral ligament, or subscapularis tendon. Tearing or inflammation of the biceps tendon may also be evident.

Laboratory Findings

Specific laboratory abnormalities are not seen. Arthrocentesis of the shoulder joint will show changes compatible with osteoarthritis.

DIFFERENTIAL DIAGNOSES

Shoulder instability must be differentiated from OCD, biceps disease, osteoarthritis, other trauma, and neoplasia.

MEDICAL MANAGEMENT

Medical management consists primarily of rest and physical therapy and is recommended in mild cases. Shoulder stabilization orthotics that prevent thoracic limb abduction and shoulder extension/flexion are available and have been reported to be effective.[16] Rehabilitation programs are designed to maintain range of motion while strengthening the muscles around the shoulder joint to minimize instability. Physical rehabilitation for shoulder instability should focus on strengthening periarticular structures and improving comfortable active range of motion (see Chapter 11).

Body weight management in overweight dogs aids in decreasing loads on the joint and limiting the signs of osteoarthritis.

SURGICAL TREATMENT

Treatment of shoulder instability is an advanced technique that should be performed by an individual with significant experience in the management of joint disease and surgery. Treatment of shoulder instability has been performed with either arthroscopic or open surgery, and neither method has been shown to be superior.[16] Arthroscopic-assisted techniques include arthroscopic-assisted anchor and suture techniques, radiofrequency shrinkage of the attenuated ligaments and joint capsule, and tightrope (TR) stabilization of medial shoulder instability. Radiofrequency shrinkage is not recommended due to serious complications that are well documented in human medicine. Open surgery involves placement of suture anchors at the sites of origin and insertion of the medial glenohumeral ligament.

Arthroscopy for Shoulder Instability

AP *Perform standard lateral shoulder arthroscopy, and examine the medial glenohumeral ligament and subscapularis tendon and the biceps tendon and cartilage surfaces. Observe for tearing or significant laxity of the medial supporting structures (Fig. 34.32). If tearing or laxity is not apparent, switch to a craniomedial arthroscope portal for examination of the lateral supporting structures. To perform a craniomedial arthroscope portal, insert a switching stick through a standard instrument portal, advance the tip of the stick to the medial aspect of the biceps tendon, and push the skin out. Incise the skin over the switching stick and advance the stick out of the joint. Remove the arthroscope from the lateral portal and pass the arthroscope cannula over the switching stick. Evaluate the lateral collateral ligament for tears and stretching.* If a pathologic condition is identified, arthroscopic-assisted anchor and suture techniques to stabilize the joint can be considered; however, this is a highly advanced technique.

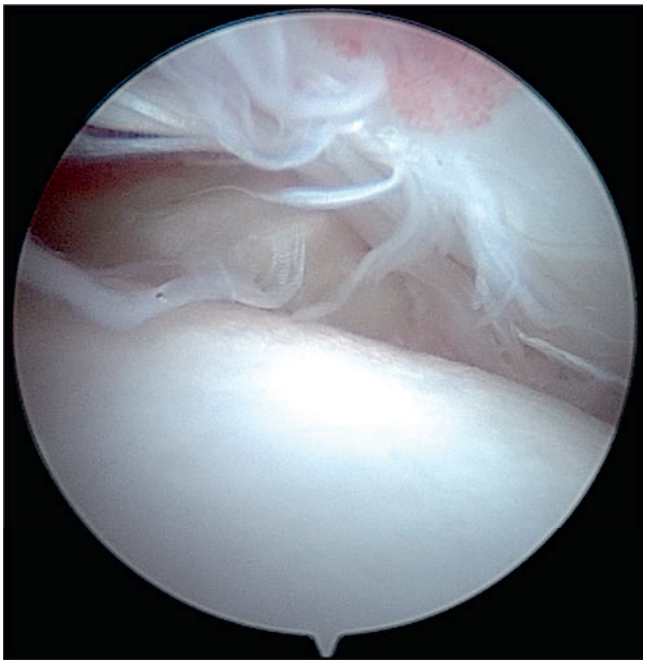


FIG. 34.32 Arthroscopic view of the medial aspect of the shoulder showing tearing of the subscapularis tendon and medial glenohumeral ligament.

Tightrope Stabilization of Medial Shoulder Instability

Position the patient with a hanging-limb prep for access to both the medial and lateral aspects of the shoulder joint. Perform standard (inverted) lateral shoulder arthroscopy, and examine the medial glenohumeral ligament and subscapularis tendon and the biceps tendon and cartilage surfaces. Observe for tearing or significant laxity of the medial supporting structures. Make a small ~2-cm incision along the cranial border of the superficial pectoral muscle in the axillary region at the level of the joint. Use blunt dissection to palpate and access the medial aspect of the shoulder joint through this incision. Insert a cannulated drill bit guide pin through the incision and into the medial compartment of the joint. With arthroscopic guidance, place the tip of the guide pin midway between the cranial and caudal origins of the medial glenohumeral ligament proximal to the rim of the glenoid. Drive the guide pin through the scapula at an angle such that it exits on the lateral aspect of the neck, proximal to the region of the suprascapular nerve and just cranial to the acromion process. Insert a second cannulated drill bit guide pin through the incision and into the medial compartment of the joint. With arthroscopic guidance, place the tip of the second guide pin on the humerus at either the insertion of the medial glenohumeral ligament or the insertion of the subscapularis tendon, depending on the predominant pathology present. Drive the guide pin through the proximal humerus at an angle such that it exits on the lateral aspect of the proximal humerus at the distal aspect of the greater tubercle at the cranial border of the acromial head of the deltoid. Make a small approach to the proximal humerus on the lateral side at the site where this guide pin exits. Overdrill both of the guide pins with the cannulated drill bit (3.55 mm),

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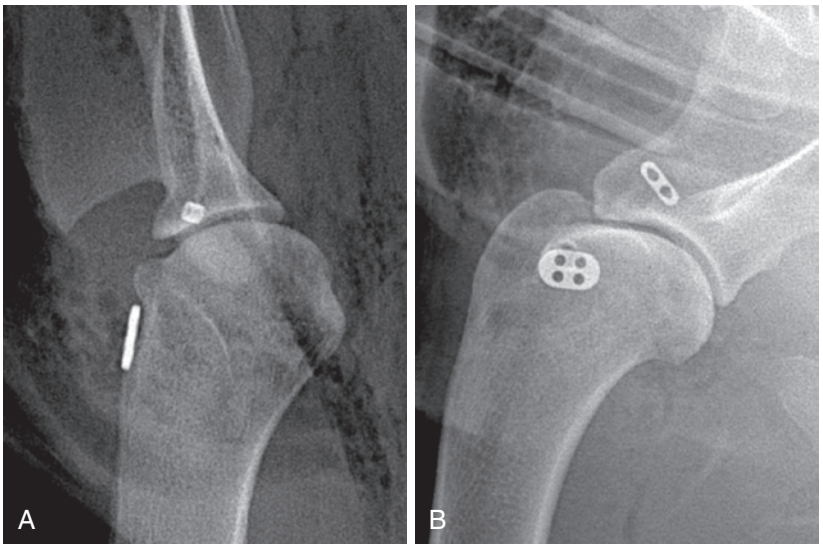


FIG. 34.33 (A) Craniocaudal and (B) lateromedial postoperative radiographs following tightrope stabilization of medial shoulder instability. (Courtesy Dr. James Cook.)

starting with the humerus from medial to lateral. When the drill bit exits the lateral side of the humerus, remove the guide pin laterally and push the tip of the TR lead needle into the lumen of the drill bit. Retract the drill bit medially while pushing the TR lead needle into the humeral tunnel, pulling it through until the TR toggle exits medially. Then drill the scapular tunnel from lateral to medial. When the drill bit exits medially, place the tip of the TR lead needle into the lumen of the drill bit to follow the drill bit through the scapular tunnel from medial to lateral as the drill bit is retracted. When the toggle exits the hole on the lateral aspect of the scapula, manipulate the fiber tape and lead suture so that the toggle is seated firmly against the lateral aspect of the scapula. A small incision may be made over the scapula to be sure that the toggle is seated properly against the bone. *Tighten the fiber tape and tie securely over the button on the lateral aspect of the humerus to eliminate the medial instability. Routinely close all incisions. Obtain craniocaudal (Fig. 34.33A) and lateromedial (Fig. 34.33B) radiographs to document location of the bone tunnels and the toggle and button.*

Open Surgery for Shoulder Instability

Perform a craniomedial approach to the medial aspect of the shoulder joint (see p. 1164). Place two bone anchors in the distal scapula at the sites of origin of the medial glenohumeral ligament (see Fig. 34.24) and a single bone anchor in the humerus at the point of insertion. Connect the humeral and scapular bone anchors with heavy gauge nylon. Tighten the sutures to minimize abduction of the joint but not so tightly as to limit joint motion. Close the surgical site routinely.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Arthroscopic management of shoulder instability requires a radiofrequency system with shrinkage probes. Thorough knowledge of the mechanism and precautions of this equipment is imperative before its use. Radiofrequency can cause severe and permanent damage to cartilage and soft tissue structures around the joint. TR repair of medial shoulder instability requires a TR implant and appropriately sized cannulated drill bits and drill guides. A power drill is also required. Open repair of shoulder instability requires bone anchors or bone screws and heavy nylon suture material or orthopedic suture for stabilization.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative care for TR stabilization requires the use of hobbles (e.g., Dogleggs Therapeutic and Rehabilitative Products) for 6 to 12 weeks. Activity should be restricted to leash walking for a minimum of 8 weeks after surgery. Passive range of motion, swimming, and other muscle building activities should be employed. The use of physical therapy in the management of this disease is highly recommended (see Chapter 11). Reevaluation of the patient should include a lameness evaluation and joint palpation for stability every 4 weeks. Return to full athletic function is initiated at 16 weeks following surgery when stability has been achieved.

Postoperative care following open surgery requires placement of the affected limb in a Velpeau sling for 1 to 2 weeks (see p. 1166). The Velpeau sling can result in significant complications, particularly contracture of the carpus, therefore extreme care and careful observation must be used. Use of physical therapy in the management of this disease may be very beneficial and is highly recommended (see Chapter 11). Reevaluation of the patient should include a lameness evaluation and joint palpation for stability every 2 to 4 weeks.

PROGNOSIS

Prognosis for treatment of shoulder instability is variable depending on the severity of the soft tissue injuries and the osteoarthritis. Reports of both open and arthroscopic techniques have suggested favorable results.[16]

BICEPS TENDON DISEASE

DEFINITIONS

Biceps tendon disease (tendinopathy, tendinitis) is a general term that incorporates various types of injury, including tearing

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of the tendon and inflammation of the tendon and surrounding synovial sheath. **Bicipital tenosynovitis** is an inflammation of the biceps brachii tendon and its surrounding synovial sheath. **Bicipital tenotomy** is a technique where the tendon is released from its origin near the superglenoid tubercle. **Bicipital tenodesis** involves surgically moving the origin of the bicipital tendon and stabilizing it to the humerus. **Supraspinatus tendinopathy** refers to disease of the insertional tendon of the supraspinatus complex that lies craniolateral to the biceps tendon.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The cause of bicipital tenosynovitis is either direct or indirect trauma to the bicipital tendon. Repetitive injury or overuse may be an inciting factor. Chronic inflammation causing synovial hyperplasia and dystrophic mineralization of the tendon may occur. The tendon may be partly or completely ruptured. Proliferative fibrous connective tissue and adhesions between the tendon and sheath limit motion and cause pain. Osteophytes may form in the intertubercular groove. Mineralization of the supraspinatus tendon may cause a secondary mechanical biceps tenosynovitis and lameness that is refractory to steroid injection but responsive to curettage of the mineralization. The biceps is a significant stabilizer of the shoulder joint, and biceps tenosynovitis may occur in conjunction with generalized shoulder instability (see p. 1167).

DIAGNOSIS

Clinical Presentation

Signalment

Dogs of any size and age may be affected. There is no predisposition in either gender. Working, active dogs are most commonly affected.

History

Intermittent or progressive forelimb lameness that worsens after exercise is common. Tenosynovitis usually presents as a chronic progressive lameness. Tearing of the tendon may be associated with an acute onset of lameness and a history of blunt trauma.

Physical Examination Findings

Lameness of one forelimb is usually evident. The animal usually stands on the limb but is lame during gaiting. Pain may be evident during palpation of the bicipital tendon, especially with concurrent flexion of the shoulder and extension of the elbow. Pain may also be evident during palpation of the biceps muscle while the dog is standing. Thickening of the biceps bursa may be palpable in some dogs. In some cases the condition is bilateral. Atrophy of the supraspinatus and infraspinatus muscles may be palpable.

Diagnostic Imaging

Radiographic views should include standard lateral projections of both shoulders. A craniocaudal projection of the humerus (skyline) with the shoulder flexed can be used to identify the bicipital groove. Calcification of the biceps tendon and osteophytes in the intertubercular groove may be evident, which should be differentiated from supraspinatus tendinopathy with mineralization (see p. 1286). Arthrography can be used to outline the tendon and reveal irregularities and filling defects suggestive of synovial hyperplasia, tendon rupture, and joint mice. Caution is indicated in interpretation of arthrography because the biceps tendon sheath is a continuation of the shoulder joint capsule, and any disease that causes inflammation of the shoulder joint may also cause synovial proliferation of the biceps tendon sheath. Radiographic findings of synovial proliferation within the sheath therefore are not diagnostic for biceps tendon disease.

Ultrasound provides excellent imaging of the biceps tendon, adjacent tendons, and the bursa and groove. Both transverse and longitudinal views should be obtained. The normal biceps tendon has an echogenic linear echo-texture in a uniform pattern. Acute trauma results in fiber pattern disruption and fluid accumulation within or around the tendon. Chronic trauma may demonstrate dystrophic mineralization. Fluid accumulation and synovial proliferation of the tendon sheath are easily appreciated, but they cannot be differentiated between those caused by bicipital disease and those associated with shoulder joint inflammation of other causes.

MRI provides excellent imaging of the biceps tendon and all the soft tissue structures surrounding the shoulder joint and can differentiate inflammation and partial and complete tears of these structures.

Arthroscopy provides direct visualization of the proximal biceps tendon and sheath and definitive diagnosis of disease in most cases (Fig. 34.34A–C). From a standard lateral arthroscopy portal, flexing the shoulder and elbow to approximately 90 degrees each maximizes observation of the tendon. The normal biceps tendon has a small cuff of proliferative tissue around the origin of the biceps tendon, which should not be interpreted as disease. Moderate to severe fibrous proliferation at the origin of the tendon or obvious tearing is compatible with disease. The joint should be carefully observed for damage to other supporting structures.

Laboratory Findings

The results of hematologic and serum biochemical analyses reflect the dog's general health. The results of arthrocentesis may suggest mild inflammation and DJD, with higher concentrations of monocytes and macrophages in the joint fluid. Evidence of sepsis is a contraindication to arthrography or intraarticular corticosteroid therapy.

DIFFERENTIAL DIAGNOSIS

Bicipital tenosynovitis must be differentiated from osteoarthritis of the shoulder, shoulder instability, supraspinatus tendonitis, OCD, early osteosarcoma or other bone tumors of the proximal humeral metaphysis, synovial osteochondromatosis, cervical disc disease, or neurofibroma of the brachial plexus. Definitive diagnosis is confirmed with arthroscopic examination of the shoulder joint (see p. 1168).

MEDICAL MANAGEMENT

Medical therapy is generally used initially to treat dogs with bicipital tenosynovitis. Methylprednisolone acetate (10–40 mg) or triamcinolone (3 mg) can be infiltrated into the bicipital tendon sheath or injected into the scapulohumeral joint. This treatment is followed by confinement for 6 weeks. After the confinement period, exercise should be increased gradually to strengthen the surrounding musculature. Oral administration of corticosteroids does not appear to be effective.

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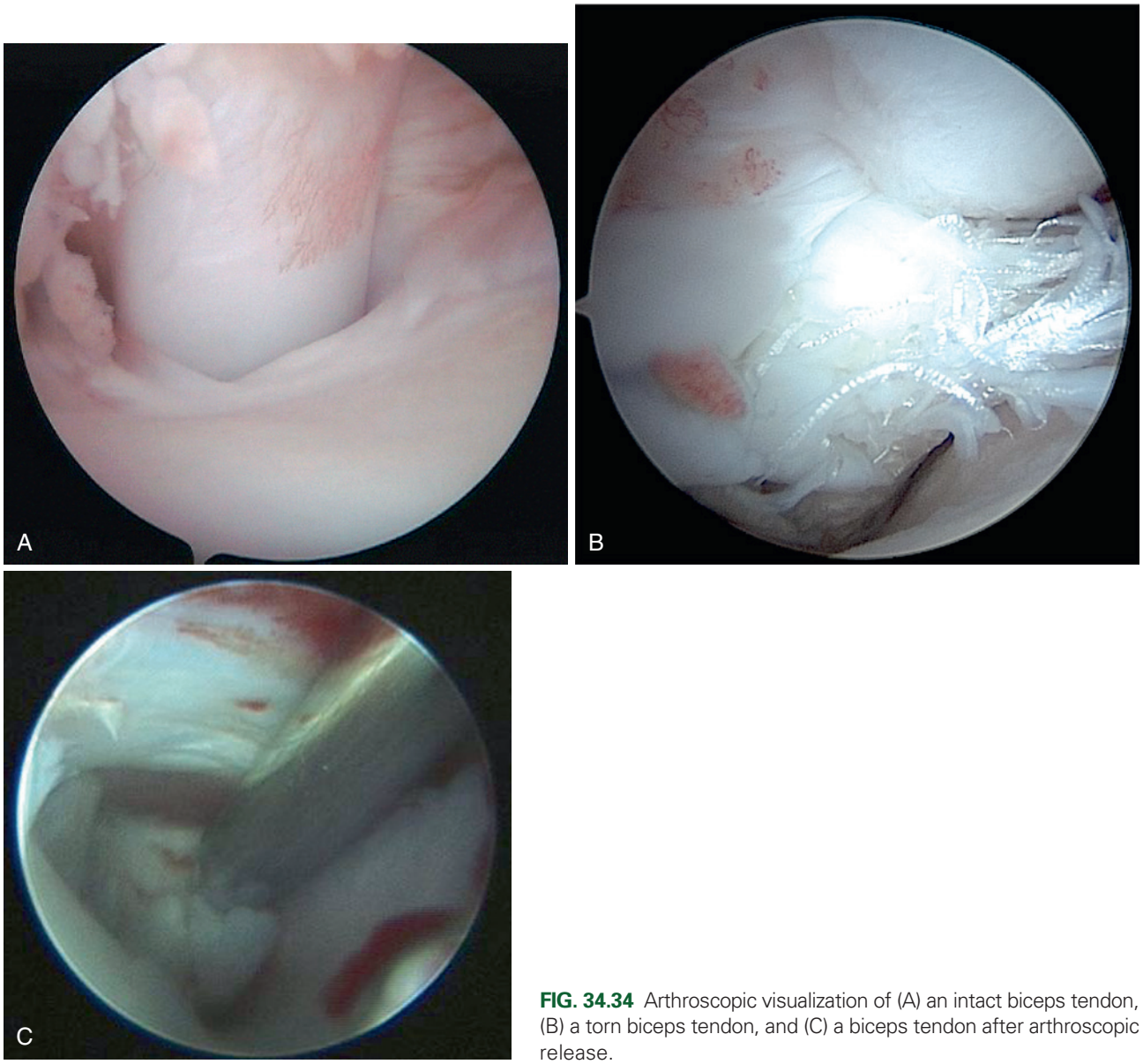


FIG. 34.34 Arthroscopic visualization of (A) an intact biceps tendon, (B) a torn biceps tendon, and (C) a biceps tendon after arthroscopic release.

Body weight management in overweight dogs aids in decreasing loads on the joint and may reduce the clinical signs associated with osteoarthritis.

SURGICAL TREATMENT

Bicipital tendon tenotomy or tenodesis is performed to eliminate movement of the biceps tendon in the inflamed tendon sheath or to release a partially torn or avulsed biceps tendon. This procedure is indicated in dogs with ruptured bicipital tendons and chronic bicipital tenosynovitis, if the condition has not responded to medical therapy. As an alternative, surgical or arthroscopic exploration and resection of mineralization and inflamed synovium may be effective. Confirmation of the disease and evaluation of the other supporting structures of the shoulder joint should be performed before surgical therapy. Tenotomy or tenodesis should only be performed when either there is confirmation of a partial tear of the biceps tendon or there is evidence of chronic biceps tenosynovitis with documentation of normalcy of the other supporting structures of the shoulder joint, including the medial and lateral glenohumeral ligaments, subscapularis tendon, and supraspinatus tendon. Tenodesis or tenotomy of the biceps tendon in a shoulder with other significant soft tissue injuries may contribute to irreversible instability of the joint.

Preoperative Management

The overall health of the animal should be determined before surgery.

Anesthesia

Anesthetic protocols for the management of dogs with orthopedic disease that are stable or that have sustained trauma are provided in Table 32.2. Perioperative analgesics are provided in Box 32.1.

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Surgical Anatomy

Surgical anatomy of the shoulder is discussed on p. 1164. The bicipital tendon arises from the supraglenoid tubercle and crosses the cranial portion of the scapulohumeral joint. The tendon courses through the intertubercular groove of the humerus and is held in position by the transverse humeral ligament. The tendon is surrounded by a synovial sheath that communicates with the scapulohumeral joint.

Positioning

For arthroscopic or open treatment, the dog is positioned in lateral recumbency. The prepared area should extend from the dorsal and ventral midlines to below the elbow.

SURGICAL TECHNIQUE

Arthroscopic Treatment

Standard lateral arthroscopy is performed on the affected shoulder joint (see p. 1156). *Examine the entire joint, paying particular attention to the biceps tendon (Fig. 34.35A), medial glenohumeral ligament, and subscapularis tendon.* If a pathologic condition of the biceps tendon (such as partial rupture or avulsion of its origin; see Fig. 34.35B–C) is identified and the remainder of the joint is normal, tenotomy of tendon may be performed. *Establish a cranial instrument portal by inserting a needle adjacent to the biceps tendon (medial or lateral). Once the needle can be visualized through the arthroscope, incise adjacent to the needle to establish the cranial portal. Insert an arthroscopic blade through a cannula or a No. 11 blade without a cannula, and transect the tendon below the site of disease. Resect the remaining origin of the tendon with a power shaver, scalpel blade, or arthroscopic punch.*

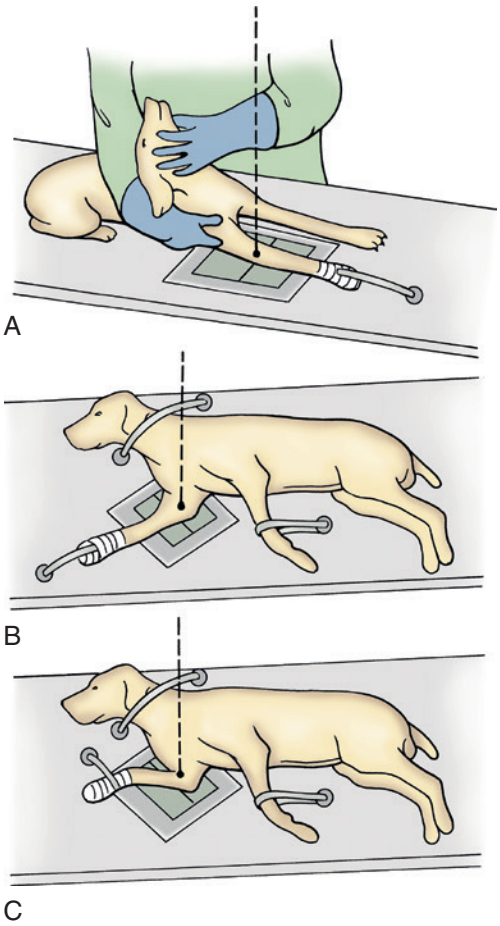


FIG. 34.35 (A) For a craniocaudal radiograph of the elbow, position the dog in sternal recumbency with the elbow flexed 30 degrees and slightly medially rotated. For (B) standard lateral and (C) flexed lateral views, position the dog in lateral recumbency with the elbow against the cassette.

Open Surgical Treatment

An approach to the cranial region of the shoulder joint is used to expose the bicipital tendon and bicipital groove (see Fig. 34.23). *Incise the transverse humeral ligament and joint capsule to expose the tendon and intertubercular groove. Transect the tendon near the supraglenoid tubercle. If desired, reattach the tendon to the humerus distal to the groove with a bone screw and spiked Teflon or stainless steel washer. As an alternative, redirect the tendon through a bone tunnel created in the humerus, and suture it to the supraspinatus muscle.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

No special arthroscopic equipment is required for arthroscopic treatment. For tenodesis, equipment for placing a screw and spiked washer is needed.

POSTOPERATIVE CARE AND ASSESSMENT

Following arthroscopic treatment, the dog should be rested without significant activity for 2 to 3 weeks. Use of a sling is not necessary. After tenodesis, the animal should be closely confined for 4 weeks, after which gradual resumption of normal activity should be encouraged. Postoperative physical therapy can aid in the rehabilitation after biceps surgery. Therapy involves cryotherapy, passive range of motion, and short leash walks followed by gradual muscle strengthening exercises.

PROGNOSIS

The results of medical treatment range from excellent to poor. The results of arthroscopic and surgical treatment are generally good to excellent. A long-term study of the outcome of arthroscopic tenotomy reported excellent subjective (client questionnaire) results at 12 to 48 months in 22 of 24 shoulders.[17] The time required for dogs to regain optimum function ranges from 2 to 9 months.

SCAPULAR LUXATION

DEFINITION

Scapular luxation or *dislocation* is characterized by upward displacement of the scapula after rupture of supporting musculature.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Scapular luxation is rare but occasionally occurs with trauma that causes the serratus ventralis, rhomboideus, and trapezius muscular insertions to be torn from their scapular attachments. This allows upward displacement of the scapula during weight

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bearing. Concurrent injuries (i.e., rib fractures, pneumothorax, pulmonary contusions) are common and may require immediate treatment.

DIAGNOSIS

Clinical Presentation

Signalment

Scapular luxation occurs more often in cats than dogs; animals of either gender or any age may be affected.

History

The animal typically has a recent history of trauma.

Physical Examination Findings

Notable dorsal displacement of the scapula is observed as the animal bears weight on the affected limb. Adduction of the limb induces lateral scapular displacement. The symmetry of scapular location and prominence should be compared with the normal limb.

Diagnostic Imaging

Survey radiographs of the thorax should be evaluated for signs of thoracic trauma, such as fractured ribs, pulmonary contusions, or pneumothorax. Usually, the scapula is not fractured but may appear displaced.

Laboratory Findings

Specific laboratory abnormalities are not seen. Traumatized animals undergoing surgery should have sufficient measurement of blood values to determine the optimum anesthetic regimen.

DIFFERENTIAL DIAGNOSIS

Scapular luxation must be differentiated from a scapular fracture (see p. 1047).

MEDICAL MANAGEMENT

Surgical stabilization of the scapula is usually required for a good cosmetic and functional result; however, closed reduction with placement of a Velpeau bandage (see p. 1166) has been reported to be successful in cats with acute luxation.

SURGICAL TREATMENT

Although the torn muscles' bellies occasionally can be identified and reattached to the scapula, this repair is usually insufficient to allow weight bearing. Generally, the scapula is held in its normal position by wire suture, which is placed around an adjacent rib and through holes drilled in the caudal scapular border.[18]

Preoperative Management

Thoracic radiographs and electrocardiographic analysis are warranted to detect any concurrent thoracic or cardiovascular trauma in acute injuries. Prophylactic antibiotic therapy is usually unnecessary.

Anesthesia

If the animal is healthy and has no significant concurrent injuries, a number of anesthetic regimens can be used safely (see Table 32.1); however, if evidence of concurrent thoracic or cardiovascular injury is noted, anesthesia should be induced and monitored with care (see Table 32.2). Equipment should be available for ventilating the animal if the chest is opened inadvertently (see Chapter 12).

Surgical Anatomy

See p. 1047 for a detailed discussion of the anatomy of the scapula. Important landmarks for surgical repair of a scapular luxation are the dorsocaudal scapular border and the fifth, sixth, or seventh ribs.

Positioning

The animal is positioned in lateral recumbency with the affected side up. The entire scapular region from midcervical region to midthorax should be prepared for aseptic surgery.

SURGICAL TECHNIQUE

After returning the scapula to its normal position, incise through the skin and subcutaneous tissue along the dorsal caudal scapular margin to expose the caudal scapular border. If possible, identify the torn edges of the muscles. Elevate a small portion of the teres major muscle from the caudal scapular border and identify the underlying rib. Elevate the periosteum from the rib surface, taking care to avoid penetrating the parietal pleura. Use a wire passer to position a piece of wire around the rib. Use 18- to 20-gauge wire to secure the scapula to the rib. Drill two holes in the caudal scapular border adjacent to the exposed rib, and pass the free ends of the wire through these holes in a medial to lateral direction. Tighten the wire by twisting the ends to secure the scapula to the rib. If the torn muscle edges have been identified, reattach them before closing the subcutaneous tissue and skin.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Necessary equipment includes 18- or 20-gauge wire, a wire passer, a periosteal elevator, Steinmann pins, and a hand chuck. Absorbable (polydioxanone or polyglyconate) or nonabsorbable (polypropylene or nylon) suture can be used to reattach the muscles to their scapular insertions.

POSTOPERATIVE CARE AND ASSESSMENT

The patient's respiratory rate and effort should be observed carefully postoperatively to determine if the animal is able to ventilate normally. Ventilatory difficulties may indicate a pneumothorax if the pleura was inadvertently penetrated during passage of the wire. Thoracic radiographs should be taken and thoracentesis performed in such patients. A spica splint (see p. 1166) should be applied to immobilize the limb for 3 weeks after surgery (the bandage may need to be changed every 5–7 days). Owners should be instructed to confine the animal until the bandage is removed; normal exercise may be resumed at 6 weeks.

PROGNOSIS

Chronic luxation will not resolve without surgical intervention. Possible complications of surgery include pneumothorax, fixation failure, iatrogenic infection, and eventual wire fatigue and breakage with migration; however, complications are rare.[18] Most animals bear weight normally on affected limbs after surgery.

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ELBOW JOINT

CANINE ELBOW DYSPLASIA

Elbow dysplasia likely represents the leading cause of forelimb lameness in the dog. Several diseases have been designated as components of inherited canine elbow dysplasia (Table 34.6). These diseases (e.g., joint incongruity, FCP, OCD, UAP, medial compartment disease [MCD]) may differ in terms of pathophysiology, but all are causes of elbow arthrosis. IOHC is not usually included in the category of elbow dysplasia, although it is a developmental disease that may cause similar clinical signs. Breed dispositions to the development of these diseases indicate that they are genetically influenced, and evidence now indicates that canine elbow dysplasia, like hip dysplasia, is a polygenic trait with both hereditary and environmental influences playing a part in its development (Box 34.14). Selective breeding in Sweden has reduced the prevalence of elbow arthrosis in Rottweilers and Bernese mountain dogs. Several groups have been organized to review elbow radiographs to determine the presence of elbow dysplasia (similarly to hip radiographs for hip dysplasia).

NOTE Because all forms of canine elbow dysplasia are hereditary, breeding dogs identified with this condition should be strongly discouraged.

TABLE 34.6 Categorizations and Surgical Options for Developmental Elbow Disease

Fragmented coronoid process	Arthroscopic fragment removal—S Open fragment removal—S Subtotal coronoidectomy—S Bicipital tendon release—A
Radial-ulnar incongruence	Proximal ulnar osteotomy—S Distal ulnar osteotomy—S Proximal radial osteotomy—A
Medial compartment disease	Proximal ulnar osteotomy—S Distal ulnar osteotomy—S Sliding humeral osteotomy—A Proximal radial osteotomy—A Total elbow replacement—A Subtotal coronoidectomy—S Bicipital tendon release—A
Osteochondritis dissecans	Arthroscopic flap removal—S Open flap removal—S Osteochondral autogenous transfer—A Synthetic cartilage plug—A Sliding humeral osteotomy—S, A
Ununited anconeal process	Fragment removal—S Proximal ulnar osteotomy—S Lag screw fixation—S Combined osteotomy/lag screw fixation—S
Incomplete ossification of the humeral condyle	Lag/compression screw fixation—S Lag screw and bone bridge—A

A, Advanced surgical technique; *S,* surgical description in text.

MEDIAL CORONOID DISEASE AND FRAGMENTED CORONOID PROCESS

DEFINITION

Medial coronoid disease occurs as cartilage degeneration, osteonecrosis of the coronoid, or fissures within the medial coronoid that may not have led to complete fragmentation. **FCP** is a common type of medial coronoid disease and usually occurs as a separation of a small portion of the medial coronoid process of the ulna. Synonyms include *ununited coronoid process, fractured coronoid process,* and *jump-down syndrome (acute onset of FCP after low-grade trauma)*. **MCD** refers to moderate to severe cartilage erosion limited to the medial aspect of the canine elbow joint, and is commonly a consequence of elbow dysplasia, including medial coronoid disease.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The etiology of medial coronoid disease is unknown.[19,20] However, several theories have been proposed: (1) medial coronoid disease/FCP may result from an osteochondrosis lesion in which disruption of endochondral ossification of the coronoid process leaves it vulnerable to cartilage degeneration, necrosis, and fissure formation; (2) developmental elbow incongruity may play a role in the pathophysiology of FCP: asynchronous growth of the radius and ulna (radioulnar incongruence [RUI]) may increase weight-bearing forces on the medial coronoid process (focally increased humeroulnar contact pressure, humeroulnar conflict), which precipitates fissuring and fragmentation; (3) trochlear notch dysplasia or malformation, resulting in joint incongruity, may contribute to mechanical overloading and fragmentation of the coronoid (humeroulnar incongruence, ulnar notch incongruence); (4) incongruence between the radial head and the radial notch of the ulna (radioulnar conflict) has also been proposed as a cause of fragmentation; (5) FCP may result from vascular abnormality of the medial coronoid that leads to osteonecrosis and fragmentation; (6) FCP may be related to

BOX 34.14 Important Considerations for Treatment of Elbow Dysplasia

- Elbow dysplasia is a set of diseases that include osteochondrosis, fragmented coronoid process, medial compartment disease, and ununited anconeal process.
- Incomplete ossification of the humeral condyle is another developmental disease of the canine elbow that may cause similar clinical signs or may lead to fracture of the humeral condyle.
- There is strong evidence of a hereditary component in the etiology of elbow dysplasia.
- Loss of elbow range of motion is evidence of degenerative joint disease; in immature large dogs, this finding usually indicates the presence of elbow dysplasia.
- Careful attention to positioning for radiographs is essential for the diagnosis of subtle lesions and for differential diagnosis.
- Both elbows should be radiographed.
- Surgical removal of bone and cartilage pieces usually results in improved limb function.

Watch for neutropenia; do periodic complete blood cell counts to check.

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tension from the insertion of the biceps brachii on the proximal ulna[21]; (7) FCP may occur secondary to low-grade trauma when a dog lands from a jump (jump-down syndrome).[22]

Medial coronoid disease may be characterized by complete fragmentation and separation (FCP), partial fissuring of the coronoid, or osteonecrosis. Fissures that develop in the coronoid may or may not progress to fragmentation. Fissures may still be a source of pain and lameness without fragmentation. When fragmentation occurs, it results in a separated piece of cartilage and trabecular bone that may be attached to the annular ligament with fibrous tissue. Separation occurs through calcified trabeculae that are then covered in part with a thin layer of fibrous tissue. The end result is coronoid fragmentation and development of DJD. The loose coronoid fragment may erode cartilage of the opposing medial humeral condyle ("kissing lesion"). The degree of cartilage damage associated with medial coronoid disease and FCP varies tremendously, and the specific cause of the cartilage wearing is uncertain. Some cartilage damage may be due to abrasion against the loose fragment; however, elbow joint pathology with severe cartilage loss cannot be explained by this mechanism alone. The greatest cartilage damage is almost always concentrated in the medial portion of the joint (medial coronoid and medial portion of the humeral condyle) and is commonly described as *MCD* (discussed later).

NOTE Fissuring of the subchondral bone of the medial coronoid cannot be diagnosed by radiography or computed tomography but may be a source of pain and lameness. If suspected, it should be treated by subtotal coronoidectomy.

DIAGNOSIS

Clinical Presentation

Signalment

Large dogs (Labrador retrievers, rottweilers, Bernese mountain dogs, Newfoundlands, golden retrievers, German shepherds, and chows) are usually affected. The disease process starts when the animal is immature, with clinical signs first becoming apparent at 5 to 7 months of age.[19] However, dogs may be brought in at any age for osteoarthritis secondary to FCP and elbow dysplasia.

History

Forelimb lameness that worsens after exercise may be acute or chronic. Owners frequently complain that the dog is stiff in the morning or after rest. There may be a coincidental history of trauma.

Physical Examination Findings

Lameness of one forelimb is usually evident. The gait may appear stiff or stilted if bilateral lameness is present because the animal may walk with shortened steps. Palpation of the dog standing may demonstrate symmetric or asymmetric muscle atrophy associated with chronic pain and decreased muscle use. Joint effusion and periarticular soft tissue swelling may be palpable and are most easily felt while the dog is standing. Palpation of the elbows should include an evaluation of range of motion. Pain on hyperextension of the elbow joint may be one of the earliest signs of FCP. Decreased ability to flex the elbow is indicative of more severe osteoarthritis. Crepitation during elbow flexion and extension may be felt if advanced osteoarthritis is present. Manipulation of the joint is often painful. It is important that the shoulder not be inadvertently flexed and extended during elbow manipulation to prevent mistaking shoulder pain for elbow pain.

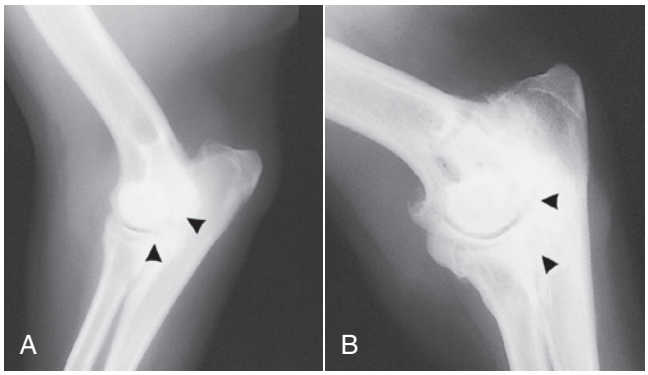


FIG. 34.36 (A) Preoperative radiographs of a dog with fragmented coronoid process (FCP) and degenerative joint disease. There is subchondral bone sclerosis caudal and distal to the trochlear notch of the ulna *(arrowheads)*. (B) Radiographs of the same dog 8 years after surgical removal of the FCP. Note the progression of degenerative changes *(arrowheads)*.

Diagnostic Imaging

In many cases, a diagnosis of early medial coronoid disease and FCP may be difficult with radiographs.[19,23] Radiographic views should include a standard craniocaudal view (see Fig. 34.35A), a standard lateral of the elbow (see Fig. 34.35B), and flexed lateral (joint positioned at 45 degrees' flexion) to expose the anconeal process (see Fig. 34.35C). Radiographs of both elbows should be made because bilateral disease is common. The earliest radiographic sign is often sclerosis of the distal aspect of the trochlear notch (Fig. 34.36A). This is visible as a loss of the fine trabecular pattern and an increase in opacity. Blunting of the medial coronoid process is also an early radiographic finding. These radiographic findings may be subtle, and evaluation by a radiologist or orthopedic surgeon may be helpful. Visible fragments are rarely seen. Later, a diagnosis of medial coronoid disease and FCP is made on the basis of radiographic signs of DJD; osteophytes associated with the coronoid process and tip of the anconeus may be visible (see Fig. 34.36B). Joint incongruence may be evaluated on radiographs, but evidence suggests a high rate of false positives and negatives for incongruence less than 3 mm.

NOTE Radiographs in dogs with elbow dysplasia may have very subtle changes (occult elbow dysplasia) even in the presence of significant elbow arthrosis.

CT is helpful for identifying FCP and can be used to evaluate other aspects of the articular surfaces for defects and incongruity (Fig. 34.37). CT scans are more accurate for identifying FCP than survey radiographs.[23] CT cannot, however, diagnose fissuring and microcracks of the coronoid. This diagnosis can only be made histologically. CT has the advantage over arthroscopy of being able to diagnose incomplete fragmentation of the medial

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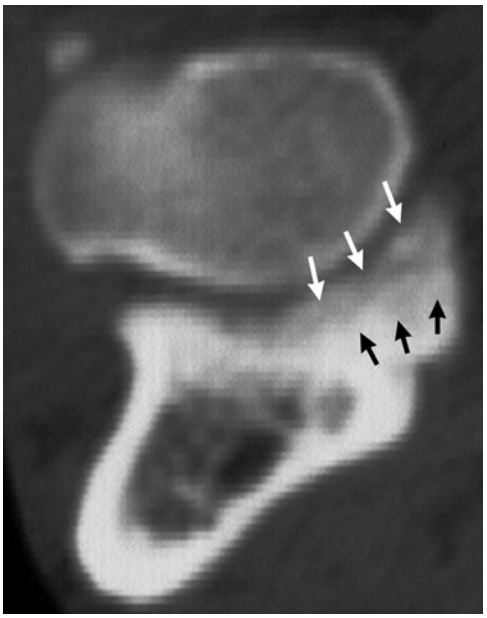


FIG. 34.37 Computed tomography scan showing in situ osteomalacia of the medial coronoid process *(arrows)*.

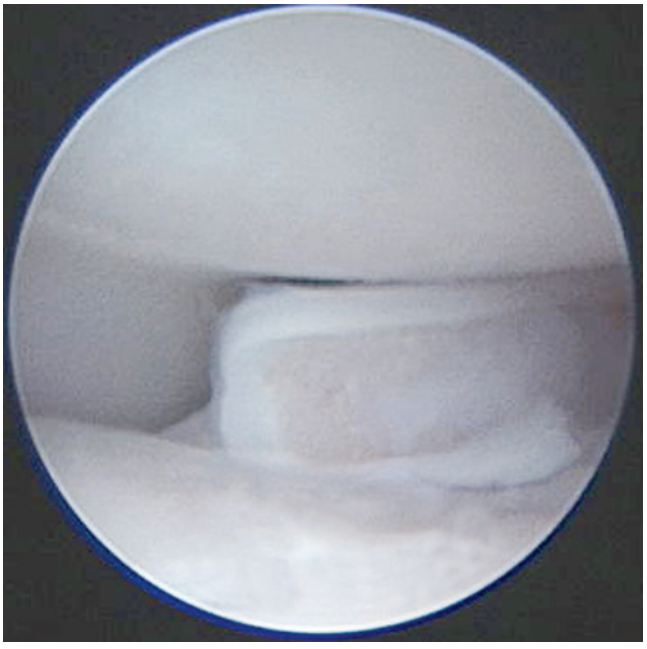


FIG. 34.38 Arthroscopic view of the elbow demonstrating an osteochondral fragment of the medial coronoid process.

coronoid that does not reach the cartilage surface. CT may aid in evaluating the elbow joint for incongruence.

Bone scintigraphy appears to be sensitive for the detection of chondromalacia, fissures, and fragmentation of the medial coronoid process.

Arthroscopy is the most valuable tool for diagnosing medial coronoid disease and FCP (Fig. 34.38). Unlike other modalities, arthroscopy enables direct visualization and assessment of the cartilage surface. Fragmentation is clearly visible, except with incomplete fragments that do not reach the cartilage surface. Osteonecrosis may be diagnosed by firm palpation of the medial coronoid process. Synovial proliferation is a nonspecific finding that is indicative of elbow joint pathology.

Laboratory Findings

Results of hematologic and serum biochemical analyses are normal in most affected animals. Results of arthrocentesis may include decreased synovial fluid viscosity, increased fluid volume, and increased numbers of mononuclear phagocytic cells (up to 6000–9000 WBC/μL).

DIFFERENTIAL DIAGNOSIS

Other conditions affecting the elbow of immature dogs (OCD [see p. 1186], UAP [see p. 1188], combined UAP and FCP, IOHC [see p. 1192], and MCD [see p. 1183]) may produce similar clinical signs and must be differentiated from FCP. Other diseases that affect the forelimb of young growing dogs (OCD of the shoulder, panosteitis) should also be differentiated from FCP. These conditions usually can be differentiated radiographically.

MEDICAL MANAGEMENT

Dogs with FCP may be treated conservatively; however, owners should be cautioned that delay in surgical removal of a loose fragment may allow continued cartilage damage by the fragment. Exploration of the elbow joint with arthroscopy should be encouraged, particularly for young patients with mild to moderate osteoarthritis. Patients that benefit most from surgical treatment are those that have only fragmentation, but no significant cartilage damage. In these patients, fragment removal can completely resolve clinical signs and eliminate the possibility of cartilage damage caused by abrasion from the fragment. If surgery is to be performed, it should be done as early in the disease process as possible to minimize progression of osteoarthritis.

Alternatively, because cartilage damage cannot be attributed to the fragment alone in all cases, surgery may not necessarily affect the progression of osteoarthritis. The value of surgical or arthroscopic treatment of dogs with advanced osteoarthritis is questionable, as the pain coming from the osteoarthritis is likely greater than that caused by the presence of the fragment.

When conservative management is elected, it should include focusing on the five principles of medical management of osteoarthritis (see p. 1137), particularly weight management, nutritional supplementation, exercise moderation, and antiinflammatory medication. Many dogs with radiographic signs of osteoarthritis are asymptomatic and remain so for several years. Although owners should be apprised of the problem, they should also be reassured that the dog may function as a pet with minimal treatment (Box 34.15).

SURGICAL TREATMENT

Affected animals are candidates for surgery when they have intermittent or chronic lameness. Surgical treatment for medial coronoid disease may include fragment removal, debridement of necrotic bone, and subtotal coronoidectomy. Animals with advanced osteoarthritis (MCD) and persistent lameness not amenable to medical therapy may benefit from joint exploration and removal of a loose fragment. However, surgical treatment does not halt the progression of osteoarthritis, and continued medical therapy or additional surgical management (see p. 1184) may be required in these patients.

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BOX 34.15 Important Information for Clients With Dogs That Have Elbow Dysplasia

- There is strong evidence for a hereditary component in the etiology of fragmented coronoid process and osteochondrosis.
- Bilateral elbow radiographs should be made because of the frequency of bilateral disease; however, unless the animal shows clinical signs, surgery of the opposite limb is not performed.
- Surgical removal of the bone and cartilage pieces may improve function of the limb if the dog is treated before extensive secondary degenerative changes occur in the joint.
- After surgery, the dog is confined and exercise is limited to short walks on a leash for 2 to 4 weeks.
- Surgical treatment does not appear to alter the progression of degenerative joint disease (osteoarthritis); if the elbow is incongruent, changes may be moderate or severe, and the dog may require medical therapy after surgery.
- Dogs with osteoarthritis of the elbow usually function well as pets, but they may not be appropriate as working or competitive sporting dogs.

The basis of treatment of FCP is fragment removal by either arthroscopy or open arthrotomy. Arthroscopic treatment of FCP has numerous advantages over open surgery. Arthroscopy provides superior visualization and magnification of the joint, is less invasive, has lower postoperative morbidity, and provides greater opportunity for topical treatment of osteoarthritic lesions.

If arthroscopy or arthrotomy fails to demonstrate a fragment when FCP is suspected, subtotal coronoidectomy may be performed based on suspicion of fissuring and incomplete fragmentation of the coronoid.[19,24] Subtotal coronoidectomy may also be performed in addition to fragment removal if there is concern for fissuring or future fragmentation.

Release of the biceps insertion on the ulna has been described as a treatment for FCP.[25] The biceps exerts significant proximal forces on the radius and ulna, and release of the ulnar insertion may decrease transarticular forces between the distal medial humeral condyle and the medial coronoid process. Advocates suggest that bicipital ulnar release may aid in healing of microcracks in the coronoid and decrease the risk of MCD. Both arthroscopic and open bicep ulnar insertion releases have been described.

Additional surgical treatments for dogs with medial coronoid disease depend on the specific lesions diagnosed, including incongruence, MCD, and OCD.

Preoperative Management

Most affected animals are young and require minimal preoperative workup (see Chapter 4).

Anesthesia

These dogs usually are young and healthy; thus a variety of anesthetic regimens may be used (see Table 32.1).

Arthroscopic Treatment of Fragmented Coronoid Process

Indications

AP Elbow arthroscopy is most commonly used for diagnosis and treatment of elbow dysplasia. Further indications for arthroscopic surgery of the elbow include diagnosis of IOHC, biopsy of inflamed synovium, and biopsy of abnormal intraarticular tissue.

Instrumentation

A 30-degree foreoblique 1.9-mm arthroscope is commonly used in the elbow joint. It is easily inserted into the joint space without introducing significant damage to the articular cartilage. The surgeon must be aware of the diameter of the arthroscope sheath (cannula) because it too must enter the joint. The arthroscope cannula can be fitted with a blunt obturator or a sharp obturator (trocar point). Although either can be used to enter the joint, the blunt obturator is preferred because it is safer.

An assortment of hand instruments is necessary for elbow arthroscopy (see Chapter 14). Instruments to assist in the inspection of intraarticular structures (probes), grasping forceps for removal of free bodies (FCP, OCD), biopsy forceps, and instruments for surface arthroplasty are recommended. Instruments commonly used for surface arthroplasty are handheld burrs, curettes, or a motorized shaver. Instruments commonly used for microfracture are micropicks and a mallet. Instruments used for subtotal coronoidectomy include a power burr or osteotome and mallet. Some instruments can be inserted into the joint through an open instrument port, instrument cannulas, or a combination of the two. If one chooses to work through an instrument cannula, different size cannulas and switching sticks are necessary.

Portal Sites and Technique

Clip and prepare the medial aspect of the elbow joint. Beginning arthroscopists should clip and prepare the area as if an open arthrotomy were to be performed, so that the arthroscopy procedure can be aborted and an open arthrotomy performed if necessary. Advanced arthroscopic techniques, including use of caudal and cranial portals, also require circumferential preparation of the limb. One of two methods for limb preparation may be used, depending on the limb maneuverability desired. *If you wish to have maximum maneuverability of the limb during surgery, use a hanging-limb preparation.* As you become more experienced and adept at elbow arthroscopy, less maneuverability is needed, and you may wish to perform a medial limb preparation for each limb. *Use two or three portal sites for elbow arthroscopy, depending on the purpose of arthroscopic intervention. If visual exploration of the elbow joint is all that is required, use an egress portal and an arthroscope portal. If tissue biopsy or treatment of a pathologic joint condition is undertaken, use an additional instrument portal. Position the dog in dorsal recumbency because both elbows may need intervention. As one elbow is operated on, lean the body toward that limb. Position the limb such that the elbow joint can be centered over the edge of the table (padded) or over an elbow brace (Fig. 34.39).* This allows the assistant to externally rotate the paw and apply a valgus stress to the limb. These maneuvers help open the medial joint space, allowing increased mobility of the arthroscope and instruments. The joint may also be positioned with self-retaining retractors. This allows the surgeon to move freely around the limb and prevents problems of movement by the assistant.

As with other joints, accurate placement is essential for the egress, arthroscope, and instrument portals. *Infuse and distend the joint first, then establish the arthroscope portal (Fig. 34.40), the egress, and finally the instrument portal. Infuse the joint by inserting an 18- or 20-gauge needle perpendicular to the joint line approximately 1 cm distal and 0.5 cm caudal to the medial epicondyle. Once the needle is in place, aspirate joint fluid to ensure proper positioning of the needle in the joint. Instill lactated Ringer's solution through the needle to distend the joint. Use a No. 11*

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FIG. 34.39 Positioning for elbow arthroscopy.

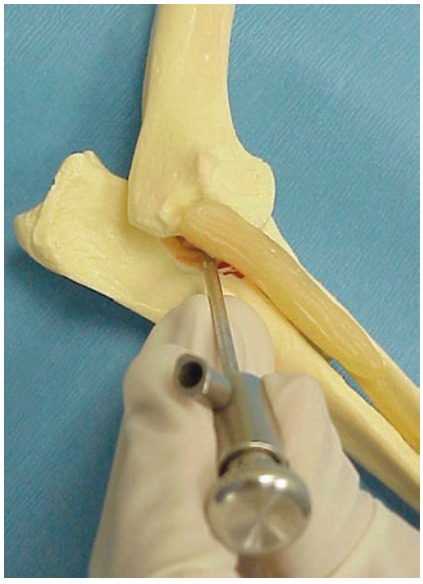


FIG. 34.40 Specimen showing the location of the arthroscope portal site. The position is caudal to the medial epicondyle at the level of the joint line.

Bard-Parker blade to make a small entry wound through the skin and superficial soft tissue at the site of the previous needle insertion. It is not advisable to enter the joint with the scalpel blade because extravasation of fluid from the joint cavity is more likely if the blade enters the joint. *Remove the needle and insert the arthroscope cannula with the attached blunt obturator. Once in the joint, remove the obturator from the cannula.* Fluid will flow freely from the cannula, confirming correct placement. *Attach the fluid ingress line to the cannula and insert the arthroscope.*

If treatment of a pathologic joint condition (FCP, OCD) or biopsy of intraarticular structures is required, establish an instrument portal approximately 1 to 2 cm cranial to the arthroscope portal. This distance is only an approximation; it will vary with the size of the dog. *Learn to triangulate the instrument portal relative to the arthroscope portal for best results. To triangulate the position for the instrument portal, position the arthroscope to view the medial coronoid process. Insert a 20-gauge needle at the estimated position for the instrument portal. Insert the needle perpendicular to the skin surface, and maintain this orientation as it passes through the soft tissue. As the needle enters the joint, observe it on the monitor.* The most common reason for not seeing the needle as it enters the joint is crossing the needle over the scope. This is caused either by inserting the needle too close to the arthroscope portal or by inserting the needle such that the point is angled toward the arthroscope. *Once the position of the instrument portal has been confirmed by the triangulation needle, make a 3-mm incision through the skin and superficial soft tissue. If working through an open instrument portal, lengthen the incision (4–6 mm) and carry it into the joint. If working through an instrument cannula, do not lengthen the incision and do not carry it into the joint. Instead, insert a cannula with appropriate obturator into the joint. Use switching sticks to place increasing sized cannulas and instruments. Remove in situ or free fragments through the open or cannulated instrument portal (Fig. 34.41).*

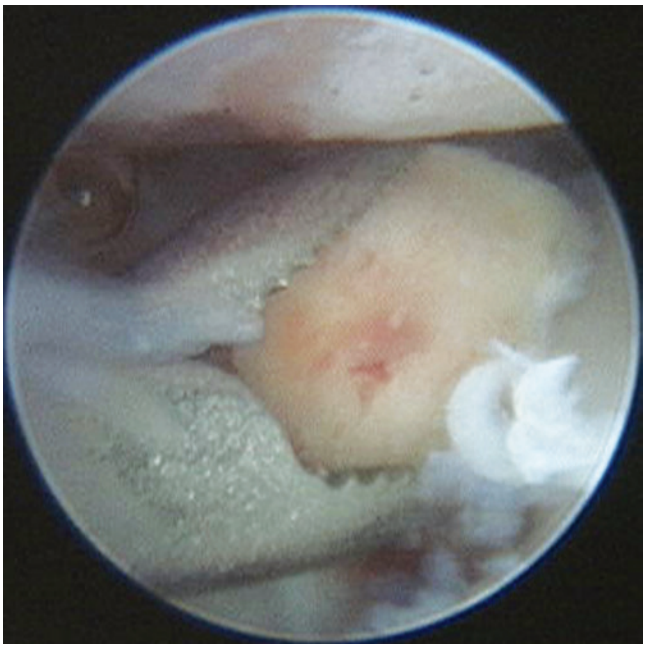


FIG. 34.41 Arthroscopic view showing a fragmented medial coronoid process.

Subtotal Coronoidectomy

Arthroscopic Subtotal Coronoidectomy by Power Burr

Instrument the joint with an arthroscope and establish the instrument portal as described previously. Insert a motorized shaver with a burr attachment into the instrument portal. With suction applied to the motorized shaver, begin grinding and removing the medial coronoid process beginning at the cranial tip, being careful not to damage the radial head or humerus. Remove the coronoid process until a line connecting the medial border and the most caudal extent of the radial incisure is reached (Fig. 34.42). Angle the plane of removal distocranially. Lavage the joint thoroughly to remove any remaining bone fragments.

Arthroscopic-Assisted Subtotal Coronoidectomy by Osteotome

Instrument the joint with an arthroscope and establish the instrument portal as described previously. Insert an osteotome and position it at the medial border of the medial coronoid. Aim the osteotome at the most caudal extent of the radial incisure. Angle the osteotome distocranially. Use a mallet to tap the osteotome until the fragment is free. Remove the fragment with grasping forceps.

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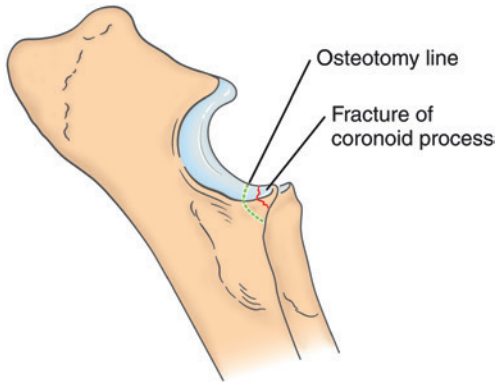


FIG. 34.42 Osteotomy line for subtotal coronoidectomy.

Postoperative Care and Assessment

Bandaging is not indicated following elbow arthroscopy. Rest and minimal weight bearing are recommended for up to 8 weeks to allow optimal cartilage healing. Physical rehabilitation may be of significant benefit postoperatively (Table 34.7). Gradual return to full activity is recommended.

OPEN SURGICAL TREATMENT OF FRAGMENTED CORONOID PROCESS

Affected animals are candidates for surgery when they are persistently lame and have mild degenerative changes. Occasionally, animals with advanced DJD and persistent lameness not amenable

TABLE 34.7 Sample Rehabilitation Protocol for Patient With Elbow Dysplasia Postarthroscopy

Treatments/ Modalities	Day 1–3	Day 4–14	2–4 WK	4–8 WK	8–12+ WK
Pain medications	As directed	As directed	PRN	PRN	PRN
Cryotherapy	10–15 min 3 times daily to affected elbow	To affected elbow after exercise for 15 min	To affected elbow after exercise for 15 min	To affected elbow after exercise for 15 min	To affected elbow after exercise for 15 min
Heat therapy		Apply heat to the thoracic muscles before exercise	PRN	PRN	PRN
Massage	Gentle effleurage to shoulder musculature twice daily	Continue twice daily	Twice daily	PRN	PRN
PROM	Gentle PROM to thoracic limb with attention to elbow flexion and extension, 8–10 reps twice daily	Gentle PROM to thoracic limb with attention to elbow flexion and extension, 10–15 reps twice daily	Gentle PROM to thoracic limb with attention to elbow flexion and extension, 10–15 reps twice daily	As before only if range of motion is not normal	PRN
Laser therapy	Daily	Daily until day 7 then 3 times weekly	Twice weekly	Twice weekly	Only if needed
Walks	5-min leash walk 2–3 times daily for toileting only with a supported harness	5-min leash walk 2–3 times daily with a supported harness Work up to 15-min walks over a few weeks	10-min leash walk 2–3 times daily with a supported harness Work up to 15-min walks over a few weeks	15-min leash walk 2–3 times daily with a supported harness	15- to 20-min walks 2–3 times daily with a harness
NMES	10 min twice daily	10 min twice daily	PRN	PRN	
Balancing			5 min twice daily on a foam pad, transitioning to balance board	5–10 min twice daily on balance board or disc	5–10 min on balance board, disc, or egg ball
Cavalettis				5–10 reps twice daily, low rails	10–20 reps twice daily
Shaking paw/ digging				8–10 reps twice daily or 3–5 minutes of digging	10–15 reps twice daily or 5–8 minutes of digging
Hills				Zigzag low, slow hills 5 min up and down	Increase to 10 min twice daily
Underwater treadmill			5–10 min daily	10–15 min once to twice daily, introducing weights or floatation device to affected limb	20–30 min twice weekly, moderate and variable speed until released from rehab
Swimming				10–15 min 3 times weekly with breaks and floatation device	15–20 min 3 times weekly with flotation device

NMES, Neuromuscular electrical stimulation; *PRN,* as needed; *PROM,* passive range of motion.

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to medical therapy may benefit from joint exploration and removal of a loose fragment. However, surgical treatment does not halt the progression of DJD, and continued medical therapy may be required in these patients. There does not appear to be any correlation between the severity of lameness before treatment, severity of radiographic signs, and type of lesion found at surgery.

Preoperative Management

Most affected animals are young and require minimal preoperative workup.

Anesthesia

These dogs usually are young and generally healthy, and a variety of anesthetic regimens may be used (see Table 32.1).

Surgical Anatomy

Landmarks for the surgical incision include the medial epicondyle, epicondyloid crest, and proximal radius. The median nerve and brachial artery and vein course cranially to the medial epicondyle. The ulnar nerve courses caudally to the medial epicondyle over the anconeus muscle. The median nerve and brachial artery and vein are visible in the surgical field and must be identified and protected.

Positioning

The limb is prepared from shoulder to carpus. The dog is positioned in dorsal recumbency with the affected limb suspended for draping. The limb is then released to allow access to the medial surface of the elbow.

OPEN SURGICAL TECHNIQUE

Exposure of the medial coronoid process can be obtained with one of several techniques. Tenotomy of the pronator teres muscle and incising the medial collateral ligament offer good exposure but at the expense of the supporting structures. A muscle-splitting technique has been proposed that preserves the joint's supporting tendons and ligaments but limits exposure. Osteotomy of the medial epicondyle provides the best exposure but requires implantation of a lag screw or wire to replace the epicondyle and is associated with more postoperative complications than other procedures.

Exposure of the Medial Coronoid Process via Transection of the Pronator Teres Muscle

Make an incision on the medial surface of the joint starting at the medial epicondylar crest and extending distally over the medial epicondyle to the proximal radius (Fig. 34.43A). Protect the median nerve and brachial artery (Fig. 34.43B). Transect the pronator teres tendon (Fig. 34.43C). Make an incision to the humeral condyle through the joint capsule and collateral ligament to expose the joint. Identify the coronoid process and any lesions on the medial humeral condyle. Remove the coronoid fragment (Fig. 34.43D). When removing the coronoid fragment, the line of cleavage of the fragment may not be readily visible and may require prying with an elevator to allow identification. In chronic cases, osteophytes may obscure the line of cleavage and must be removed with a rongeur to allow identification of fragments.

Open Subtotal Coronoidectomy

Insert an osteotome and position it at the medial border of the medial coronoid. Aim the osteotome at the most caudal extent of the radial incisure. Angle the osteotome distocranially. Use a mallet to tap the osteotome until the fragment is free. Remove the fragment with grasping forceps. Close the wound by suturing the joint capsule with simple interrupted absorbable sutures. Place several nonabsorbable sutures in the collateral ligament. Reattach the pronator teres tendon with a Bunnell's or locking-loop nonabsorbable suture (see p. 1283). Suture the fascia, subcutaneous tissue, and skin in separate layers.

Exposure of the Medial Coronoid Process via Muscle Splitting

Incise the skin and subcutaneous tissue as described previously. Identify the demarcation between the flexor carpi radialis and superficial digital muscles, and separate and retract them. Expose the joint capsule and incise it parallel to the muscle-splitting incision to expose the coronoid process (Fig. 34.44). Remove the fragmented coronoid as described previously and/or perform a subtotal coronoidectomy as described previously. Suture the joint capsule with interrupted absorbable sutures. Suture the fascia, subcutaneous tissue, and skin in separate layers.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

An Ochsner forceps is useful for grasping the fragment, and an osteotome and mallet are required for a subtotal coronoidectomy.

POSTOPERATIVE CARE AND ASSESSMENT

The limb may be bandaged after surgery for up to 1 week to provide soft tissue support, and the animal should be confined for 4 weeks. Physical rehabilitation is beneficial in regaining strength and range of motion and decreasing overall lameness. For a sample physical rehabilitation exercise protocol for this condition, refer to Table 34.7.

COMPLICATIONS

Possible surgical complications of fragment removal include iatrogenic infection and nerve damage. The risk of infection is likely decreased with arthroscopy. Possible surgical complications of coronoidectomy include fracture of the ulna.

PROGNOSIS

Prognosis for return to full function depends on the severity of cartilage disease. In cases with fragmentation only in which the remainder of the joint cartilage appears healthy, the prognosis after fragment removal is good to excellent. Minimal osteoarthritis may progress, but in most cases it does not become severe. In cases with significant cartilage damage (MCD), the prognosis is guarded because of progressive osteoarthritis. The use of physical rehabilitation may dramatically improve joint and limb function in cases of elbow dysplasia (see Box 34.15). Recurrent lameness can be treated with antiinflammatory drugs and analgesics (see Table 34.3).

RADIAL-ULNAR INCONGRUENCE (ELBOW JOINT INCONGRUITY)

DEFINITION

RUI in elbow dysplasia refers to elevation of the coronoid above the level of the radial head.

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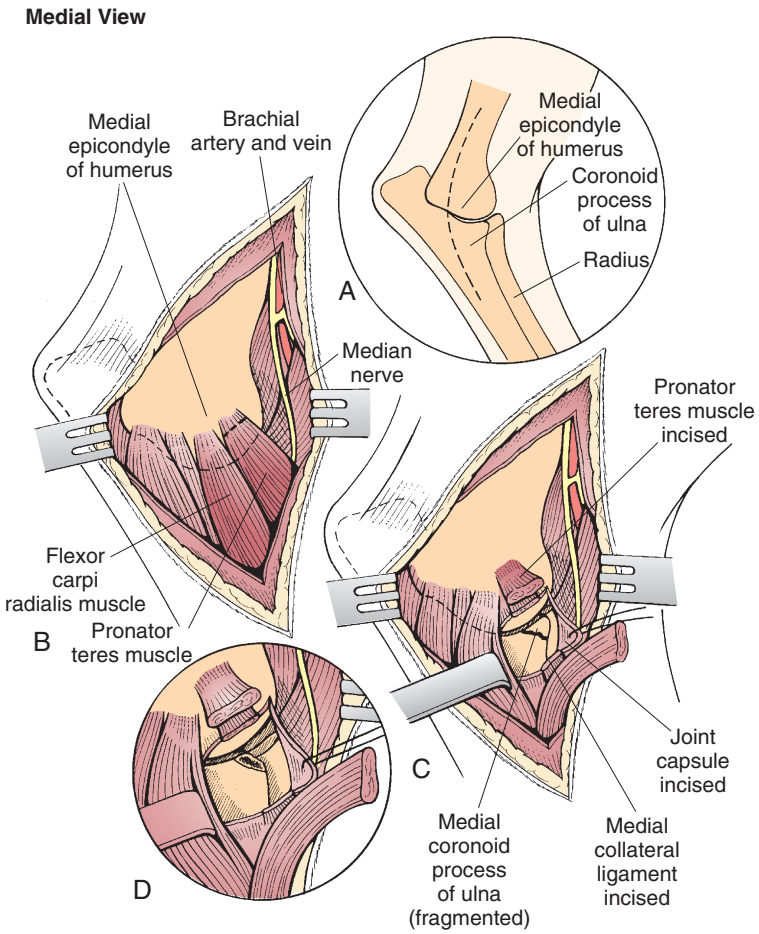


FIG. 34.43 (A) To expose the medial aspect of the elbow joint by means of transection of the pronator teres muscle, make a skin incision on the medial surface of the joint, starting at the medial epicondylar crest and extending distally over the medial epicondyle. (B) Gently retract the median nerve and brachial artery and vein cranially. (C) Transect the pronator teres tendon and make an incision parallel to the humeral condyle through the joint capsule and collateral ligament to expose the fragmented coronoid process. (D) Remove the fragment.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

RUI has been suggested to be a cause of medial coronoid disease (FCP) and MCD. Asynchronous growth between the radius and ulna may lead to increased forces across the medial compartment of the elbow joint, leading to fragmentation and cartilage damage of the medial coronoid process. Elbow subluxation due to premature closure of the distal ulnar or radial physes is discussed later in this chapter (see p. 1197).

DIAGNOSIS

Clinical Presentation

Signalment

Large dogs (Labrador retrievers, Rottweilers, Bernese mountain dogs, Newfoundlands, and golden retrievers) are usually affected. The disease process starts when the animal is immature, with clinical signs first becoming apparent at 5 to 7 months of age. However, dogs may be brought in at any age for osteoarthritis secondary to elbow dysplasia.

History

Forelimb lameness that worsens after exercise may be acute or chronic. Owners frequently complain that the dog is stiff in the morning or after rest. There may be a coincidental history of trauma.

Physical Examination Findings

Lameness of one forelimb usually is evident. The gait may appear stiff or stilted if bilateral lameness is present because the animal may walk with shortened steps. Palpation of the dog standing may demonstrate symmetric or asymmetric muscle atrophy associated with chronic pain and decreased muscle use. Joint effusion and periarticular soft tissue swelling may be palpable and are most easily felt while the dog is standing. Palpation of the elbows should include an evaluation of range of motion. Pain on hyperextension of the elbow joint may be one of the earliest signs of elbow dysplasia. Decreased ability to flex the elbow is indicative of more severe osteoarthritis. Crepitation during elbow flexion and extension may be felt if advanced osteoarthritis is present. Manipulation of the joint often causes signs of pain. It is important that the shoulder not be inadvertently

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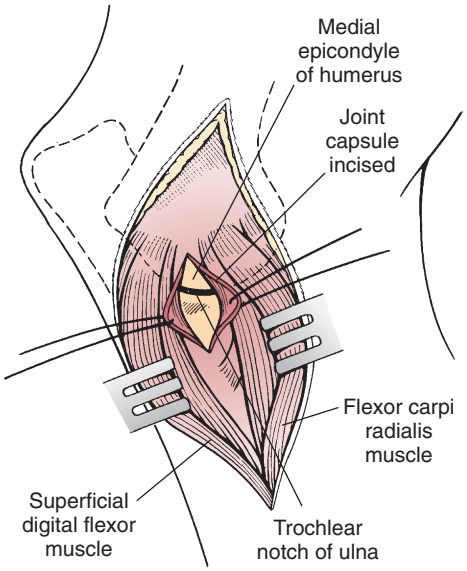


FIG. 34.44 To expose the medial aspect of the elbow joint using a muscle-splitting approach, identify the demarcation between the flexor carpi radialis and the superficial digital muscles and separate and retract them. Expose the joint capsule and incise it parallel to the muscle-splitting incision to expose the coronoid process.

flexed and extended during elbow manipulation to prevent mistaking shoulder pain for elbow pain.

Diagnostic Imaging

Incongruence of the joint may be investigated with either plain film radiography or CT. In severe cases (>4 mm), RUI is recognized on a lateral or craniocaudal view of the elbow.[26,27] The medial coronoid process will appear proximal to the radial head. Routine radiography using standard medial-lateral positioning has been reported to be an inaccurate means of diagnosis of mild incongruity; however, incongruence may be more accurately evaluated on flexed lateral views. CT is a more accurate means of evaluating incongruence. Arthroscopy has been reported to be an accurate means of diagnosing RUI.[26,27]

Laboratory Findings

Results of hematologic and serum biochemical analyses are normal in most affected animals. Results of arthrocentesis may include decreased synovial fluid viscosity, increased fluid volume, and increased numbers of mononuclear phagocytic cells (up to 6000–9000 WBC/μL).

DIFFERENTIAL DIAGNOSIS

Other conditions affecting the elbow of immature dogs (OCD, UAP, IOHC) may produce similar clinical signs and must be differentiated. Other diseases that affect the forelimb of young growing dogs (OCD of the shoulder, panosteitis) should also be differentiated from RUI. These conditions can usually be differentiated radiographically.

MEDICAL MANAGEMENT

Dogs with RUI may be treated conservatively; however, owners should be cautioned that a delay in treatment may allow continued cartilage damage. Exploration of the elbow joint with arthroscopy should be encouraged. If surgery is to be performed, it should be done as early in the disease process as possible to minimize progression of osteoarthritis.

The value of surgical or arthroscopic treatment of dogs with advanced osteoarthritis is questionable as the pain coming from the osteoarthritis is likely greater than that caused by the presence of the fragment.

When conservative management is elected, it should include focusing on the principles of medical management of osteoarthritis (see p. 1137), particularly weight management, nutritional supplementation, exercise moderation, and antiinflammatory medication. Many dogs with radiographic signs of osteoarthritis are asymptomatic and remain so for several years. Although owners should be apprised of the problem, they should also be reassured that the dog may function as a pet with minimal treatment (see Box 34.15).

SURGICAL TREATMENT

Surgical treatment of RUI is aimed at restoring the normal elbow joint congruence. Accomplish this by allowing the proximal ulna segment to move into a more appropriate position, dictated by the action of soft tissues, articular interface interaction and loading forces (dynamic proximal ulnar osteotomy),[28] or shortening the ulna or lengthening the radius (see p. 1199).

Preoperative Management

Most affected animals are young and require minimal preoperative workup.

Anesthesia

These dogs usually are young and generally healthy, and a variety of anesthetic regimens may be used (see Table 32.1).

Surgical Anatomy

The landmark for an ulnar osteotomy is the caudal border of the ulna, which is easily palpated.

Positioning

For a caudal approach to the ulna, the animal is positioned in lateral recumbency with the affected limb uppermost. The limb should be suspended for draping to facilitate limb manipulation during surgery.

Dynamic Proximal Ulnar Osteotomy

See Fig. 33.50 for the approach for ulnar osteotomy. *In the proximal third of the ulna, perform an oblique osteotomy; the angle of the osteotomy should be caudoproximal to craniodistal (Fig. 34.45A) and craniolateral to caudomedial (Fig. 34.45B). Reappose the periosteum with 3-0 absorbable suture in a simple continuous pattern. Close the deep fascia in a simple continuous pattern with absorbable suture. Apply a bandage for 3 to 5 days to aid in patient comfort.*

Proximal Ulnar Segmental Ostectomy

In the proximal third of the ulna, perform a segmental osteotomy removing approximately 0.5 cm of bone. The angle of the osteotomy should be caudoproximal to craniodistal (Fig. 34.45A) and craniolateral to caudomedial (Fig. 34.45B). Reappose the periosteum with 3-0 absorbable suture in a simple continuous pattern. Close the deep fascia in a simple continuous pattern with absorbable suture. Apply a bandage for 3 to 5 days to aid in patient comfort.

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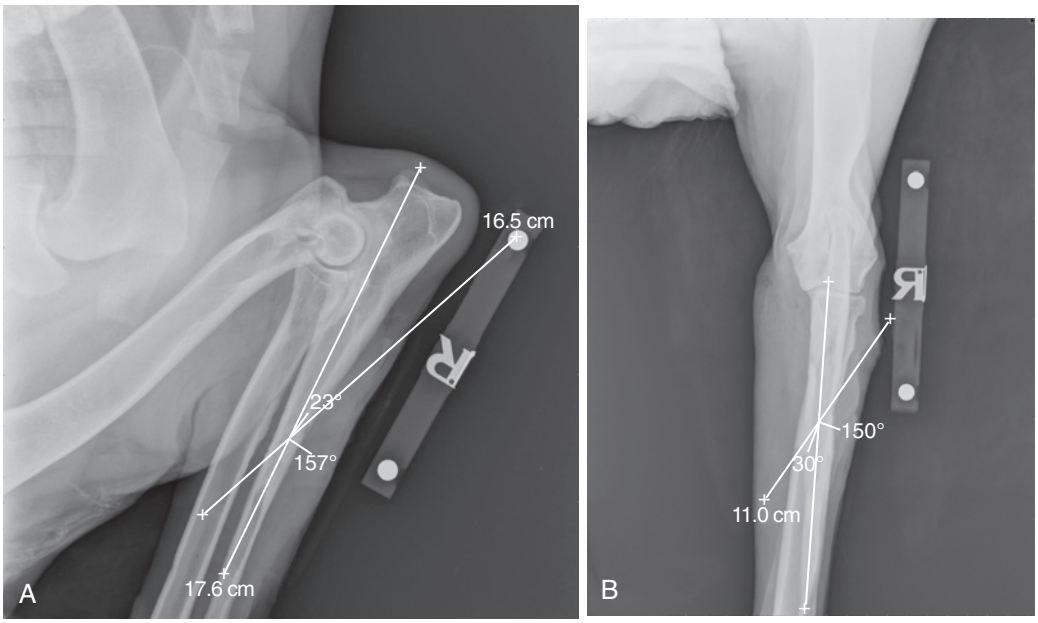


FIG. 34.45 (A) Mediolateral and (B) craniocaudal radiographs demonstrating the osteotomy lines for proximal ulnar osteotomy. (Courtesy Dr. Noel Fitzpatrick.)

Distal Segmental Ulnar Ostectomy

Make a 3-cm longitudinal incision over the lateral aspect of the distal third of the ulna, ending at the distal ulnar physis. Dissect between the tendon of the lateral digital extensor muscle and the tendon of the ulnaris lateralis muscle to expose the diaphysis of the ulna. Incise and elevate the periosteum, and isolate the ulna with Hohmann retractors. Remove a 5-mm-length section of the ulna with an osteotome, rongeur, or bone saw. Reappose the periosteum with 3-0 absorbable suture in a simple continuous pattern. Close the deep fascia in a simple continuous pattern with absorbable suture. Apply a soft bandage for 3 to 5 days to aid in patient comfort. Various locations for the distal ulnar osteotomy have been used. Elevation of the interosseous ligament off the ulna aids in distal migration of the ulna; however, it may lead to significant hemorrhage.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

A Gigli wire, osteotome and mallet, or oscillating saw is needed for ulnar osteotomy.

POSTOPERATIVE CARE AND ASSESSMENT

Immediate postoperative radiographs should be made. The limb may be bandaged after surgery for up to 1 week to provide soft tissue support, and the animal should be confined for 4 to 8 weeks. Repeat radiographs are recommended 6 weeks after surgery. Physical rehabilitation may be beneficial in management of osteoarthritis and in improving the range of motion (see Table 34.7). After surgery, cryotherapy, passive range of motion, and controlled leash walks are the primary methods of physical rehabilitation.

COMPLICATIONS

Complications of ulnar osteotomy include nonunion and infection.

PROGNOSIS

Prognosis for return to full function depends on the severity of cartilage disease. In animals with significant cartilage damage (MCD), the prognosis is guarded because of progressive osteoarthritis. The use of physical rehabilitation may dramatically improve joint and limb function in cases of elbow dysplasia (see Box 34.15). Recurrent lameness can be treated with antiinflammatory drugs and analgesics (see Table 34.3).

MEDIAL COMPARTMENT DISEASE

DEFINITION

MCD refers to moderate to severe cartilage erosion limited to the medial aspect of the canine elbow joint. The regions commonly affected include the medial portion of the coronoid process, medial distal aspect of the humeral condyle, and in some cases, the medial-most portion of the radial head.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The etiology of MCD is unknown; however, the most likely cause is mechanical overload or incongruity of the elbow joint.[29] Cartilage loss from abrasion by a fragmented coronoid is an unlikely cause based on the location and the severity of the cartilage damage and because MCD may be identified with or without concurrent FCP. The severity of cartilage loss is graded using a modified Outerbridge scale (Box 34.16). MCD is often the end-stage form of elbow dysplasia, where the inside part of the joint collapses, with eventual grinding of bone on bone. Interestingly and importantly, the larger lateral part of the elbow joint appears normal in the vast majority of patients.

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BOX 34.16 Modified Outerbridge Scale for Arthroscopic Grading of Osteoarthritis

I—chondromalacia
II—partial-thickness fibrillation and fissuring
III—full-thickness fissuring
IV—full-thickness cartilage loss
V—eburnated cartilage

DIAGNOSIS

Clinical Presentation

Signalment

Large-breed dogs are usually affected although MCD may be diagnosed in any size dog. The age of onset of the disease process is unknown, as MCD has been diagnosed in dogs as young as 6 months of age but is frequently not diagnosed until several years of age.[29]

History

Forelimb lameness that worsens after exercise may be acute or chronic. Owners frequently complain that the dog is stiff in the morning or after rest.

Physical Examination Findings

Lameness of one or both forelimbs is usually evident. The gait may appear stiff or stilted if bilateral lameness is present because the animal may walk with shortened steps. Palpation of the dog standing may demonstrate symmetric or asymmetric muscle atrophy associated with chronic pain and decreased muscle use. Joint effusion and periarticular soft tissue swelling or lateral osteophytosis may be palpable and are most easily felt while the dog is standing. Palpation of the elbows should include an evaluation of range of motion. Decreased ability to flex the elbow is indicative of severe osteoarthritis. Manipulation of the joint is often painful.

Diagnostic Imaging

In many cases, diagnosis of MCD is suspected on the basis of radiographic signs of DJD. Radiographic findings are variable, and severe cartilage damage may be present with minimal radiographic changes. Osteophytes associated with the coronoid process and tip of the anconeus may be visible. In some cases, severe diffuse osteophytosis may be observed. The severity of radiographic changes does not correlate with the severity of cartilage damage.

Arthroscopy

Arthroscopy is the most definitive tool for diagnosing MCD (Fig. 34.46). Unlike other modalities, arthroscopy enables direct visualization and assessment of the cartilage surface.[29] Arthroscopy is superior to open surgery because it allows less invasive examination of a greater portion of the joint and the severity and extent of cartilage damage is more easily recognized and documented.

Laboratory Findings

Results of hematologic and serum biochemical analyses are normal in most affected animals. Results of arthrocentesis may include decreased synovial fluid viscosity, increased fluid volume, and increased numbers of mononuclear phagocytic cells (up to 6000–9000 WBC/μL).

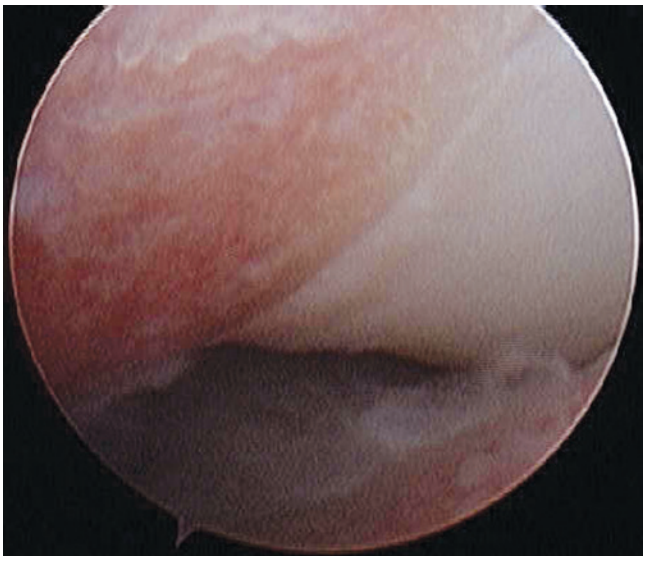


FIG. 34.46 Arthroscopic view of the elbow demonstrating medial compartment disease with full-thickness (grade IV) cartilage damage.

DIFFERENTIAL DIAGNOSIS

Other conditions affecting the elbow of dogs (OCD, UAP, combined UAP and FCP, and IOHC) may produce similar clinical signs and must be differentiated from MCD. When MCD is suspected in older dogs, it must be differentiated from neoplasia of the bone or surrounding soft tissues.

MEDICAL MANAGEMENT

Medical management should primarily focus on the five principles of management of osteoarthritis (see p. 1137), particularly weight management, nutritional supplementation, exercise moderation, and antiinflammatory medication. Additional nonsurgical treatments include stem cell therapy (see Chapter 31) and hyaluronan injections (see p. 1139).

SURGICAL TREATMENT

Many patients with MCD also have FCP; however, the benefit of fragment removal alone is questionable, as the pain associated with the cartilage damage will remain. Arthroscopic techniques for the treatment of arthritic lesions include microfracture and abrasion arthroplasty. These techniques are intended to create channels for revascularization of the arthritic lesion from the bone marrow. This may aid in recruiting stem cells to the area to aid in healing. However, these techniques are likely of limited benefit as the mechanical forces that led to the cartilage erosion also prevent healing of the cartilage lesion.

Surgical treatments of MCD are intended to decrease pain and joint inflammation by decreasing stimulation of nerve endings located in the subchondral bone. This may be accomplished by removal of the coronoid (subtotal coronoidectomy), decreasing transarticular loads (sliding humeral osteotomy [SHO], radial osteotomy, ulnar osteotomy), or replacing the bearing surface (total elbow replacement).

Subtotal coronoidectomy may decrease the pain associated with MCD by removing the damaged region of the coronoid.

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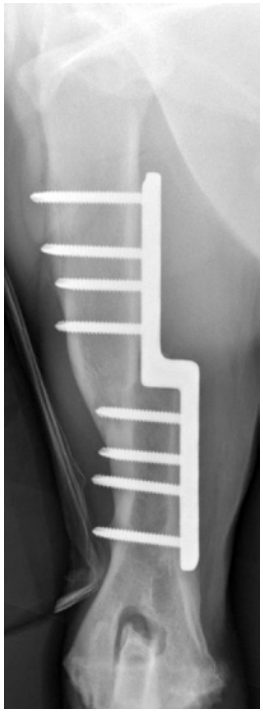


FIG. 34.47 Postoperative radiograph of sliding humeral osteotomy.

Diseased cartilage remains on the opposing medial humeral region. Although the efficacy of subtotal coronoidectomy has been demonstrated for FCP, it has not been evaluated for MCD.[24]

The proximal and distal ulnar osteotomies, sliding proximal radial osteotomy, abducting proximal ulnar osteotomy, and SHO are intended to decrease transarticular loads across the medial compartment of the elbow joint. The SHO and proximal ulnar osteotomy have been demonstrated to decrease medial compartment loads in laboratory specimens, and the radial osteotomy has not been evaluated.[28,30,31] The SHO (Fig. 34.47) has been reported to decrease lameness in dogs with arthroscopically diagnosed MCD.[30,31] SHO is a highly advanced procedure and should be performed only by experienced surgeons trained in this technique. The efficacy of ulnar and radial osteotomies for management of MCD has not been reported.

Release of the biceps insertion on the ulna has been described as a treatment for FCP.[25] The biceps exerts significant proximal forces on the radius and ulna and release of the ulnar insertion may decrease transarticular forces between the distal medial humeral condyle and the medial coronoid process. Advocates suggest that bicipital ulnar release may aid in healing of microcracks in the coronoid and decrease the risk of MCD. Both arthroscopic and open bicep ulnar insertion releases have been described.[25]

Since the early 1990s, several total elbow joint replacements have been described for use in dogs. Most have been abandoned due to high complication rates, including joint subluxation, fracture, loosening, and infection. Unicompartmental elbow arthroplasty is a partial replacement procedure using humeral and ulnar implants, which are press-fit into bone to resurface pathologic weight-bearing areas of the medial compartment of the elbow (Fig. 34.48).[32] Elbow replacement is a highly advanced procedure and should be performed only by experienced surgeons trained in this technique.

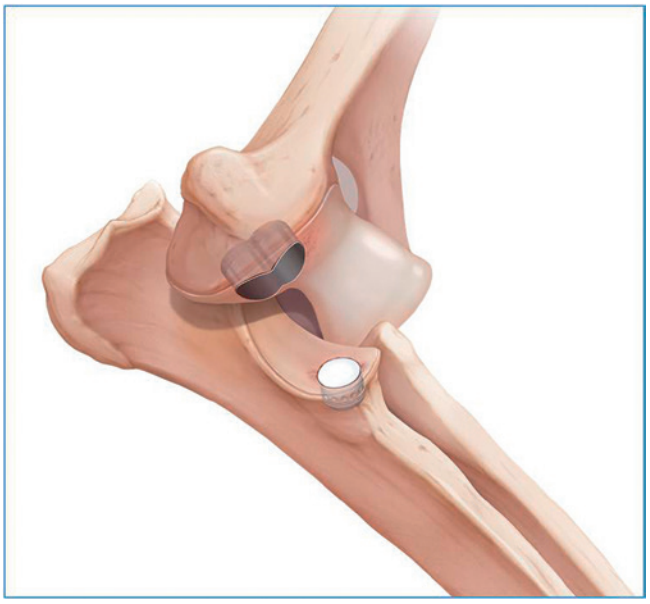


FIG. 34.48 Unicompartmental elbow arthroplasty using press-fit implants into pathologic weight-bearing areas of the medial compartment of the elbow.

Preoperative Management

Preoperative workup depends on the patient's age and the presence of other diseases (see Chapter 4).

Anesthesia

The anesthetic regimen used depends on the patient's age and the presence of other diseases (see Tables 32.1 and 32.2).

Arthroscopic Treatment of Medial Compartment Disease

Perform standard medial elbow arthroscopy (see p. 1177).

Abrasion Arthroplasty

Insert a power burr or hand burr, and evenly abrade any exposed subchondral bone, removing 1 mm of bone at a time. Check for bleeding by shutting off the inflow fluids. If bleeding is inadequate, remove more subchondral bone. If bleeding is adequate, flush the joint and close routinely.

Microfracture

Insert an angled micropick (Fig. 34.49). Position the tip of the pick on the subchondral bone and force the tip into the bone. Have an assistant hit the external end of the micropick lightly with a mallet to form the microfractures. Repeat this technique across the surface of the exposed subchondral bone every few millimeters. Check for bleeding by shutting off the inflow fluids. If bleeding is inadequate, perform more microfractures. If bleeding is adequate, flush the joint and close routinely.

Subtotal Coronoidectomy

See p. 1178 for the description of the subtotal coronoidectomy.

Proximal Ulnar Osteotomy

See p. 1183 for the description of the proximal ulnar osteotomy.

Distal Ulnar Ostectomy

See p. 1082 for the description of the distal ulnar osteotomy.

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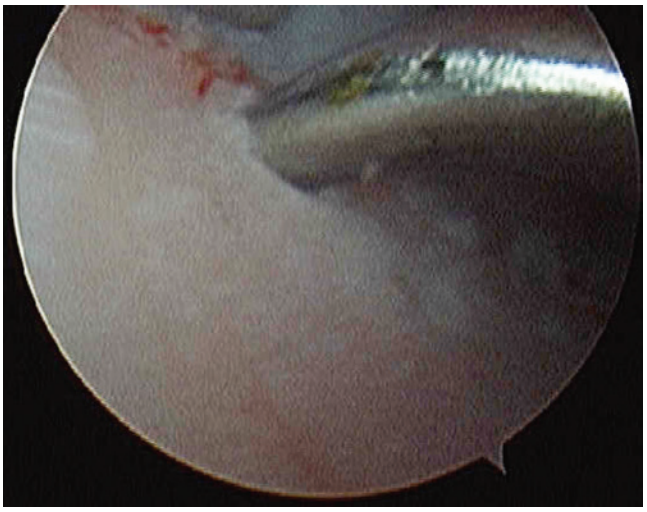


FIG. 34.49 Arthroscopic microfracture of the medial coronoid process of the elbow.

POSTOPERATIVE CARE AND ASSESSMENT

Bandaging or postoperative cryotherapy can aid in the minimization of swelling and pain postoperatively. Rest and minimal weight bearing are recommended for up to 8 weeks to allow optimal cartilage healing. Physical rehabilitation may be of significant benefit postoperatively. A sample physical rehabilitation exercise regimen can be found in Table 34.7. Gradual return to full activity is recommended.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

A power burr is required for abrasion arthroplasty, and a micropick and mallet are required for microfracture. A bone saw, osteotome, or Gigli wire are required for an ulnar osteotomy. Either an osteotome or a power shaver with a burr attachment is necessary for a subtotal coronoidectomy.

COMPLICATIONS

Possible surgical complications of an ulnar osteotomy are nonunion or infection.

PROGNOSIS

Prognosis for return to full function is guarded due to the high frequency of severe cartilage damage and associated joint arthrosis. SHO has been reported to decrease lameness in dogs with MCD by 26 weeks postoperatively. Reports of the efficacy of ulnar and radial osteotomies in the management of MCD are anecdotal. Canine unicompartmental elbow arthroplasty has been reported to be effective (91.3% full or acceptable outcomes) in clinical patients with symptomatic MCD by 24 weeks postoperatively.

OSTEOCHONDRITIS DISSECANS OF THE DISTAL HUMERUS

DEFINITIONS

Osteochondrosis is a disturbance in endochondral ossification that leads to retention of cartilage; it occurs commonly in the shoulder, elbow, stifle, and hock of immature large dogs. OCD occurs when fissure formation in the abnormal cartilage leads to development of a flap of cartilage.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Osteochondrosis begins with a failure of endochondral ossification in either the physis or the articular epiphyseal complex, which is responsible for formation of metaphyseal bone. Osteochondrosis has been implicated in the pathophysiology of OCD, FCP, and UAP of the elbow. The origin is unknown, but genetic factors, rapid growth, overnutrition, trauma, ischemia, and hormonal factors are possible causes.[33] Failure of endochondral ossification leads to cartilage thickening. Because developing cartilage is nourished initially by synovial fluid and later by vascularization through subchondral bone, increased cartilage thickness may result in malnourished, necrotic chondrocytes. Chondrocyte loss deep in the cartilage layer leads to formation of a cleft at the junction of calcified and noncalcified tissues. Subsequent trauma caused by normal activity can lead to development of vertical fissures in the cartilage; eventually the fissures communicate with the joint, and a cartilage flap is formed. This communication allows cartilage degradation products to reach the synovial fluid, where they may cause joint inflammation. OCD does not cause apparent clinical signs until a loose flap of cartilage develops. This loose body of cartilage does not ossify. DJD (osteoarthritis) of the elbow is the final outcome.

With OCD of the distal humerus, a flap of cartilage covering a defect in the surface of the trochlear ridge of the medial humeral condyle usually is seen. Opposing joint cartilage of the coronoid process may be eroded. Histologic examination of the lesion confirms that the flap consists of cartilage, that subchondral bone trabeculae in the defect are thickened, and that bone marrow fibrosis is present.

DIAGNOSIS

Clinical Presentation

Signalment

Affected dogs are usually large (Labrador retrievers, golden retrievers). The usual age of onset of lameness is 5 to 7 months.

History

Forelimb lameness that worsens after exercise may be acute or chronic. Owners frequently complain that the dog is stiff in the morning or after rest. There may be a coincidental history of trauma.

Physical Examination Findings

Lameness of one forelimb is usually evident. The gait may be stiff or stilted if forelimb steps are shortened because of bilateral lameness. Pain may be elicited on elbow extension and on lateral rotation of the forearm. Palpation of the elbows should include an evaluation of range of motion. A decrease in the animal's ability to flex the elbow is indicative of secondary osteoarthritis. Crepitation during elbow flexion and extension, joint effusion, and periarticular swelling may be noted if osteoarthritis is present. It is important that the shoulder not be inadvertently flexed and extended during elbow manipulation to avoid confusing shoulder pain and elbow pain.

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FIG. 34.50 Radiograph of an elbow with osteochondritis dissecans of the medial portion of the humeral condyle. There is a radiolucent defect in the humeral condyle *(arrow)*.

Diagnostic Imaging

Radiographic views should include a standard lateral view of the elbow, a flexed lateral view to expose the anconeal process, and a craniocaudal view (Fig. 34.50). Radiographs of both elbows should be made because bilateral disease is common. Definitive radiographic diagnosis of OCD is made when a radiolucent concavity is observed on the distal aspect of the medial humeral condyle (see Fig. 34.50). This lesion is most often identified on the craniocaudal view. Radiographic signs of secondary osteoarthritis are similar to those seen with FCP (see Fig. 34.36).

Arthroscopy

Arthroscopy permits confirmation of the diagnosis of elbow OCD (Fig. 34.51). Unlike other modalities, arthroscopy enables direct visualization and assessment of the cartilage surface, measurement of the size of the OCD, assessment of the opposing cartilage, and more accurate prognostication.

Laboratory Findings

Results of hematologic and serum biochemical analyses are normal in most affected animals. Results of arthrocentesis may include decreased synovial fluid viscosity, increased fluid volume, and increased numbers of mononuclear phagocytic cells (up to 6000–9000 WBCs/μL).

DIFFERENTIAL DIAGNOSIS

OCD must be differentiated from FCP (and kissing lesions from FCP), UAP, combined UAP and FCP, and other diseases of young, growing dogs that affect the forelimb (e.g., OCD of the shoulder and panosteitis) and may produce similar clinical signs. These diseases are usually differentiated radiographically. Rarely, OCD and FCP may be identified in the same elbow.

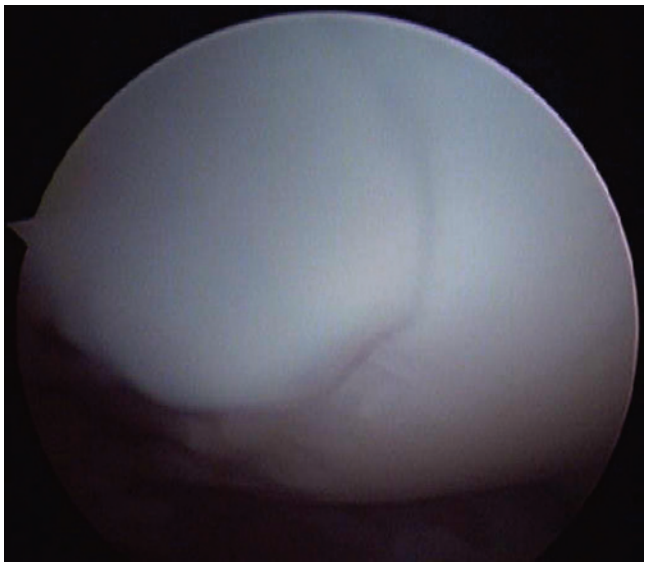


FIG. 34.51 Arthroscopic view of osteochondritis dissecans of the elbow.

MEDICAL MANAGEMENT

Dogs that have radiographic abnormalities but no symptoms do not require treatment. This is most commonly seen in cases of osteochondrosis that have not progressed to OCD. Dogs with true OCD may be treated conservatively; however, owners should be cautioned that delay in surgical removal of a loose cartilage flap may encourage additional damage to the opposing cartilage. Patients that benefit the most from surgical treatment are those that have only OCD but no significant cartilage damage to the opposing surfaces (kissing lesions). If surgery is going to be performed, it should be done as early in the disease process as possible to minimize the progression of osteoarthritis.

When conservative management is elected, it should include focusing on the principles of medical management of osteoarthritis, particularly weight management, nutritional supplementation, exercise moderation, and antiinflammatory medication. After lameness has subsided or resolved, exercise should gradually be increased to strengthen the surrounding musculature.

SURGICAL TREATMENT

Surgical removal of the cartilage flap is recommended in young animals when the disease is diagnosed before the onset of osteoarthritis and can also be helpful in treating animals with chronic moderate to severe lameness. Arthroscopic treatment of OCD has numerous advantages over open surgery. Arthroscopy provides superior visualization and magnification of the joint, is less invasive, and provides greater opportunity for topical treatment of osteoarthritic lesions. Advanced treatments of OCD include SHO, osteochondral autogenous transfer, and synthetic osteochondral resurfacing.[30,31,34] SHO decreases loads on the medial compartment of the elbow joint and subsequently the subchondral bone at the OCD site. SHO may decrease pain and aid in fibrocartilaginous healing of the lesion following flap removal. Osteochondral autogenous transfer and synthetic osteochondral resurfacing are used to replace the hyaline cartilage that is lost in OCD and provide a high-quality bearing surface

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to eliminate pain and decrease the progression of osteoarthritis.[34] SHO, osteochondral autogenous transfer, and synthetic osteochondral resurfacing are highly advanced procedures and should be performed only by experienced surgeons trained in these techniques.

Preoperative Management

Most affected animals are young and require minimal preoperative workup (see Chapter 4).

Anesthesia

These animals are usually young and healthy; thus a variety of anesthetic regimens can be used (see Table 32.1).

Surgical Anatomy

For pertinent anatomy of the elbow joint, see p. 1181.

Positioning for Open Surgery

The limb is prepared from shoulder to elbow. The dog is positioned in dorsal recumbency with the affected limb suspended for draping. The limb is then released to allow access to the medial surface of the elbow.

SURGICAL TECHNIQUE

Arthroscopy

Arthroscopy of the elbow joint is discussed on p. 1177. Moving the arthroscope portal slightly caudally and proximally from the standard positioning permits improved visualization of the OCD lesion. Inspect the entire elbow joint, including the medial coronoid process area. *Remove in situ or free cartilage fragments (Fig. 34.52). Using a curette, make the "shoulders" of the lesion right angles to the joint surface. Perform surface arthroplasty through the open or cannulated instrument portal using a hand burr.*

Open Surgery

Exposure of the medial portion of the elbow joint can be obtained with several techniques. Tenotomy of the pronator teres muscle and incision of the medial collateral ligament offer good exposure at the expense of the supporting structures (see p. 1181). A muscle-splitting technique preserves the joint's supporting tendons and ligaments but limits exposure (see pp. 1181–1182). *Regardless of the technique used for exposure, examine the medial condyle of the humerus and remove the flap of cartilage. Curette only to remove fragments of cartilage from the edges of the lesion. Repeatedly flush the joint to remove any small fragments. Inspect the coronoid process and remove it if fragmented.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

An Ochsner forceps is useful for grasping the flap, and a bone curette is needed to curette the defect.

POSTOPERATIVE CARE AND ASSESSMENT

The limb may be bandaged after surgery for up to 1 week to provide soft tissue support, and the animal should be confined for 4 weeks. Gradual return to normal activity is recommended.

Bandaging is not indicated following elbow arthroscopy. Rest and minimal weight bearing are recommended for up to 8 weeks to allow optimum cartilage healing. Physical rehabilitation may be of significant benefit postoperatively. The primary focus is to retain range of motion and musculature immediately after the surgical intervention (see Chapter 11). After surgery, cryotherapy, passive range of motion, and controlled leash walks are the primary methods of physical rehabilitation.



FIG. 34.52 Arthroscopic removal of osteochondritis dissecans of the elbow.

PROGNOSIS

Dogs treated conservatively for OCD of the distal humerus usually have continued intermittent lameness and progressive osteoarthritis. Dogs treated surgically for OCD of the distal humerus usually have improved limb function; however, lameness may be evident after exercise. Osteoarthritis is usually present and requires medical treatment (see the previous discussion), but most dogs are functional pets and are only intermittently lame. Iatrogenic infection is a possible surgical complication.

NOTE Be sure to warn owners that many affected dogs will have progressive osteoarthritis despite surgery.

UNUNITED ANCONEAL PROCESS

DEFINITION

UAP or *elbow dysplasia* is a disease of large growing dogs in which the anconeal process does not form a bony union with the proximal ulnar metaphysis.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The anconeal process arises as a secondary center of ossification in the elbow at 11 to 12 weeks of age. It does not fuse to the ulna until 4 to 5 months of age; therefore the diagnosis of UAP cannot be made before that age. Several theories have been

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proposed for the etiology of UAP. One is that the UAP may be a manifestation of osteochondrosis in which failure of timely endochondral ossification of the attachment of the anconeal process to the ulna leads to thickened cartilage, necrosis, and fissures. The stress of weight bearing on this abnormal cartilage then causes failure of ulnar unification. Other possible etiologic factors include heredity, hormonal influences, nutrition, and acute or chronic trauma.

Developmental joint incongruity has been theorized to cause increased pressure or trauma to the anconeal process. Trochlear notch malformation and disparate radius and ulnar lengths (i.e., a longer radius in relationship to the ulna) have both been incriminated in the development of UAP. Pressure of the trochlear notch against the humeral condyles may cause a shearing stress on the anconeal process, resulting in a fracture, or simply may provide enough stress to prevent fusion. The UAP may be free within the joint, but in most cases, it is attached to the ulna with fibrous tissue. Ununited or fractured anconeal processes are unstable and result in secondary DJD. Pathologic changes include joint effusion, chondromalacia, periarticular fibrosis, and osteophyte formation.

DIAGNOSIS

Clinical Presentation

Signalment

Large- to giant-breed male dogs are most commonly affected. German shepherds are overrepresented. The usual age of presentation is 6 to 12 months. Some older animals may be seen for lameness caused by secondary osteoarthritis.

History

The history is generally intermittent lameness of one or both forelimbs that worsens after exercise. Owners frequently complain that the dog is stiff in the morning or after rest.

Physical Examination Findings

Lameness of one forelimb is usually evident. The gait may be stiff or stilted because elbow range of motion is decreased. This causes the elbow to circumduct laterally during the swing phase of the gait. The dog may sit and stand with the paw externally rotated. If osteoarthritis is present, there may be crepitation during elbow flexion and extension. Joint effusion and periarticular soft tissue swelling may be palpable. The animal may experience pain during joint manipulation, particularly during palpation over the anconeal process. It is important that the shoulder not be inadvertently flexed and extended during elbow manipulation to avoid mistaking shoulder pain for elbow pain.

Diagnostic Imaging

Radiographic views include a standard lateral view of the elbow, a flexed lateral view to expose the anconeal process, and a craniocaudal view of the elbow made while the elbow is flexed 30 degrees and slightly rotated medially (see Fig. 34.33). Radiographs of both elbows should be made because bilateral disease occurs in 20% to 35% of cases. UAP is visible as a lucent, indistinct line separating the anconeal process from the ulna. It is best visualized on the flexed lateral view (Fig. 34.53). Concurrent FCP can occur. Signs of osteoarthritis may include subchondral bone sclerosis, articular and periarticular osteophyte formation, joint space narrowing, joint effusion, and an increase in periarticular soft tissue.



FIG. 34.53 A flexed lateral radiograph of an elbow with an ununited anconeal process. There is a lucent, irregular line between the anconeal process and the olecranon *(arrow),* with subchondral bone sclerosis of the olecranon.

NOTE Be sure the animal is old enough for physeal closure before diagnosing ununited anconeal process.

Laboratory Findings

The results of hematologic and serum biochemical analyses are normal in most affected animals. The results of arthrocentesis may include decreased synovial fluid viscosity, increased fluid volume, and increased numbers of mononuclear phagocytic cells (up to 6000–9000 WBC/μL).

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include OCD, FCP, combined UAP and FCP, and other diseases of young, growing dogs that affect the forelimb (i.e., OCD of the shoulder and panosteitis). These diseases can usually be differentiated radiographically.

MEDICAL MANAGEMENT

Medical therapy is generally used to treat older dogs with established osteoarthritis. Surgical removal of the anconeal process after extensive development of osteoarthritis does not stop the progression of osteoarthritis. Conservative management should include implementation of the five principles of medical management of osteoarthritis: weight management, nutritional supplementation, exercise moderation, physical rehabilitation, and antiinflammatory medication. After lameness has subsided or resolved, exercise should be gradually increased to strengthen surrounding musculature.

SURGICAL TREATMENT

Surgical removal of the anconeal process has been the standard treatment if UAP is diagnosed before the onset of extensive osteoarthritis, despite concerns regarding loss of normal joint congruity. Surgical reduction and lag screw fixation of the anconeal process have been reported sporadically since the 1980s, but the outcome is not uniformly successful and implant failure can occur. Ulnar osteotomy has been recommended to relieve pressure on the anconeal process, allowing spontaneous healing of the fragment to the ulna in immature dogs. Ulnar osteotomy

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may be used concurrently with lag screw fixation of the anconeal process. The combined technique has been reported to result in a higher rate of radiographic union than ulnar osteotomy alone.[35] These procedures should only be recommended in dogs under 1 year of age with minimal degenerative changes in the joint and at the site of nonunion.

Preoperative Management

Most affected animals are young and require minimal preoperative workup.

Anesthesia

These animals are usually young, and a variety of anesthetic regimens can be used (see Table 32.1).

Surgical Anatomy

Landmarks for the skin incision when performing a lateral approach for removal of a UAP include the lateral humeral epicondyle, epicondylar crest, and proximal radius. The deep branch of the radial nerve runs under the proximal cranial border of the extensor carpi radialis muscle. The superficial branch of the radial nerve lies between the lateral head of the triceps and the brachialis muscle and can be visualized in the proximal portion of the incision. Landmarks for the combined medial approach are the medial epicondyle, epicondylar crest, and proximal radius. The median nerve and brachial artery and vein course cranial to the medial epicondyle and run under the pronator teres muscle. These structures must be identified and protected. The ulnar nerve courses caudal to the medial epicondyle, over the anconeus muscle, and is visualized during the medial approach to the anconeal process. The landmark for ulnar osteotomy is the caudal border of the ulna, which is easily palpated.

Positioning

For a lateral approach to the elbow, the animal is positioned in lateral recumbency, whereas for a medial approach and for ulnar osteotomy, the animal is positioned in dorsal recumbency. The limb should be suspended for draping to facilitate limb manipulation during surgery.

SURGICAL TECHNIQUE

Removal of the Anconeal Process: Lateral Approach

Make an incision in the skin, starting proximal to the lateral humeral epicondyle. Curve the incision to follow the epicondylar crest, and end it over the proximal portion of the radius. Incise the subcutaneous tissue to expose the cranial border of the lateral head of the triceps muscle. Retract the cranial border of the triceps muscle caudally to expose the anconeus muscle. Incise the anconeus muscle and joint capsule along the epicondylar crest, and retract them caudally to expose the anconeal process (Fig. 34.54). Grasp the process with towel forceps or an Ochsner forceps and remove it. Fibrous tissue attachments to the ulna may have to be incised before the anconeal process can be mobilized. *If necessary, smooth the remaining bone surface with a file. Flush the joint. Suture the joint capsule and anconeus muscle to the extensor carpi radialis muscle. Suture the subcutaneous tissue and skin in separate layers.*

Removal of the Anconeal Process and Medial Coronoid Process: Combined Medial Approach

Expose the medial coronoid process using one of the techniques described on p. 1182. After closing the craniomedial compartment of the elbow, approach the caudomedial compartment by retracting the ulnar nerve cranially and exposing the cranial border of the medial head of the triceps muscle. Retract the triceps muscle caudally to expose the caudal border of the medial epicondylar crest and origin of the anconeus muscle. Incise the anconeus muscle and joint capsule parallel to the medial epicondylar crest, leaving 2 to 4 mm of tissue attached to the crest for closure (Fig. 34.55). Remove the anconeal process. Suture the anconeus muscle and joint capsule in one layer to close the joint.

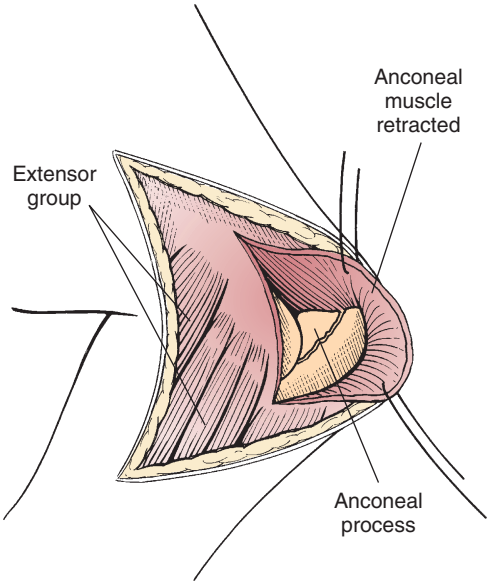


FIG. 34.54 For a lateral approach to the caudal compartment on the elbow, make a skin incision starting proximal to the lateral humeral epicondyle and curving to follow the epicondylar crest. End the incision over the proximal portion of the radius. Incise the anconeus muscle and joint capsule along the epicondylar crest and retract them caudally to expose the anconeal process.

Ulnar Osteotomy

Make a skin incision along the caudal border of the ulna, beginning medial to the tuber olecranon and ending at the middiaphysis of the ulna. Incise the subcutaneous tissue and fascia along the same line. Incise the attachments of the flexor carpi ulnaris and ulnaris lateralis muscles along the medial and lateral borders of the ulna, and elevate the muscles to expose the joint capsule. Make an osteotomy of the ulna 20 to 30 mm distal to the trochlear notch with an oscillating saw. The angle of the osteotomy should be caudoproximal to craniodistal and craniolateral to caudomedial (see Fig. 34.45). A gap should occur at the osteotomy site. *If necessary, elevate the interosseous ligament to free the proximal ulna so that it can move into position. Remove a 2- to 4-mm section of the ulna if there is concern that the osteotomy will heal before the dog has stopped growing. Suture the flexor carpi ulnaris fascia to the ulnaris lateralis fascia over the caudal border of the ulna. Suture the subcutaneous tissue and skin separately.*

Lag Screw Fixation of the Ununited Anconeal Process

AP This technique may be performed open or closed with arthroscopic guidance. Screw placement is critical, as there is only limited bone stock available in the ununited portion and inadvertent placement of the screw in the joint will lead to hemarthrosis and crippling osteoarthritis. Results with

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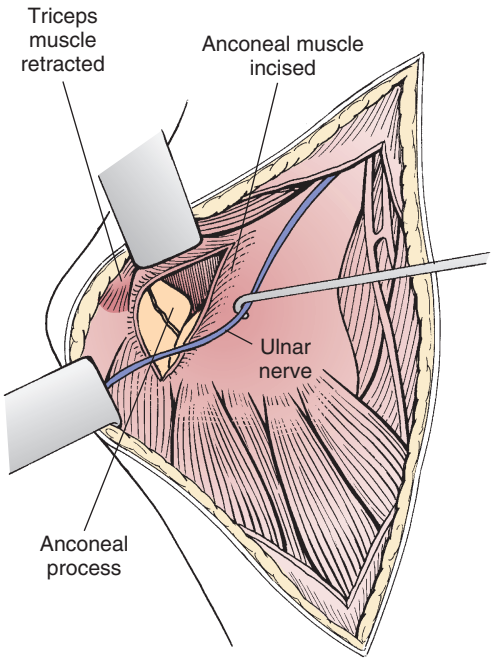


FIG. 34.55 For a medial approach to the caudal compartment of the elbow, retract the ulnar nerve cranially and the triceps muscle caudally to expose the caudal border of the medial epicondylar crest and the origin of the anconeus muscle. Incise the anconeus muscle and joint capsule parallel to the medial epicondylar crest to expose the anconeal process.



FIG. 34.56 Postoperative radiograph of lag screw fixation of an ununited anconeal process.

this technique are best when combined with a proximal ulnar ostectomy. *To perform the procedure open, begin with a medial or lateral approach to the olecranon (see the previous discussion). Roll the skin incision caudally to expose the proximodistal aspect of the ulna. Drive a small K-wire beginning at the caudal aspect of the ulna perpendicular to the nonunion line and into the ununited portion. Drill a 3.5-mm hole parallel to the K-wire, from the caudal aspect of the ulna toward the tip of the anconeus, stopping when the drill reaches the nonunion site. Place a centralizing drill guide in the hole, and drill through to the tip of the anconeus with a 2.5-mm drill bit. Measure the depth of the hole, and place a 3.5-mm screw several millimeters shorter in the hole and observe for compression of the nonunion site.* It is important to use a screw several millimeters shorter than the measured length to prevent exposure of the tip of the screw as the nonunion site compresses (Fig. 34.56). *Close the joint routinely and perform a proximal ulnar osteotomy.*

To perform lag screw fixation with arthroscopic guidance, begin with standard arthroscopy portals and visualize the nonunion site. Make a small incision on the caudal aspect of the proximal ulna at the level of the anconeus. Place a lag screw as described previously.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

An Ochsner forceps is useful for extracting the UAP. Rongeurs and bone files are used to smooth the area. A Gigli wire or oscillating saw is needed for ulnar osteotomy. Bone screws and a power drill are required for lag screw fixation.

POSTOPERATIVE CARE AND ASSESSMENT

Immediate postoperative radiographs should be made if implants are used. The limb may be bandaged after surgery for up to 1 week to provide soft tissue support, and the animal should be confined for 4 weeks. Repeat radiographs are recommended 6 weeks after surgery if an osteotomy or lag screw fixation was performed. Physical rehabilitation may be beneficial in management of osteoarthritis and in improving the range of motion associated with UAP (see Table 34.7). After surgery, cryotherapy, passive range of motion, and controlled leash walks are the primary methods of physical rehabilitation.

COMPLICATIONS

Possible surgical complications include iatrogenic infection, nonunion of the ulnar osteotomy, nonunion of the anconeal process, and implant complications.

PROGNOSIS

Prognosis for dogs with UAP is guarded for normal limb function because secondary osteoarthritis will occur. The prognosis for limb function is good in most dogs less than 1 year old when treated surgically. However, despite treatment, loss of range of motion, crepitation, and progressive osteoarthritis occurs.

NOTE Counsel owners about the probability of progressive osteoarthritis in these dogs, even after surgery.

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INCOMPLETE OSSIFICATION OF THE HUMERAL CONDYLE

DEFINITION

IOHC is a failure of bony union between the medial and lateral portions of the humeral condyle.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The underlying cause of IOHC remains uncertain. Normal ossification of the humeral condyle starts at 2 weeks of age and is complete between 8 and 12 weeks. Elbow incongruence causing an increased humeroulnar joint space may be a factor, based on identification of radioulnar incongruence in CT examinations of dogs with IOHC.[36] The overrepresentation of spaniel breeds suggests a genetic cause. The incidence of bilateral disease is reportedly approximately 90%.[36,37] Abnormalities of the medial coronoid process may be identified in the elbows of dogs with IOHC.

DIAGNOSIS

Clinical Presentation

Signalment

Any breed of dog may be affected, but spaniel breeds are overrepresented. The age of presentation varies from young to middle-aged dogs, although there is an increased incidence in middle-aged male dogs.

History

Dogs present for a history of weight-bearing lameness that is worse after exercise. IOHC may lead to a lateral humeral condylar fracture, in which case the patient will present with a non–weight-bearing lameness. Dogs with humeral condylar fractures that are possibly the result of IOHC (spaniel breeds) should have the contralateral limb radiographed to evaluate for contralateral IOHC.

Physical Examination Findings

Lameness of one forelimb is usually evident. The gait may be stiff or stilted. Pain can be elicited during elbow joint manipulation.

Diagnostic Imaging

IOHC may be detected on a craniocaudal view of the elbow joint (Fig. 34.57); however, the radiographic beam must be lined up with the sagittal plane of the lesion or the lesion may be missed. In addition, the ulna is superimposed over the lesion, making it more difficult to see. Sclerosis of the lateral epicondylar crest may be seen radiographically. CT is more sensitive than radiography in the detection of IOHC.[36,37] In addition to the area of incomplete ossification, new bone formation may be seen on the lateral humeral epicondyle. An increased humeroulnar joint space may also be seen on CT of elbows with IOHC.

Arthroscopy

Arthroscopic examination of elbows with IOHC reveals a fissure line in the cartilage at the center of the humeral condyle (Fig. 34.58). Based on the pathophysiology of IOHC, it is possible that the disease may occur without associated fissuring of the cartilage; however, the sensitivity of arthroscopy in the diagnosis of IOHC has not been studied.



FIG. 34.57 Radiograph of incomplete ossification of the humeral condyle. Note the fissure line.



FIG. 34.58 Arthroscopic image of incomplete ossification of the humeral condyle.

Laboratory Findings

The results of hematologic and serum biochemical analyses are normal in most affected animals.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include OCD, FCP, combined UAP and FCP, and other diseases of young, growing dogs that affect the forelimb (i.e., OCD of the shoulder and panosteitis). These diseases can usually be differentiated radiographically.

MEDICAL MANAGEMENT

Medical management is generally ineffective in the management of IOHC because the lesion can often lead to a complete fracture of the humeral condyle.

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SURGICAL TREATMENT

Surgical treatment is aimed at stabilizing the medial and lateral humeral condyles. Lag screw fixation is the treatment most commonly described, but screw failure due to cyclic loading may occur months or years postoperatively.[36,37] Placement of an autogenous corticocancellous autograft core has been described to create a biologic bone bridge between the medial and lateral condyles.[38] Autogenous bone core grafting is a highly advanced procedure and should be performed only by experienced surgeons trained in this technique.

Preoperative Management

Preoperative workup depends on the patient's age and the presence of other diseases (see Chapter 4).

Anesthesia

The anesthetic regimen used depends on the patient's age and the presence of other diseases (see Tables 32.1 and 32.2).

Surgical Anatomy

Landmarks for placement of a lag screw are the medial and lateral epicondyles. The center of the humeral condyle is just slightly cranial and distal to the epicondyles.

Positioning

The patient is positioned in lateral recumbency with the affected limb uppermost.

SURGICAL TECHNIQUE

This technique is performed closed, with or without arthroscopic visualization. Screw placement is critical, as there is only limited bone stock available in the humeral condyle and placement of the screw in the joint will lead to hemarthrosis and crippling osteoarthritis. *On a cranial caudal radiograph, measure the distance from the medial epicondyle to the fissure line. Palpate the lateral epicondyle and make a limited approach to the region. Drill a 3.5-mm hole beginning just cranial and distal to the lateral epicondyle and aiming toward a similar point on the medial epicondyle. Check the depth of the hole frequently and stop when the depth is the same as that which was measured on the radiograph. Place a centralizing drill guide in the hole, and drill through to the target just cranial and distal to the medial epicondyle. Measure the depth of the hole and place a 3.5-mm screw in the hole. If arthroscopy is available, observe for compression of the fissure as the screw is tightened.*

> **NOTE** The largest diameter screw possible should be placed to minimize the risk of screw failure.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Bone screws and a power drill are required for lag screw fixation.

POSTOPERATIVE CARE AND ASSESSMENT

Immediate postoperative radiographs should be made to evaluate implant positioning (Fig. 34.59). The limb may be bandaged after surgery for up to 1 week to provide soft tissue support, and the animal should be confined for 4 weeks. Physical rehabilitation may be beneficial in management of osteoarthritis and in improving the range of motion (see Table 34.7). After surgery, cryotherapy, passive range of motion, and controlled leash walks are the primary methods of physical rehabilitation.



FIG. 34.59 Postoperative radiograph following lag screw fixation of incomplete ossification of the humeral condyle.

COMPLICATIONS

Breakage of the lag screw and recurrence of clinical signs may occur months to years postoperatively.

PROGNOSIS

Placement of a lag screw generally results in resolution of lameness after surgery; however, breakage of the lag screw and recurrence of clinical signs may occur months to years postoperatively.

TRAUMATIC ELBOW LUXATION

DEFINITION

Traumatic elbow luxation (or *dislocated elbow*) is usually associated with blunt trauma of the elbow joint, causing lateral displacement of the radius and ulna with respect to the humerus.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Elbow trauma that results in rupture or avulsion of one or both collateral ligaments allows luxation of the radius and ulna (Box 34.17).[39] The radius and ulna usually luxate laterally because the large medial condyle of the humerus generally prevents medial luxation. The bone to which the collateral

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BOX 34.17 Important Considerations in Traumatic Elbow Luxation

- Closed reduction of the elbow should be attempted as soon as possible.
- The stability of the elbow should be carefully evaluated after reduction.
- The elbow's response to trauma is degenerative joint disease and diminished range of motion.
- The prognosis is good if the elbow is quickly reduced and stabilized.

ligaments attach may be avulsed, or rupture of the ligaments may occur. In severe injuries, origins of the extensor or flexor muscles may be ruptured or avulsed from the humeral condyle as well. Cartilage damage may occur at the time of injury. Chronic luxation results in chondromalacia, articular cartilage destruction, and secondary DJD.

DIAGNOSIS

Clinical Presentation

Signalment

Any age or breed of dog may be affected, although traumatic elbow luxation is rare in cats. Immature animals tend to have physeal fractures rather than joint luxation.

History

The history usually includes trauma, such as a vehicular or dogfight encounter. The animal is usually acutely lame on the affected limb.

Physical Examination Findings

Affected dogs are unable to bear weight on the affected limb, and the elbow is carried in a flexed position. The forelimb is abducted and externally rotated. Palpation of the elbow reveals a prominent radial head, indistinct lateral humeral condyle, and lateral displacement of the olecranon. Most animals are in pain and resist elbow extension.

Diagnostic Imaging

Lateral displacement of the radius and ulna is apparent on a craniocaudal view of the elbow (Fig. 34.60A). The lateral view (see Fig. 34.60B) shows an uneven joint space between the humeral condyle and the radius and ulna. Avulsion fractures of the medial or lateral condyle of the humerus may be evident. Because of the traumatic cause, thoracic radiographs before surgery are indicated.

Laboratory Findings

Significant laboratory abnormalities are not associated with elbow luxation but may be associated with the trauma that caused the injury.

DIFFERENTIAL DIAGNOSIS

Traumatic elbow luxations must be differentiated from articular fractures and Monteggia fractures.

MEDICAL MANAGEMENT

Most luxated elbows can be reduced by closed manipulation if treated within the first few days after injury. However, dogs with avulsion fractures may be candidates for open reduction and stabilization of the fractured bone to provide greater immediate stability. Closed reduction may be facilitated by suspending the limb from an IV pole for 5 to 10 minutes and using the dog's body weight to distract the joint and encourage muscle relaxation; the dog is then placed in lateral recumbency with the affected limb up. The objective during manual reduction is to hook the anconeal process between the condyles. *To reduce the elbow, determine the position of the anconeal process in relation to the humeral condyles (Fig. 34.61A). Flex the elbow to approximately 100 degrees and inwardly rotate the antebrachium (Fig. 34.61B). After the anconeal process hooks over the lateral condyle, extend the elbow slightly. Abduct and inwardly rotate the antebrachium while placing medial pressure over the head of the radius to force it under the humeral capitulum and into the reduced position (Fig. 34.61C). After the elbow has been reduced, evaluate stability provided by the collateral ligaments by flexing the elbow and paw to 90 degrees and rotating the paw medially and laterally.* If the lateral collateral ligament is intact, the paw can be rotated medially to approximately 70 degrees (140 degrees if it is ruptured). If the medial collateral ligament is intact, the paw can be rotated laterally to 45 degrees; rupture allows the paw to be rotated laterally to approximately 90 degrees.

After the elbow has been reduced, radiographs should be performed to document the location of the radius and ulna and to evaluate joint stability. Mild subluxation, or widening of the joint space, usually responds to immobilization. Severe subluxation is an indication for open reduction and internal stabilization. If reduction is achieved, the limb should be positioned with the elbow in extension and supported with a soft, padded bandage and spica splint or a cast that prevents elbow flexion for 10 to 14 days (see p. 995). After removal of the bandage, range of motion exercises should be performed daily. Exercise should be limited for 3 to 4 weeks after bandage removal. Complications of closed reduction are rare but may include reluxation and osteoarthritis.

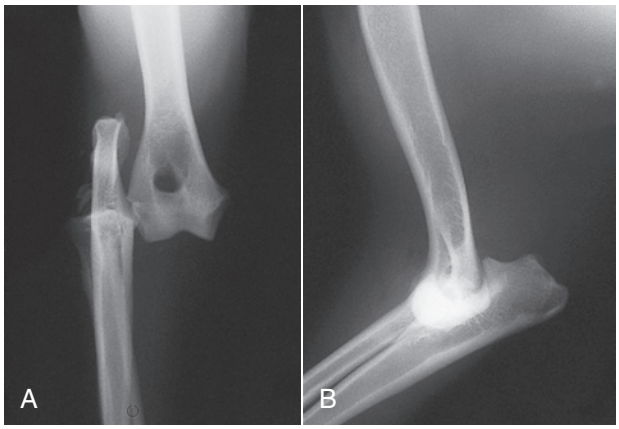


FIG. 34.60 Craniocaudal (A) and lateral (B) radiographs of a luxated elbow in a dog. The radius and ulna are luxated laterally. On the lateral view, there is loss of the humeroradial joint space.

SURGICAL TREATMENT

Open reduction of a luxated elbow is indicated when it is impossible to achieve closed reduction. This is most common when the luxation is chronic. Open reduction should also be considered

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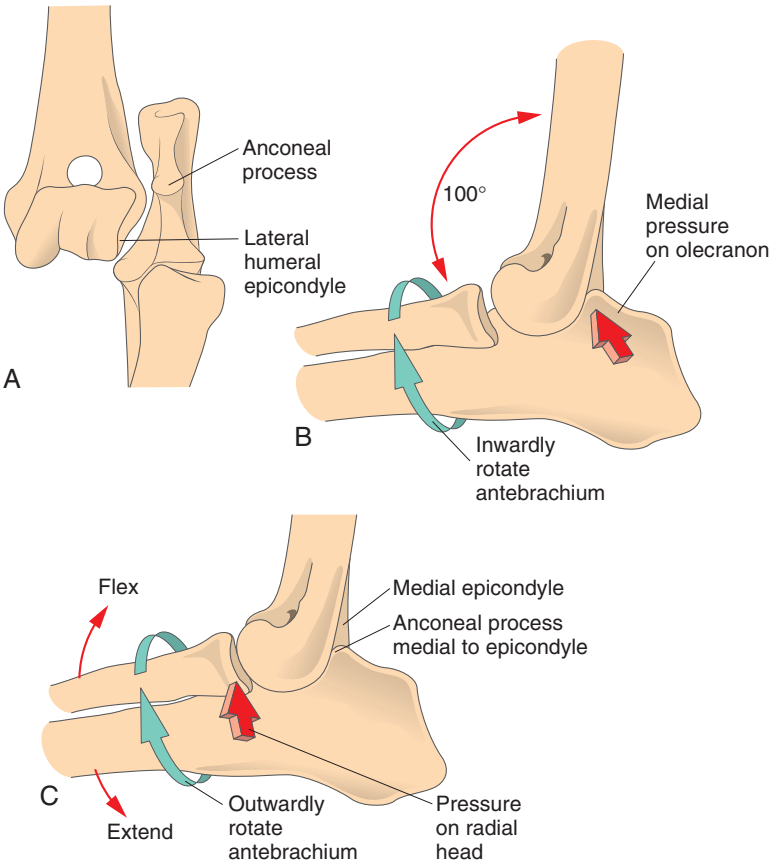


FIG. 34.61 (A) and (B) For closed reduction of a laterally luxated elbow, flex the elbow and inwardly rotate the antebrachium to hook the anconeal process into the olecranon fossa. (C) Then extend the elbow slightly and abduct and outwardly rotate the antebrachium while placing pressure on the radial head.

when the elbow is profoundly unstable after closed reduction or when stabilization of an avulsion fracture will improve joint stability.[40] Elbow arthrodesis should only be considered with severe damage to the cartilage from the trauma of luxation. Elbow arthrodesis limits the dog's function because a normal range of motion in this joint is essential for a normal gait. It is a salvage procedure that is done as a last resort and as an alternative to amputation.

NOTE Excessive instability of the joint indicates the need for open stabilization of surrounding joint tissue. If the joint is stable despite collateral ligament damage, immobilization will allow periarticular fibrosis and some degree of stability; however, this may not provide sufficient stability for large, active dogs.

Preoperative Management

The extent of the preoperative workup depends on the animal's overall health. Laboratory evaluation, thoracic radiographs, and an electrocardiogram evaluation may be indicated before traumatized animals are anesthetized. Stabilization of the trauma patient's condition is more important than reduction of the elbow. However, as soon as the patient's condition has been stabilized, reduction should be attempted.

Anesthesia

General anesthesia is necessary for closed reduction to achieve the profound muscle relaxation necessary for manipulating the elbow into position. Neuromuscular blockage may be helpful in recent fractures or luxations. Special care should be exercised in anesthetizing older animals or those with concurrent injuries (see Table 32.2).

Surgical Anatomy

Anatomic landmarks for open reduction of an elbow luxation are the radial head, olecranon and anconeal processes, and lateral humeral condyle. The deep branch of the radial nerve runs under the proximal cranial border of the extensor carpi radialis muscle. The superficial branch of the radial nerve lies between the lateral head of the triceps and the brachialis muscle and may be exposed in the cranial portion of the incision; it must be identified and protected during surgery. The median nerve and brachial artery and vein course cranially to the medial epicondyle, and the ulnar nerve courses caudally to the medial epicondyle, over the anconeus muscle. These structures must be visualized and protected if a medial approach is made to repair the medial collateral ligament.

Positioning

The dog is positioned in lateral recumbency with the affected leg up. The leg should be prepared from shoulder to carpus.

SURGICAL TECHNIQUE

Open Reduction of an Elbow Luxation

Perform a lateral approach to the caudal compartment of the elbow (see p. 1064). Reduce the elbow as described previously for closed reduction. Protect the articular cartilage during reduction. If muscle contraction and subsequent overriding are severe, use a blunt

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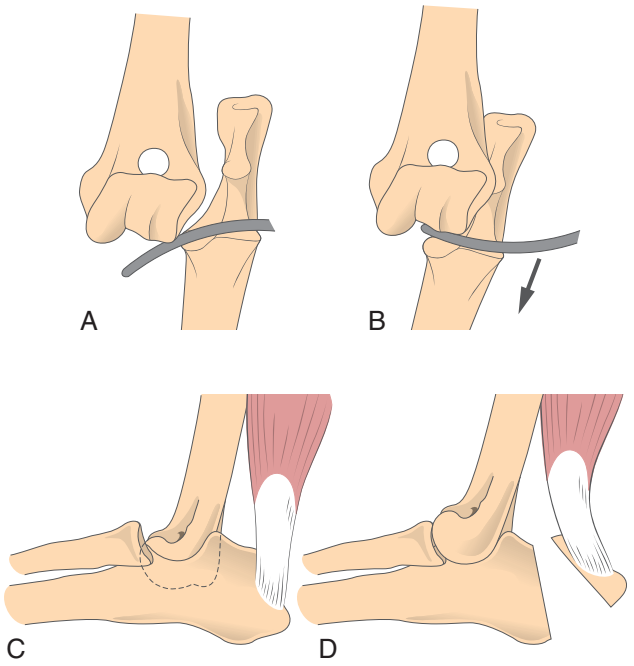


FIG. 34.62 (A) and (B) Open reduction of the elbow may be accomplished using a blunt, curved instrument as a lever. (C) and (D) As an alternative, perform an olecranon osteotomy to eliminate the pull of the triceps muscle during reduction.

instrument to gently lever the radial head into position (Fig. 34.62A–B). If reduction is not achieved, perform an olecranon osteotomy to eliminate the pull of the triceps muscle (Fig. 32.62C–D). After reduction, flush the joint and assess its stability. Stability may be enhanced by primary repair of the lateral collateral ligament. *Identify the ends of the ligaments, and appose them with nonabsorbable sutures in a locking-loop or Bunnell pattern (see Fig. 35.4). If the ligament has torn from its attachment to the bone, secure it to the bone with a screw and spiked washer or use a suture anchor. If the collateral ligament is beyond repair, replace it with two screws and a figure-eight wire, or heavy (No. 1 or No. 2) nonabsorbable suture, or two suture anchors and orthopedic suture (Fig. 34.63A–B). Reduce avulsion fractures of the humeral condyle and secure them with a lag screw (Fig. 34.63C–D). Suture the torn muscles. Suture the fascia, subcutaneous tissue, and skin in separate layers. If additional stability is necessary, expose the medial surface of the elbow and repair or replace the medial collateral ligament.*

Elbow Arthrodesis

AP *Predetermine the angle of arthrodesis by measuring the standing angle of the opposite elbow. Make a caudolateral approach to the joint (see Fig. 33.37), and osteotomize the olecranon (Fig. 34.64A). Expose the joint by incising the lateral collateral ligament and elevating the origins of the extensor muscles. Remove the cartilage from the distal humerus, radial head, and trochlear notch with a high-speed burr. Follow the contours of the joint. Temporarily stabilize the elbow in the correct position with a pin placed through the humerus and into the ulna. Contour a plate to fit onto the caudal surface of the humerus, over the joint, and onto the caudal surface of the ulna. Place at least three screws in the humerus and three in the ulna. Use additional screws as lag screws to gain compression where they cross the arthrodesis site*

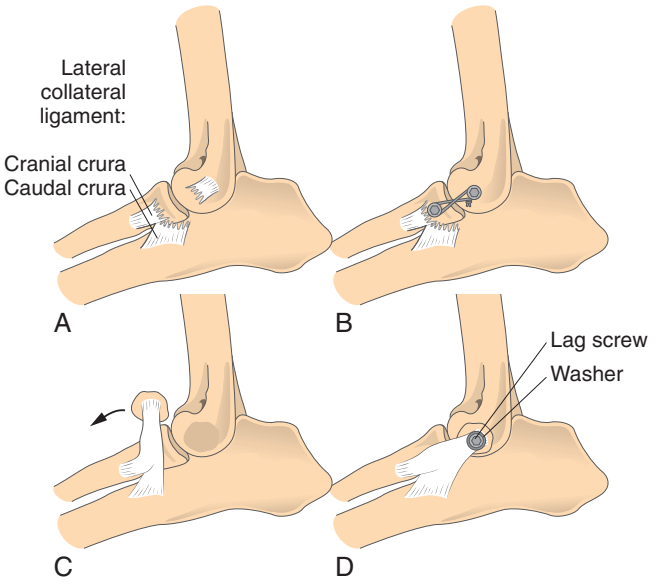


FIG. 34.63 (A–B) To stabilize the elbow, replace collateral ligaments by using two screws and a figure-eight wire. (C–D) To secure an avulsed fragment *(arrow)*, use a lag screw with a spiked Teflon washer.

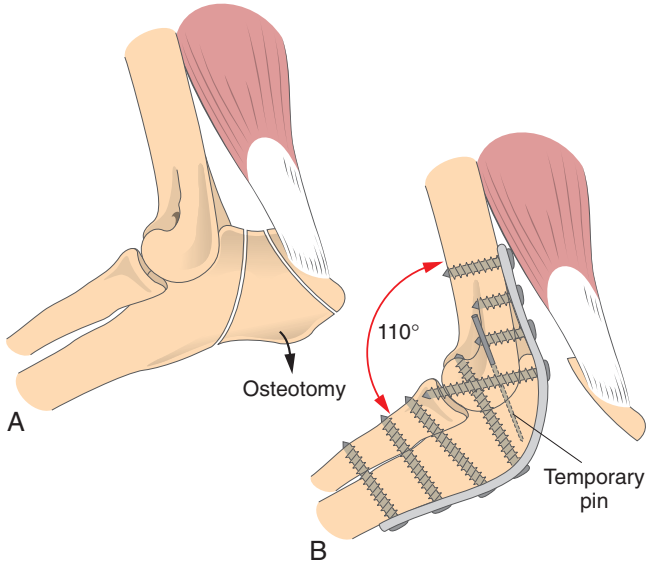


FIG. 34.64 Arthrodesis of the elbow. (A) Make two osteotomies and remove a portion of the proximal ulna. Temporarily stabilize the elbow in the correct position with a pin placed through the ulna and into the humerus. (B) Contour a plate to fit onto the caudal surface of the humerus and the caudal surface of the ulna. Place at least three screws in the humerus and three in the ulna. Use additional screws as lag screws to gain compression across the arthrodesis site.

(see Fig. 34.64B). Check limb alignment, rotation, and angulation before securing fixation. Harvest and place a cancellous bone graft at the arthrodesis site. The most accessible site for cancellous bone harvest is the ipsilateral proximal humerus (see p. 992). *Reattach the olecranon to the ulna on either side of the plate with a lag screw (see p. 1019).*

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NOTE Be sure to remove articular cartilage and rigidly stabilize the joint, or arthrodesis will not occur.

NOTE Locking bone screws and plates increase the stiffness of the arthrodesis construct and limit complications of implant failure or nonunion.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Instruments needed for open reduction of the elbow include suture anchors or bone screws and heavy nonabsorbable suture or orthopedic suture. For arthrodesis, a high-speed drill, burrs, and plating equipment are necessary.

POSTOPERATIVE CARE AND ASSESSMENT

After open reduction, postoperative radiographs should be made to document the location of the radius and ulna and to evaluate the implant's position. After surgery, the limb should be positioned with the elbow in extension and supported with a soft, padded bandage for several days. If questionable stability was achieved, a spica splint can be applied to the limb for 2 weeks. After removal of the bandage, passive range of motion should be performed daily, but exercise should be limited for 3 to 4 weeks.

After arthrodesis, radiographs of the elbow should be taken to evaluate the implant's position and limb alignment. A soft, padded bandage should be applied for a few days after surgery. If there is concern about stability of the implants, a spica splint can be applied for 6 weeks or until radiographic evidence of bone healing is seen. Activity should be limited until the arthrodesis has healed.

COMPLICATIONS

Possible surgical complications include recurrent luxation, infection, decreased range of motion of the elbow, irritation or migration of implants, and secondary osteoarthritis.[40] Complications of arthrodesis include iatrogenic infection, delayed union or nonunion, implant migration, implant irritation of soft tissue, fracture of the bone at either end of the plate, and increased degenerative changes in the limb's distal joints, which are forced to compensate for the loss of elbow range of motion.

PROGNOSIS

The prognosis is good for normal limb function after a stable closed reduction; however, variable development of osteoarthritis and limited range of motion will occur. The prognosis is fair for normal limb function after an unstable closed reduction. Smaller, less active dogs have a better prognosis because the periarticular fibrosis that occurs is likely to be adequate to maintain the elbow in position, whereas it may not be adequate in large or active dogs. Osteoarthritis and limited range of motion are also more likely to occur in larger dogs. The prognosis after surgical reduction and stabilization of the elbow depends on chronicity of the luxation and severity of joint damage. Most dogs have good limb function after surgery. Smaller, less active dogs have a better prognosis than larger, more active dogs. Varying degrees of osteoarthritis and limited range of joint motion commonly develop after surgery.

The prognosis following arthrodesis depends on the size and activity level of the dog. Smaller dogs have fewer complications and less noticeable gait abnormalities. Larger dogs have a higher rate of complications, including delayed union or nonunion and implant failure. The gait abnormalities tend to be more apparent in larger dogs. Active dogs will tend to use the limb at a walk and while standing, but they may hold the limb to the side when running. Elbow arthrodesis in very active dogs may be a source of exercise restriction when compared with amputation.

ELBOW SUBLUXATION CAUSED BY PREMATURE CLOSURE OF THE DISTAL ULNAR OR RADIAL PHYSES

DEFINITIONS

Elbow subluxation may be associated with asynchronous radial or ulnar growth caused by premature closure of one of the distal physes. An **osteotomy** is a cut made through bone. An **ostectomy** is removal of a section of bone.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Two syndromes are grouped under elbow subluxation or incongruity. One syndrome is observed as a part of the pathophysiology of premature closure of the distal ulnar or radial physes after trauma in immature dogs (see p. 1079). The other occurs primarily in chondrodystrophic breeds and is caused by asynchronous growth of the radius and ulna with no overt injury to the growth plate. This asynchronous growth results in incongruity of the elbow joint because either the radius or ulna is inappropriately short. Untreated, elbow incongruity causes instability of the joint and development of secondary DJD (Box 34.18).

When the ulna is too short, the trochlear notch is pulled distally and the anconeal process impinges on the humeral trochlea. In some dogs, this may be associated with a UAP (see Fig. 34.53). In other dogs, it may lead to cranial subluxation or luxation of the radial head. It is often associated with angular limb deformity (see p. 1079), including radius curvus, valgus, and external rotation. When the radius is too short, the radial head is pulled distally and does not articulate with the humeral capitulum (Fig. 34.65). The trochlea of the humerus then rests directly on the coronoid process of the ulna. This incongruity has been implicated in the etiology of FCP. Shortening of the radius is not usually associated with angular limb deformity.

Clinical Presentation

Signalment

This condition occurs in immature dogs with open physes. Any breed of dog affected with premature closure of the physes may

BOX 34.18 Important Considerations in Elbow Incongruity

- Elbow incongruity is the most likely cause of elbow pain and lameness in dogs with asynchronous radial and ulnar growth.
- Elbow incongruity may contribute to fragmented coronoid process and an ununited anconeal process.
- Elbow incongruity leads to secondary degenerative joint disease.

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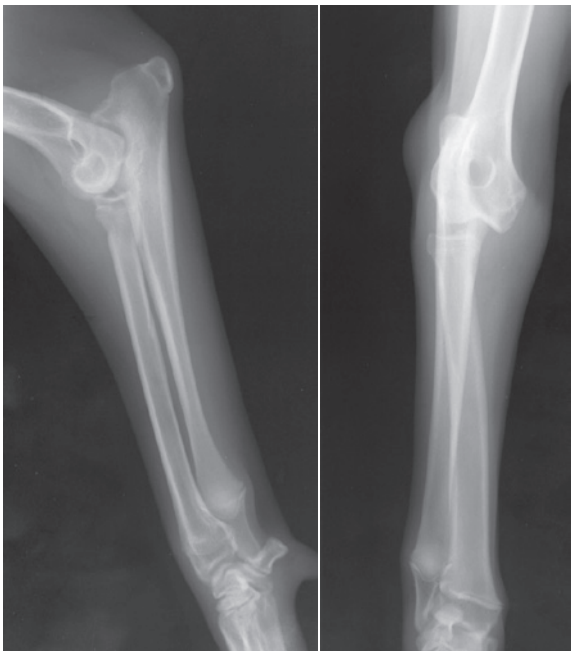


FIG. 34.65 Radiograph of elbow incongruence due to radial shortening.

have subluxation of the elbow. Basset hounds and other chondrodystrophic dogs are more frequently affected with asynchronous growth of the radius and ulna that is not attributable to trauma. The animal may be presented for examination at the onset of the problem or as an older animal with established secondary osteoarthritis.

History

Affected animals often have a history of intermittent lameness.

Physical Examination Findings

Affected dogs show varying degrees of lameness. A gross deformity of the limb may be present, depending on the physeal plate affected and the relationship of the injury to the animal's growth. Even if there is no limb deformity, the animal is usually lame and sensitive to joint manipulation because of the elbow incongruence. Crepitation and limited range of motion may be present during elbow manipulation.

Diagnostic Imaging

Radiographs of the radius and ulna should include the carpus and elbow to determine the exact configuration and cause of the elbow incongruity. Radiographs of the contralateral forelimb can often can be used to compare the affected limb with a normal one; however, bilateral disease may be present.

Laboratory Findings

Specific laboratory abnormalities are not found with this condition.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses in large dogs include FCP, UAP, or OCD. Other conditions that may show similar clinical signs in small dogs include trauma and osteoarthritis.

MEDICAL MANAGEMENT

Medical treatment should address the five principles of medical management of osteoarthritis; however, response to medical therapy is unpredictable, and correction of the subluxation should be attempted to minimize additional joint damage, slow the progression of osteoarthritis, and decrease lameness.

SURGICAL TREATMENT

Surgical treatment is directed at restoring elbow congruity by performing a corrective osteotomy of either the radius or the ulna. Ulnar-lengthening osteotomy is indicated when the cause of elbow incongruity is an ulna that is too short. Ulnar-shortening ostectomy or radial-lengthening osteotomy are performed when radial shortening has caused subluxation (widening) of the humeroradial joint. Radial lengthening may also be performed by distraction osteogenesis using a linear or circular external fixator and motors. Distraction osteogenesis is a highly advanced procedure and should be performed only by an experienced surgeon trained in this technique.

Preoperative Management

Affected animals are young and require minimal preoperative workup (see Chapter 4).

Anesthesia

These animals are usually young and healthy; thus a variety of anesthetic regimens can be used (see Table 32.1).

Surgical Anatomy

Landmarks for ulnar osteotomy include the caudal border of the ulna, which is palpable, and the trochlear notch, which can be exposed surgically by muscle elevation and must be avoided when the osteotomy is performed. Landmarks for radial-lengthening osteotomy depend on the size of the bone plate to be applied.

Positioning

The dog is positioned in dorsal recumbency with the affected leg suspended for draping. The leg is prepared from shoulder to carpus.

SURGICAL TECHNIQUE

Ulnar-Lengthening Osteotomy

Make a skin incision along the caudal border of the ulna, beginning medial to the tuber olecranon and ending at the ulnar middiaphysis. Incise the subcutaneous tissue and fascia along the same line. Incise the attachments of the flexor carpi ulnaris and ulnaris lateralis muscles along the medial and lateral borders of the ulna, and elevate the muscles to expose the joint capsule. Incise the joint capsule on both sides of the ulna to expose the distal trochlear notch area. Make an oblique osteotomy of the ulna distal to the trochlear notch with an oscillating saw (Fig. 34.66). The angle of the osteotomy should be caudoproximal to craniodistal and craniolateral to caudomedial (see Fig. 34.45). A gap should form at the osteotomy site. The gap may be increased by prying apart with an osteotome. If necessary, elevate the interosseous ligament to free the proximal ulna so that it can move into position.

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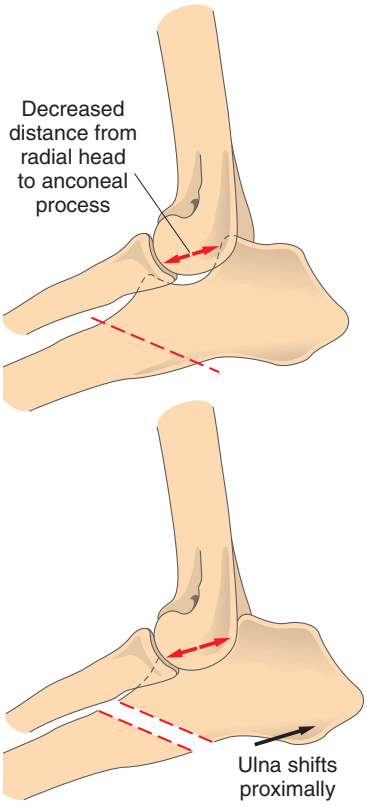


FIG. 34.66 For an ulnar-lengthening osteotomy, make an oblique osteotomy of the ulna distal to the coronoid process.

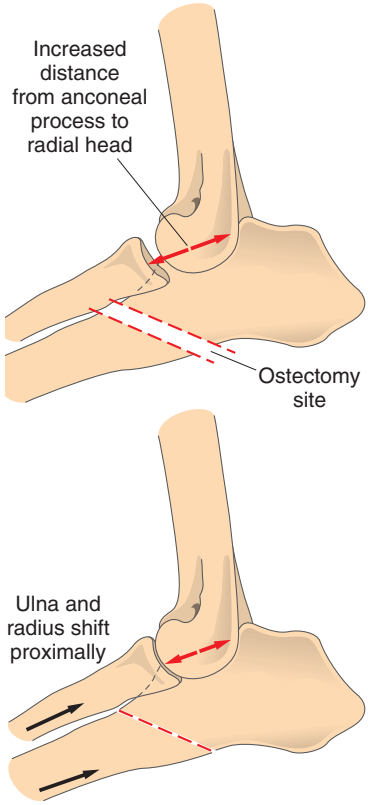


FIG. 34.67 For an ulnar-shortening ostectomy, make two oblique osteotomies of the ulna distal to the coronoid process. Remove a length of bone sufficient to allow reduction of the radial head.

NOTE A small smooth pin or K-wire may be driven from the tuber olecranon down the medullary canal across the fracture gap and into the medullary canal of the distal ulna. The smooth pin allows the forces of the surrounding musculature to exert a dynamic effect on the proximal ulna, causing it to distract. However, there is a high complication rate of implant failure and nonunion when a pin is used.

Suture the joint capsule. Suture the flexor carpi ulnaris fascia to the ulnaris lateralis fascia over the caudal border of the ulna. Suture the subcutaneous tissue and skin separately.

Ulnar-Shortening Ostectomy

Shortening the ulna allows the radial head to come into contact with the humeral capitulum.

Approach the ulna as described earlier for the ulnar-lengthening osteotomy. Using an oscillating saw, resect a segment of ulna that is longer than the measured distance from the radial head to the humeral capitulum (Fig. 34.67).

NOTE See the previous note regarding the use of a small smooth pin or K-wire.

Radial Lengthening

AP Radial lengthening is performed by transposing a segment of ulna into an osteotomy site on the radius to lengthen the radius while permitting the ulna to shorten.

Perform a caudal approach to the central third of the ulna. Dissect down to the bone and around the diaphysis. Using a bone saw, remove a segment of bone equal to or just shorter than the length discrepancy of the radius. Close the ulnar incision routinely. Perform a cranial approach to the radius over the proximal and central thirds of the diaphysis. Operative planning for the site of the radial osteotomy requires determining the length and location of an appropriate-sized bone plate. In small dogs, a 2.7-mm bone plate may be used, whereas in large dogs, a 3.5-mm bone plate may be used. The osteotomy should be planned so that three holes of the bone plate may be placed proximal to the osteotomy. *Perform a transverse osteotomy of the radius at the location that permits stabilization with a bone plate. Using an osteotome, lever the radial osteotomy apart, and insert the ulnar bone fragment or a portion of it. Secure the osteotomy with a 2.7- or 3.5-mm bone plate using standard technique.* As an alternative, an oblique osteotomy of the radius may be performed using a bone sliding technique. *Perform an oblique osteotomy in the radius, then slide the proximal segment further proximally. Secure temporarily with a K-wire or bone clamp. Secure the osteotomy with a 2.7- or 3.5-mm bone plate using the standard technique of the Association for the Study of Internal Fixation.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Instruments for ulnar osteotomy include an oscillating saw, Steinmann pins, and a pin chuck or power drill for pin insertion. Instruments for radial lengthening include a power saw and bone plating equipment.

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POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiographs should be taken to evaluate the position of the ulna and congruence of the joint. Obvious changes in position may not be evident until several days after surgery. A soft, padded bandage should be applied to the limb postoperatively. Early motion of the elbow is important for dynamic reduction of the subluxated elbow; therefore leash activity should be encouraged. However, excessive activity may lead to implant failure and nonunion. Serial radiographs should be made until the area of osteotomy or ostectomy has healed. The implant can be removed after the bone has healed. Following healing, rehabilitation consists of passive range of motion, muscular strengthening, and aquatic therapy. Physical rehabilitation can be key in maintaining range of motion of the other joints of the limb after this type of procedure, along with decreasing lameness and improving overall function. For a sample physical rehabilitation exercise protocol, please refer to Table 34.7.

NOTE Radiographs taken 48 to 72 hours after surgery can demonstrate the effect of motion on the position of the ulna.

COMPLICATIONS

Possible surgical complications include iatrogenic infection, implant migration, implant failure, delayed union or nonunion of the osteotomy, and implant irritation of soft tissue. If the intramedullary pin irritates the triceps tendon, it should be removed.

PROGNOSIS

Without surgery, abnormal anatomy of the elbow will cause osteoarthritis and lameness. If surgery is performed before osteoarthritis is established, the prognosis is good for relatively normal limb function, although some osteoarthritis usually occurs. The amount of correction after surgery varies and appears to be directly related to the amount of displacement; that is, less displacement results in better correction. Despite the lack of radiographic correction, most dogs appear to be more comfortable after surgery. After osteoarthritis is established, periodic treatment with analgesics and antiinflammatory drugs may be necessary (see Table 34.3).

CONGENITAL ELBOW LUXATION

DEFINITION

Congenital elbow luxation results in lateral rotation of the proximal ulna and subluxation or luxation of the humeroulnar joint. Synonyms include *congenital elbow luxation* and *congenital elbow malformation*.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The etiology of congenital elbow luxation is unknown. The bone malpositioning occurs at a young age, and because the bones do not articulate normally, congruent joint surfaces do not form. At approximately 3 months of age, secondary remodeling occurs and degenerative changes begin to develop. Pathologic conditions vary with the chronicity of the condition. The olecranon is rotated

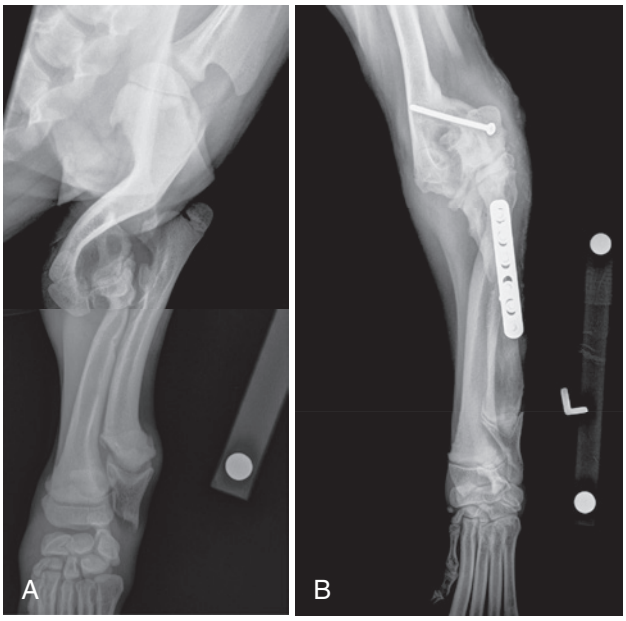


FIG. 34.68 (A) Preoperative craniocaudal radiograph of the forelimb of a dog with congenital elbow luxation with lateral rotation of ulna. (B) Craniocaudal radiograph of the forelimb after reduction and stabilization of the elbow luxation.

lateral to the distal humerus, and the trochlear notch does not contact the humeral condyles (Fig. 34.68). The result or results may be (1) hypoplasia and remodeling of the trochlea and trochlear notch, (2) hypoplasia of the medial humeral condyle with stretching of the medial collateral ligament and joint capsule, (3) hyperplasia of the lateral humeral condyle with contracture of the lateral joint capsule and lateral collateral ligament, (4) contracture and displacement of the triceps muscle, and (5) degenerative changes of the articular cartilage. Caudolateral luxation of the radial head is a different type of congenital elbow luxation (Fig. 34.69).

DIAGNOSIS

Clinical Presentation

Signalment

Small breeds of dogs are affected: pugs, Yorkshire terriers, Boston terriers, miniature poodles, Pomeranians, Chihuahuas, cocker spaniels, and English bulldogs are overrepresented. The condition can be unilateral or bilateral and is usually recognized when the puppy begins to walk at 3 to 6 weeks of age.

History

The history describes the dog's inability to extend one or both front legs and difficulty walking because of the crouching position. Smaller dogs tolerate the deformity better and in some cases, show minimal or no gait abnormality or pain.

Physical Examination Findings

Puppies with this condition carry the affected forelimb in flexion and internal rotation (external rotation of the elbow). If the condition is bilateral, the puppy supports weight on the caudomedial aspect of the forelimbs. The elbows cannot be extended. The olecranon is located on the lateral aspect of the limb and may be mistaken for the lateral humeral condyle. The condition is not usually painful.

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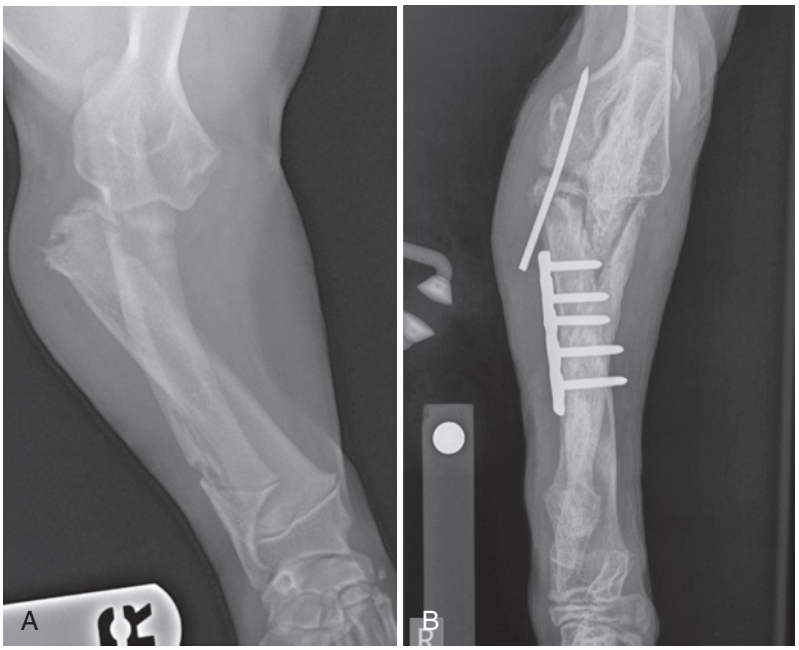


FIG. 34.69 (A) Preoperative craniocaudal radiograph of the forelimb of a dog with congenital elbow luxation with lateral displacement of radius. (B) Craniocaudal radiograph of the forelimb after reduction and stabilization of the elbow luxation.

Diagnostic Imaging

Lateral and craniocaudal radiographs of the elbow show lateral displacement and rotation of the olecranon with varying degrees of contact between the ulna and the humerus (see Fig. 34.68A). The radial head is usually in contact with the humerus, although this may change as the animal matures. Secondary DJD is apparent in chronic cases.

Laboratory Findings

Abnormal laboratory findings are uncommon.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include caudolateral luxation of the radial head (see Fig. 34.69), hemimelia (congenital segmental deficiency of the radius or ulna), ectrodactyly (congenital splitting of the limb), and previous fracture and malunion of the limb.

MEDICAL MANAGEMENT

Conservative therapy (splints and bandages) does not alter the course of the disease. Joint reduction and stabilization should be performed as soon as possible, before secondary degenerative changes and joint remodeling occur (usually before the animal is 4 months of age). The type of reduction technique used depends on the pathologic condition present. Closed reduction can be successful if the joint can be manipulated into position. Closed reduction is indicated in dogs that have only mild changes in bone and soft tissue. The olecranon should be rotated medially into position and secured by placing a transarticular pin from the caudal aspect of the olecranon, through the olecranon, and into the humerus. The pin is left in place for 10 to 14 days (Fig. 34.70).

SURGICAL TREATMENT

Open reduction and corrective osteotomy are used when the joint cannot be manually replaced (Box 34.19). Techniques for

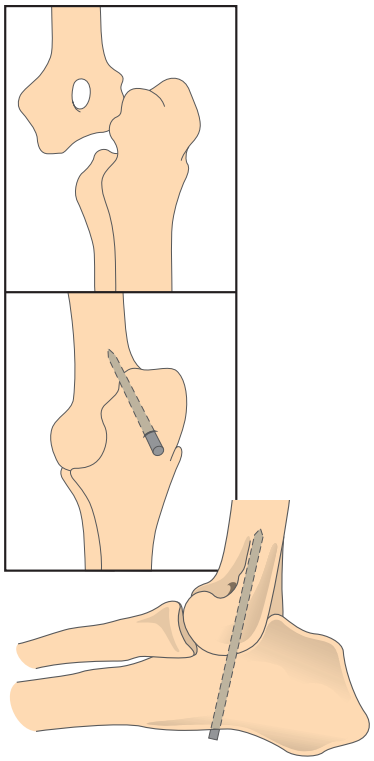


FIG. 34.70 A transarticular pin can be placed to hold the ulna in position after manual reduction of a congenitally luxated elbow.

BOX 34.19 Important Considerations in Congenital Elbow Luxation

- Immediate surgical treatment provides the best outcome.
- Repositioning the ulna results in improved limb function because the pull of the triceps muscle is redirected.
- Limited range of motion and degenerative joint disease occur even with surgery.

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open reduction and stabilization vary, depending on the severity of the pathologic condition. Techniques used include lateral release of soft tissue (including the joint capsule and anconeus muscle), medial support of the olecranon using capsular imbrication and stay sutures, olecranon or ulnar osteotomy and transposition to reconstruct the joint, and redirection of the pull of the triceps muscle to allow joint extension. The osteotomy is stabilized with K-wires and, if necessary, a tension band wire (see Fig. 34.69B).

Preoperative Management

Affected animals are young and require minimal preoperative workup.

Anesthesia

General anesthesia is required for both closed and open reduction. Pediatric anesthesia techniques should be used. For anesthetic management of animals with orthopedic disease see Tables 32.1 and 32.2.

Surgical Anatomy

Anatomic landmarks are the olecranon and the medial and lateral humeral condyles. The ulnar nerve is on the medial aspect of the surgical site and should be identified and protected.

Positioning

The dog is placed in dorsal recumbency with the affected limb prepared from shoulder to carpus.

SURGICAL TECHNIQUE

Make an incision on the lateral surface of the joint, starting on the lateral epicondylar crest and extending distally over the lateral epicondyle to the proximal radius, as described on p. 1064. Incise the subcutaneous tissue, and retract the skin medially to expose both the medial and lateral surfaces of the elbow. Incise the soft tissue on the lateral aspect of the humeroulnar joint (including the anconeus muscle and joint capsule) to expose the joint and reposition the ulna. If repositioning is possible, stabilize the ulna by imbricating the medial joint capsule and placing a large (No. 0 to No. 2) nonabsorbable suture from the proximal ulna to the humeral condyle through tunnels drilled in the bone with the needle or a small K-wire. Perform an olecranon osteotomy, and transpose the osteotomized bone to a position on the ulna that best redirects the pull of the triceps muscle to extend the joint. Stabilize the osteotomy with a plate or K-wires and possibly a tension band wire (see p. 1075). Reposition the skin to the lateral aspect of the elbow, and close the subcutaneous tissue and skin with interrupted sutures.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Instruments needed for open reduction include a general surgery pack, pin chuck, K-wires, and orthopedic wire.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiographs should be made to evaluate the location of the ulna with respect to the humerus and the position of any implants. The limb should be bandaged to support the fixation for 2 to 3 weeks. Splint the elbow in a functional position. Activity should be restricted to leash walking for 4 to 6 weeks. The K-wires should be removed when the osteotomy has healed. Physical rehabilitation can be key in maintaining muscle mass, range of motion, and comfort. See Table 34.7 for a recommended physical rehabilitation exercise protocol.

COMPLICATIONS

Possible surgical complications include loss of joint reduction, iatrogenic infection, implant migration, and irritation of soft tissue.

PROGNOSIS

Without surgery, the dog may learn to compensate for loss of normal forelimb function by using the rear limbs for support and locomotion; however, ambulation will always be abnormal. The prognosis is good for the return of satisfactory function after surgery but poor for the development of a normal joint.

CARPAL JOINT

CARPAL LUXATION OR SUBLUXATION

DEFINITIONS

Carpal luxation or **subluxation** results from loss of palmar ligamentous support of the antebrachial, carpal, middle carpal, and/or carpometacarpal joints. Carpal subluxation may also result from severe end-stage polyarthritis. **Pancarpal arthrodesis** involves fusion of all three carpal joints, whereas **partial arthrodesis** is a selective fusion of one or more of the carpal joints.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Carpal hyperextension injuries are sorted into three categories based on injury location. *Category I injuries* are subluxations or luxations of the antebrachial carpal joint. *Category II injuries* include subluxation of the middle carpal and carpometacarpal joints and are associated with disruption of the accessory carpal ligaments, palmar fibrocartilage, and palmar ligaments of the middle carpal and carpometacarpal joints. Dorsal displacement of the free end of the accessory carpal and ulnar carpal bones occurs. *Category III injuries* are disruptions of the accessory carpal ligaments, carpometacarpal ligaments, and palmar fibrocartilage. However, in these injuries subluxation of the carpometacarpal joint occurs without disruption and displacement of the accessory carpal and ulnar carpal bones.

Polyarthritis often affects smaller joints, such as the carpus or tarsus (see p. 1151). Chronic joint inflammation may lead to severe cartilage and soft tissue damage with progressive collapse of the joint to a palmigrade stance.

DIAGNOSIS

Clinical Presentation

Signalment

Any age or breed and either gender of dog or cat may be affected with traumatic luxation or subluxation. Smaller dogs, particularly Shetland sheepdogs, are overrepresented for idiopathic or immune-mediated collapse of the carpal joints.

History

Acutely affected animals usually have a non–weight-bearing lameness. Because the animal is not using the limb, the extent of hyperextension may not be apparent initially. Most animals return to using the leg within a week, but they are lame and

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have a plantigrade stance. Chronically affected dogs with idiopathic or immune-mediated polyarthritis and carpal joint collapse have a history of progressive degeneration of the carpi.

Physical Examination Findings

Acute injuries show clear indications of swelling, pain, and instability. With category I injuries, the animal usually remains unable to bear weight until definitive treatment is given. With category II or III injuries, the animal may begin to bear weight on the limb after the injury. However, as increased weight is placed on the limb, collapse and hyperextension of the carpus become evident.

Dogs with idiopathic or immune-mediated polyarthritis and carpal subluxation have a plantigrade stance and may have other visible deformity of the carpi (Fig. 34.71). There is mild to moderate pain on manipulation of the joints. The disease is often bilateral.

Diagnostic Imaging

Standard craniocaudal and medial-to-lateral radiographs are needed to detect bone fractures or joint malalignment associated with complete luxation of the joints. However, stress radiographs should be made to assess carpal integrity accurately and identify the joint level of the hyperextension injury (Fig. 34.72). With the patient under heavy sedation or general anesthesia, it should be positioned in lateral recumbency with stress applied to the foot to place the carpus in maximum extension. A medial-to-lateral radiograph is produced with the carpus in maximal extension. If the contralateral limb is normal, similar images may be made for comparison. Isolated instability at the level of the antebrachial carpal joint allows an increase in extension without altering the relationships of the carpal and metacarpal bones. Instability at the middle carpal joint, including loss of integrity of the accessory carpal bone, results in widening of the space between the palmar process of the ulnar carpal bone and the base of metacarpal V. The accessory carpal bone deviates dorsally. The proximal carpal bones may appear to override the distal row of carpal bones with instability at the carpometacarpal level. In chronic cases, radiographic evidence of osteoarthritis is evident.

Laboratory Findings

Cytology from arthrocentesis of dogs with carpal collapse may be indicative of DJD (see p. 1143) or immune-mediated joint disease (p. 1151).

DIFFERENTIAL DIAGNOSIS

Differential diagnoses of traumatic carpal injuries include acute sprains, collateral ligament tears, distal radial fractures, and fractures of the metacarpal bones. These conditions can be differentiated from carpal luxation or subluxation radiographically by use of standard and stress views.

Differential diagnoses for progressive collapse of the carpal joints include any form of polyarthritis or joint collapse due to chronic steroid administration.

MEDICAL MANAGEMENT

Medical or conservative management of carpal hyperextension injuries is often unrewarding. External coaptation may be tried in younger patients, but gradual hyperextension often occurs as weight bearing returns.[41]





FIG. 34.71 (A) Carpal hyperextension. (B) Severe carpal collapse in a dog with immune-mediated polyarthritis.

For medical management of polyarthritis, see p. 1151. Medical management of end-stage carpal collapse due to idiopathic or immune-mediated joint disease is often unrewarding, as the animals usually have severe problems ambulating because of biomechanical failure of the carpal joints.

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FIG. 34.72 Stress radiograph of a dog with a category II carpal hyperextension injury. Stress radiographs should be obtained to help classify hyperextension injuries.

SURGICAL TREATMENT

Carpal luxations are best treated with pancarpal arthrodesis. Pancarpal arthrodesis is most commonly performed with a dorsally placed bone plate; however, application of a specially designed medial bone plate has also been described.[42,43] Pancarpal arthrodesis may also be performed with an external fixator. Carpal hyperextension injuries are best treated with either pancarpal arthrodesis or partial arthrodesis. Antebrachial carpal injuries should be treated with a pancarpal arthrodesis accomplished by removing articular cartilage at all joint levels, placing a cancellous bone autograft at the joint surfaces, and stabilizing with a bone plate or external fixator. Pancarpal arthrodesis or partial carpal arthrodesis may be used to manage injuries of the middle carpal joint; however, partial arthrodesis that does not reestablish the accessory carpal moment arm may eventually fail because of breakdown of the radiocarpal joint. Partial arthrodesis for treating this injury should include fusion of the middle carpal and carpometacarpal joints with selective fusion of the accessory carpal–ulnar carpal articulation. This procedure may be accomplished with a bone plate, cross pins, or placement of longitudinal metacarpal pins. With isolated carpometacarpal instability, a partial arthrodesis is recommended. Although the accessory carpal ligaments may be compromised, the ligaments between the base of the accessory carpal bone, ulnar carpal bone, and fifth metacarpal bone are intact, preserving integrity of the accessory carpal moment arm. Articular cartilage should be removed, cancellous bone placed in the fusion site, and the site stabilized with a T plate or intramedullary pins.

End-stage carpal joint collapse as a result of idiopathic or immune-mediated joint disease is best treated with pancarpal arthrodesis. It is important to verify with arthrocentesis and cytology that the inflammatory stage of the disease is no longer active.

Preoperative Management

An external coaptation splint or bandage should be applied to protect the limb until definitive surgical treatment is undertaken. The animal's activity should be strictly limited to prevent further joint damage.

Anesthesia

Recommendations for the anesthetic management of animals with orthopedic disease are provided in Tables 32.1 and 32.2.

Surgical Anatomy

The carpus consists of seven bones arranged in two rows. The radiocarpal and ulnar carpal bones make up the proximal row, and the first, second, third, and fourth carpal bones make up the distal row. The accessory carpal bone lies caudally and articulates with the ulnar carpal bone. The radiocarpal and ulnar carpal bones articulate with the radius and styloid process of the ulna to form the antebrachial carpal joint. The middle carpal joint, formed by articulation of the proximal and distal rows of carpal bones, has the greatest movement—accounting for 10% to 15% of carpal motion. Very little motion occurs in the carpometacarpal and intercarpal joints. Palmar support is from the flexor retinaculum proximally and palmar fibrocartilage distally. Multiple small ligaments cross the intercarpal articulations between carpal bones to provide additional collateral and palmar support. Two accessory ligaments originate from the free end of the accessory carpal bone and insert onto the palmar surface of the fourth and fifth metacarpal bones. The caudal position of the free end of the accessory carpal bone, in conjunction with the accessory carpal ligaments, acts as a moment arm to balance the vertical force produced when the paw strikes the ground.

Positioning

For arthroscopy, the animal should be positioned in sternal recumbency with the limb that is to be operated pulled forward and off the side of the table. For open surgery, the animal should be positioned in dorsal recumbency. A hanging-leg preparation allows maximum manipulation of the limb during surgery (see Chapter 5). The leg should be clipped and prepared from the proximal humeral region to the tips of the toes. The proximal humerus serves as the donor site for cancellous bone.

SURGICAL TECHNIQUE

Considerable collagenous and bony tissue proliferation often occurs with carpal luxation or subluxation. Increased vascularity, which accompanies the fibrous proliferation, can make surgical dissection difficult. Use of a tourniquet is helpful. The joint is approached dorsally, where proliferative tissue is pronounced. Sharp dissection through scar tissue and the joint capsule creates the least trauma.

Approach to the Carpal Bones

Make a skin incision over the midline of the dorsal surface of the carpus, extending from 4 cm proximal to the radiocarpal joint line to 4 cm distal to the carpometacarpal joint line (Fig. 34.73A). Incise the subcutaneous tissue, proliferative fibrous tissue, and joint capsule overlying the radiocarpal, middle carpal, and carpometacarpal joints (Fig. 34.73B). The proliferative fibrous tissue will be confluent with the joint capsule proximally and distally. *Using sharp dissection, reflect the synovial joint capsule incision from the cranial face of the carpal bones both medially and laterally. Place Gelpi retractors to maintain exposure of the joint surfaces, and position a small Hohmann retractor between joint surfaces to help visualize the articular cartilage of each joint. Use a low-speed power burr to remove articular cartilage from the surface of the carpal bones in each joint. Attempt to preserve the tendon of the extensor carpi radialis muscle as it crosses the craniolateral aspect of the joint. Harvest a cancellous bone graft from the proximal humerus (see p. 992) and insert the graft within the denuded surfaces of*

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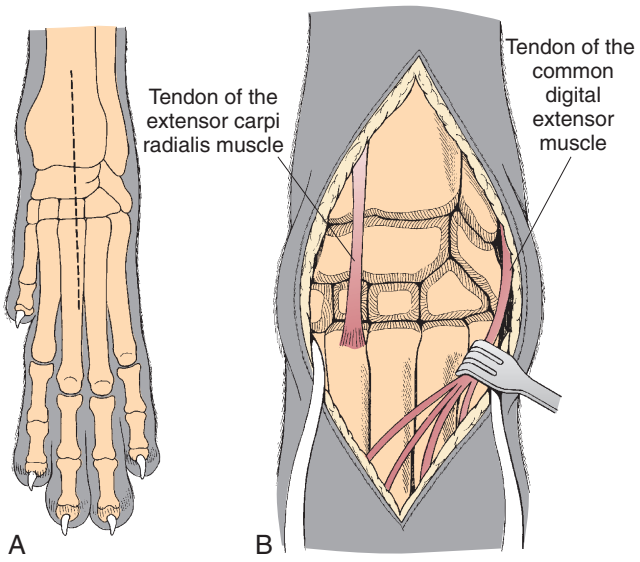


FIG. 34.73 (A) To approach the dorsal surface of the carpus, make a skin incision over the midline, extending from 4 cm proximal to the radiocarpal joint line to 4 cm distal to the carpometacarpal joint line. (B) Incise the subcutaneous tissue, proliferative fibrous tissue, and joint capsule overlying the radiocarpal, middle carpal, and carpometacarpal joints. Remove articular cartilage (*shaded areas*).

each joint. Stabilize the arthrodesis with an implant as discussed later in the chapter. Suture the wound in layers; tension may be needed to appose the tissue over a plate. If tension is excessive, perform release incisions on the medial and/or lateral aspects of the limb to permit closure of the primary incision. Excessive tension during closure of the primary incision can lead to severe limb swelling, wound dehiscence, and, in severe cases, necrosis and death of the foot. Performing full-thickness incisions on the medial or lateral surfaces (release incisions) results in formation of a bipedicle flap that permits closure of the primary wound without tension. The release incisions will heal quickly by secondary intention.

Pancarpal Arthrodesis

Expose the joint surfaces of the radiocarpal, middle carpal, and carpometacarpal articulations, and remove articular cartilage as described previously. Use a small K-wire to drill multiple holes through the distal radial epiphysis into the marrow cavity to aid vascularization of the fusion. Stabilize the fusion with a bone plate applied as a compression plate to the dorsal surface of the radius. Contour the plate to reflect a 10-degree angle of extension of the carpus. Apply the plate so that the proximal three plate screws enter the distal radius and the distal three plate screws enter the third metacarpal bone. Place one intermediate plate screw in the radiocarpal bone and the others where bone stock is available. Because the plate is not positioned on the tension surface of the joint, it may be supported with small Steinmann cross pins or external coaptation or both. If pins are selected, place one pin from medial to lateral, entering the bone near the head of the second metacarpal and exiting through the distal ulna. Place a second pin from lateral to medial, entering the bone near the head of the fifth metacarpal and exiting through the distal radius (Fig. 34.74).

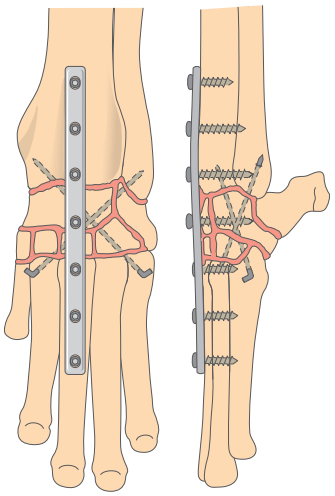


FIG. 34.74 Pancarpal arthrodesis for stabilization of category I carpal hyperextension injuries. Note the cranial application of a bone plate and caudal placement of cross pins. The plate should be contoured 10 degrees in extension.

Pancarpal Arthrodesis With an External Fixator

Expose the joint surfaces of the radiocarpal, middle carpal, and carpometacarpal articulations, and remove articular cartilage as described previously. Harvest a cancellous bone graft from the proximal humerus (see p. 992) and insert the graft within the denuded surfaces of each joint. Close the wound as described previously. For a type II external fixator, place two to three threaded pins from medial to lateral in the metacarpal bones and two to three pins in the radius. Connect the pins with clamps and rods or acrylic. For a type Ib external fixator place pins at an angle (craniomedial and craniolateral) into two of the metacarpal bones and the distal radius. Connect the pins with clamps and rods or acrylic.

Partial Carpal Arthrodesis

Expose the joint surfaces of the middle carpal and carpometacarpal joints, and remove articular cartilage as described previously. Harvest and insert a cancellous bone graft. Stabilize the joints with a small T plate attached to the distal dorsal surface of the radiocarpal bone (as far distally as possible to avoid interference with the antebrachial carpal joint) and the dorsal surface of metacarpal III. Cut the tendon of insertion of the extensor carpi radialis muscle from metacarpal III to facilitate placement of the plate, and suture it to the similar tendon of insertion on metacarpal II (Fig. 34.75).

NOTE Be sure to remove articular cartilage and rigidly stabilize the joint, or arthrodesis will not occur.

Partial Carpal Arthrodesis With Intramedullary Pins

Expose the joint surfaces of the middle carpal and carpometacarpal joints, and remove articular cartilage as described previously. Harvest and insert a cancellous bone graft. To stabilize the fusion, create a slot in the dorsal cortex of metacarpals III and IV, and drive a pin proximally into the medullary canal of each bone. Reduce the carpus by flexing it 90 degrees and placing proximal and palmar pressure on the metacarpal bones. Drive the pins proximally into the radiocarpal bone, but do not penetrate the proximal articular surface.

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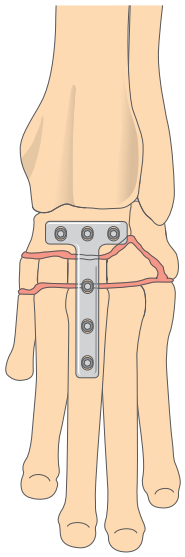


FIG. 34.75 Partial carpal arthrodesis can be stabilized with a T plate.

Bend the distal ends of the pins into a hook, cut the excess pin, and turn the hook into the bone.

Partial Carpal Arthrodesis With Cross Pins and a Lag Screw

Expose the joint surfaces of the middle carpal and carpometacarpal joints, and remove articular cartilage as described previously. Harvest and insert a cancellous bone graft. Stabilize the fusion by placing a pin from medial to lateral, entering the bone near the head of the second metacarpal, and penetrating the radiocarpal bone. Place a second pin from lateral to medial, entering the bone near the head of the fifth metacarpal, and penetrating the ulnar carpal bone. Fuse the accessory carpal–ulnar carpal articulation by removing articular cartilage between joint surfaces of the accessory carpal bone and ulnar carpal bone, placing a cancellous bone graft, and stabilizing it with a compression screw. Make an incision lateral to the base of the accessory carpal bone. Incise the subcutaneous tissue and deep fascia adjacent to the lateral accessory carpal ligament. The medial and lateral accessory carpal ligaments will be torn. *Continue the incision lateral to the abductor digiti quinti muscle to the joint capsule. Incise the joint capsule to expose and remove articular cartilage from the ulnar carpal and accessory carpal bones. Insert a cancellous bone graft, and stabilize the fusion with a compression screw and wire. Insert the compression screw from the cranial face of the ulnar carpal bone into the accessory carpal bone. Place a wire from the base of the accessory carpal bone through the head of the fifth metacarpal bone.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Instruments for arthrodesis include a power drill, burrs, bone curette, bone plates, and screws or external fixation pins, clamps, bars, or acrylic.

POSTOPERATIVE CARE AND ASSESSMENT

A soft, padded bandage with a coaptation splint should be applied postoperatively to reduce swelling and support the internal fixation. Healing of an arthrodesis takes 12 to 16 weeks, and an external splint should be worn for 6 to 8 weeks. Once the splint is removed, conservative physical rehabilitation should begin immediately to combat muscle atrophy and loss of range of motion. External splinting is not used with external fixator stabilization. Uncontrolled activity should be strictly controlled until union has occurred.

COMPLICATIONS

Occasionally, soft tissue irritation or screw loosening is an indication for removal of the implant. Infection may occur due to limited soft tissue covering. Metacarpal bone fractures may occur in a small percentage of patients with pancarpal arthrodesis, especially if the plate extends only a short distance on the metacarpal bone. Return to function after partial carpal arthrodesis may be compromised if additional instabilities are overlooked or if the plate interferes with the antebrachial carpal joint.

PROGNOSIS

Pancarpal and partial carpal arthrodeses result in excellent limb function in approximately 80% of patients treated for hyperextension injuries.[42,43] The remaining 20% are substantially improved after surgery but may have varying degrees of limb dysfunction (lameness) after exercise. A large percentage of working dogs may return to full activity. A small percentage exhibit continued, slight, weight-bearing lameness, but limb function is vastly improved relative to preoperative function.

CARPAL SUBLUXATION RESULTING FROM COLLATERAL LIGAMENT INJURY

DEFINITIONS

Carpal subluxation may result from loss of collateral ligamentous support of the antebrachial carpal joint. **Pancarpal arthrodesis** involves fusion of all three carpal joints. A **prosthetic ligament** involves replacing the damaged ligament with a synthetic suture.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Carpal collateral ligament injuries are always due to trauma. Either the medial or lateral collateral ligament may be involved. Most injuries involve the radiocarpal joint. Collateral injuries involving other joints of the carpus or other ligaments of the carpus should be treated as described previously for carpal luxations.

DIAGNOSIS

Clinical Presentation

Signalment

Any age or breed and either gender of dog or cat may be affected.

History

Acutely affected animals usually have a non–weight-bearing lameness.

Physical Examination Findings

Acute injuries show clear indications of swelling, pain, and instability. Palpation of the carpus demonstrates the ability to "break open" the joint either medially or laterally.

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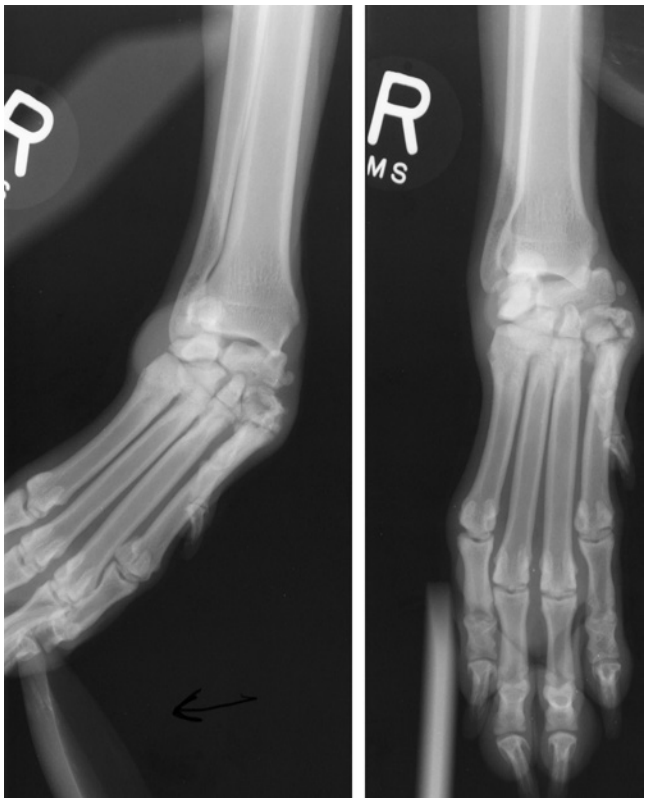


FIG. 34.76 Radiographs of collateral ligament instability of the carpal joint.

Diagnostic Imaging

Standard craniocaudal and medial-to-lateral radiographs are needed to detect bone fractures or joint malalignment associated with complete luxation of the joints. However, stress radiographs should be made to assess carpal integrity accurately and identify the level of the collateral ligament injury (Fig. 34.76). Integrity of the collateral ligaments is determined by obtaining craniocaudal views of the carpus while a medial and lateral stress is applied to the foot.

Laboratory Findings

No consistent laboratory abnormalities are seen.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include acute sprains, carpal luxation or subluxation, distal radial fractures, and fractures of the metacarpal bones. These conditions can be differentiated from collateral ligament injuries radiographically by use of standard and stress views.

MEDICAL MANAGEMENT

Medical or conservative management of carpal collateral ligament injuries is often unrewarding. External coaptation may be tried in younger patients, but lameness often persists as weight bearing returns.[41]

SURGICAL TREATMENT

Collateral ligament injuries involving primarily the radiocarpal joint can be treated with prosthetic ligament techniques. A pancarpal arthrodesis may be used to manage these injuries primarily or if the prosthetic ligament fails.

Preoperative Management

An external coaptation splint or bandage should be applied to protect the limb until definitive surgical treatment is undertaken. The animal's activity should be strictly limited to prevent further joint damage.

Anesthesia

Recommendations for the anesthetic management of animals with orthopedic disease are provided in Tables 32.1 and 32.2.

Surgical Anatomy

Collateral ligamentous support arises from the short radial collateral ligament medially and from the short ulnar collateral ligament laterally. In addition, sleeves of collagenous tissue that house tendons provide medial and lateral collateral support. Multiple small ligaments cross the intercarpal articulations between carpal bones to provide additional collateral and palmar support.

Positioning

The animal should be positioned in dorsal recumbency. A hanging-leg preparation allows maximum manipulation of the limb during surgery (see Chapter 5). The leg should be clipped and prepared from the humeral region to the tips of the toes.

SURGICAL TECHNIQUE

Considerable collagenous and bony tissue proliferation often occurs with collateral ligament injury. Increased vascularity, which accompanies the fibrous proliferation, can make surgical dissection difficult. Use of a tourniquet is helpful. The joint is approached medially or laterally depending on the site of injury. Sharp dissection through scar tissue and the joint capsule creates the least trauma.

Approach to the Carpal Collateral Ligaments

Make a skin incision over the medial or lateral aspect of the radiocarpal joint extending 4 cm proximal and distal to the radiocarpal joint. Incise the subcutaneous tissue, proliferative fibrous tissue, and joint capsule overlying the radiocarpal joint. The proliferative fibrous tissue will be confluent with the joint capsule proximally and distally. *Using sharp dissection, reflect the synovial joint capsule incision from the medial or lateral face of the distal radius and carpal bone. Place Gelpi retractors to maintain exposure of the joint surfaces. If the remnants of the collateral ligament can be sutured, repair the ligament with interrupted sutures or a locking loop pattern. To place a prosthetic ligament with bone tunnels, drill a small hole from dorsal to ventral through the medial or lateral styloid process and a similar hole through the radiocarpal bone or ulnar carpal bone, being careful to avoid the articular surfaces.* For lateral collateral ligament injuries, the suture may be passed between the distal ulna and the distal radius. *Thread orthopedic suture or heavy gauge monofilament nylon through the holes in a figure-eight pattern, and tie to eliminate the collateral instability (Fig. 34.77). Do not tie the suture so tight that range of motion in the joint is sacrificed. To place a prosthetic ligament with bone anchors, insert the bone anchors in the medial or lateral styloid process and the radial or ulnar carpal bone. Pass the suture through the bone anchors in a figure-eight pattern, and tie to eliminate instability.*

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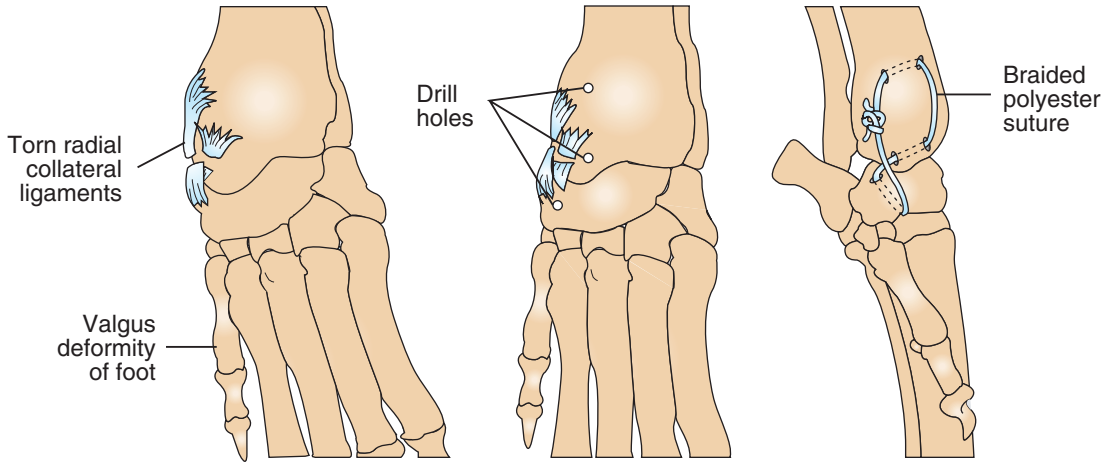


FIG. 34.77 Collateral ligament damage of the carpal joint may be treated by replacement of the ligament with a suture.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Instruments for a prosthetic ligament include a drill and orthopedic suture and bone anchors if desired. Instruments for arthrodesis include a battery- or air-driven power drill, burrs, bone curette, Steinmann pins, bone plates, and screws or external fixation.

POSTOPERATIVE CARE AND ASSESSMENT

A soft, padded bandage with a coaptation splint should be applied postoperatively to reduce swelling and support the internal fixation. An external splint should be worn for 6 to 8 weeks. Once the splint is removed, conservative physical rehabilitation should begin immediately to combat muscle atrophy and loss of range of motion (see Chapter 11). Uncontrolled activity should be strictly controlled until union has occurred.

PROGNOSIS

Prosthetic ligament repair results in excellent limb function in most patients if other injuries to the carpus are not present. Failure of fixation necessitates pancarpal arthrodesis.

CARPAL LAXITY SYNDROME

DEFINITIONS

Carpal laxity syndrome is hyperextension of the carpus in young growing dogs.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Carpal laxity syndrome is thought to be due to malnutrition, poor footing, and improper exercise in young puppies. The result is weakness and irregular tension between the extensor and flexor muscle groups and secondary carpal laxity.

DIAGNOSIS

Clinical Presentation

Signalment

Young puppies (6–8 weeks old) of any breed may be affected.



FIG. 34.78 Carpal laxity syndrome in a puppy.

Physical Examination Findings

Orthopedic examination demonstrates carpal and in some cases tarsal laxity and a palmigrade stance (Fig. 34.78). There is no significant swelling or pain of the joints.

Diagnostic Imaging

Radiographs of the joints are normal.

Laboratory Findings

No consistent laboratory abnormalities are seen.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include acute traumatic hyperextension injuries.

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MEDICAL MANAGEMENT

Medical management is composed of proper nutrition, good footing, and exercise. Splints should not be applied as this may contribute to muscle weakness.

SURGICAL TREATMENT

Surgical treatment is not indicated.

PROGNOSIS

The prognosis is excellent, with most puppies improving within 1 to 4 weeks.[44]

COXOFEMORAL JOINT

HIP DYSPLASIA

DEFINITIONS

Hip dysplasia is the abnormal development of the coxofemoral joint characterized by subluxation, or complete luxation, of the femoral head in younger patients and mild to severe DJD in older patients. **Luxation of the coxofemoral joint** is a complete separation between the femoral head and the acetabulum, whereas **subluxation** is a partial or incomplete separation. The **angle of inclination** is the angle formed between the long axis of the femoral neck and the femoral diaphysis in the frontal plane. The **angle of anteversion** is the angle formed between the long axis of the femoral neck and the transcondylar axis. The **angle of subluxation** is the angle between the femur and the pelvis at which the hip subluxates during limb abduction. The **angle of reduction** is the angle between the femur and the pelvis at which the hip reduces during limb adduction. The **angle of ventroversion** is the angle between the vertical plane and the face of the acetabular cup.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The causes of hip dysplasia are multifactorial; both hereditary and environmental factors play a part in the development of abnormal bone and soft tissue. However, hereditary factors are the primary determining factors. Rapid weight gain and growth through excessive nutritional intake may cause a disparity of development of supporting soft tissue, contributing to hip dysplasia. Factors that cause synovial inflammation (i.e., mild, repeated trauma) may also be important. Synovitis leads to an increased volume of joint fluid, which abolishes joint stability derived from the suction-like action produced by a thin layer of normal synovial fluid between the articular surfaces. These factors contribute to development of hip joint laxity and subsequent subluxation, which are responsible for early clinical signs and joint changes. Subluxation stretches the fibrous joint capsule, causing pain and lameness. Acetabular cancellous bone is easily deformed by continual dorsal subluxation of the femoral head. The piston-like action of the femoral head dynamically subluxating from the acetabulum with each step causes tilting of the acetabular articular surface from a normal horizontal plane to a more vertical plane. It also reduces the surface area of articulation, which concentrates the stress of weight bearing over a small area in the hip joint. Fractures of the acetabular trabecular cancellous bone may occur and exacerbate the pain and lameness. Physiologic responses to joint laxity (subluxation) are proliferative fibroplasia of the joint capsule and increased trabecular bone thickness. These changes relieve pain associated with capsular sprain and trabecular fractures. However, the surface area of articulation is still reduced, leading to premature wear of articular cartilage, exposure of subchondral pain fibers, and lameness.

NOTE Hip dysplasia is painful in juvenile dogs because wear of articular cartilage exposes pain fibers in subchondral bone and laxity causes stretching of soft tissue. In older dogs, hip dysplasia causes pain through osteoarthritis.

DIAGNOSIS

Clinical Presentation

Signalment

The incidence of hip dysplasia is highest in large-breed dogs. History and clinical signs vary with the patient's age. Two populations of animals are affected: juvenile patients with hip laxity and mature patients with osteoarthritis. Hip dysplasia is rare in cats.

History

Symptoms in young patients include difficulty rising after rest, exercise intolerance, and intermittent or continual lameness. As animals mature they may develop additional signs attributable to hip joint pain. Progressive DJD in these patients results in difficulty rising, exercise intolerance, lameness after exercise, atrophy of the pelvic musculature, and/or a waddling gait attributed to abnormal movement of the rear limbs. Patients are often seen for evaluation of lameness that has suddenly worsened during or after increased activity or injury.

NOTE Exercise intolerance is the most common sign of hip dysplasia.

Physical Examination Findings

Juvenile patients with lameness associated with hip dysplasia are typically first evaluated at 5 to 10 months of age. Physical findings in these patients include pain during extension, external rotation, and abduction of the hip joint, and poorly developed pelvic musculature. Hip examination performed under general anesthesia shows increased laxity of the hip joints as evidenced by abnormal angles of reduction and subluxation (Fig. 34.79). Many juvenile dogs spontaneously improve with increasing age after conservative management. This is due to elimination of subluxation by scar tissue formation around the joint.

Physical examination findings in older animals include pain during hip joint extension, reduced range of motion, and atrophy of pelvic musculature. There is generally no detectable joint laxity because of the proliferative fibrous response, but crepitus may be detected on joint manipulation. It is important to note that clinical signs do not always correlate with radiographic findings. A correct diagnosis of hip dysplasia as the cause of clinical problems is based on age, breed, history, physical findings, and radiographic changes.

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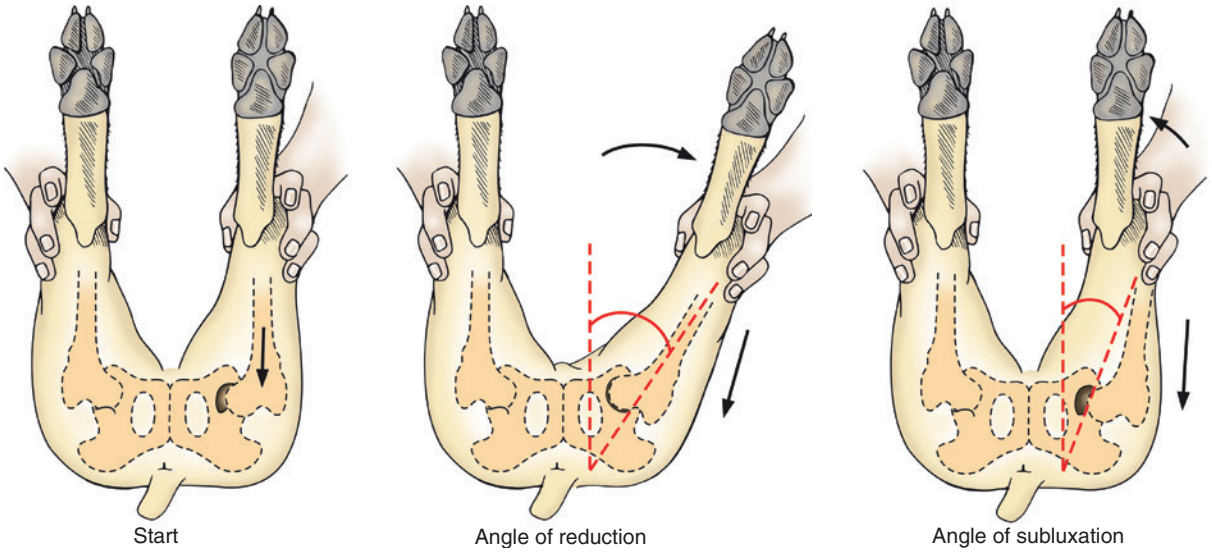


FIG. 34.79 The *angle of reduction* is the measured point where the femoral head slips back into the acetabulum when the limb is abducted. The *angle of subluxation* is the measured point where the femoral head slips out of the acetabulum when the limb is adducted.



FIG. 34.80 For standard Orthopedic Foundation for Animals–type radiographs to evaluate hip conformation, extend the hips and internally rotate the tibias until the patella lies directly over the trochlear grooves. Be sure the pelvis is straight, with symmetric obturator foramina.

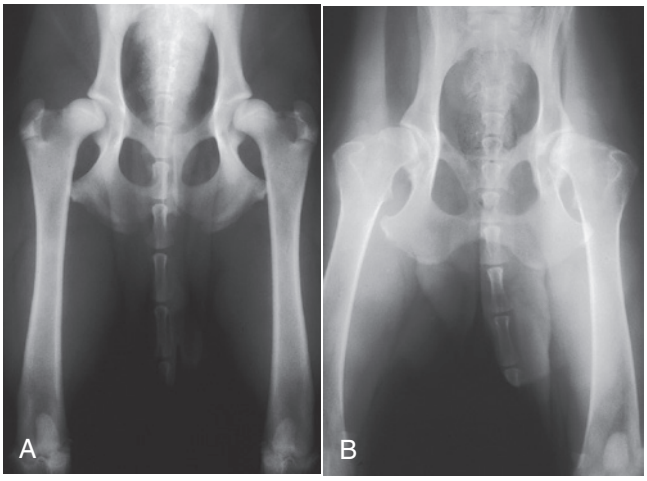


FIG. 34.81 (A) Ventrodorsal radiograph of an immature dog with subluxation of the femoral heads and minimal evidence of degenerative joint disease, typifying a candidate for triple pelvic osteotomy. (B) Ventrodorsal radiograph of a dog with advanced hip dysplasia and osteophyte formation. This dog may be a candidate for total hip replacement or femoral head ostectomy if clinical signs cannot be managed medically.

Diagnostic Imaging

The standard radiographic view for diagnosis of hip dysplasia is the ventrodorsal view of the pelvis with rear limbs extended symmetrically and rotated inward to center the patellae over the trochlear grooves (Figs. 34.80 and 34.81A). The dog must be heavily sedated or anesthetized for proper relaxation and positioning. The Orthopedic Foundation for Animals will certify a dog over 2 years of age within seven established grades for categorizing radiographic congruity between the femoral head and acetabulum. Hips determined to be "normal" radiographically may be further classified as excellent, good, fair, or near normal; animals with dysplasia are categorized as mild, moderate, or severe. Stress radiographs can be used to detect breed susceptibility to hip dysplasia as early as 4 months (Penn Hip). Stress radiographs require that the dog be under deep sedation or light anesthesia to eliminate muscle tension. Radiographic views are taken with the hips in both a neutral stance position and distracted (obtained by levering a custom-designed distractor between the legs). A distraction index is calculated from these views and is used to predict the likelihood of development of radiographic DJD secondary to hip laxity. Individual logistic regression curves that predict the risk of the development of radiographic DJD have been developed for different breeds because it appears that some breeds are more "laxity tolerant" than others. Specialty centers have been certified nationally to determine a patient's distraction index. Older dogs with established DJD do not require perfect positioning or special studies for a diagnosis of hip dysplasia (see Fig. 34.81B).

NOTE Clinical signs often do not correlate with radiographic findings. Some dogs with moderate or severe dysplasia are asymptomatic.

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FIG. 34.82 Arthroscopic view of a normal hip joint.

Arthroscopy

AP Arthroscopy of the coxofemoral joint permits direct visualization of cartilage, ligament of the femoral head, and acetabular labrum (Fig. 34.82). Assessment of arthroscopic findings may be of assistance in decision making relative to the appropriateness of a pelvic osteotomy to the treatment of juvenile hip dysplasia; although specific criteria have not been established, arthroscopic evidence of moderate or severe cartilage damage worsens the prognosis for success of a pelvic osteotomy. Thorough knowledge of hip joint anatomy is important, as the joint is deep within the musculature, providing few palpable landmarks for instrumentation of the joint. In most cases, a long 2.7-mm arthroscope is necessary to reach the joint. *Position the patient in lateral recumbency with the affected limb uppermost. Have an assistant hold the limb horizontally, and apply distal distraction of the limb to help open the joint space dorsally. Insert a large 3.5-inch (90-mm) spinal needle or catheter needle just proximal to the greater trochanter and perpendicular to the limb until it enters the joint and contacts the medial wall of the acetabulum. Aspirate joint fluid to confirm that the needle is in the joint, and then infuse saline or lactated Ringer's solution to fill the joint to moderate pressure. Remove the needle and make a 7-mm–long incision at the same location, inserting the blade deeply into the muscles, but do not enter the joint capsule. Insert the arthroscope cannula with a blunt obturator through the incision and into the joint. On a right hip, insert the spinal needle into the joint at the 5 o'clock position (on the left hip insert the needle at the 7 o'clock position) as an outflow cannula. Explore the joint completely.*

Laboratory Findings

Consistent laboratory abnormalities are not seen. Cytology of the joint fluid may indicate osteoarthritis.

DIFFERENTIAL DIAGNOSIS

A number of neurologic and orthopedic problems cause similar clinical signs. In younger dogs, lameness caused by panosteitis, osteochondrosis, physeal separation, hypertrophic osteodystrophy, and partial or complete CCL injury must be differentiated from hip dysplasia. In older patients, neurologic conditions (e.g., cauda equina) and orthopedic conditions (rupture of the CCL, polyarthritis, bone neoplasia) must be ruled out before attributing clinical signs to hip dysplasia.

MEDICAL MANAGEMENT

Treatment depends on the patient's age and degree of discomfort, physical and radiographic findings, and client's expectations and finances.[1] Conservative and surgical options are available for juvenile and mature animals with hip pain secondary to hip dysplasia. Although early surgical intervention may increase the prognosis for long-term acceptable clinical function, approximately 75% of young patients treated conservatively return to acceptable clinical function with maturity. The remaining patients require further medical or surgical management at some point in life. Surgery is indicated in older patients when conservative treatment is not effective or in young patients when athletic performance is desired or the owner wishes to slow the progression of DJD and enhance the probability of good long-term limb function.

Pain relief and clinical improvement associated with conservative treatment are derived from fibrous proliferation of the joint capsule, which strengthens the capsule and prevents further capsular sprain. Concurrently, increased thickness of the cancellous trabeculae in the subchondral bone strengthens the bony trabeculae and prevents microfractures. However, these patients are still afflicted with hip dysplasia and have a diminished surface area of hip joint articulation. Clinical signs that develop as the animal matures are attributable to articular cartilage wear and progressive osteoarthritis. Conservative treatment is divided into short- and long-term phases. Initially, these patients should be treated for an acute sprain. Complete rest is mandatory and must be enforced for 10 to 14 days.

Adjunct physical rehabilitation is helpful in maintaining range of motion and providing comfort during this period. Intensive physical rehabilitation should concentrate on strengthening periarticular structures, which can decrease lameness and discomfort (see Chapter 11). Antiinflammatory drugs are indicated to relieve pain and make administration of physical therapy more tolerable. However, antiinflammatory drugs are likely to make the patient more comfortable, which may make enforcement of rest more difficult. Clients must be advised to continue the rest period even if the patient appears to have returned to normal function.

NOTE Be sure to stress to the owners of an animal with an acute injury that they must enforce rest, even if the animal feels like exercising.

A number of NSAIDs are available over-the-counter and through prescriptions. NSAIDs most commonly used in veterinary medicine are presented in Table 34.3. Clients should be advised to avoid giving their pets NSAIDs without the recommendation

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of a veterinarian. When administering NSAIDs, begin with the lowest possible therapeutic dosage and administer with small amounts of food.

NOTE Administer nonsteroidal antiinflammatory drugs at the lowest effective dose.

Long-term conservative treatment for pain associated with DJD includes application of the five principles of medical management of osteoarthritis (see p. 1137). Weight management is the single most important aspect of these principles. The animal should be weighed weekly and its caloric intake determined. Feeding bulk diets low in fat and protein may be beneficial. Nutritional supplementation, including omega-3 fatty acids and glucosamine/chondroitin, may be of benefit in many cases. Exercise (e.g., swimming and long walks) is important to maintain an appropriate weight. High-intensity activity should be allowed only for short durations after an adequate warm-up period. Antiinflammatory drugs should be administered only as needed and should not take the place of weight control and a moderate exercise program. Physical rehabilitation may involve sit-to-stand exercises and aquatic therapy, specifically on an underwater treadmill (see Chapter 11).

NOTE Stress to owners that weight control is a critical part of managing chronic hip dysplasia. If pain associated with hip dysplasia prevents the animal from exercising normally, the owners should reduce the animal's caloric intake to prevent weight gain.

SURGICAL TREATMENT

In puppies less than 20 weeks of age, **juvenile pubic symphysiodesis** (JPS) may be performed to alter growth of the pelvis and degree of ventroversion of the acetabulum.[45] Most puppies of this age do not show clinical signs of hip dysplasia, so diagnosis depends on use of a screening technique to determine which animals may be candidates for the procedure. Although specific criteria for application of JPS have not been developed, puppies under 20 weeks of age that have palpable and radiographic evidence of laxity on a hip distracted view should be considered for the procedure. Risks of complications with the procedure are low, and failure of the procedure to reduce hip subluxation does not preclude further surgical treatment in the future.

In immature dogs, a decision must be made early to perform **pelvic osteotomy** for maximum benefits, but this decision must be balanced against the observation that many dogs diagnosed with hip dysplasia at a young age do not have clinical signs at long-term follow-up.[46] Pelvic osteotomy is useful in younger patients to axially rotate and lateralize the acetabulum in an effort to increase dorsal coverage of the femoral head. This procedure is indicated in patients leading athletic lives (e.g., working breeds) or when the client wants to arrest or slow the progress of osteoarthritis. The most favorable prognosis for pubic osteotomy is in patients with (1) radiographic evidence of hip subluxation with minimal degenerative changes, in conjunction with (see Fig. 34.81A) (2) an angle of reduction less than 30 degrees and an angle of subluxation less than 10 degrees (see Fig. 34.79), (3) a solid feeling of reduction of the femoral head into the acetabulum (see Fig. 34.79), and (4) minimal cartilage damage as visualized on hip arthroscopy (see Fig. 34.82). The angle of subluxation increases as the acetabular rim is lost to wear. In addition, the loss of the acetabular rim can be appreciated as a grating or crepitant feeling as the femoral head slides over the acetabular rim into the acetabulum. The angle of reduction is an indication of the degree of capsular laxity, which increases with increased subluxation of the hip but decreases over time as the capsule becomes thickened with fibrosis. Reduction of the head should produce a solid "clunk." An indistinct reduction indicates filling of the acetabulum. A canine pelvic osteotomy plate is the most effective method of obtaining axial rotation and acetabular lateralization. With this procedure, the amount of axial rotation is set by choosing an angled plate based on previously determined angles of reduction (maximum amount of rotation) and subluxation (minimum degree of rotation). The angle of acetabular rotation commonly used is slightly less than the measured angle of reduction, usually not exceeding 30 degrees. Twenty to 30 degrees of acetabular rotation effectively achieves the maximum increase in articular contact area of the hip. Pelvic osteotomy may be performed as a triple pelvic osteotomy (TPO) or double pelvic osteotomy (DPO). The proposed advantages of a DPO include elimination of the ischial cut, which may be a source of pain and seroma formation. The DPO requires additional screw fixation to minimize mechanical failure and a greater angle of rotation to achieve the same amount of femora head coverage.

THR is a highly advanced procedure and should be performed only by experienced surgeons trained in this technique.[47–49] THR is considered a salvage procedure in that the coxofemoral joint cannot be repaired and so the joint is removed and replaced. Most commonly, THR is performed when medical management of osteoarthritis of the hip can no longer maintain function of the limb and quality of life of the patient. Generally, the use of the five principles of medical management should be exhausted before electing THR because of the costs, risks, and complications of the surgery. Traditionally, THR is done as late in life as possible. This philosophy is based on a similar practice in human hip replacement and is based on the concept that a prosthetic joint replacement is in a constant state of degeneration and placement as late in life as possible diminishes the potential need to revise or replace the original hip prosthesis. Some owners and surgeons may elect hip replacement instead of medical management for patients with significant complications from NSAIDs or for clients who are extremely concerned with potential complications from NSAIDs. The advent of cementless THRs has also altered the philosophy of when THR is indicated. Because cementless prostheses are much less inclined to loosen with time, their use in younger patients is more common and acceptable. At a minimum, THR should be delayed until body weight reduction is attempted, particularly in obese patients. Correction of body weight may eliminate or delay the need for THR and will decrease the risks of complications when THR is performed.

Contraindications to THR include septic arthritis and significant or progressive neurologic disease. Sepsis of the coxofemoral joint, although rare, is an absolute contraindication to cemented THR. Cementless THRs have been used for the revision of septic loosening of cemented THRs.

Implant Designs

Canine THRs are broadly divided into cemented and cementless designs. Cemented THRs (Fig. 34.83A) have been used significantly

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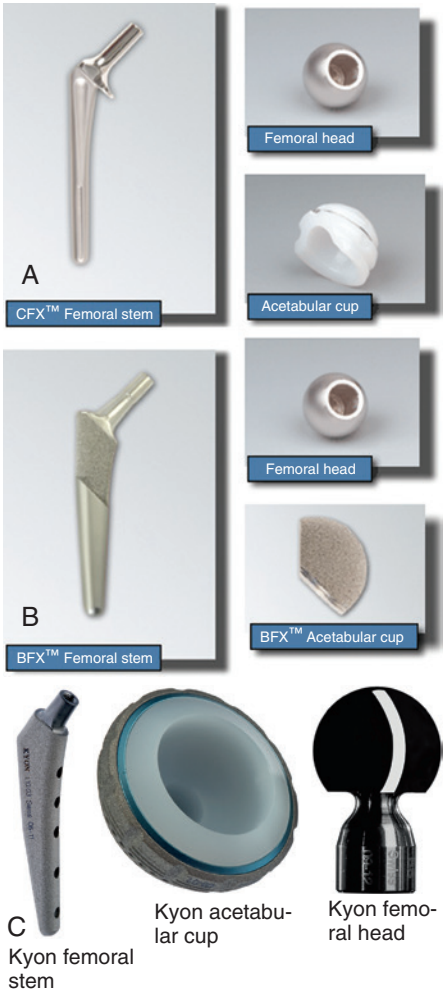


FIG. 34.83 (A) Cemented canine total hip replacement (THR) implants. (B) Cementless canine THR implants. (C) Kyon THR. (A–B, Courtesy BioMedtrix LLC. C, Courtesy Kyon Pharma, Inc.)

longer in dogs than cementless designs. The cement serves as a mortar between the implant and the bone. Medical grade bone cement is polymethylmethacrylate and bonds as a cohesive versus an adhesive (i.e., it locks into irregularities in the implant and bone versus forming a chemical bond) (Fig. 34.84A). Most contemporary cemented implants are modular, meaning that they have interchangeable parts, allowing the surgeon to adjust stem size, neck length, head diameter, and cup size to fit the individual patient. Generally, the preparation of the bone (acetabulum and femur) is simpler with a cemented THR than a cementless THR. The use of cement, however, requires strict attention to asepsis, handling characteristics, and implant positioning. Cemented THRs are generally strongest within 2 days of implantation, but then may weaken and loosen over time because the cement cannot adapt to changes in the bone or alteration of loads.

Cementless THRs are either press fit–ingrowth (see Figs. 34.83B and 34.84B) or stabilized by monocortical screws (see Fig. 34.83C). All canine cementless acetabular cups are press fit–ingrowth, meaning that initial stabilization is achieved by impacting the cup into an acetabular bed that has been prepared to have a smaller diameter than the cup, resulting in very high friction between the cup and bone after it has been forced (impacted) into the bone bed. Long-term stabilization is achieved by bone growth into the shell of the cup. Most canine THR cups include a metallic shell and a polyethylene liner. The shell is designed to encourage bone ingrowth that locks the implant in place.

The femoral component of cementless THRs is initially stabilized either by press fit or monocortical screws into the medial femoral cortex. Selection between the different designs is based primarily on surgeon preference and experience. In either case, long-term stabilization is achieved by bony ingrowth into the implant or around the screws.

Development of cementless implants was based primarily on concern regarding aseptic loosening in cemented implants. For surgeons using both cemented and cementless varieties, age often plays a major role in the decision-making process. Older patients often receive cemented implants, as their limited life expectancy and lower level of activity suggest that the cemented implant will outlast the patients' lifetime. Older patients may also have slower rates of bone ingrowth and a higher risk of fracture with cementless implants. Younger patients are more appropriate for cementless implants because their higher bone metabolism may result in more rapid and secure bone ingrowth, whereas their longer life expectancy and higher activity levels may lead to aseptic loosening of cemented THRs. Hybrid THR (e.g., cementless acetabular cup and cemented femoral stem) can also be chosen based on surgeon preference and experience.[47]

In general, bone preparation for cemented THRs is less exacting than that for press fit cementless THRs. Because the cement serves as a mortar between the implant and the bone, the acetabulum and femoral canal do not need to be prepared as precisely, whereas with a press fit implant, the implant must fit precisely to prevent implant migration during the initial period before bone ingrowth. Cementless femoral components relying on screw fixation also do not require as exacting preparation of the femoral canal as do press fit cementless designs.

Cement preparation requires thorough understanding of the chemistry and mechanics of bone cement. Issues of temperature, hardening rates, and handling characteristics are critical to successful cemented THRs. The complexities of polymethylmethacrylate and the inability to modify the cement after hardening are other reasons for the increased popularity of cementless implants.

Femoral head and neck excision (**FHO**) limits bony contact between the femoral head and acetabulum and allows formation of a fibrous false joint (Fig. 34.85). This procedure can be used when conservative treatment has failed and the patient is in severe pain, and when financial, medical, or size constraints preclude alternative methods of surgical intervention. Caution should be used in treating juvenile animals with this procedure because a significant percentage of them improve with maturity. Because fibrous pseudoarthrosis is an unstable joint, clinical function postoperatively is unpredictable.[50] For this reason, most surgeons consider femoral head and neck excision to be a salvage procedure. However, many patients with painful arthritic hips undergoing femoral head and neck excision have improved limb function after FHO. Although smaller patients routinely have better results than larger patients, this procedure may yet be indicated in large- and giant-breed dogs in which other treatments have been ineffective or are not feasible.

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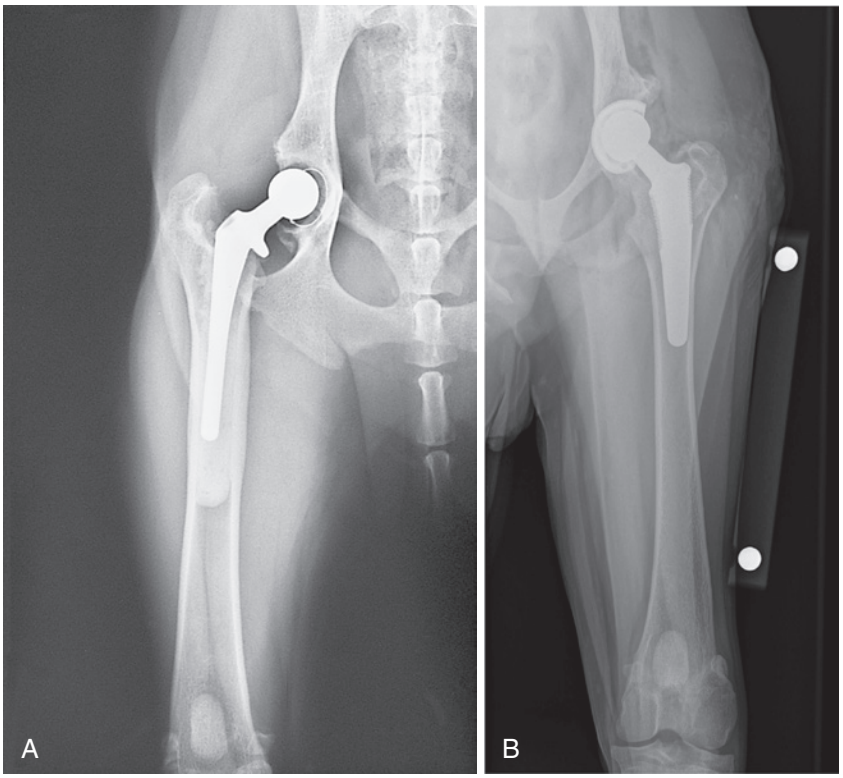


FIG. 34.84 (A) Radiograph of a dog after cemented total hip replacement (THR). Note the radiopaque cement mantle surrounding the femoral and acetabular prostheses. (B) Radiograph of a dog after cementless THR.

Preoperative Management

A complete orthopedic and neurologic examination should be performed to correctly attribute the clinical problem to hip dysplasia. Perioperative systemic antibiotics should be administered when pelvic osteotomy or THR is performed.

Anesthesia

Dogs treated with pubic symphysiodesis or pelvic osteotomy are generally young and healthy and can be anesthetized with a variety of protocols. Older dogs treated with THR or femoral head and neck excision should be carefully evaluated and the anesthetic protocol altered if necessary. Epidural administration of analgesics (see Chapter 13 and Table 32.3) preoperatively is useful for lowering the anesthetic dose requirement and for reducing postoperative discomfort.

Surgical Anatomy

Special anatomic considerations for pubic symphysiodesis include identification of the pubic symphysis and protection of the urethra and colon. Special anatomic considerations for pelvic osteotomy include identifying the pectineus muscle as a landmark for the ventral approach to the pubis and avoiding the obturator nerve at the caudal limit of the pubic osteotomy. Surgical anatomy encountered during the approach to the iliac body is described on p. 1097. It is important to protect the sciatic nerve ventral to the iliac body during the osteotomy. Special anatomic considerations for FHO are important in patients with hip dysplasia. In patients with atrophied hip musculature, the joint capsule is obvious as soon as the deep gluteal muscle is reflected. If subluxation of the hip joint is moderate or severe, the joint capsule usually appears thickened and bulges outward. In mature patients, thickening of the joint capsule is even more pronounced. To obtain adequate exposure of the femoral head and neck, the vastus lateralis muscle must be released and reflected ventrally. The femoral head and neck are often short and deformed. To perform an FHO in these patients, the juncture of the femoral neck and femoral shaft must be clearly visualized. In young patients, the round ligament may be intact and must be severed; the ligament is usually absent in older patients.

NOTE Before performing pelvic osteotomy, review the relationship between the gluteal musculature and iliacus muscle, the position of the sciatic nerve, and the course of the internal pudendal artery and nerve.

Positioning

The patient is positioned in dorsal recumbency for pubic symphysiodesis. The patient is positioned in lateral recumbency for hip arthroscopy or pelvic osteotomy. Clip and prepare the leg for aseptic surgery from the midline to the tarsal joint on both medial and lateral limb surfaces. For femoral head and

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FIG. 34.85 Radiograph of a dog after femoral head and neck ostectomy. Note the complete removal of the femoral neck.

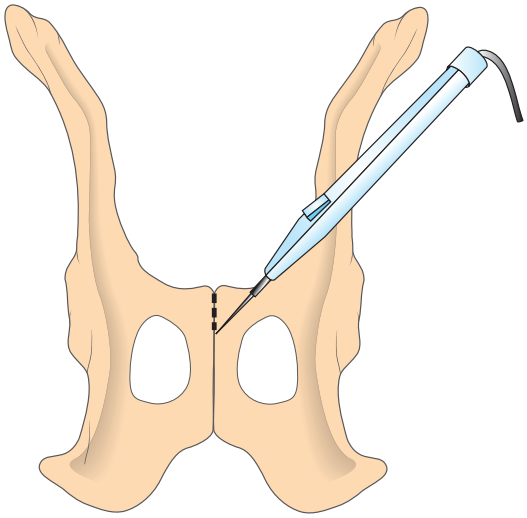


FIG. 34.86 Expose the pubic symphysis with a ventral midline incision over the pubis. Using a spatula electrode set at 40 W, perform ablation by placing the electrode against the symphysis for approximately 10 seconds, repeating every 2 to 3 mm along the symphysis.

neck excision, the patient is positioned in lateral recumbency and the area from the dorsal midline to the stifle is clipped and prepared for aseptic surgery. The patient should be draped in a manner that allows manipulation of the limb during surgery (see Chapter 5).

SURGICAL TECHNIQUE

Juvenile Pubic Symphysiodesis

In many cases, the patient is spayed or neutered at the same time. The surgical incision should be adjusted as necessary to combine the two procedures. For female dogs, spay them first and then extend the incision over the pubis and perform JPS before closing the abdomen. This enables palpation in the pelvis. In male dogs, perform JPS first as it is a cleaner surgery than the neuter. *With the patient in dorsal recumbency, make a ventral midline incision over the pubic symphysis. In male dogs, make the skin incision parallel to the penis, extending from the scrotum to 3 cm cranial to the pubis. Incise subcutaneous fascia, and ligate any collateral branches of the pudendal artery and vein. Retract the penis past midline. Expose the pubic symphysis by incision of the deep fascia followed by subperiosteal elevation of the adductor and gracilis muscles. Introduce a thin ribbon malleable retractor or finger into the pelvic canal to protect the rectum and urethra from thermal injury (Fig. 34.86). Using a spatula electrode set at 40 W, perform ablation by placing the electrode against the symphysis for approximately 10 seconds, repeating every 2 to 3 mm along the symphysis. Perform the ablation primarily on the cranial half of the symphysis.* Alternatively, a needle electrode or metal needle may be inserted into the symphysis every 2 to 3 mm using 40 W for up to 30 seconds. *Perform routine closure in layers starting with reapposition of the adductor and gracilis muscles across the pubic symphysis.*

Pelvic Osteotomy

DPO requires that an incision be made through the pubic brim and iliac body. A TPO requires an additional incision be made through the ischial floor. The optimum position for pubic osteotomy is adjacent to the medial wall of the acetabulum. *With the patient in lateral recumbency, abduct the leg while keeping the femur perpendicular to the acetabulum. Locate the origin of the pectineus muscle, and center a 6-cm skin incision over this point. Incise the subcutaneous tissue to further isolate the origin of the pectineus muscle at the iliopectineal eminence. Release the origin of the pectineus muscle to expose the cranial brim of the pubis.* The pectineus muscle may be allowed to retract or may be resected. In some cases, it may be possible to perform the osteotomy without release of the pectineus muscle. *Reflect the periosteum from the cranial, lateral, and caudal pubic surfaces. To protect soft tissue during the osteotomy, place spoon Hohmann retractors cranial to the pubis and within the obturator foramen caudally. Ostectomize a portion of the pubis adjacent to the medial wall of the acetabulum with an osteotome or rongeurs (Fig. 34.87A). Suture soft tissue and skin using standard methods.*

For a TPO, perform an osteotomy of the ischial floor next. For a DPO, perform an iliac osteotomy instead. Make a skin incision midway between the medial prominence of the ischium and the lateral tuberosity. Make the incision in the vertical plane, beginning 4 cm proximal to the ischial floor and extending 3 cm distally. Incise the subcutaneous tissue and deep fascia. Make a 3-cm incision through the periosteal insertion of the internal obturator muscle at the dorsal crest of the ischial floor. Elevate the internal obturator muscle cranially to the obturator foramen. Then incise the periosteal origin of the external obturator muscle at the ventral crest of the ischial floor and reflect the muscle from the ventral surface of the ischium cranially to the obturator foramen. Place two spoon Hohmann retractors to protect the soft tissue; insert one into the

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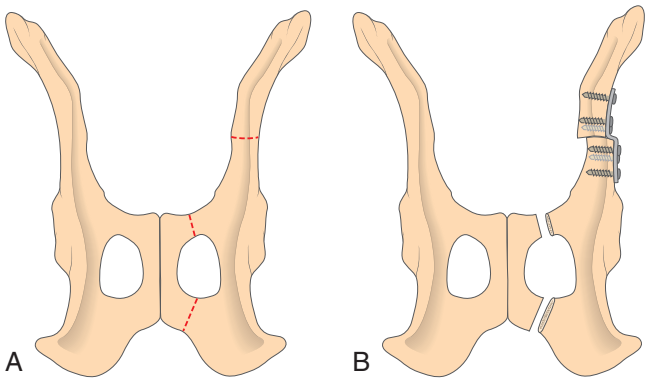


FIG. 34.87 Position of osteotomies for completion of (A) a triple pelvic osteotomy and (B) stabilization with a bone plate. Note the axial rotation and lateralization of the hemipelvis.

obturator foramen dorsally and one into the foramen ventrally. Direct an osteotome caudal to cranial in line with the center of the Hohmann retractors; this centers the osteotomy line into the obturator foramen. As an alternative, use a reciprocating saw or Gigli wire to perform the osteotomy. Close the incision after the osteotomy of the ilium is completed. At that time, if desired, drill two small holes on either side of the osteotomy adjacent to each other. Place orthopedic wire through the holes and twist them in a figure-eight fashion to stabilize the osteotomy. Suture the fascia of the internal obturator muscle to that of the external obturator muscle, then close the subcutaneous tissue and skin using standard methods.

Perform an osteotomy of the ilium to allow axial rotation of the acetabulum (see Fig. 34.87A). Make an incision from the cranial extent of the iliac crest caudally, 1 to 2 cm beyond the greater trochanter. Center the incision over the ventral third of the iliac wing. Incise the subcutaneous tissues and gluteal fat along the same line to visualize the intermuscular septum between the superficial gluteal muscle and the short part of the tensor fasciae latae muscle. Incise the muscular septum to separate the tensor fasciae latae muscle and middle gluteal muscle cranially and the tensor fasciae latae and superficial gluteal muscles caudally. Cranially, use sharp dissection to separate the middle gluteal muscle and long head of the tensor fasciae latae muscle. Palpate the ventral border of the ilium and make an incision to the bone near the ventral insertion of the middle and deep gluteal muscles. Isolate and ligate the iliolumbar vessels and reflect the deep gluteal muscle from the lateral surface of the ilium. Incise the origin of the iliacus muscle at the ventral border of the ilium and reflect the muscle from the ventral surface. Elevate the periosteum from the medial surface of the ilium with a periosteal elevator. Place two spoon Hohmann retractors to protect soft tissue during the osteotomy; place one medial to the ilium to reflect the iliacus muscle and one over the dorsal crest of the ilium to retract the gluteal muscle mass. Judge the cranial position of the osteotomy by placing the osteotomy plate such that the most caudal plate hole is just cranial to the acetabulum. Perform the iliac osteotomy with an oscillating saw tilted 20% cranially to a line perpendicular to the long axis of the hemipelvis. Lateralize the caudal segment with bone-holding forceps, and secure an appropriate osteotomy plate to this segment.

NOTE If the acetabular segment is difficult to rotate with the double osteotomy technique, release of the insertion of the sacrotuberous ligament aids in rotation.

Next, reduce the osteotomy and apply plate screws in the cranial segment (see Fig. 34.87B). If the cranial plate screws penetrate the sacrum, they should penetrate well within the sacrum to prevent premature screw loosening. *Remove the sharp spike of ilium dorsal to the plate, fragment it, and use it as bone graft at the osteotomy site. To close the incision, place sutures between the fascia of the middle gluteal muscle and that of the tensor fasciae latae muscle cranially and between the superficial gluteal muscle and the tensor fasciae latae caudally. Approximate the gluteal fascia and tensor fasciae latae, subcutaneous tissue, and skin, using standard methods.*

NOTE Double and triple pelvic osteotomy plates differ in angle and screw orientation. Double pelvic osteotomy plates have 5 degrees more angulation to achieve a similar degree of acetabular ventroversion. Additionally, double pelvic osteotomy plates have divergent screw holes to increase stiffness.

Femoral Head and Neck Ostectomy

Make a craniolateral approach to the hip joint and luxate the hip (Fig. 34.88A–B). If the round ligament is intact, incise it. Incising the round ligament is facilitated by placing lateral traction on the greater trochanter with bone-holding forceps and subluxating the femoral head. This allows curved scissors to be placed into the joint to cut the ligament. *Perform the ostectomy by externally rotating the limb to where the joint line of the stifle is parallel to the operating table. Identify the line of ostectomy perpendicular to the operating table at the junction of the femoral neck and the femoral metaphysis (Fig. 34.88C). To ensure the accuracy of the bony cut, predrill a series of three or more holes along the line of the ostectomy. Use an osteotome and mallet to complete the cut.* Ventral reflection of the vastus lateralis muscle facilitates proper placement of an osteotome or saw blade during this procedure. *Once the femoral neck and head have been removed, palpate the cut surface of the femoral neck for irregularities.* The most common finding is a shelf of femoral neck left on the caudal surface of the femur. *Remove any edges with rongeurs. Suture the joint capsule over the acetabulum if possible. To close, suture the vastus lateralis and deep gluteal muscles, tensor fasciae latae, subcutaneous tissue, and skin using standard methods.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

A 2.7-mm–long arthroscope is necessary for hip arthroscopy. An electrocautery unit is necessary for JPS. An oscillating saw, reciprocating saw, or osteotome and mallet, self-retaining retractors, Hohmann retractors, and instrumentation for insertion of a bone plate and screws are necessary for pelvic osteotomy. FHO requires an osteotome and mallet or oscillating saw.

POSTOPERATIVE CARE AND ASSESSMENT

No restrictions are necessary after diagnostic hip arthroscopy. After prepubic symphysiodesis, the puppy should be restricted until skin sutures are removed. After that time, no special postoperative care is required. Determination of the efficacy of the procedure may be made by follow-up radiographs at skeletal maturity; however, accurate determination of the effectiveness of the procedure in inducing ventroversion requires CT. Complications are rare and include damage to the urethra or colon or failure to achieve symphysiodesis.

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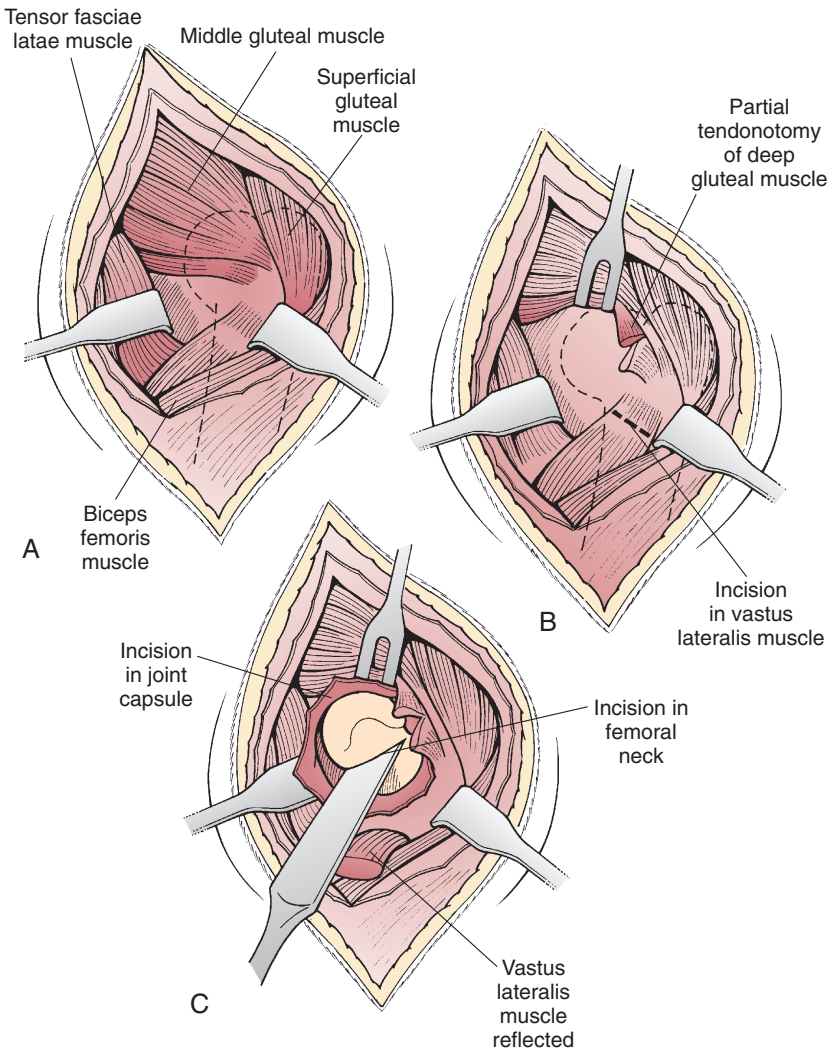


FIG. 34.88 To expose the femoral head for ostectomy, make a craniolateral skin incision centered over the hip joint. (A) Retract the biceps femoris muscle caudally and the tensor fasciae latae muscle cranially. (B) Incise the vastus lateralis muscle and reflect it ventrally. (C) Incise the joint capsule and perform the ostectomy by externally rotating the limb such that the joint line of the stifle is parallel to the operating table. Identify the line of ostectomy perpendicular to the operating table at the junction of the femoral neck and femoral metaphysis.

After pelvic osteotomy, the patient should be restricted to leash exercise until radiographic evidence of healing of the osteotomies is complete, generally in 6 weeks. The duration of exercise (leash walks) should be increased gradually according to the patient's tolerance. If the contralateral side is to have surgical treatment, the second surgery should be performed when discomfort associated with the first surgery is well tolerated by the dog. Reported complications include implant failure, loss of limb abduction, and pelvic outlet narrowing.

After THR, strict exercise restriction should be maintained for 8 to 12 weeks, followed by gradual return to normal activity over 4 to 8 weeks (Table 34.8).

Early active use of the limb is beneficial after FHO. A recommended physical therapy protocol for FHO patients is provided in Table 34.9. Good return of active limb function depends on the length of time the pathologic hip joint condition was present and on the severity of the degenerative changes. Patients with chronic disease (muscle atrophy and proliferative DJD) are slower to return to function than patients with acute lameness.

COMPLICATIONS

Complications of canine THR vary with the type of implant placed. Complications that are common to all designs include luxation, fracture, infection, and aseptic loosening. The incidence of luxation after canine THR is approximately 5%.[47–49] Luxations may be due to intrinsic laxity of the hip, preexisting luxation, incorrect implant selection or positioning, or trauma. Treatments for luxation include closed reduction and sling application, open reduction with surgical stabilization (see section on hip luxation), implant modification (alteration of cup position, neck lengthening), or implant removal.

Infection is a more significant problem with cemented THRs than cementless THRs; cement appears to be more easily colonized

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TABLE 34.8 Sample Rehabilitation Protocol for Patient Following Total Hip Replacement

Treatments/ Modalities	Day 1–14	2–12 WK	12–16 WK	16+ WK
Pain medications	As directed	As directed	PRN	PRN
Cryotherapy	15–20 min 3 times daily for first 72 hours then as needed after exercise First session immediately after surgery	Use after exercise for 15–20 minutes as needed	15–20 min PRN to hip after exercise	15–20 min PRN to hip after exercise
Massage	5 min 3 times daily gently, avoiding incision—hip, thigh, and lumbosacral area	PRN	PRN	PRN
PROM	8–10 reps flexion and extension up to 4 times daily of stifle and tarsus ONLY of affected limb Avoid abduction or adduction of hip; therapist can do gentle PROM in sagittal plane only	8–10 reps flexion and extension with bicycling twice daily before activities Avoid abduction or adduction of hip	Range of motion should be near normal at this point	
Laser therapy	Daily	PRN	PRN	PRN
Walks	5 min supported leash walk 2–3 times daily; outside for toileting only	Slow leash walking with sling support 3 times daily Work up to 10 minutes per walk	Slow leash walk 15–20 min twice daily By week 10 can incorporate 3 min of light trotting on flat surface	15–20 min twice daily; incorporate zigzag hill walking across hills Can increase minutes of slow trotting on flat surface
NMES	Quadriceps and hamstring muscles 15 min daily beginning at day 7	Quadriceps and hamstring muscles 15 min daily until week 5 then every other day if needed		
Balancing	At day 5 begin with assisted sling on soft foam pad 2–3 min twice daily	5 min daily with sling support as needed	5 min daily with sling support as needed	Balance and weight shifting exercise; may need leg weights for atrophy
Cavalettis (begin 4–6 wk low then work up to higher rails at 8 wk)		Low: 8–10 reps BID, until 8 weeks, then 8–10 reps BID higher rails	10–15 reps twice daily	5 min twice daily
Sit to stand (begin 8–10 wk postoperatively)		8–10 reps twice daily elevated hindquarters	10–15 reps twice daily	15–20 reps per session
Crawling			5–10 reps per session twice daily	5–10 reps per session twice daily
Underwater treadmill (begin 3–4 wk postoperatively)		Daily for 3–5 min working up to 10–15 minutes	15–20 min 3–4 times weekly	15–20 min 2–4 times weekly variable speed
Swimming			5–10 min daily working up to 20 min with breaks	20–30 min once daily with breaks

Weeks 2–12, do only one to two exercises per session, done by trained therapist.
Strict exercise restriction, other than rehabilitation by trained therapist, should be maintained for 8–12 wk following this procedure. Week 12–16 do only two to three exercises per session. Doing all exercises may result in pain and lameness. Week 16 and beyond, gradually return to normal activity but avoid jumping and sharp turns until full function has returned.
BID, Twice daily; *NMES,* neuromuscular electrical stimulation; *PRN,* as needed; *PROM,* passive range of motion.

by bacteria than metallic implants. Most surgeons mix antibiotic powder with the bone cement; however, this does not prevent infection in a small percentage of cases. Treatment of septic loosening in cemented THRs invariably requires removal of the implants and cement to resolve the infection; however, some cases have been treated by replacement of the infected cemented prosthesis with a cementless design. The incidence and specific treatment of infection of cementless THRs have not been reported.[47–49]

Aseptic loosening of cemented THRs may occur on the acetabular or femoral side or both. The cause for aseptic loosening of cemented implants may be either biomechanical or cytologic. Biomechanical causes occur when bone remodeling leads to loosening of the cement-bone interface or when cement cracking initiates loosening between the cement and the bone. Cytologic causes are usually initiated by wear particles of polyethylene, metal, or rarely, cement, which cause macrophage migration and upregulation of bone resorption. Aseptic loosening of cemented THRs usually occurs years after implantation, but rarely can occur within months of implantation.

Aseptic loosening of cementless implants most commonly occurs during the press fit stage of stabilization. Acetabular

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TABLE 34.9 Sample Rehabilitation Protocol for Patient Post–Femoral Head and Neck Osteotomy

Treatments/ Modalities	Day 1–7 Toe Touching	Day 7–21 Early Weight Bearing	3–4 WK	5–8 WK	8+ WK
Pain medications	As directed	As directed	PRN	PRN	PRN
Cryotherapy	15–20 min 3 times daily before walks or exercises First session immediately after surgery	Use after exercise for 15–20 min up to 3 times daily	PRN	PRN	PRN
Heat therapy		Apply heat to the thigh and hip muscles before exercise	PRN	PRN	PRN
Massage	Gentle massage around the surgical site, lumbosacral area and thigh	Continue twice daily	Once to twice daily	PRN	PRN
PROM	10 repetitions 3–4 times daily, focus on extension of hip	PROM and hip stretches (10–20 reps) 3 times daily Evoke flexor reflex and bicycle affected leg (2–5 min)	Continue 15–20 reps flexion and extension of all joints of affected leg once to twice daily	PRN	PRN
Laser therapy	Daily	Every other day for 1 week then twice weekly	Twice weekly	PRN	PRN
Walks	5-min supported leash walk 2–3 times daily	Increase each walk by 2–3 min each week	Increase by 5 min each week	Increase by 5 min each week	15- to 20-min walks 2–3 times daily
NMES	10 min twice daily	10 min twice daily	Discontinue if dog is doing well		
Balancing		5 min twice daily on soft pad	5 min 2–3 times daily, can use egg ball or disc	One-leg standing up to 5 min 2 times daily or balancing on inflatable ball	10 min balancing on inflatable ball
Cavalettis/ obstacles/sit to stand			5 min twice daily for cavalettis Sit to stand do 10–15 reps BID	5 min twice daily for cavalettis Sit to stand do 15–20 reps BID	5 min twice daily for cavalettis Sit to stand do 15–20 reps BID
Jogging/Stairs			1 flight stairs once daily, 3 min light jogging	2–4 flights of stairs 1–2 times daily, 3–5 min light jogging on flat surface	2–5 flights of stairs 1–2 times daily, 3–5 min light jogging on flat surface
Hills				Zigzag low slow hills 5 min up and down	Increase to 10 min twice daily
Underwater treadmill		Start after day 14: 5–10 min total daily	10 min daily or every other day	15–30 min twice weekly	15–30 min twice weekly until released from rehab
Swimming			2–3 days per week	2–5 days per week	As desired as part of home exercise program

BID, Twice daily; *NMES,* neuromuscular electrical stimulation; *PRN,* as needed; *PROM,* passive range of motion.

implants may rotate or avulse out of the bony bed. Femoral press fit components may rotate or subside and may lead to severe fractures of the femoral diaphysis. Femoral screw fixation components may avulse from the medial wall, or fractures may occur through the screw holes or access holes. The incidence of delayed loosening of cementless implants has not been reported, but appears to be low.

Results after FHO vary. The prognosis is highly dependent on the patient's size and on postoperative physical therapy. In large patients, approximately 50% of animals have good function.[50] The remaining animals have varying degrees of lameness, but function is usually improved compared with the preoperative status. Medium and small patients usually have good limb function.

PROGNOSIS

Prognosis after JPS is determined by the age at treatment and severity of the dysplasia.[45] Dogs treated after 20 weeks of age have a poor prognosis for increased ventroversion. The degree of ventroversion in dogs treated before 20 weeks is variable and somewhat unpredictable.

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Prognosis after pelvic osteotomy is largely determined by case selection.[46] Best results are obtained in patients with acceptable physical findings (see the previous discussion) and few or no degenerative changes. Long-term function is good to excellent. Although degenerative changes are radiographically evident after this procedure, they are less than what would be expected without surgery.

THR results in excellent return to normal function unless complications occur. Reported rates of successful outcomes of THRs vary from 75% to 95%.[47–49] In the majority of cases, the outcome of THR is either complete resolution of lameness or failure of the technique, resulting in implant explantation. In a small percentage of cases, there may be chronic lameness following THR.

COXOFEMORAL LUXATION

DEFINITION

Coxofemoral or **hip luxation** is a traumatic displacement of the femoral head from the acetabulum.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Coxofemoral luxation typically results in craniodorsal displacement of the femoral head relative to the acetabulum. Most affected animals have sustained trauma, such as motor vehicle accidents. Ventrocaudal displacements, in which the femoral head may lodge within the obturator foramen, occur less often. This type of luxation may be associated with fracture of the greater trochanter. Spontaneous luxation secondary to hip dysplasia carries a poor prognosis. The amount of soft tissue damage surrounding the hip joint depends on the trauma incurred. The round ligament of the femoral head always fails completely; it may be an interstitial rupture or an avulsion of the ligament from the fovea capitis. The fibrous joint capsule must also be completely torn to permit dislocation of the femoral head. The tear in the joint capsule may be a small rent through which the femoral head is dislodged, or complete fraying of the entire capsule may occur.

Hip luxation should be treated as quickly as possible to prevent continued damage of the soft tissue surrounding the hip joint and degeneration of articular cartilage. Articular cartilage derives its nutrients from synovial fluid, which is pumped into the matrix during normal articular movement. Early reduction allows rapid return of the nutrient source of the articular cartilage. Because coxofemoral luxations are usually associated with trauma, as many as half of these patients have a major injury in addition to the hip luxation. A careful physical examination should be performed before induction of anesthesia and treatment of the luxated hip to identify concurrent trauma.

NOTE Early reduction of luxated hips is essential. Do not delay in treating these animals.

DIAGNOSIS

Clinical Presentation

Signalment

Any age or breed and either gender of dog or cat may be affected.

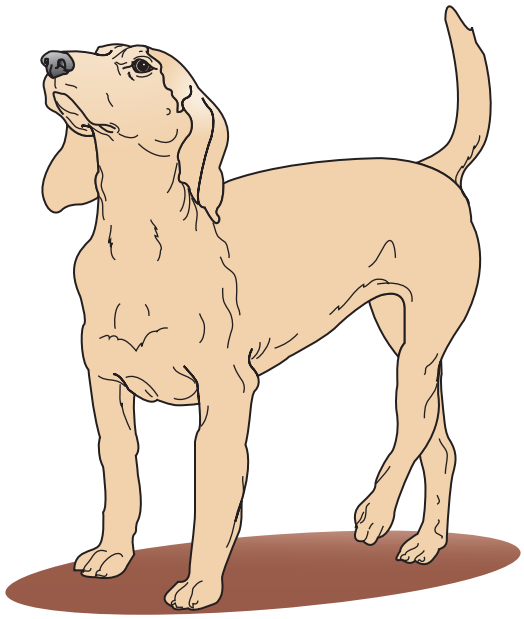


FIG. 34.89 Typical carriage of the limb in a patient with a craniodorsal coxofemoral luxation. Note the position of the paw beneath the body and the external rotation of the stifle.

History

Affected animals usually show a unilateral non–weight-bearing lameness. The traumatic episode may or may not be witnessed by the owner.

Physical Examination Findings

Animals with hip luxation are usually presented for evaluation of a non–weight-bearing lameness associated with trauma. When the femur is displaced craniodorsally, the limb is carried adducted, with the stifle externally rotated (Fig. 34.89). When it is displaced caudoventrally, the limb is carried abducted, with the stifle internally rotated. Manipulation of the limb causes crepitus or pain. A palpable lack of symmetry is noted between the tuber ischii and greater trochanter on the affected side compared with the normal limb. With craniodorsal displacement, the greater trochanter is dorsal to an imaginary line drawn from the crest of the ilium to the tuber ischii, and the distance between the tuber ischii and greater trochanter is greater than in the normal limb (Fig. 34.90). With a ventrocaudal luxation, the greater trochanter is displaced ventrally, and the space between the tuber ischii and greater trochanter may be narrowed. Hip luxation also causes limb length discrepancy. Craniodorsal luxations cause the affected limb to be shorter than the normal limb, whereas the opposite is true with ventral luxations.

Diagnostic Imaging

Diagnosis of hip luxation should be confirmed with ventrodorsal and lateral radiographs (Fig. 34.91). Before a treatment method is chosen, radiographs should be carefully evaluated for evidence of avulsion of the fovea capitis, associated hip joint fractures, and degenerative changes secondary to hip dysplasia.

Laboratory Findings

Consistent laboratory abnormalities are not seen.

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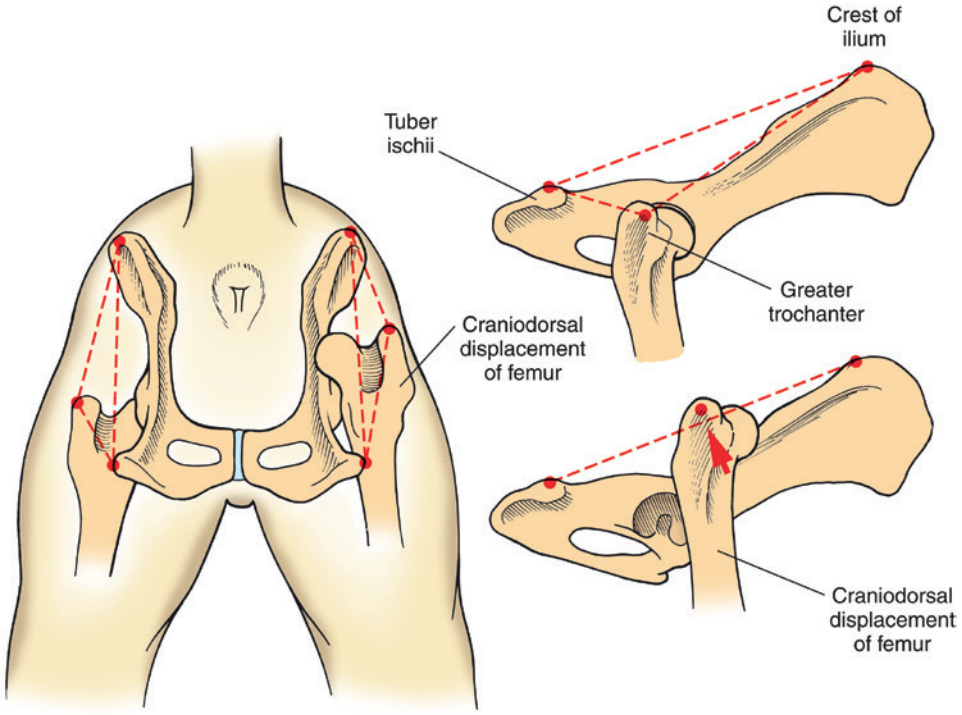


FIG. 34.90 With craniodorsal displacement of the femur, the greater trochanter is dorsal to an imaginary line drawn from the crest of the ilium to the tuber ischii, and the distance between the tuber ischii and the greater trochanter is greater than in the normal limb.



FIG. 34.91 Ventrodorsal radiograph of an animal with a craniodorsal coxofemoral luxation.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include acute subluxation or luxation of the hip joint secondary to hip dysplasia, femoral capital physeal fracture, femoral neck fracture, and acetabular fracture.

MEDICAL MANAGEMENT

Coxofemoral luxation may be managed with closed manipulation to replace the femoral head within the acetabulum or with open surgical manipulation. Closed reduction should be attempted before open reduction in most animals, unless there is radiographic evidence of hip dysplasia or a fracture. The animal should be anesthetized for a closed reduction.

Closed Reduction of a Craniodorsal Luxation

Place the patient in lateral recumbency under general anesthesia. Place a rope in the groin of the affected limb, over the back, and secure it to the table to provide resistance. Grasp the affected limb with one hand near the tarsal joint and place the other hand on the greater trochanter to direct the proximal femur (Fig. 34.92A). Externally rotate the limb, and pull it distally to position the femoral head over the acetabulum (Fig. 34.92B). When the femoral head lies lateral to the acetabulum, internally rotate the limb to seat the femoral head within the acetabulum (Fig. 34.92C). Apply medial pressure to the greater trochanter while flexing and extending the joint to help expel debris from the acetabular cup. This maneuver is critical to maintaining reduction and should be performed for 10 to 15 minutes. *Place the limb in an Ehmer sling (Fig. 34.93).* If the hip is very stable, or if the animal's conformation or multiple limb trauma prohibits the use of a sling, cage confinement may be adequate. *Limit the animal to cage confinement and controlled activity on a leash until the bandage is removed in 7 to 10 days. After removal of the bandage, limit activity to controlled leash walking for an additional 2 weeks.*

Ehmer Slings

Ehmer slings prevent weight bearing of the pelvic limb (see Fig. 34.93). The most common use for Ehmer slings is to support the closed or open reduction of hip luxations. *Place a thin layer of cast padding around the metatarsal area and wrap nonadherent gauze (e.g., Kling) multiple times over the padding. Maximally flex the stifle and wrap the gauze around the thigh by bringing it medially between the body wall and limb. Pull the gauze firmly and bring it over the front of the thigh to maintain flexion. Then*

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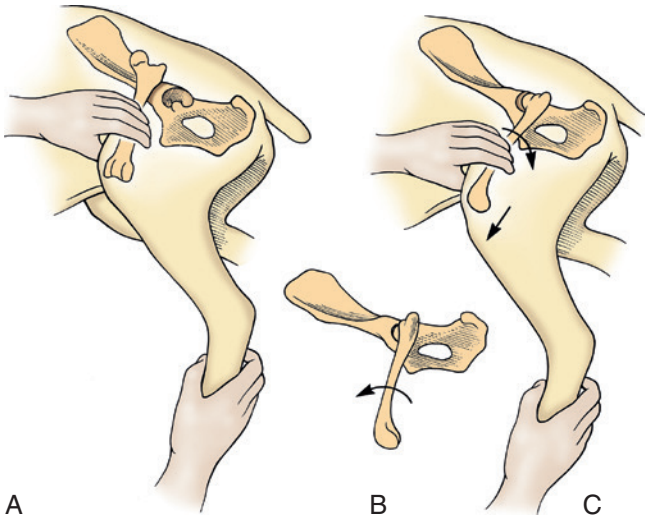


FIG. 34.92 Closed reduction of a craniodorsal hip luxation. (A) Grasp the affected limb near the tarsus with one hand and place the other hand under the limb against the body wall to provide resistance. (B) Externally rotate the limb and pull it caudally to position the femoral head over the acetabulum. (C) When the femoral head lies lateral to the acetabulum, internally rotate the limb to seat the femoral head within the acetabulum.



FIG. 34.93 Ehmer sling applied to rear limb of dog to prevent adduction and weight bearing (e.g., after craniodorsal hip luxation).

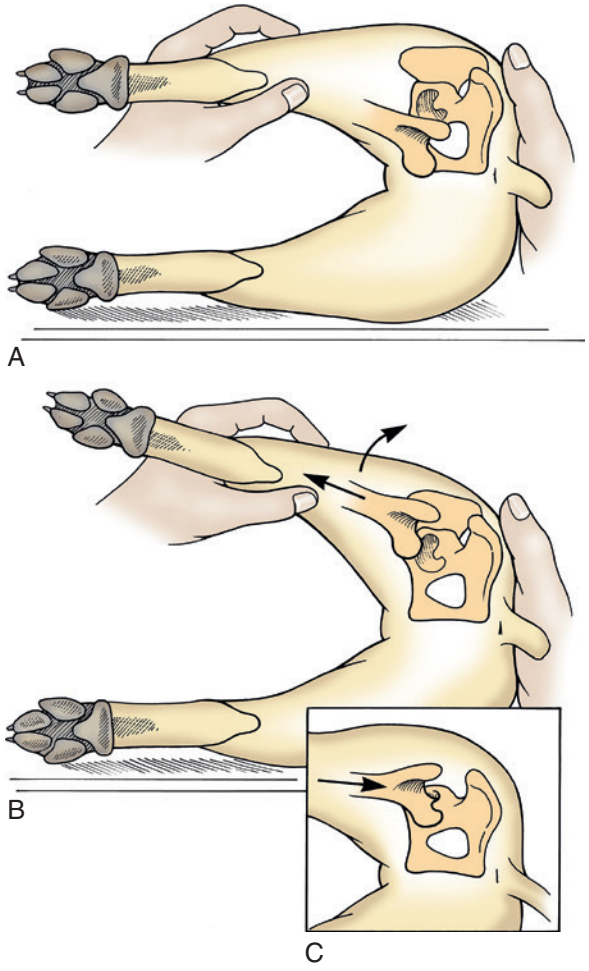


FIG. 34.94 Closed reduction of a caudoventral hip luxation. (A) Place the patient in lateral recumbency with the limb held perpendicular to the spine. Grasp the limb near the tarsal joint with one hand and use the other hand to stabilize the body. (B) Place traction on the limb while simultaneously abducting the leg to pull the femoral head beyond the medial rim of the acetabulum. (C) Once the femoral head has cleared the acetabular rim, exert lateral pressure medial to the hip joint to position the femoral head lateral to the acetabulum. Push proximally and allow the femoral head to fall into the acetabulum.

wrap the gauze over the lateral surface of the thigh and bring it distally medial to the tarsus and over the padded metatarsal area. Repeat the wrapping three or four times. Finish the bandage by applying elastic adhesive material in the same manner. Ehmer slings should not be used for more than 2 weeks. Long-term limb immobilization in a flexed position makes rehabilitation difficult.

Closed Reduction of a Caudoventral Luxation

Place the patient in lateral recumbency with the limb held perpendicular to the spine. Grasp the limb near the tarsal joint with one hand, and place the other hand to stabilize the body (Fig. 34.94A). Place traction on the limb while simultaneously abducting the leg to pull the femoral head beyond the medial rim of the acetabulum (Fig. 34.94B). Once the femoral head has cleared the acetabular rim, exert lateral pressure to position the femoral head lateral to the acetabulum. Push proximally and allow the femoral head to fall into the acetabulum (Fig. 34.94C). After reduction, place the patient in hobbles to prevent abduction of the limb. Limit activity to controlled walking on a leash until the bandage is removed in 4 to 7 days. Limit activity to leash walking for an additional 2 weeks after the hobbles have been removed.

Hind Limb Hobbles

Hobbles prevent abduction of the limbs while allowing weight bearing and walking (Fig. 34.95). The most common use for hind limb hobbles is to support the closed or open reduction of ventral hip luxations. Hind limb hobbles can be applied at the level of the tarsus or stifle.

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FIG. 34.95 Hobbles applied to rear limb of dog to prevent abduction (e.g., after caudoventral hip luxation).

SURGICAL TREATMENT

Open reduction is indicated with avulsion of the fovea capitis or when closed reduction has failed to reduce the hip or maintain hip reduction. The hip joint should be explored to assess the soft tissue injury and the likelihood of maintaining reduction with a reconstructive procedure. If stability of the hip joint can be achieved through a reconstructive procedure, there are a number of techniques from which to choose. If no reasonable chance exists of maintaining long-term reduction after a stabilization procedure, an alternate procedure must be considered, such as an FHO (see p. 1213) or THR.

Preoperative Management

These animals should be thoroughly examined for evidence of concurrent trauma. Surgery may need to be delayed until the animal's condition has been adequately stabilized.

Anesthesia

See Table 32.1 on for anesthetic considerations for patients with orthopedic disease.

Surgical Anatomy

Pertinent anatomy of the hip is described on p. 1217. With hip luxation, the anatomy may appear abnormal and be difficult to identify. Muscles surrounding the joint are often bruised and swollen. It is helpful to reduce the hip before starting the surgical approach to establish normal tissue relationships. The cranial edge of the greater trochanter may be used to help identify the correct plane of surgical dissection. The prominent tendinous insertion of the deep gluteal muscle can also be used for orientation. When the hip is luxated, the femoral head usually lies beneath the deep gluteal muscle. The proximal femur is usually displaced craniodorsally and may obscure visualization of the acetabulum.

Positioning

The patient is positioned in lateral recumbency with the affected limb up. The limb is suspended for surgical preparation to allow limb manipulation during surgery.

SURGICAL TECHNIQUE

Surgical stabilization of hip luxation may be accomplished by capsular reconstruction if the joint capsule is salvageable, although this is rare. In most cases, the capsule cannot be securely closed and additional stability is needed. Other reconstructive procedures must be performed to ensure hip stability for 3 to 4 weeks until capsular healing occurs.[51] Reconstructive procedures include synthetic capsular reconstruction (with suture and bone screws or suture anchors) and toggle pin placement.[52,53] Additional stability may be gained by translocation of the greater trochanter. If the dog is mildly dysplastic, pelvic osteotomy may be indicated to maintain hip reduction (see p. 1212). Pelvic osteotomy may also be useful for nondysplastic dogs when other means are insufficient.

Exploration of the Hip Joint

Perform a craniolateral exposure of the hip joint (see Fig. 34.92A, B). Reflect the deep gluteal muscle and visualize the femoral head craniodorsal to the hip joint. Visualize and remove remnants of the round ligament and debris from the femoral head and acetabulum; this allows the femoral head to completely seat within the acetabulum. Once the hip has been reduced, assess its stability by viewing the acetabular coverage of the femoral head and placing the hip joint through a complete range of motion. Perform the chosen stabilization technique (see the later discussion).

NOTE A Hohmann retractor placed within or just caudal to the acetabulum and used to lever the femur caudally improves visualization of the acetabulum. However, excess traction may lead to sciatic neuropraxia.

Capsule Reconstruction

In some cases the joint capsule may be intact, except for a small rent through which the femoral head has luxated, or an area where the capsule has torn loose from its insertion site at the femoral neck. In both situations, if after replacing the femoral head the acetabular coverage is adequate and the joint is stable through a range of motion, primary suturing of the capsule can be used as the sole reconstructive procedure. *Suture the joint capsule with nonabsorbable monofilament material using an interrupted pattern (Fig. 34.96). If the capsule has torn from its insertion site, drill small holes in the femoral neck through which the suture can pass or reattach the capsule with suture anchors.*

NOTE Reconstruction of the joint capsule as the sole means of stabilization requires that the dorsal joint capsule is identifiable and that the conformation of the hip joint is normal.

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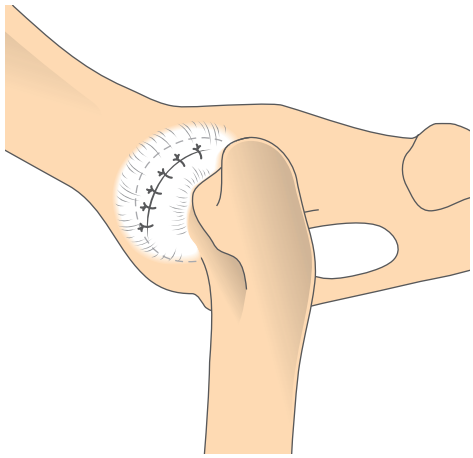


FIG. 34.96 Coxofemoral joint stabilization by means of capsulorrhaphy. Interrupted sutures have been placed to appose the joint capsule.

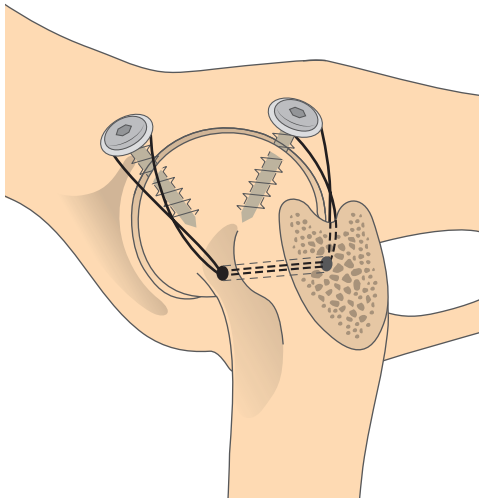


FIG. 34.97 Coxofemoral joint stabilization by placement of a prosthetic capsule. Note the strategic placement of bone anchors in the dorsolateral acetabulum. Suture material is passed from the anchors through a predrilled tunnel in the dorsal femoral neck and tightened. The presence of suture in this position prevents craniodorsal reluxation. The same procedure may be performed using suture anchors.

Joint Reconstruction

The joint capsule is often damaged to a degree that primary suturing is impossible. In such cases, a prosthetic capsule or toggle pin can be used to maintain reduction during healing of the fibrous joint capsule. A prosthetic capsule is made of suture material inserted in the craniodorsal acetabular rim and trochanteric fossa (Fig. 34.97). *Place two screws with flat metal washers or bone anchors in the dorsal rim of the acetabulum. Insert one anchor at the 10 o'clock position and one at the 1 o'clock position (on a left hip). Place a third screw and washer or anchor in the trochanteric fossa (as an alternative, drill a hole through the femoral neck in the trochanteric fossa to accept suture). Pass orthopedic suture or heavy nonabsorbable suture in a figure-eight pattern between the acetabular anchors and trochanteric fossa. Tie the sutures tight enough to maintain reduction but not so tight that they will tear with normal ambulation.*

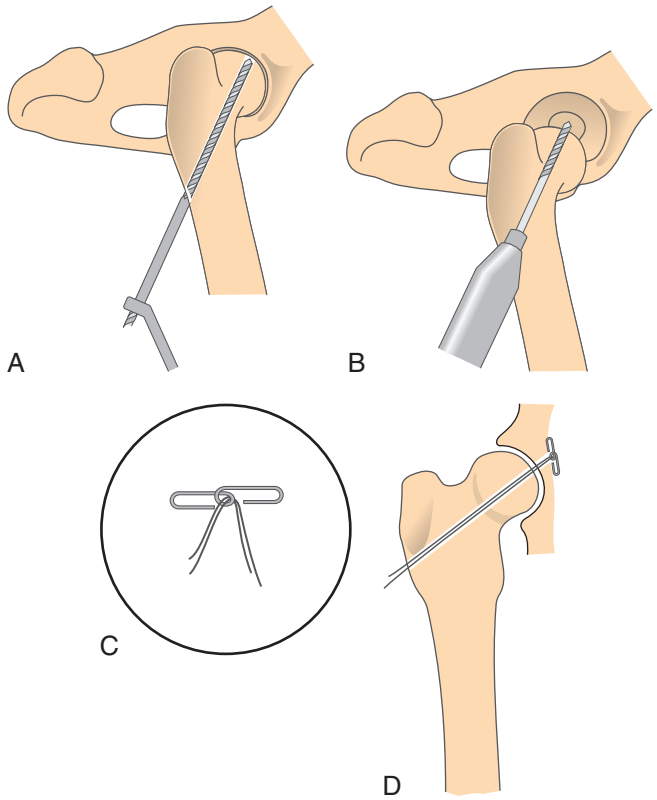


FIG. 34.98 Stabilization of the coxofemoral joint with a toggle pin suture. (A) Drill a hole centered through the femoral neck and (B) through the acetabular fossa. (C) Attach multiple strands of nonabsorbable suture to a toggle pin formed from a K-wire. Pass the toggle pin through the hole in the acetabular fossa and pull to set the pin. (D) Pass the sutures through the hole drilled in the femoral neck. Reduce the hip and secure the sutures.

A toggle pin technique may be beneficial when the capsule is severely damaged or the luxation is chronic, or if multiple limb injuries demand early use of the reconstructed hip. The synthetic round ligament created will not last indefinitely, but if properly placed, it will provide stability until capsular fibroplasia occurs. *Starting at the level of the third trochanter and centered in the femoral neck, use a C-guide to drill a 2.5-mm hole that exits at the fovea capitis (Fig. 34.98A).* Alternatively, the hole may be drilled retrograde from the fovea capitis to the lateral aspect of the femur at the level of the third trochanter. *Drill a 3.5-mm hole through the dorsal aspect of the acetabular fossa (Fig. 34.98B). Attach multiple strands of large nonabsorbable suture to a commercial toggle rod or a toggle pin made by bending a K-wire to form a loop and two wings (Fig. 34.98C).* Alternatively, a TR system (Arthrex Vet Systems) (Fig. 34.99) may be used as it includes the toggle pin, orthopedic suture, and surgical button. *Pass the toggle rod through the acetabular hole and rotate it by pulling on the suture until the pin is secured against the medial acetabular wall. Pass the suture through the femoral tunnel, reduce the hip, and pull the sutures taut (Fig. 34.98D). Secure the suture by passing one pair of sutures through a drill hole created through the lateral femoral cortex and tying it to the opposite suture pair. As an alternative, pass the suture through a surgical button and tie the suture. Tie the suture tight enough to maintain hip reduction but not so tight that it will rupture with normal ambulation.*

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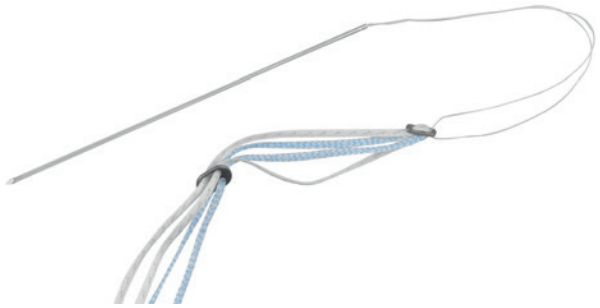


FIG. 34.99 Arthrex tightrope system for use in toggle pin technique. (Courtesy Arthrex Vet Systems.)

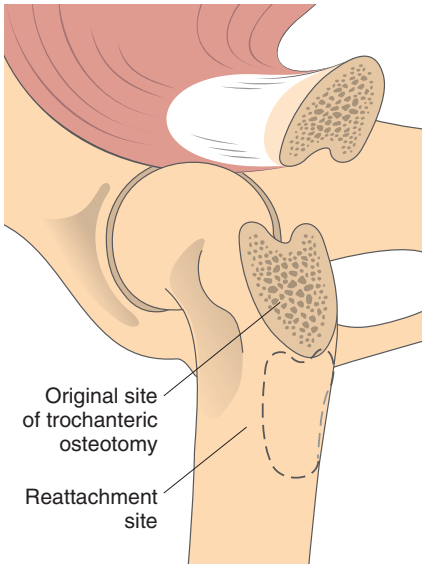


FIG. 34.100 Stabilization of the coxofemoral joint by translocating the greater trochanter. Prepare a new site distal and slightly caudal to the normal anatomic position. Stabilize the greater trochanter in this position with small pins and orthopedic wire (tension band).

Translocation of the Greater Trochanter

If the hip is unstable and if the gluteal musculature is not compromised, a trochanteric osteotomy can be performed to translocate the greater trochanter distally and slightly caudal to add stability to the repair. Relocation of the greater trochanter allows contraction of the gluteal muscles to abduct and internally rotate the femoral head. *Perform a trochanteric osteotomy (see Fig. 33.80), and reflect the gluteal musculature proximally. Once the hip has been cleaned of debris and reduced, place the limb in abduction. Use an osteotome and mallet to create a new surface caudal and distal to the point where the greater trochanter normally seats (Fig. 34.100). Then replace the greater trochanter at its new attachment site, and secure it in position with a pin and tension band technique.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Either orthopedic suture or No. 1 or No. 2 nonabsorbable suture is used for capsule reconstruction. An osteotome, mallet, pins, and wire are needed for translocation of the greater trochanter. Prosthetic capsule reconstruction requires screws and stainless steel washers or suture anchors. Toggle rods are commercially available, or toggle pins may be made of small K-wires; a drill and drill guide are needed to place the toggle pin and suture.

POSTOPERATIVE CARE AND ASSESSMENT

Patients may be placed in an Ehmer bandage to assist hip reduction in the early postoperative period. The bandage is removed 4 to 7 days after reduction. Cage confinement may be adequate for dogs with stable hips. Once the Ehmer sling has been removed, it is beneficial to begin very controlled physical rehabilitation exercises (see Chapter 11). Reexamination 3 days after removal of the Ehmer bandage and before resumption of unsupervised activity is advised.

PROGNOSIS

The success rate for maintaining reduction and regaining good to excellent limb function with closed reduction is approximately 50%. The prognosis is poor in patients with poor conformation of the hip joint secondary to hip dysplasia or previous trauma. Clinical studies indicate that the success of surgical intervention after failure of closed reduction is not different from the success rate in patients undergoing surgical reduction as a primary treatment.[51–53] Therefore it is reasonable to attempt closed reduction in patients with a hip luxation. The success rate for maintaining reduction with good to excellent limb function after open reduction is approximately 85% to 90%.[51–53] The results do not appear to favor any one reconstruction technique.

LEGG-PERTHES DISEASE

DEFINITION

Legg-Perthes disease is a noninflammatory aseptic necrosis of the femoral head that occurs in young patients before closure of the capital femoral physis. Synonyms include *OCD of the femoral head* and *avascular necrosis of the femoral head*.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Legg-Perthes disease results in collapse of the femoral epiphysis because of an interruption of blood flow. The reason for the loss of blood flow is not known for certain, but several theories have been proposed, including hormonal influence, hereditary factors, anatomic conformation, intracapsular pressure, and infarction of the femoral head. The vascular supply to the femoral head in young animals with open proximal femoral physes is derived solely from epiphyseal vessels; metaphyseal vessels do not cross the physis to contribute to femoral head vascularity. Epiphyseal vessels course extraosseously along the surface of the femoral neck, cross the growth plate, and penetrate bone to supply nourishment to the femoral epiphysis. Synovitis or sustained abnormal limb position may increase intraarticular pressure enough to collapse the fragile veins and inhibit blood flow. An autosomal recessive gene has been proposed as a genetic cause for the development of aseptic necrosis of the femoral head. After cell death occurs, the reparative processes begin. The bone substance is weakened mechanically during the revascularization period, and normal physiologic weight-bearing forces may cause collapse and fragmentation of the femoral epiphysis. When this happens, incongruence of the femoral epiphysis and acetabulum

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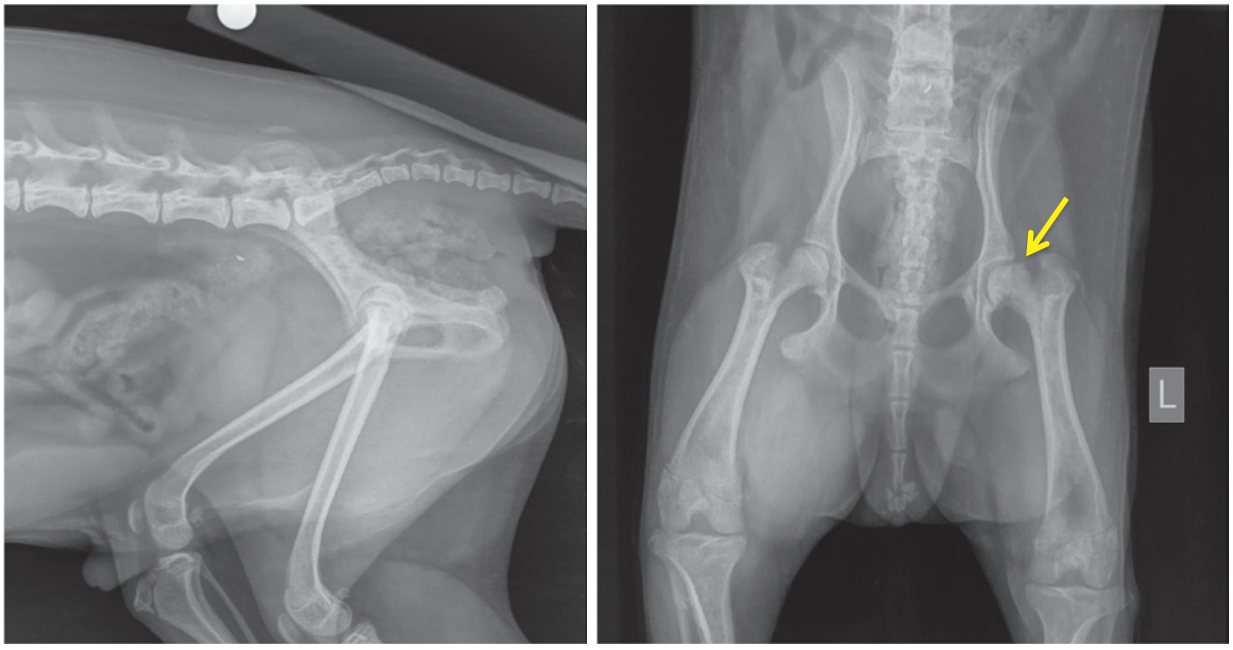


FIG. 34.101 Radiograph of a young dog with Legg-Perthes disease. The femoral head appears moth-eaten and is misshapen *(arrow)*.

results in DJD. Fragmentation (fractures) of the femoral epiphysis and osteoarthrosis cause pain and resulting lameness.

NOTE Because this condition has been linked to an autosomal recessive gene, be sure to advise owners to have affected animals neutered.

DIAGNOSIS

Clinical Presentation

Signalment

Legg-Perthes disease is diagnosed in young, small-breed dogs (i.e., <10 kg). The peak incidence of onset is 6 to 7 months with a range of 3 to 13 months, and males and females are equally affected. This condition occurs bilaterally in 10% to 20% of affected animals.

History

Affected animals are usually presented for evaluation of a slow onset, weight-bearing lameness that worsens over a 6- to 8-week period. Lameness may progress to non–weight bearing. Some clients report an acute onset of clinical lameness. In these patients, sudden collapse of the epiphysis may cause acute exacerbation of an already present but imperceptible lameness. Other clinical signs can include irritability, reduced appetite, and chewing at the skin over the affected hip.

Physical Examination Findings

Manipulation of the hip joint consistently elicits pain in affected animals. Limited range of motion, muscle atrophy, and crepitus may be present with advanced disease.

Diagnostic Imaging

Radiographs show deformity of the femoral head, shortening and/or lysis of the femoral neck, and foci of decreased bone opacity within the femoral epiphysis (Fig. 34.101). A flexed hip ("frog leg") view is often useful to detect abnormal shape of the affected femoral head (see Fig. 34.101). A CT scan may become necessary to identify lysis or cavitation within an affected femoral head that maintains a relatively normal shape.

Laboratory Findings

Consistent laboratory abnormalities are not seen.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include physeal trauma and luxation of the medial patella. Small dogs may have concurrent bilateral medial patellar luxation (see p. 1254), and the stifle joint must be examined carefully to diagnose this condition.

MEDICAL MANAGEMENT

Because the condition is not painful in most dogs during the early stages of the disease, the diagnosis is often made after collapse and fragmentation have resulted in joint incongruity and DJD. Conservative treatment with antiinflammatory medication and leash-limited or non–weight-bearing exercise, such as swimming, may provide pain relief in a small percentage of dogs, but most dogs require surgical intervention to relieve the lameness. In rare cases the diagnosis is made before collapse of the femoral head, and treatment consists of limiting weight bearing on the limb during the revascularization period to prevent collapse of the femoral head.

SURGICAL TREATMENT

Options for management of Legg-Perthes disease include femoral head and neck excision and THR. Miniature THRs have been reported to have a high success rate in small-breed dogs.[54]

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Preoperative Management

Activity is limited until definitive surgical treatment has been performed. Antiinflammatory medications may be administered for pain relief.

Anesthesia

Recommendations for the anesthetic management of animals with orthopedic disease are provided in Tables 32.1 and 32.2.

Surgical Anatomy

In animals with Legg-Perthes disease, the joint capsule appears thickened and more vascular than normal. The femoral head and neck often appear misshapen. The bone may be soft and may fragment when the head and neck are excised, requiring that rongeurs be used to remove small fragments.

Positioning

The animal is positioned in lateral recumbency with the affected leg up. A hanging-limb preparation may be used to facilitate manipulation of the limb during surgery. The leg should be prepared from the dorsal midline to midtibia.

SURGICAL TECHNIQUE

Excision of the femoral head and neck is often the treatment of choice. See p. 1213 for a description of the technique.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Instruments necessary to remove the femoral head and neck in small dogs include an osteotome and mallet, bone cutters, and rongeurs.

POSTOPERATIVE CARE AND ASSESSMENT

The animal should be encouraged to use the limb immediately after surgery. This should include immediate rehabilitation exercises as outlined in Table 34.9. NSAIDs should be administered to reduce pain and encourage early function. Passive flexion and extension of the hip joint should be performed twice daily as soon as the animal tolerates it. Physical therapy should be initiated with small movements and the range of motion gradually increased over 5 to 10 minutes.

PROGNOSIS

Prognosis for normal limb use is good after femoral head and neck excision because of the small size of affected dogs; however, owners should be warned that slight, intermittent lameness may still occur in damp weather or after heavy exercise or a period of inactivity. Poor results occur occasionally and have been related to non–weight bearing before surgery, severe preoperative muscle atrophy, and incorrect surgical technique.

STIFLE

CRANIAL CRUCIATE LIGAMENT RUPTURE (CRUCIATE DISEASE)

DEFINITIONS

CCL injuries are complete or partial tears of the ligament or avulsions of the origin or insertion. **Cranial drawer** is a term used to describe excessive craniocaudal movement of the tibia relative to the femur as a result of cruciate ligament injury. **Cranial tibial thrust** (CTT) is defined as cranial movement of the tibial tuberosity in the cranial cruciate–deficient stifle when the hock is flexed and the gastrocnemius muscle contracts. **Translation** is defined as movement of a bone parallel to an axis or plane. **Pivot shift** is cranial movement of the tibia combined with internal rotation of the tibia. **Tibial plateau angle (TPA)** is the angle between a line perpendicular to the long axis of the tibia and a line parallel to the tibial plateau. **Medial buttress** is a palpable thickening of the medial aspect of the stifle. **Imbrication** is defined as the tightening of a structure. **Isometry** is defined as maintaining equal distance or tension throughout a range of motion.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The CCL functions primarily to limit cranial translation of the tibia relative to the femur (Fig. 34.102). The CCL can be divided into craniomedial and caudolateral bundles, which have different insertion points on the tibial plateau. The craniomedial band is taut during all phases of flexion and extension; the caudolateral band is taut in extension, but it becomes lax in flexion. The CCL also limits internal rotation of the tibia; as the stifle is flexed, the cranial and caudal cruciate ligaments twist on each other, limiting the degree of internal rotation of the tibia relative to the femur. Interaction of the cranial and caudal cruciate ligaments during flexion also provides a limited degree of varus-valgus support to the flexed stifle joint.

Mechanoreceptors and afferent nerve endings have been identified in the interfiber layers of the CCL. Innervation of the ligament serves as a proprioceptive feedback mechanism to prevent excessive flexion or extension of the stifle joint. This protective action is accomplished through stimulation or relaxation of muscle groups that lend support to the joint.

NOTE The craniomedial band of the cranial cruciate ligament is the primary check against craniocaudal drawer motion.

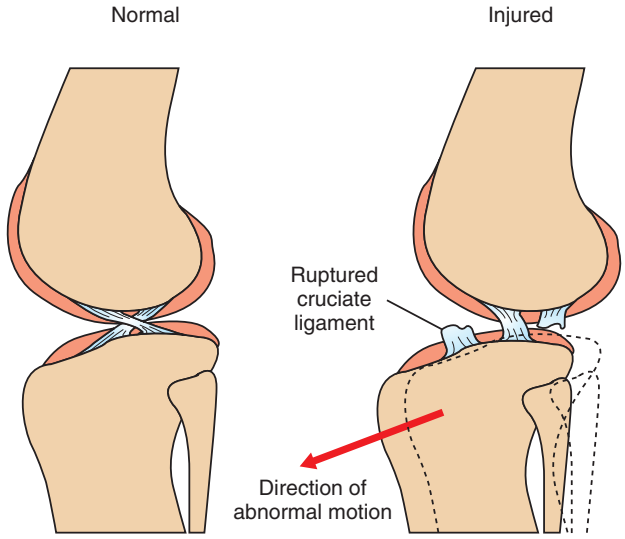


FIG. 34.102 The cranial cruciate ligament prevents cranial translation of the tibia.

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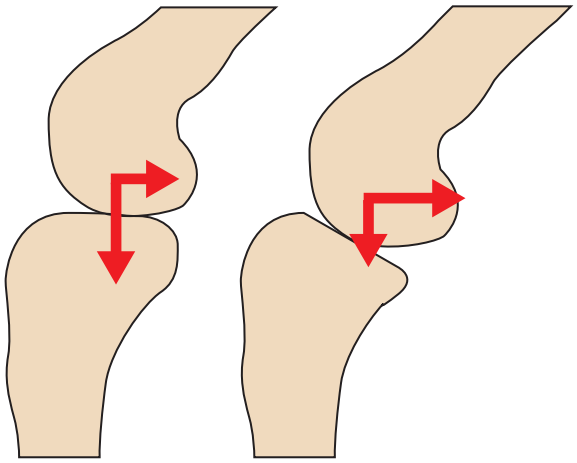


FIG. 34.103 A larger tibial plateau angle results in a greater cranial force on the tibia during weight bearing.

CCL failure can result from degenerative and traumatic causes. The categories are interrelated because ligaments weakened by degeneration are more susceptible to trauma. The high incidence of CCL failure in dogs suggests that there is an underlying cause of premature degeneration of the cruciate ligament in most cases.[55,56] Degeneration of the ligament is associated with aging (especially in large-breed dogs), conformational abnormalities (straight rear limbs), and immune-mediated arthropathies. Degeneration of the ligament has also been associated with an increased TPA, although not all studies have identified this correlation.[55–57] An increased TPA has been theorized to place chronic excessive loads on the CCL, leading to eventual mechanical failure (Fig. 34.103). In cats, excessive body weight may significantly increase the risk of CCL rupture.

The mechanism of CCL traumatic injury is primarily a reflection of its function as a constraint to joint motion. Acute injury is most commonly associated with the hyperextension and internal rotation of the leg that occurs when a dog's foot becomes caught in a hole or fence. Jumping can also cause cruciate ligament rupture if the force of the CTT exceeds the breaking strength of the ligament. With ligament degeneration, even repetitive normal activities can cause progressive rupturing of the ligament.[57] In many cases, the underlying pathologic condition is present in both stifles and a high percentage of dogs either have bilateral cruciate ligament rupture, or they rupture the contralateral ligament within 1 to 2 years. Partial rupture of the CCL results in lameness with minimal detectable stifle instability and progressive radiographic signs of osteoarthritis. Partial rupture generally proceeds to complete ligament rupture with time.

CCL injury with stifle instability is part of a cascade of events that include progressive osteoarthritis and medial meniscus injury. Stifle instability results in synovitis, articular cartilage degeneration, periarticular osteophyte development, and capsular fibrosis. The immobile medial meniscus is subject to injury in the unstable joint (see p. 1243). Progressive osteoarthritis occurs after CCL rupture regardless of the treatment method.

DIAGNOSIS

Clinical Presentation

Signalment

Either gender and any age or breed of dog may be affected; however, most dogs brought in for treatment of CCL injury are young, active, large-breed dogs. CCL injury is uncommon in cats.

History

Acute injury, chronic injury, and partial tears are three clinical presentations associated with CCL injury. Patients with acute tears show a sudden onset of non–weight-bearing or partial weight-bearing lameness. Lameness usually decreases somewhat within 3 to 6 weeks after injury without treatment, particularly in patients that weigh less than 10 kg. An exception is dogs with associated meniscal injury. These dogs usually maintain a minimally weight-bearing or non–weight-bearing lameness until surgical intervention takes place.

Patients with chronic injury have a prolonged weight-bearing lameness. There may or may not be a history of acute non–weight-bearing lameness followed by gradual improvement to moderate weight bearing. Patients may have a history of difficulty rising and sitting. The owners may report that the dog sits with the affected limb out to the side of the body. Lameness is typically worse after exercise or after sleeping. Chronic lameness is associated with the development of DJD.

Partial CCL tears are difficult to diagnose in the early stages of injury. Initially, affected animals have a mild weight-bearing lameness associated with exercise; the lameness resolves with rest. This stage of the disease may last for many months. As the ligament continues to tear and the stifle becomes increasingly unstable, degenerative changes worsen, and lameness becomes more pronounced and does not resolve with rest.

Dogs of any age may have bilateral subacute or chronic bilateral CCL rupture. These dogs may have a presumptive neurologic disease because the dog may be unable or unwilling to support weight in either hind limb. The owner may also report that the dog is unable to sit normally but sits on elevated surfaces, such as a stool or step.

Physical Examination Findings

Animals with acute complete tears are often apprehensive during examination of the stifle joint. Instability can be difficult to elicit because of the patient's apprehension and the resulting muscle contraction. Joint effusion may be palpable adjacent to the patellar tendon. A positive tibial compression test may be easier to elicit than a positive drawer test.

Patients with chronic tears may have thigh muscle atrophy (compared with the normal limb), and crepitus may be evident when the stifle is flexed and extended. When the joint is extended from a flexed position, a clicking or popping may be heard and felt; this is commonly associated with a meniscal tear. However, the absence of joint noise does not eliminate the possibility of meniscal injury. An enlargement along the medial joint surface (medial buttress) can often can palpated and is caused by osteophyte formation along the trochlear ridges and fibrous tissue formation along the medial condyle and proximal tibia. Craniocaudal instability can be difficult to elicit, particularly in large or apprehensive patients with chronic tears, because of proliferation of the fibrous joint capsule.

With partial tears, early instability is difficult to detect because a portion of the ligament is intact and inhibits craniocaudal movement. Tearing of the caudolateral band alone does not produce instability because the intact craniomedial band is taut in both flexion and extension. If an isolated injury to the craniomedial band occurs (caudolateral band remains intact), the joint is stable in extension because the caudolateral band remains taut; however, instability is present in flexion because the caudolateral band normally becomes lax during flexion. Initially, pain, synovial

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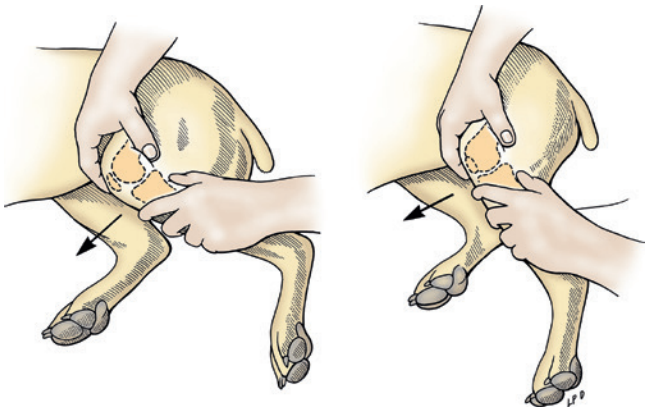


FIG. 34.104 To examine for cruciate ligament injury, place the thumb of one hand over the lateral fabella and the index finger over the patella. Stabilize the femur with this hand. Place the thumb of the opposite hand caudal to the fibular head with the index finger on the tibial tuberosity. With the stifle flexed and then extended, attempt to move the tibia cranially and distally to the femur.

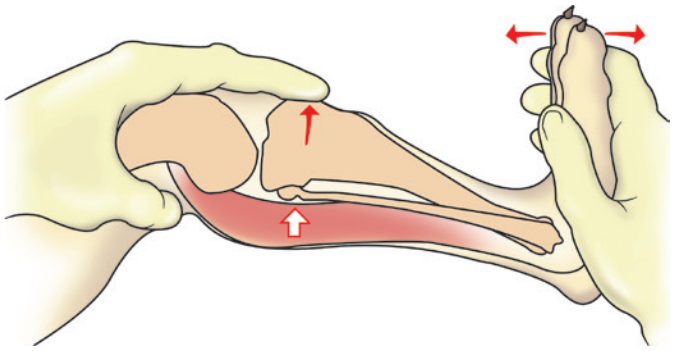


FIG. 34.105 To perform a tibial compression test, grasp the distal quadriceps with one hand from the cranial surface so that the index finger can be extended down over the patella and the tip of the finger is on the tibial crest. Use the second hand to grasp the foot at the metatarsal region from the plantar surface. With the limb in moderate extension, flex the hock with the lower hand and prevent stifle flexion with the upper hand. Feel for cranial movement of the tibia, which is an indication of cruciate ligament damage.

effusion, and crepitus are absent, but signs of instability and DJD eventually become evident. Pain on hyperextension of the joint is commonly present in dogs with partial tears.

NOTE Always compare the limb that has the suspected injury with the opposite limb if the instability and stifle swelling are questionable.

Cranial drawer movement is diagnostic of cruciate ligament injury (Video 34.5). The **cranial drawer test** is performed with the patient in lateral recumbency. Lack of adequate patient relaxation is a common cause of failure to elicit cranial drawer movement. Therefore if lameness caused by cruciate ligament injury is suspected, general anesthesia or heavy sedation may be necessary to negate the influence of muscle tension (see p. 961). Once the dog is in lateral position, the examiner stands to the patient's rear and positions the thumb and forefinger of one hand on the femur (Fig. 34.104). The thumb is placed directly behind the fabella and the forefinger over the patella. The remaining fingers are wrapped around the thigh. The other hand is placed on the tibia with the thumb directly behind the fibular head and the forefinger over the tibial crest. The three remaining fingers are wrapped around the tibial shaft. The femur is stabilized with the one hand while the second hand moves the tibia forward and back in a direction parallel to the transverse plane of the tibial plateau. The pressure to move the tibia forward should be applied through the thumb behind the fibular head.

The tibia must be held in neutral position, as determined by the position of the fingers on the patella and tibial tuberosity, and not be allowed to rotate internally. If this occurs, internal rotation of the joint may appear as cranial drawer movement. The examiner must test for signs of instability with the stifle joint in extension, in the normal standing angle, and in 90 degrees of flexion. If the degree of movement is questionable, comparison with the opposite limb is helpful. A positive test result is craniocaudal movement beyond the 0 to 2 mm found in normal stifle joints. In younger patients, normal craniocaudal translation may be as great as 4 to 5 mm, but ligament rupture is confirmed by the absence of an abrupt stop at the cranial extent of movement. Because most isolated cruciate ligament tears involve the CCL, craniocaudal instability is usually associated with injury of this ligament. If a partial tear is present, the cranial drawer sign may reveal only 2 to 3 mm of instability when the test is done with the stifle flexed and no instability with the stifle in extension. Mild partial tears may have no cranial drawer sign in any position. On completion of the cranial drawer test, the stifle should be flexed and extended through a range of normal movement. With the leg in extension, collateral stability should be assessed.

NOTE Juvenile dogs have increased joint laxity (4–5 mm) but maintain a distinct end point when the tibia is moved cranially.

The **tibial compression test (CTT test)** is performed with the patient standing or in lateral recumbency. The examiner stands to the patient's rear and grasps the distal quadriceps with one hand from the cranial surface so that the index finger can be extended down over the patella and the tip of the finger is on the tibial crest (Fig. 34.105). The second hand grasps the foot at the metatarsal region from the plantar surface. The limb is positioned in moderate extension, and as the lower hand flexes the hock, the upper hand prevents stifle flexion. The index finger of the upper hand is used to feel for cranial movement of the tibial crest while the hock is being flexed. In the normal knee, the upper hand will feel pressure from the patella on the index finger. With CCL rupture, the tibial crest will be advanced forward as the hock is flexed (CTT). The technique should be repeated in different degrees of stifle flexion to test for a partial CCL rupture.

Diagnostic Imaging

With acute tears, radiographs are helpful in ruling out other causes of stifle joint lameness. Radiographic findings in patients with chronic ligament tears or partial tears include compression

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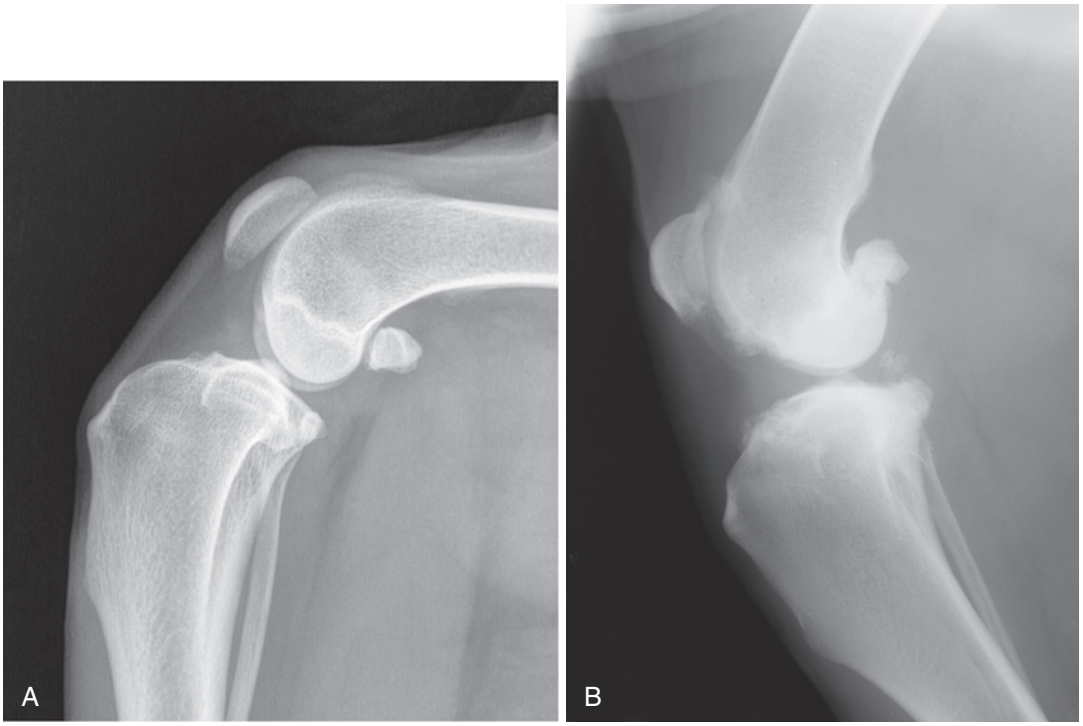


FIG. 34.106 Lateral radiograph of a dog with a cruciate ligament rupture. (A) Note the loss of fat pad definition and distention of the caudal joint capsule. (B) Note the loss of fat pad definition and distention of the caudal joint capsule and the osteophyte formation along the trochlear ridge and the subchondral bone sclerosis of the tibial plateau.

of the fat pad in the cranial aspect of the joint and extension of the caudal joint capsule caused by joint effusion and osteophyte formation along the trochlear ridge, the caudal surface of the tibial plateau, and the distal pole of the patella (Fig. 34.106).[55] Thickening of the medial fibrous joint capsule and subchondral sclerosis are also evident. Radiographic changes in patients with CCL rupture are nonspecific and can be seen with other stifle diseases, including infection, soft tissue neoplasia, and immune-mediated arthritis. Avulsion of the CCL attachment may be specific in that a bone fragment may be seen adjacent to this site.

MRI has been used for evaluation of the cruciate ligament in dogs; however, the cost and necessity for general anesthesia limit the use of this technique in the diagnosis of canine cruciate ligament injury.

Arthroscopy

AP A large percentage of the surface of the cruciate ligament can be examined arthroscopically for gross tears, fibrillation, or discoloration associated with cruciate damage. The menisci and cartilage may also be examined. Arthroscopy has been shown to decrease the short-term morbidity for dogs requiring stabilization of CCL injuries (Videos 34.6, 34.7, and 34.8).

Indications. Diagnostic and therapeutic procedures of the stifle joint are performed arthroscopically. Diagnostic arthroscopy is most commonly used to confirm the presence of partial tears of the anterior cruciate ligament and to assess the degree of osteoarthritis; therapeutic arthroscopy is most commonly applied for removal of CCL remnants, assisted reconstruction of the CCL, treatment of meniscal injury, and treatment of stifle OCD and topical treatment of osteoarthritis (abrasion arthroplasty and microfracture).

Instrumentation. A 2.7-mm arthroscope is commonly chosen for use in the stifle joint. A 4-mm arthroscope can be used in larger dogs, but the larger the arthroscope, the more difficult it is to introduce other instruments into the joint. A motorized shaver is helpful to create a viewing window through removal of an interfering fat pad. A 3.5- or 4-mm full radius shaver blade is preferred. Hand instruments useful for stifle joint arthroscopy include probes, tissue graspers, and tissue biters. Instrumentation designed for canine arthroscopy is readily available; these instruments are preferable to most human small joint instruments because the latter are often too large for use in dogs. Be aware that the smaller the instrument, the more delicate it is and thus more easily damaged.

Portal sites and technique. Two techniques are available for stifle arthroscopy. Technique 1: *infuse the joint with saline or lactated Ringer's with a syringe and 1.5-inch (40-mm) 20-gauge needle. Locate the arthroscope portal lateral to the patellar tendon, approximately one-third of the distance from the distal pole of the patella to the tibial plateau. Incise through the skin and into the joint with a No. 15 blade. Insert the arthroscopy cannula with a blunt obturator into the incision and into the medial compartment of the joint. Remove the obturator and insert the arthroscope.*

Technique 2: *locate the arthroscope portal lateral to the patellar tendon, approximately midway between the tibial tuberosity and the distal pole of the patella, by incising through the skin and completely into the joint with a No. 15 blade. Pass the obturator of an egress cannula into the arthroscope portal and under the patella so that it tents the skin medial and proximal to the patella.*

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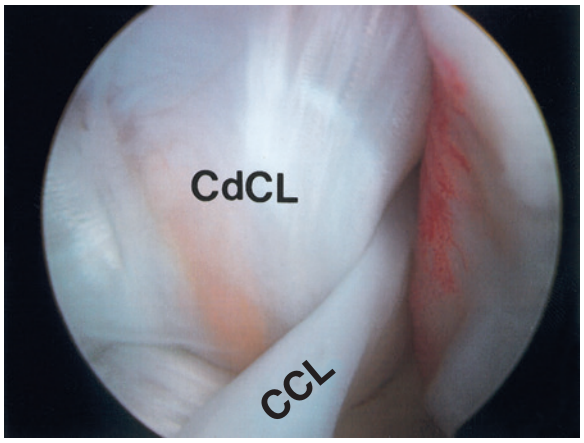


FIG. 34.107 Cranial view of the intercondylar notch. The caudal cruciate ligament *(CdCL)* is intact. A small cranial medial band is all that remains of the cranial cruciate ligament *(CCL)*.

Incise over the obturator and advance it through the skin. Pass the egress cannula over the obturator until the end is approximately under the center of the patella. Remove the obturator, and then move the tip of the egress cannula into the medial joint pouch by extending the stifle joint and levering the cannula over the medial trochlear ridge. Once the egress port has been established, insert the arthroscope in the scope portal. Place the instrument port at the same proximodistal level but medial to the patellar tendon. Systematically examine the suprapatellar compartment and trochlear ridges. As the joint is flexed, position the arthroscope lateral to the intercondylar notch. Further examination of the joint may be limited by the presence of the fat pad, which is generally inflamed and obscures viewing of the cruciate ligaments and menisci. A higher arthroscope portal decreases the obstruction by the fat pad. *Make a viewing window through the fat pad so that you can thoroughly examine the ligaments and menisci. Use a motorized shaver to remove the inflamed fat pad. Position the arthroscope to view the intercondylar notch and top of the fat pad. Establish an instrument port as described previously, and insert the shaver blade. Visualize the shaver blade and position the shaver window away from the lens of the arthroscope. Remove tissue via the suction and cutting action of the shaver blade. Visualize the cruciate ligaments (often remnants of the CCL) as a viewing window is established through removal of the fat pad (Fig. 34.107). Once a viewing window has been established, examine the cruciate ligaments and menisci. Remove remnants of a torn CCL with the motorized shaver or hand punch. Examine both the lateral and medial menisci for fraying or tears.* The medial meniscus is most commonly injured (bucket handle tear, radial tears, or fraying). *To view the posteromedial compartment (medial meniscus), place a Hohmann or joint retractor through the instrument portal or a separate portal above the instrument or arthroscope portal. Pass the tip of the retractor caudal to the tibial plateau and lever the tibia cranially and the joint open. As an alternative, apply a valgus stress to open the medial compartment of the joint. Examine the meniscus for fraying (Fig. 34.108A) or complete tears (Fig. 34.108B). Use small hand instruments, such as a grasper or probe, to hold a torn section of meniscus, then remove the damaged meniscus with tissue biters. Once the menisci have been inspected and torn sections removed, stabilize the CCL-deficient joint.*

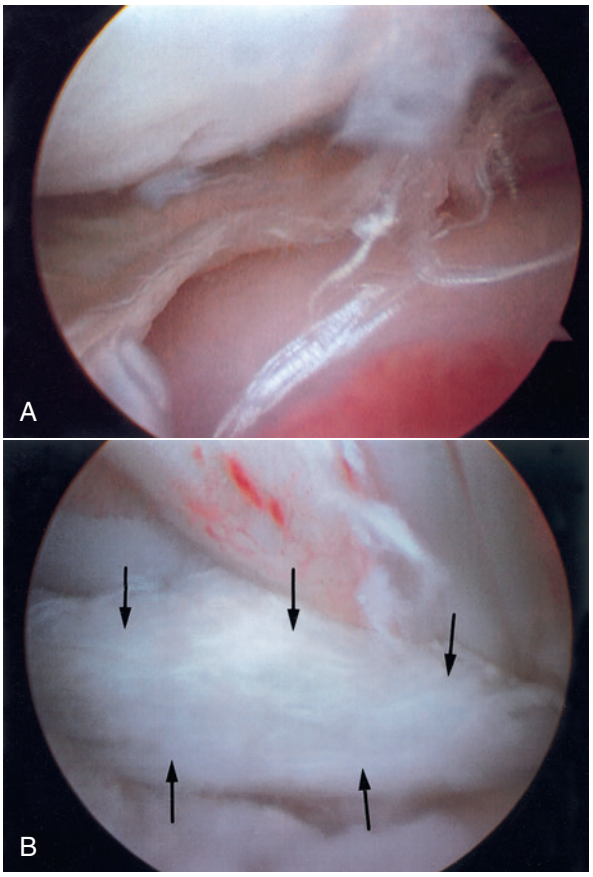


FIG. 34.108 (A) Cranial view of a frayed medial meniscus associated with a torn cranial cruciate ligament. (B) Cranial view of a bucket handle tear of the medial meniscus. The torn meniscus is folded cranially *(arrows)*.

Laboratory Findings

If joint palpation and radiographs are inconclusive, joint centesis and synovial fluid examination are helpful. In cases of partial ligament tear, centesis is particularly helpful in identifying stifle joint involvement as the cause of the lameness. Increased amounts of joint fluid and a two- to threefold increase in cell numbers (6000–9000 WBC/μL; primarily mononuclear cells) indicate secondary DJD (see also p. 1135).

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include joint neoplasia, immune-mediated arthritis, septic arthritis, mild joint sprains or muscle strains, patellar luxation, primary meniscal injury, long digital extensor (LDE) tendon avulsion, and primary or secondary arthritis.

MEDICAL MANAGEMENT

Conservative treatment is generally unsuccessful in dogs.[58,59] Lameness typically improves over time, but the animal does not return to preinjury activity without evidence of recurring lameness. Surgical stabilization is recommended in patients of any size to ensure optimum function. Lameness often decreases within 6 weeks in small patients managed conservatively (i.e., rest and antiinflammatory drugs). These patients appear to function

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normally on the injured leg; however, instability persists and secondary DJD frequently develops. Despite the fact that the animal appears to function adequately after the initial injury, body weight is often merely shifted to the uninjured leg. Abnormal stress, coupled with the increasing mechanical weakness of the cruciate ligament associated with aging, may lead to rupture of the cruciate ligament in the opposite stifle joint within 12 to 18 months. Because these patients are then nonambulatory, they are often incorrectly diagnosed as having an acute neurologic problem. An accurate history and physical examination should alert the clinician to the fact that bilateral cruciate ligament injury, rather than neurologic disease, is the problem. Treatment of patients with bilateral cruciate ligament ruptures is less successful than in animals with only one injured stifle joint.

NOTE Injury of the contralateral cruciate ligament occurs in 40% of patients. The percentage increases (60%) if radiographic changes are visible in the uninjured joint.

SURGICAL TREATMENT

Surgical therapy is divided into intracapsular and extracapsular reconstruction techniques, corrective osteotomy, or primary repair with augmentation (Table 34.10). The surgical method chosen is a matter of the surgeon's preference, patient size and function, and cost of the procedure, because most retrospective studies have shown the success rate to be near 90% regardless of the technique used.[60,61]

Intracapsular and extracapsular surgical procedures have focused on recreation of the passive constraints of the stifle joint (CCL, joint capsule fibrosis). **Intracapsular reconstruction** consists of passing autogenous tissue through the joint using the "over-the-top" method or passing the tissue through predrilled holes in the femur or tibia or both. The most commonly used material is autogenous fascia lata. Synthetic materials are rarely used because of eventual stretching or rupture and the risk of an inflammatory reaction or infection. Allografts with and without bone plugs are available, but they are not in widespread use for cruciate ligament reconstruction. An advantage of intracapsular techniques is that they most closely mimic the original position and biology of the original CCL.[62] Disadvantages of these techniques are their invasiveness and the tendency of the graft to stretch or fail.

TABLE 34.10 Categorization of Cruciate Repairs

Primary Repair With Augmentation	Repair of Avulsion of Insertion
Intracapsular	Under and over fascial strip Bone tunnel fascial strip Allograft Synthetic graft
Extracapsular	Lateral fabellar suture Bone anchors Fibular head transposition Tightrope Fascial imbrication
Osteotomy	Tibial plateau leveling osteotomy Tibial wedge osteotomy Tibial tuberosity advancement

Extracapsular reconstruction involves placement of sutures outside the joint. Extracapsular reconstruction with sutures is often incorrectly referred to as imbrication sutures. Numerous patterns and combinations of suture origins and insertions have been described. Location of the origin and insertion of an extracapsular suture can have significant effects on the isometry of the joint, thereby affecting the amount of drawer motion throughout the normal range of motion of the stifle joint.[63] Extracapsular sutures may also be secured from bone anchors or bone tunnels.[64] Materials used in extracapsular sutures include monofilament nylon or fishing or leader line, manufactured orthopedic wire, or braided orthopedic suture. Sutures may be tied or connected with a crimp. Crimping suture alters the biomechanical properties of the loop.

True **imbrication techniques** have also been described for treatment of CCL rupture, usually in addition to one of the other techniques. Imbrication for cruciate ligament rupture is performed by tightening the fascia lata either through an advancement technique using a vest-over-pants suture pattern or by partial excision and closure. These techniques are usually performed as part of the routine closure of the fascia lata.

The **tibial plateau leveling osteotomy** (TPLO) changes the mechanics of the stifle to achieve stabilization by active constraint of the joint.[59,65] The stifle joint is normally stabilized by both passive constraints (ligaments, menisci, joint capsule) and active constraints (muscles and tendons). The CCL functions as a passive constraint to cranial tibial translation and internal rotation of the tibia. Ground reaction forces and muscle forces generate compressive loads on the articular surface of the tibia during weight bearing. As a result of the caudally directed slope of the tibial plateau, when the tibia is loaded, a shear force is generated that induces abnormal tibial translation in CCL-deficient stifle joints. The shear component of the compressive force is referred to as the CTT and is passively constrained by the CCL (see Fig. 34.103). The CTT is also proportional to the slope of the tibial plateau. If the slope of the plateau decreases, the CTT also decreases. The tibial plateau slope can be reduced such that tibial thrust changes from a craniproximal direction to a neutral or caudal direction. At the point where the tibial thrust changes direction to a caudal thrust, there is increased reliance on the caudal cruciate ligament as a passive constraint to abnormal caudal translation of the tibia. The intent of TPLO surgery is to attain a tibial plateau slope (approximately 3–7 degrees) (Fig. 34.109A–B) with which tibial thrust can be effectively controlled by the caudal cruciate ligament and the active constraints of the stifle (e.g., the quadriceps muscle group). Because the CCL also passively constrains excessive internal rotation of the tibia, we must logically question the source of these internally rotatory moments and the role of the TPLO procedure in functionally controlling them. Failure to control the internal rotation resulting in drawer in association with internal rotation is referred to as *pivot shift*. The significance of this motion to functional outcome following TPLO surgery is uncertain. TPLO is an effective procedure for dogs with a complete or partial tear of the CCL. Many surgeons prefer TPLO for treating larger, active dogs in which long-term rehabilitation and postoperative control are difficult.

Tibial wedge osteotomy (TWO) is the forebearer of the TPLO and was originally described for the treatment of severely increased TPAs in dogs.[66] The TWO is based on the same principles of

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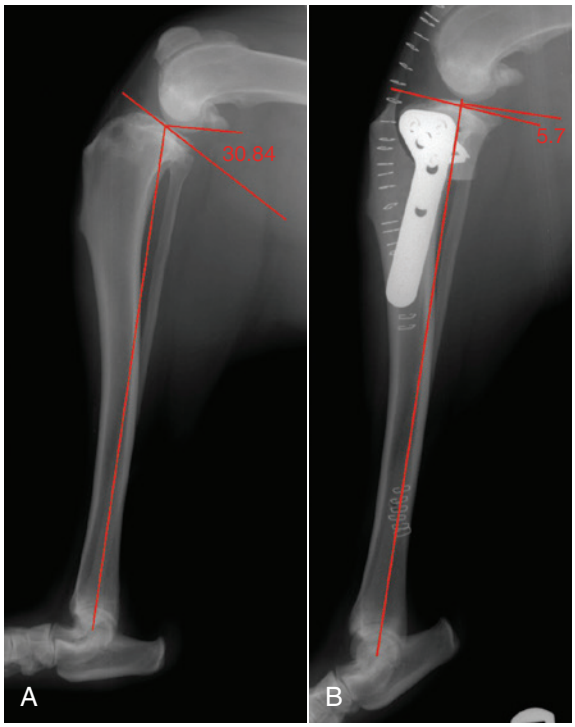


FIG. 34.109 (A) Preoperative lateral radiograph of a dog showing measurement of the tibial slope for a tibial plateau leveling osteotomy. (B) Postoperative lateral radiograph showing leveling of the tibial slope (5 degrees) for active stabilization of the cranial cruciate–deficient stifle joint.



FIG. 34.110 Radiograph of a tibia after a tibial wedge osteotomy.

biomechanics as the TPLO (Fig. 34.110); however, the lower location of the osteotomy results in alteration of the relative position of the tibial crest that may be associated with complications of the stifle extensor mechanism. The TWO remains a valuable technique for management of ruptured cranial cruciate ligament and increased TPA in young dogs with open proximal tibial physes because the TWO will not affect these physes as the TPLO may.

Tibial tuberosity advancement (TTA) involves an osteotomy of the non–weight-bearing portion of the tibia. The patellar ligament is aligned perpendicularly to the common tangent of the femorotibial joint, eliminating CTT.[67] When the paw is loaded during weight bearing, a force is created through the foot to the metatarsus and tarsus that causes the calcanean (Achilles) tendon to react with a second force to maintain the stability of the tarsus at its weight-bearing angle. A vector force (sum of the resulting forces of weight bearing) occurs in the tarsus, creating a simultaneous force through the patellar ligament necessary to stabilize the stifle. A combination of forces in the stifle results in a vector force in a plane almost parallel to the patellar ligament at the standing weight-bearing angle of the stifle (135 degrees). This is the total joint force across the stifle joint at normal weight bearing. If the slope of the tibial plateau is not anatomically oriented perpendicular to the patellar ligament on weight bearing, then the vector force does not superimpose the normal joint compressive force. A tibiofemoral shear force, known as the CTT force (in the direction of cranial drawer or tibial translation), results and is accommodated for in the normal animal by the CCL.

TTA positions the patellar ligament perpendicular to the slope of the tibial plateau by advancing its insertion in a cranial direction, eliminating the tibiofemoral shear force with weight bearing and relieving the function of the CCL.[67] The TPLO procedure accomplishes essentially the same redirection of vector force by rotating the tibial plateau to neutralize the tibiofemoral shear force. However, TPLO may increase the tension on the patellar ligament, whereas TTA theoretically reduces patellar ligament tension. TTA appears to result in less postoperative patellar ligament inflammation than the TPLO. TTA does not affect joint congruency; however, it does increase loading of the caudal cruciate. This may be partially offset by the general reduction of internal joint reactions as a result of a longer patellar ligament lever arm.

Regardless of the technique used to stabilize the stifle, the meniscus should be inspected by open arthrotomy or arthroscopy for tears or other evidence of trauma. Damage to the caudal body of the medial meniscus is seen in 50% to 75% of patients with a torn CCL.[68,69] Most of these patients have a bucket handle tear (see Fig. 34.108B), which must be excised.

Preoperative Management

Activity of patients with torn CCLs should be limited before surgery to prevent further damage to the articular cartilage or meniscus. Perioperative antibiotics (see Chapter 9) and preemptive pain management with opiates (see Box 32.1), NSAIDs (see Table 32.4), or epidural analgesia (see Table 32.3) are indicated for dogs undergoing stifle reconstructive techniques.

Anesthesia

General anesthesia recommendations for orthopedic patients are provided in Tables 32.1 and 32.2. Opioid epidural administration reduces postoperative discomfort (see Table 32.3). Intraarticular administration of 0.5% bupivacaine (0.5 mL/kg) or morphine (0.1 mg/kg diluted with saline to a volume of 0.5 mL/kg) before skin closure also reduces postoperative discomfort.

Surgical Anatomy

Knowledge of the origin and insertion of normal ligamentous structures and menisci of the stifle joint is important when

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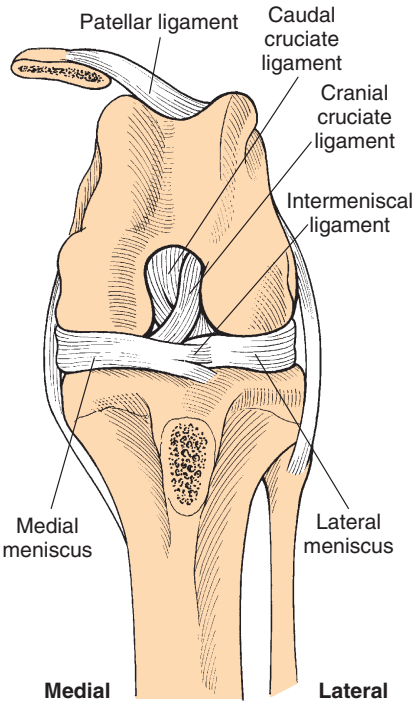


FIG. 34.111 Orientation of the cruciate ligaments and menisci.

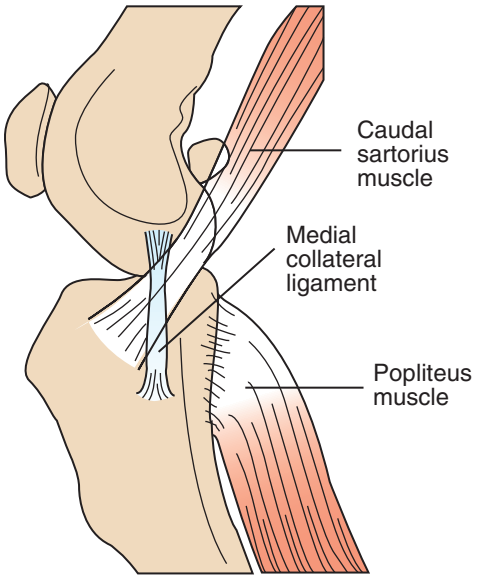


FIG. 34.112 Anatomy of the medial aspect of the stifle for tibial plateau leveling osteotomy or tibial tuberosity advancement surgery.

considering arthroscopic exploration or surgery to repair a cruciate ligament rupture. The tendon of the LDE muscle originates from the extensor fossa of the lateral femoral condyle and is located directly below the lateral arthrotomy incision. The CCL originates from the inside (medial) surface of the lateral femoral condyle (Fig. 34.111), courses distal and medial, spirals 90 degrees, and inserts onto the craniomedial surface of the tibial plateau beneath the intermeniscal ligament. The caudal cruciate ligament is visible as a broad expanse of ligament located in the intercondylar notch. The medial and lateral menisci are fibrocartilaginous discs that have a semilunar shape and are attached to the tibia and surrounding soft tissue (see p. 1243).

If an extracapsular reconstruction is chosen as a method of treatment, care must be taken to prevent injury to the peroneal nerve, which courses lateral and caudal to the stifle. The fabellae are located between the femoral condyles and the distal femoral diaphysis and sit in the origin of the gastrocnemius muscle. Osteoarthritis causes osteophytosis of the fabellae and may complicate passage of a needle and suture around the fabella. The lateral collateral originates on the lateral femoral condyle and inserts on the fibular head. The fibula is connected to the tibia proximally by the ligament of the fibular head.

The medial collateral ligament is used as an approximate landmark for the distal aspect of the TPLO osteotomy. It inserts broadly several centimeters below the tibial plateau at the caudal aspect of the tibia (Fig. 34.112). The popliteal artery and vein are located just caudal to the tibia proximally and must be protected during the osteotomy. The medial saphenous vein lies on the medial aspect of the tibia distally and should be protected during insertion of the distal jig pin.

Positioning

For arthroscopy, the patient is positioned in dorsal recumbency. The limb should be prepared in a suspended position and clipped and prepared for aseptic surgery from the hip to the tarsus. Prepare the limb as for open surgery in case an arthrotomy is required or for cruciate stabilization following the arthroscopy. The limb may be held by an assistant or placed in an arthroscopy brace to maintain the positioning.

For an intracapsular or extracapsular stabilization, the patient may be positioned in dorsal or lateral recumbency. The limb should be prepared in a suspended position to allow maximum manipulation during surgery. The leg should be clipped and prepared for aseptic surgery from the hip to the tarsus.

For TPLO, TTA, or TWO, the patient may be positioned in dorsal recumbency or dorsolateral oblique tilted toward the side to be operated. Some surgeons prefer to position the patient so that the operated limb can be placed flat on the operating table (or a Mayo stand for giant-breed dogs) when the limb is flexed with both the stifle and hock at 90 degrees. The leg should be clipped and prepared for aseptic surgery from the hip to the tarsus.

SURGICAL TECHNIQUE

Lateral Approach to the Stifle Joint (Video 34.9)

Make a craniolateral skin incision centered at the level of the patella (Fig. 34.113A). Begin the incision 5 cm proximal to the patella, and continue it distally 5 cm below the tibial crest. Incise the subcutaneous tissue along the same line to visualize the septum between the superficial leaf of the fascia lata and the biceps femoris muscle proximally and the lateral retinaculum distally. Make an incision through the fascia lata proximally, and carry the incision through the fascia lata and lateral retinaculum distally (Fig. 34.113B). Make an incision through the joint capsule, beginning 1 cm distal to the patella. Continue the incision proximally along a line adjacent to the patellar tendon and proximal to the patella. Then incise along the border of the vastus lateralis muscle toward the fabella (see Fig. 34.113C). Displace the patella medially to expose the cranial surface of the joint.

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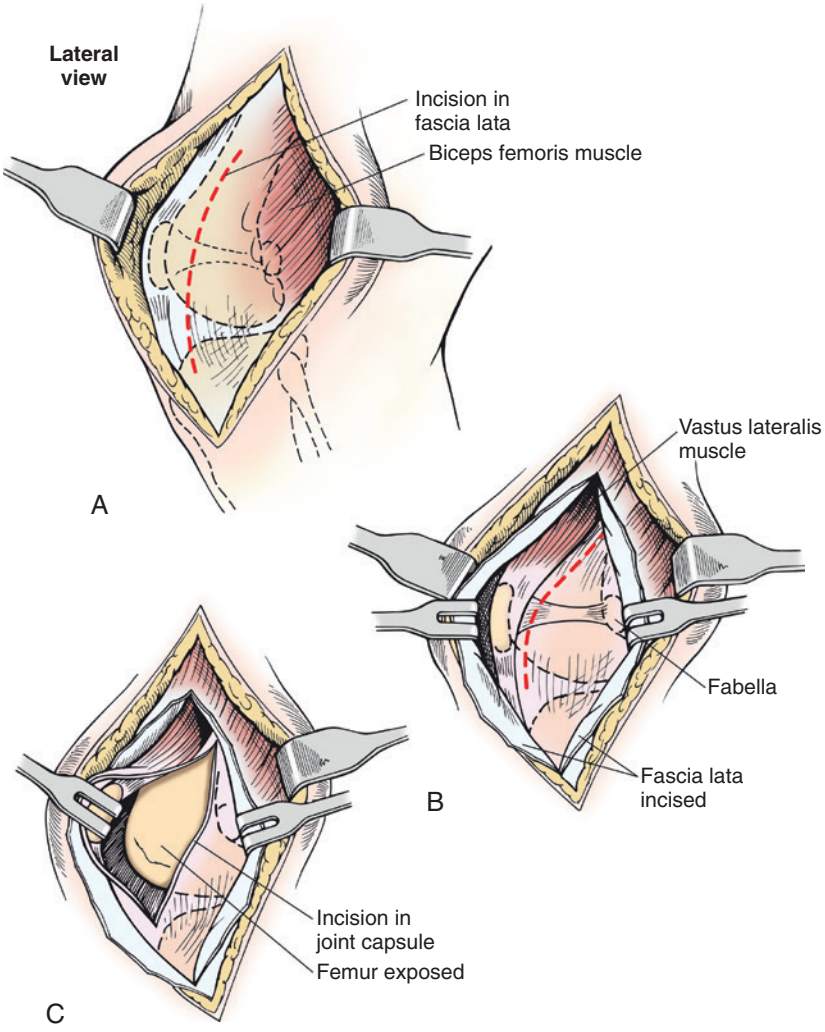


FIG. 34.113 Lateral approach to the stifle joint. (A) Make a craniolateral skin incision centered over the patella. Incise the subcutaneous tissue along the same line to visualize the septum between the superficial leaf of the fascia lata and the biceps femoris muscle proximally and the lateral retinaculum distally. (B) Make an incision through the fascia lata proximally and carry the incision through the fascia lata and lateral retinaculum distally. (C) Incise the joint capsule and continue the incision proximally adjacent to the patellar tendon. Then incise along the border of the vastus lateralis toward the fabella. Displace the patella medially to expose the cranial surface of the joint.

Medial Approach to the Stifle Joint

Make a craniomedial incision centered at the level of the patella (Fig. 34.114). Begin the incision 5 cm proximal to the patella, and continue distally 5 cm below the tibial crest. Incise subcutaneous tissue along the same line to expose the parapatellar medial retinaculum. Make an incision through the medial retinaculum and joint capsule adjacent to the medial ridge of the patellar tendon. Continue the incision proximally to the extent of the suprapatellar joint capsule and distally to the tibial tuberosity.

Lateral Extracapsular Stabilization (Videos 34.10 and 34.11)

Perform arthroscopy or an arthrotomy as described previously, resect the remnants of the CCL, and inspect the meniscus for tears or damage. If the meniscus is torn, remove the damaged portion. If the meniscus is intact, perform a meniscal release if desired, and close the arthrotomy or arthroscopy portals.

If not already done, perform the lateral approach to the stifle joint. Retract the skin laterally and make an incision through the lateral retinaculum and distal fascia lata. This incision has already been made if the stifle has been approached laterally. *Elevate the biceps femoris muscle from the lateral surface of the joint capsule to expose the gastrocnemius muscle. Pass monofilament nylon sutures, monofilament nylon leader material (No. 2 monofilament nylon suture for dogs up to 10 kg; nylon leader material [27 kg test] for dogs up to 30 kg and 36 kg test for dogs more than 30 kg) around the fabella from proximal to distal (Fig. 34.115). Alternatively, use orthopedic suture (size 2 FiberWire for dogs up to 10 kg; size 5 FiberWire for dogs more than 10 kg).* The suture may also be anchored using a suture anchor in the lateral femoral condyle (Fig. 34.116). *Place the anchor as far caudally as possible at the distal pole of the lateral fabella. Next, pass the suture behind the patellar ligament immediately proximal to the tibial tuberosity. Drill a hole of sufficient size to pass the needle through the tibial crest from medial to lateral, and pass the suture through the hole in the same direction. Alternatively, drill two holes in the tibial crest and pass the suture through the holes from medial to lateral and then lateral to medial (see Fig. 34.115). Cut the suture to remove the needle, obtaining two sutures to tie. Flex the stifle to a normal standing angle, hold the tibia caudally and rotated externally to remove drawer motion, and tie or crimp the sutures (see Fig. 34.115).* The efficacy of the crimp clamp system of securing a loop of monofilament nylon leader material was compared with knotted loops. The crimp clamp system has the potential for eliminating knot irritation and providing surgeons more consistent initial loop tensions, less loop elongation, and higher load to failure characteristics than knotted monofilament leader material for clinical stabilization of the cruciate-deficient stifle.

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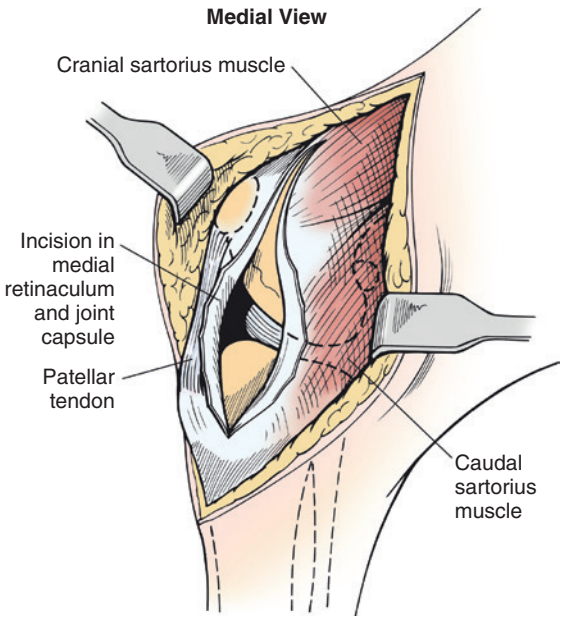


FIG. 34.114 Medial approach to the stifle joint. Make a craniomedial incision centered over the patella. Incise the subcutaneous tissue along the same line to expose the parapatellar medial retinaculum. Make an incision through the medial retinaculum and joint capsule adjacent to the medial ridge of the patellar tendon.

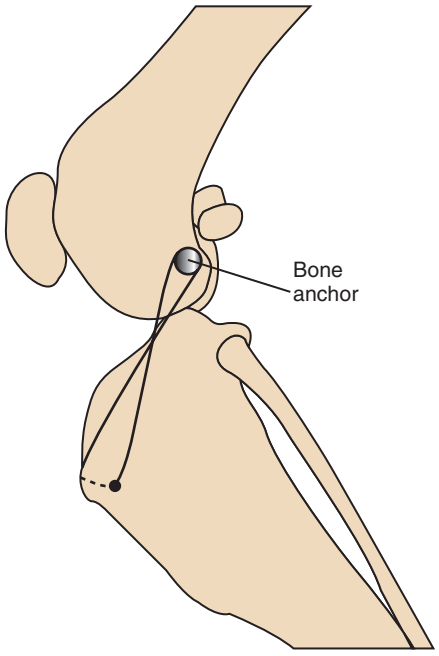


FIG. 34.116 An extracapsular reconstruction using a heavy, nonabsorbable suture and suture anchors.

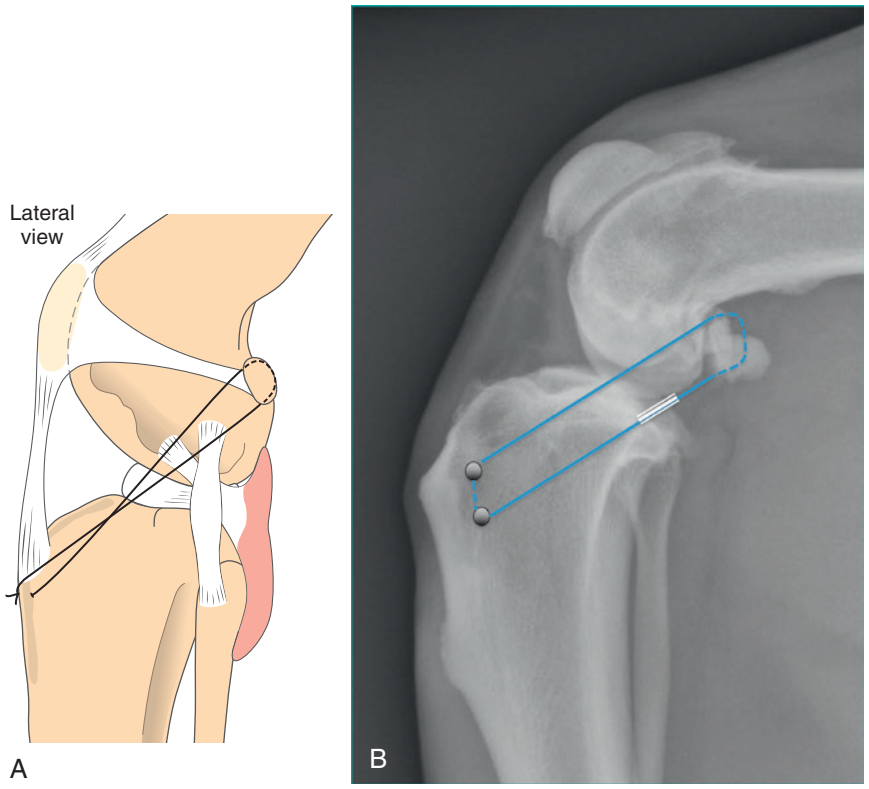


FIG. 34.115 An extracapsular reconstruction using a heavy, nonabsorbable suture (or sutures). The suture passes through deep fascia surrounding the fabella and through a predrilled hole (A) or two holes (B) in the tibial crest. Tying the suture eliminates the cranial drawer.

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In addition to the stabilizing suture, advance the lateral retinaculum cranially and distally by placing a series of vertical mattress imbricating sutures (vest over pants). Place each suture through the retinaculum caudal to the arthrotomy line, cross them superficial to the arthrotomy, penetrate in and out of the retinaculum cranial to the arthrotomy and distal to the initial position of the suture on the caudal retinaculum, and return to pass the suture through the caudal retinaculum. Preplace individual sutures and tie when all sutures in the series are in place. Proper placement of sutures draws the caudal retinaculum over the cranial retinaculum. *Close as described previously.*

Tightrope Stabilization

Perform arthroscopy or an arthrotomy as described previously, resect the remnants of the CCL, and inspect the meniscus for tears or damage. Remove the damaged meniscus, if present, or perform a meniscal release. Document all pathology in the joint and close the joint capsule.

If not already performed, expose the lateral aspect to the stifle joint and make an incision through the lateral retinaculum and distal fascia lata. This incision has already been made if the stifle has been approached laterally. *Palpate the junction of the lateral fabella with the caudal aspect of the lateral femoral condyle. Position a TR CCL guide wire to enter the femur ~2 mm distal to the lateral fabella-femoral condyle junction and within the caudal-most portion of the lateral femoral condyle (Fig. 34.117A). Take great care to ensure precise placement of the guide wire at this anatomic location. Advance the guide wire using a wire driver or drill at a proximally directed angle such that the wire traverses the distal femur and exits the distal diaphysis of the femur on the medial side immediately caudal to the vastus medialis muscle.* The wire should exit at the level of the proximal pole of the patella with the stifle at a weight-bearing angle. Optimal placement of the guide wire is ensured such that its entry point is in the caudal aspect of the lateral femoral condyle, it does not enter the joint, and its exit point allows for button or toggle placement directly onto dense cortical bone of the distal femur. *Using the 3.5-mm TR CCL cannulated drill bit, drill over the guide wire from lateral to medial. Once the drill bit exits the medial femoral cortex, remove the drill bit and*

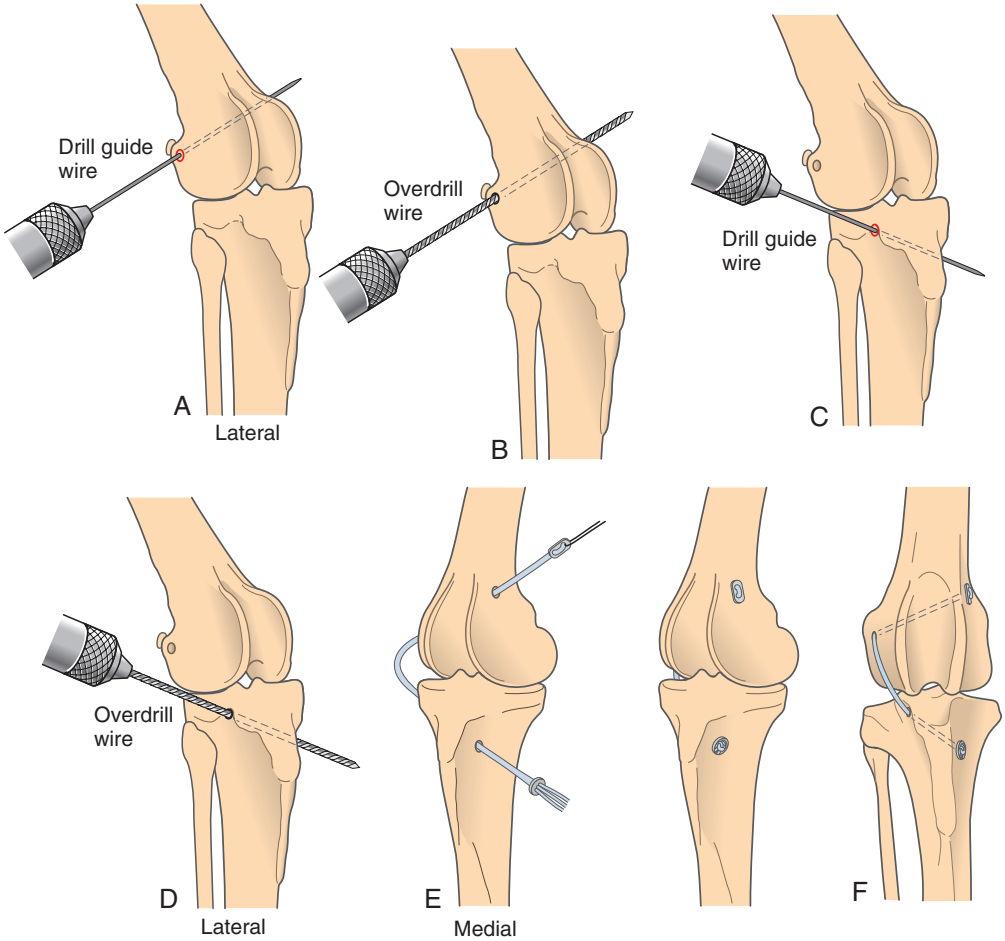


FIG. 34.117 (A) Drill a tightrope cranial cruciate ligament guide wire entering the femur ~2 mm distal to the lateral fabella-femoral condyle junction and within the caudal-most portion of the lateral femoral condyle. (B) After the wire is properly placed, overdrill the wire. (C) Retract the long digital extensor cranially and drill a second guide wire such that it starts as caudal and proximal as possible within the groove and exits the proximal tibia on the medial side. (D) After the wire is properly placed, overdrill the wire. (E) Place the tightrope beginning at the medial aspect of the tibial hole and ending at the medial aspect of the femoral hole. (F) Tighten the tightrope and secure it with multiple throws.

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guide wire and place another wire or pin into the femoral tunnel to mark its location for subsequent placement of the TR implant.

Palpate the LDE tendon within the muscular groove of the proximal lateral tibia and make a 5- to 10-mm incision in the fascia and joint capsule immediately caudal and parallel to the LDE. Retract the LDE cranially to allow for placement of the TR guide wire within the muscular groove such that it starts as caudal and proximal as possible within the groove without entering the joint (Fig. 34.117B). Advance the guide wire into the proximal tibia using a wire driver or drill at a distally directed angle such that the wire traverses the proximal tibia and exits the proximal metaphysis of the tibia on the medial side. Optimal placement of the guide wire is ensured such that its entry point is in the caudal aspect of the muscular groove at the level of the tubercle of Gerdy proximally, it does not enter the joint, and its exit point allows for toggle or button seating directly onto dense cortical bone of the proximal tibia. *Using the TR 3.5-mm cannulated drill bit, drill over the guide wire from lateral to medial. Once the drill bit exits the medial tibial cortex, remove the drill bit and guide wire and place another wire or pin into the tibial tunnel to mark its location for subsequent placement of the TR implant.*

Expose the marking wire or pin at the medial orifice of the tibial tunnel by retracting the skin incision (or making a small skin incision over this site if using a mini-incision technique after arthroscopy). Pass the TR leader needle through the tibial tunnel from medial to lateral. Apply tension laterally on the TR needle and medially on the fiber tape sutures to align the TR toggle along the axis of the tibial tunnel.

Pull the TR toggle through the tibial tunnel from medial to lateral until it exits on the lateral aspect of the stifle while the LDE is retracted cranially (Fig. 34.117C). Push the TR needle through the femoral tunnel from lateral to medial. Pull the TR needle through the orifice on the medial aspect of the femur, and apply tension on the TR needle medially and on the fiber tape sutures laterally to align the TR toggle button along the axis of the femoral tunnel. Advance the TR toggle through the femoral tunnel from lateral to medial until it exits on the medial aspect of the femur, staying deep to the vastus medialis. Flip the toggle to align it perpendicular to the femoral tunnel, and pull the fiber tape suture on the lateral side of the femur to seat the toggle firmly against the medial femoral cortex. Pull the fiber tape tight on the lateral aspect of the stifle, and remove any twists so that the strands sit flat and firmly against the lateral joint capsule deep to the fascia. Pull the fiber tape suture strands taut on the medial aspect of the tibia, and seat the TR button completely on tibial cortical bone. Hold the stifle at a weight-bearing angle in slight external rotation, and tie the free ends of the fiber tape sutures over the button. A tensioning device aids in obtaining ideal tension on the fiber tape sutures and is recommended for optimal outcomes. *Place both strands of one of the fiber tape sutures in the TR CCL tensioner channel and the ratcheting turnstile. Use the tensioner to tighten the fiber tape strands directly against the button. Place an initial tension of 10 to 12 lb on the strands and cycle the stifle through full flexion and extension ~20 times while assessing cranial drawer, tibial thrust, and internal rotation until the initial tension is maintained after cycling and the desired degrees of stability are achieved.*

Take the tensioner off the first strands and place it on both strands of the other fiber tape suture and repeat the tensioning process. Once the second suture has been pretensioned, hold the desired tension on these strands with the tensioner and tie the first suture with four to five secure throws. Remove the tensioner and tie the second suture in a similar manner. Check the stifle again for cranial drawer, internal rotation, and range of motion to ensure correct implant placement and stifle stabilization. Cut the TR pull-through suture and remove the TR needle and pull-through suture. Advance the caudal band of the sartorius over the tibial button and knots, and suture it to the cranial fascia.

In addition to the TR suture, advance the lateral retinaculum cranially and distally by placing a series of imbricating sutures (modified Mayo mattress). Place each suture through the retinaculum caudal to the arthrotomy line, cross them superficial to the arthrotomy, penetrate in and out of the retinaculum cranial to the arthrotomy and distal to the initial position of the suture on the caudal retinaculum, and return to pass the suture through the caudal retinaculum. Preplace individual sutures and tie when all sutures in the series are in place. Proper placement of sutures draws the caudal retinaculum over the cranial retinaculum. *Close as described previously.* The TR CCL device can be placed in the opposite order as described earlier, that is, medial to lateral through the femoral tunnel then lateral to medial through the tibial tunnel such that the toggle provides tibial fixation and the button provides femoral fixation. A mini–TR CCL implant system is available that allows use of this technique in smaller dogs and cats.

Tibial Plateau Leveling Osteotomy

Radiographic Assessment (Video 34.12)

With the patient under general anesthesia, position the dog in lateral recumbency with the limb to be radiographed dependent. Elevate the opposite limb out of the x-ray beam. Position a radiographic plate to include the distal femoral condyles, the entire tibia, and the tarsal joint (Fig. 34.118). Position the limb so that the femoral condyles overlap and the trochlear ridges of the talus overlap. Complete the radiograph and obtain a craniocaudal radiograph of the limb including the same structures. The most accurate measurements of TPA are made when there is less than 2 mm difference between the two femoral condyles and when the beam is centered toward the tibial plateau. *On the lateral radiograph, mark the center of the trochlea of the talus and the center of the intercondylar eminence of the tibial plateau. Connect these two points with a line (line a) (Fig. 34.119). Draw a second line (line b) to estimate the tibial plateau (see Fig. 34.119). Where these two*



FIG. 34.118 Positioning for radiography before tibial plateau leveling osteotomy or tibial tuberosity advancement surgery.

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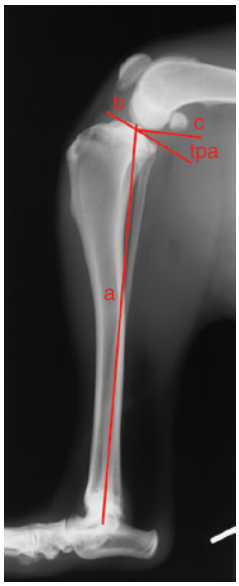


FIG. 34.119 To measure the tibial plateau angle (TPA): on the lateral radiograph, mark the center of the trochlea of the talus and the center of the intercondylar eminence of the tibial plateau. Connect these two points with a line (*line a*). Draw a second line to estimate the tibial plateau (*line b*). Where these two lines intersect, draw a third line (*line c*) perpendicular to the first line. Measure the angle between lines *b* and *c*. This is the TPA.

TABLE 34.11 Rotational Charts for Tibial Plateau Leveling Osteotomy

	ROTATION			
TPA	12 mm	18 mm	24 mm	30 mm
15	2.00	3.00	4.25	5.25
16	2.25	3.25	4.50	5.75
17	2.50	3.75	5.00	6.25
18	2.70	4.00	5.50	6.75
19	2.90	4.25	6.00	7.25
20	3.00	4.50	6.25	7.75
21	3.25	4.75	6.75	8.30
22	3.50	5.00	7.00	8.85
23	3.70	5.50	7.50	9.40
24	3.90	5.75	8.00	10.00
25	4.00	6.00	8.25	10.40
26	4.25	6.25	8.75	11.00
27	4.50	6.75	9.00	11.50
28	4.70	7.00	9.50	12.00
29	4.90	7.25	10.00	12.50
30	5.00	7.50	10.25	13.00
31	5.25	8.00	10.75	13.50
32	5.50	8.25	11.00	14.00
34	5.70	8.50	11.50	14.50
34	5.90	8.75	12.00	15.00
35	6.00	9.00	12.25	15.50
36	6.25	9.50	12.75	16.00
37	6.50	9.75	13.00	16.50
38	6.70	10.00	13.50	17.00
39	6.90	10.25	14.00	17.50
40	7.00	10.50	14.25	18.00

TPA, Tibial plateau angle.

lines intersect, draw a third line (line c) perpendicular to the first line (see Fig. 34.119). Measure the angle between lines (b) and (c). This is the TPA. Using the conversion chart appropriate to the selected osteotomy blade (Table 34.11), determine the degree of rotation. The osteotomy should be performed to allow space for the proximal aspect of the bone plate while preserving adequate tibial crest to prevent fracture. The osteotomy should exit the caudal aspect of the tibia perpendicular to the bone without an "uphill" or "downhill" exit.

Surgical Technique

Perform arthroscopy or an arthrotomy as described previously, resect the remnants of the CCL, and inspect the meniscus for tears or damage. If the meniscus is torn, remove the damaged portion. If the meniscus is intact, perform a meniscal release if desired, and close the arthrotomy or arthroscopy portals. *Make a medial skin incision centered at the level of the proximal tibia (Fig. 34.120A). Begin the incision 3 cm proximal to the tibial plateau and continue it distally 5 cm below the level of the tibial crest. Incise the subcutaneous tissue along the same line sharply or with electrocautery to visualize the insertion of the cranial head of the sartorius. Incise the insertion of the sartorius, and reflect the muscle caudally to visualize the medial collateral ligament and the caudal aspect of the proximal tibia (Fig. 34.120B). Sharply incise the origin of the popliteus muscle from the caudomedial aspect of the tibia (Fig. 34.120C). Bluntly dissect the origin of the muscle from the caudal aspect of the tibia to the lateral border. Once elevated, place a moistened sponge in between the muscle and the bone to protect the muscle and popliteal artery and vein during the osteotomy. Be certain to keep the periosteal elevator against the bone to prevent damage to the popliteal artery and vein. Insert a jig pin perpendicular to the sagittal and parallel to the transverse planes, starting at the proximal-caudal point that represents the center of rotation for the osteotomy. Advance the jig pin until it engages both cortices of the tibia. Assemble the jig on the proximal jig pin and determine the position for the distal pin. Make a 1-cm skin incision over the center of the tibial diaphysis at the location for the distal jig pin, taking care to protect the medial saphenous vein. Drive the distal jig pin through the jig and through the center of the tibia, engaging both medial and lateral diaphyses. Cut the proximal jig pin short while leaving the distal jig pin long (Fig. 34.121A–B).* Placing the jig pins may be performed with the dog in dorsal recumbency and the limb held up or the dog in oblique recumbency so that the limb may be placed flat on the operating table. *Position the appropriate-sized biradial saw at the location for the osteotomy.* In most cases, the distal aspect of the saw intersects the distal aspect of the insertion of the medial collateral ligament. The patellar tendon may be protected by placing a Hohmann retractor caudal to the ligament and having an assistant retract it cranially. Additional protection may be afforded by elevating the cranial tibial muscle off the tibia on the lateral side and placing a sponge between the muscle and the bone. *Start the osteotomy by placing the saw at an oblique angle to the bone so the saw initially engages only the edges of the bone. Then bring the saw to a position*

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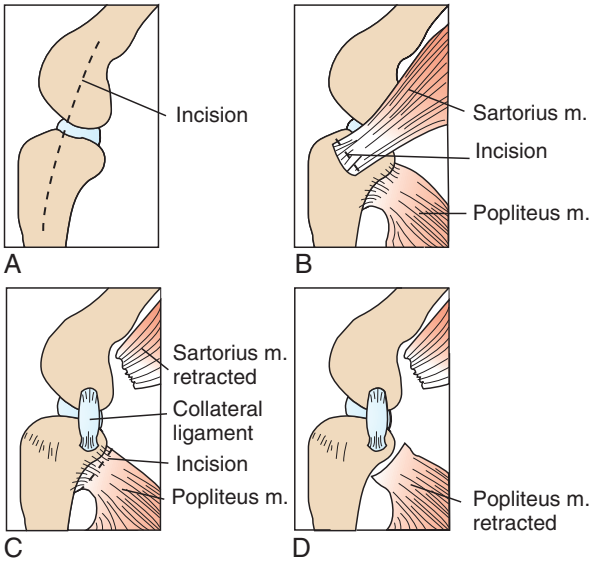


FIG. 34.120 Medial approach to the proximal tibia. (A) Make a medial skin incision centered at the level of the proximal tibia. Begin the incision 3 cm proximal to the tibial plateau and continue it distally 5 cm below the level of the tibial crest. Incise the subcutaneous tissue along the same line sharply or with electrocautery to visualize the insertion of the cranial head of the sartorius muscle. (B) Incise the insertion of the sartorius muscle and reflect it caudally to visualize the medial collateral ligament and the caudal aspect of the proximal tibia. (C) Sharply incise the origin of the popliteus muscle from the caudomedial aspect of the tibia. Bluntly dissect the origin of the muscle from the caudal aspect of the tibia to the medial border. (D) Once elevated, place a moistened sponge in between the muscle and the bone to protect the muscle and popliteal artery and vein during the osteotomy.

perpendicular to the bone and parallel to the jig pins. The saw should be roughly centered over the proximal jig pin and parallel to both pins. *Make an initial superficial cut with the saw, then stop to evaluate the osteotomy position (Fig. 34.121C). Evaluate the thickness of the tibial crest, the area available for the bone plate, and the angle of the osteotomy as it exits the caudal aspect of the tibia.* The bone may be cut with the dog in dorsal recumbency and the limb held up or the dog in oblique recumbency so that the limb may be placed flat on the operating table. The cut should preserve enough tibial crest to prevent fracture, allow ample room for the bone plate, and exit the bone caudally perpendicular to the bone. *After confirming the correct position of the osteotomy, continue the osteotomy while lavaging the saw blade with cool saline until the tibia is cut approximately 50%. Using an osteotome, make marks on either side of the osteotomy the appropriate distance apart as determined by the TPA, the saw size, and the appropriate rotation chart (Fig. 34.121D). Complete the osteotomy and remove the protective gauze sponges.*

Insert a large pin into the cranial proximal medial aspect of the proximal bone segment, directing the pin distally, caudally, and laterally. This is the rotation pin. *Rotate the proximal segment distally and caudally so that the marks line up (Fig. 34.121E).* It may be necessary to pass an elevator between the osteotomy segments and to pry slightly to permit easier rotation. *Do not translate the proximal segment medially to line up the cortices because this may contribute to limb malalignment. Insert a small pin through the proximal tibial crest and into the proximal osteotomy fragment to secure the two bone pieces in the new position. Test for tibial thrust.* It is important to test for tibial thrust before placing the bone plate to ensure that thrust has been eliminated. Adjustments to the degree of rotation may be necessary if thrust still exists at this stage of the procedure. The distance of rotation can be measured at this point by measuring the amount of exposed osteotomy surface at the caudal aspect of the tibia. *Apply the appropriate-sized bone plate beginning with screws in the distal segment followed by screws in the proximal segment (Fig. 34.121F).* Contouring of the bone plate may be made simpler by removing excessive fibrous tissue on the medial aspect of the stifle joint (medial buttress). When drilling for screw placement in the proximal segment, it is useful to have the drill parallel to the jig pin to avoid accidentally entering the stifle joint (Fig. 34.122). The second and third holes of the bone plate may be used in a compressive manner to compress the osteotomy line. *If locking screws are used, apply a nonlocking screw first if contact between the plate and the bone is intended.*

Suture the insertion of the cranial head of the sartorius muscle to the deep fascia of the tibia with absorbable suture in a continuous pattern. Suture the remainder of the deep fascia with absorbable suture in a continuous pattern. Suture the superficial fascia and subcutaneous tissues with absorbable suture in continuous patterns. Suture the skin with nonabsorbable suture in a simple interrupted pattern or use skin staples.

Tibial Wedge Osteotomy

Radiographic Assessment

With the patient under general anesthesia, position the dog in lateral recumbency with the limb to be radiographed dependent. Elevate the opposite limb out of the x-ray beam. Position a radiographic plate to include the distal femoral condyles, the entire tibia, and the hock joint. Position the limb so that the femoral condyles overlap and the trochlear ridges of the talus overlap. Complete the radiograph and obtain a craniocaudal radiograph of the limb including the same structures. The most accurate measurements of TPA are made when there is less than 2 mm difference between the two femoral condyles and when the beam is centered toward the tibial plateau. *On the lateral radiograph, mark the center of the trochlea of the talus and the center of the intercondylar eminence of the tibial plateau. Connect these two points with a line (a) (Fig. 34.123). Draw a second line (b) to estimate the tibial plateau. Where these two lines intersect, draw a third line (c) perpendicular to the first line. Measure the angle between lines (b) and (c).* This is the TPA. *Make a line (d) perpendicular to line (a) at the base of the flare of the tibial crest. Make another line that intersects line (d) at the caudal aspect of the tibia and creates an angle equal to the TPA minus 6 degrees.* The osteotomy should be performed to allow adequate length for the proximal aspect of the bone plate (a minimum of three screws) while being as close to the stifle joint as possible. Placing the osteotomy as close to the stifle joint as possible maximizes the biomechanical effect and decreases the risk of fracture.

Surgical Technique

Perform arthroscopy or an arthrotomy as described earlier, resect the remnants of the CCL and inspect the meniscus for tears or damage. Remove the damaged meniscus, if present, and close the arthrotomy or arthroscopy portals. *Make a medial skin incision centered at the level of the proposed osteotomy. Begin the incision*

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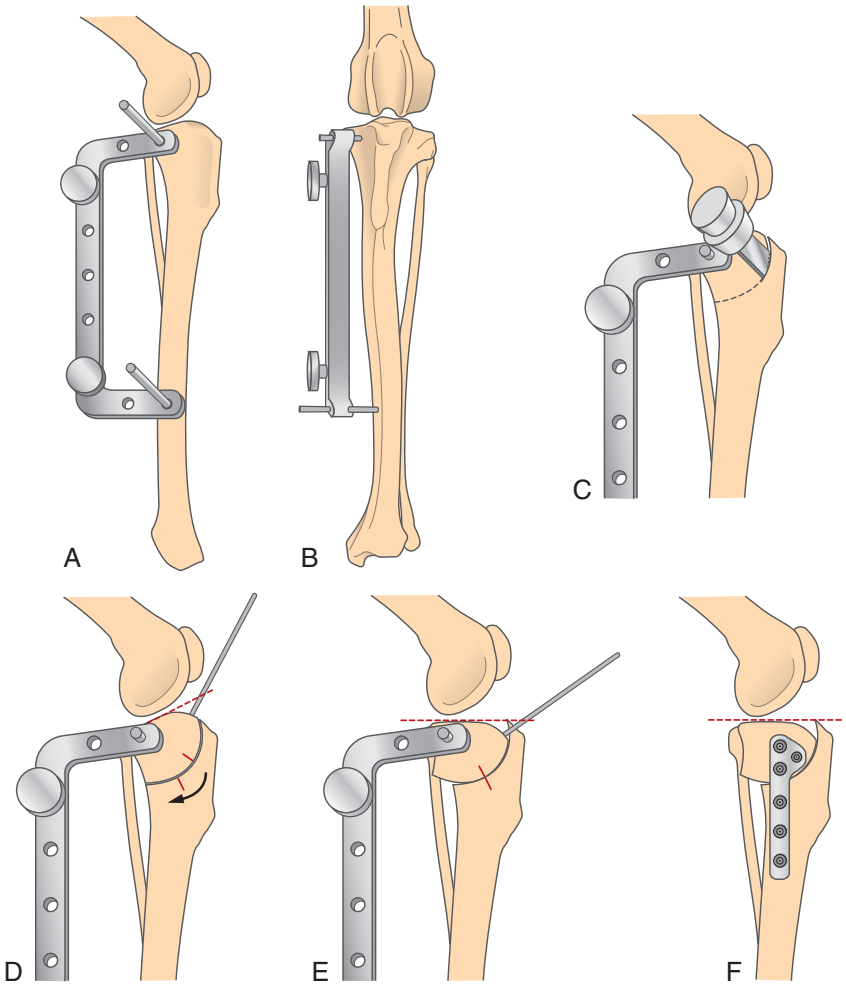


FIG. 34.121 To perform a tibial plateau leveling osteotomy: (A) and (B) Position the jig perpendicular to the long axis of the tibia. (C) Perform the osteotomy to a depth of one-third of the bone, keeping the saw parallel to the jig pins. (D) Mark the bone for rotation. (E) Rotate the proximal segment to align the marks. (F) Secure the osteotomy with the appropriate-sized bone plate.

5 cm proximal to the osteotomy site and continue it distally 5 cm below the level of the osteotomy. Incise the subcutaneous tissue along the same line sharply or with electrocautery to expose the tibia. Dissect circumferentially around the tibia and place Hohmann retractors cranially and caudally to protect the lateral structures.

Using a straight saw blade, mark both the lines of the osteotomy. Alternating between the two cuts, complete the osteotomy and remove the wedge of bone. Reduce the bone segments and apply an appropriate-sized bone plate (Fig. 34.124). In larger dogs it may be necessary to apply two parallel bone plates to achieve adequate stability. *Suture the deep fascia with absorbable suture in a continuous pattern. Suture the superficial fascia and subcutaneous tissues with absorbable suture in continuous patterns. Suture the skin with nonabsorbable suture in a simple interrupted pattern or use skin staples.*

Tibial Tuberosity Advancement

Radiographic Assessment

With the dog under general anesthesia, position it in lateral recumbency with the limb to be radiographed adjacent to the table. Elevate the opposite limb out of the x-ray beam. Position a radiographic plate to include as much of the femur as possible and the proximal half of the tibia. Ensure that the tibia is in a neutral position (i.e., it is not cranially subluxated). Position the limb so that the femoral condyles overlap. Place the stifle in a weight-bearing angle (for most dogs approximately 135 degrees of extension). Complete the radiograph and obtain a craniocaudal radiograph of the limb including the same structures. Utilize the common tangent (the tangent common to the curvature of the femoral and tibial condyles at the femorotibial contact point) method to determine the amount of advancement necessary.

Surgical Technique

Perform arthroscopy or an arthrotomy as described previously, resect the remnants of the CCL, and inspect the meniscus for tears or damage. Remove any damaged portion of the meniscus and close the arthrotomy or arthroscopy portals. Make a medial skin incision centered at the level of the proximal tibia. Expose the medial tibial crest by reflecting the insertion of the caudal belly of the sartorius, and periosteum. Elevate the fascial insertion of the sartorius muscle and related fascia proximally to the level of the patella.

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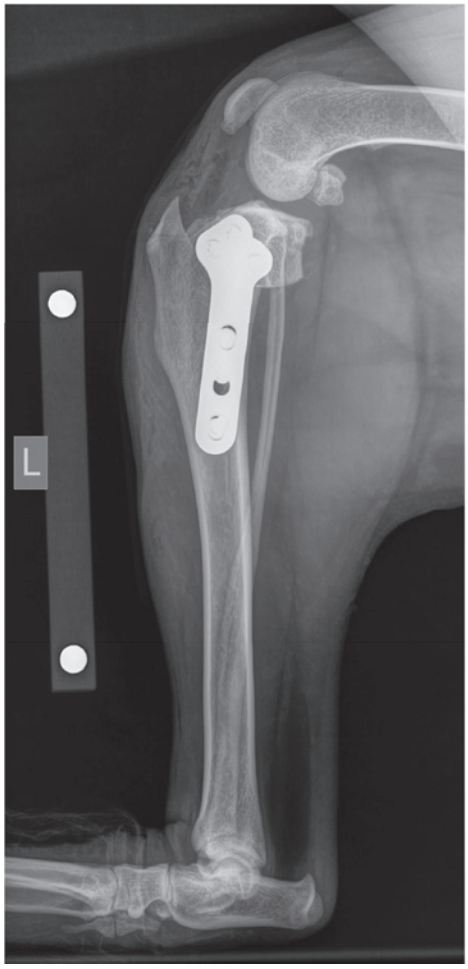


FIG. 34.122 Postoperative radiograph of a tibial plateau leveling osteotomy.

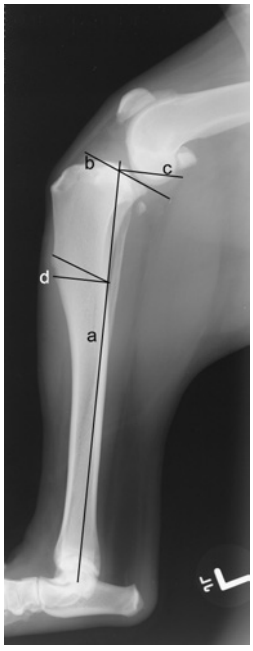


FIG. 34.123 Radiographic measurement for tibial wedge osteotomy. On the lateral radiograph, mark the center of the trochlea of the talus and the center of the intercondylar eminence of the tibial plateau. Connect these two points with a line (*line a*). Draw a second line to estimate the tibial plateau (*line b*). Where these two lines intersect, draw a third line perpendicular to the first line (*line c*). Measure the angle between lines *b* and *c*. This is the tibial plateau angle. Make a line perpendicular to line *a* at the base of the flare of the tibial crest (*line d*). Make another line that intersects line *d* at the caudal aspect of the tibia and creates an angle equal to the tibial plateau angle minus 6 degrees.

NOTE Elevation should begin approximately 1 cm caudal to the tibial crest to ensure the tissue is of adequate length for closure over the implants.

Separate the fascial attachment of the cranial tibial muscle at the level of its attachment to the cranial tibia, just distal to the termination of the tibial crest, and insert a moistened gauze sponge. Place the appropriate-sized plate over the tibial crest to assess its proper location, and ensure the fork will fit the tibial crest appropriately; the number of tines of the fork is the same as the number of holes in the plate (Fig. 34.125A).

NOTE The plate should extend beyond the cranial border of the tibia in the "nonadvanced" state, or prior to advancement, to ensure it is aligned with the tibia following advancement and does not extend beyond following advancement.

Determine the exact point for the proximal fork hole in the crest (proximal and caudal to the patellar tendon insertion on the tibial tuberosity). Position the fork drill guide on the tibia and secure it with a pointed reduction forceps. Then palpate with a drill bit to ensure that the drill holes lie over bone in appropriate locations. Note that the drill guide's holes are angled, so the hole location at the bone is distal to that observed at the surface of the guide facing the surgeon. Drill the proximal hole and insert the stabilizing pin (Fig. 34.125B). Drill the hole that corresponds to the distal hole in the plate and insert another guide pin. Drill all remaining interposed holes and remove the jig. Identify the proximal and distal crest osteotomy points. The proximal osteotomy point is the cranial bony prominence (tubercle of Gerdy) of the palpable depression on the lateral surface of the tibia (extensor groove) at the one-third and two-thirds junction between the tibial tuberosity and caudomedial tibia. The distal osteotomy point is where the crest terminates by tapering caudally onto the tibial shaft. Make sure the distal point of the proposed osteotomy does not extend distally to the level of the first screw hole in the plate. *Perform a transverse partial crest osteotomy, leaving the lateral cortex intact in the proximal one-third of the osteotomy (Fig. 34.125C).* The patellar ligament must be carefully protected when carrying the osteotomy proximally, and it is important to make the osteotomy coplanar with the frontal plane. *Remove the moist sponge from underneath the cranial tibial muscle. Contour and assemble the plate and fork, and seat the fork in the tibial crest holes using the fork inserter and mallet. Complete the osteotomy (Fig. 34.125D). Release soft tissue attachment at the portion of the tubercle of Gerdy remaining on the crest. Measure the width of the proximal tibial crest, at the osteotomy site on the tibial shaft at the location that the cage will be placed, to determine the proper cage length. Open the osteotomy*

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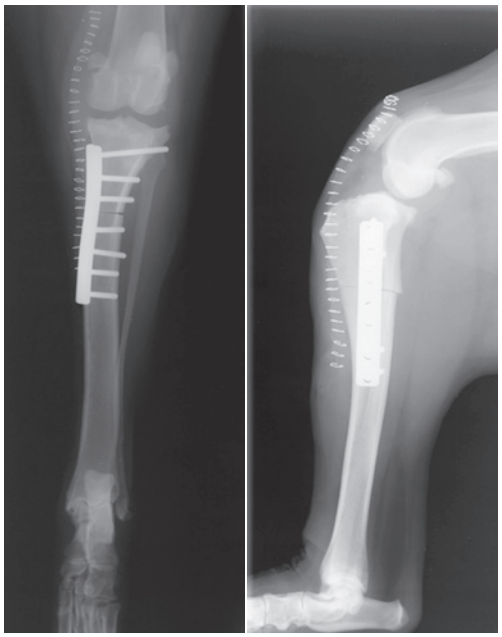


FIG. 34.124 Technique for tibial wedge osteotomy. Using a straight saw blade, mark both the lines of the osteotomy. Alternating between the two cuts, complete the osteotomy and remove the wedge of bone. Reduce the bone segments and apply an appropriate-sized bone plate.

gap, and allow the tibial tuberosity to move proximally half the advancement distance (cage size in millimeters) and insert the cage at the level of the proximal portion of the tibial osteotomy, approximately 2 to 3 mm distal to the proximal extent of the osteotomy (Fig. 34.125E). Drill the hole through the caudal cage ear (1.8 mm, directed slightly caudal and slightly distal), and place the first 2.4-mm screw in the caudal ear of the spacer. Reduce the tibial crest, and hold the distal end of the crest in contact with the tibial shaft using a pointed reduction forceps. Drill, measure, and insert self-tapping plate screws (2.7 mm for 3, 4, 5 hole/fork plate; 3.5 mm for 6, 7, 8 hole/fork plate), placing the most distal screw in the plate first (Fig. 34.125F).

Recheck the position and stability of the patella to ensure it does not luxate. Insert a 2.4-mm screw in the cranial ear of spacer cage cranially through the tibial crest (see Fig. 34.125G). Fill the osteotomy gap and cage with a cancellous bone graft obtained from the distal femur, the proximal tibia, or demineralized bone allograft. Suture the deep fascia with absorbable suture in a continuous pattern. Suture the superficial fascia and subcutaneous tissue with absorbable suture in continuous patterns. Suture the skin with nonabsorbable suture in a simple interrupted pattern or use skin staples. Obtain postoperative radiographs to confirm correct implant placement.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Small Hohmann retractors are useful for inspecting the medial meniscus. Wallace stifle retractors also aid in distracting the stifle joint and visualizing the meniscus. With intracapsular repairs, instruments to drill holes in the tibia, a periosteal elevator, and an osteotome are also needed. For extracapsular repairs, use No. 2 monofilament nylon suture, nylon leader material, or orthopedic suture. Special needles are designed for encircling the fabella. A crimp clamp system may be helpful for securing the large-diameter nylon leader material. Bone anchors may be used to secure the suture proximally. Steinmann pins and wire are used to secure the fibula in a fibular head advancement procedure.

Performance of a TR technique requires a cannulated drill bit and guide pins, a power drill, and a TR or similar system. Performance of a TPLO requires a TPLO jig, a biradial saw blade and saw, TPLO bone plates and plating equipment, and a power drill. Performance of a TWO requires a sagittal saw, straight bone plates, plating equipment, and a power drill. TTA requires a sagittal saw, a power drill, and TTA implants.

POSTOPERATIVE CARE AND ASSESSMENT

After intracapsular or extracapsular repairs, a soft bandage with a lateral splint may be maintained for 24 to 48 hours. Alternatively, the limb may be left unbandaged and treated with a cold compression system or microcurrent electrical therapy. Physical rehabilitation should begin within the first 24 to 48 hours of surgical fixation. These exercises focus on safe weight bearing of the limb and improving usage. A sample physical rehabilitation protocol can be found in Table 34.12.

Immediate use of a cold compression system postoperatively can dramatically decrease swelling and pain following a TPLO, TWO, or TTA (see Chapter 11). Strict exercise restriction is required until radiographs demonstrate adequate healing. In young dogs, healing may occur within 4 weeks, whereas in older dogs, bony union may not occur until 12 weeks postoperatively. Exercise is limited to specific physical rehabilitation exercises and leash walking for several weeks followed by a gradual return to normal activity (see Table 11.2). A rigorous progressive physical rehabilitation program may enhance recovery after stifle surgery.

COMPLICATIONS

Complications of CCL surgery include infection, lack of stabilization, meniscal injury, implant complications, and progressive osteoarthritis.[60,61,65] Additional complications of TPLO are patellar tendon desmitis and tibial crest fracture. Additional complications of TTA are tibial crest fractures and patellar luxation.[65] Patients having poor outcome following CCL repair should undergo systematic evaluation for each of these potential complications.

PROGNOSIS

Long-term function for patients that have undergone a reconstructive procedure is good, and results are conflicting regarding the influence of the method of reconstruction. Most published assessments of outcome state that 85% to 90% of dogs improve after surgery.[60,61,65] DJD progresses regardless of treatment. The long-term outcome includes a decline in activity over time, an increasing level of disability, an adverse response to cold weather, and stiffness after inactivity related to progressive DJD.

MENISCAL INJURY

DEFINITIONS

Meniscal injury *(tear)* occurs when excessive crushing or shearing forces associated with stifle injury result in meniscocapsular detachment or separation in the substance of the meniscus. **Radial tears** are those that run in an axial to abaxial direction. **Circumferential tears** are those that follow the curvature of the meniscus.

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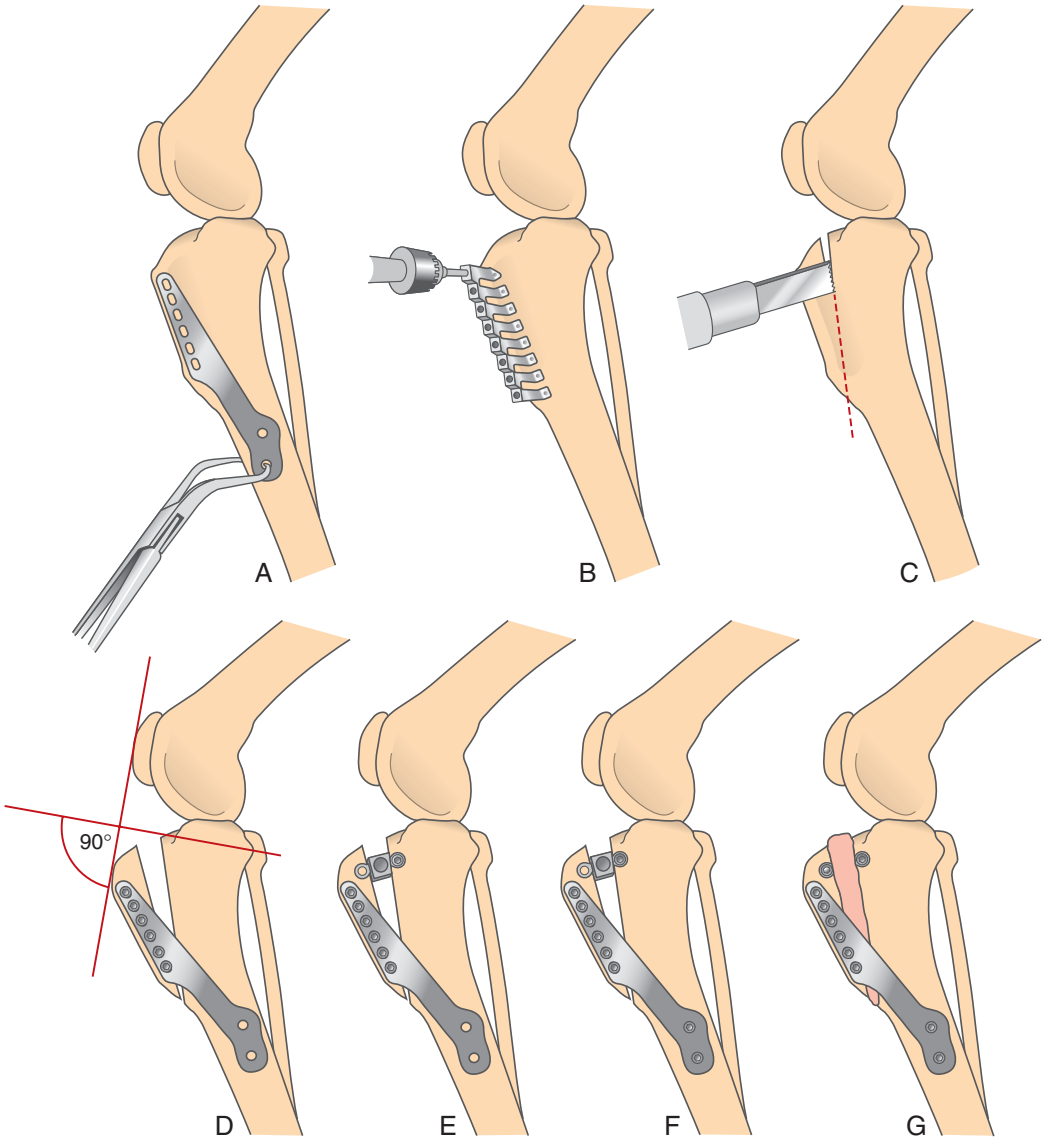


FIG. 34.125 Technique for tibial tuberosity advancement. (A) Position the proper sized plate on the tibial crest to assess its selection. (B) Place the fork template over the crest and drill the holes beginning with the proximal pilot hole, the most distal hole, and then the remaining holes. (C) Perform a transverse partial crest osteotomy, leaving the lateral cortex intact. (D) Seat the plate into the tibial crest, then complete the osteotomy. (E) Open the osteotomy gap and insert the cage at the level of the proximal aspect of the osteotomy and secure with a screw through the caudal cage ear. (F) Insert the screws through the plate beginning with the most distal screw. (G) Insert the cranial cage ear screw, fill the gap with bone graft, and close the surgical site.

Bucket handle tears are circumferential or transverse tears with separation of the meniscus at the site of the tear. **Meniscal release** is a midbody or meniscotibial incision of the medial meniscus intended to prevent future meniscal impingement and damage.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The menisci are important intraarticular structures. They function in load transmission and energy absorption, help provide rotational and varus-valgus stability, lubricate the joint, and render joint surfaces congruent. Isolated meniscal injuries are uncommon in dogs. Most meniscal tears causing lameness in dogs occur in conjunction with CCL ruptures.[68,69] These tears usually involve the caudal body of the medial meniscus because the craniocaudal instability associated with CCL rupture displaces the medial femoral condyle caudally during stifle joint flexion. The caudal body of the medial meniscus becomes wedged between the femur and the tibia and is crushed on weight bearing and joint extension. The most common type of injury is a bucket handle tear. This is a circumferential or transverse tear in the caudal body of the medial meniscus that extends from medial to lateral. The free portion of the meniscus frequently is folded forward (Fig. 34.126). Peripheral meniscal tears are the second

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TABLE 34.12 Sample Rehabilitation Protocol for Patient With a Cranial Cruciate Ligament Rupture Extracapsular Repair

Treatments/ Modalities	Day 1–3	Day 4–14	2–6 wk	6–12 wk	12+ wk
Pain medications	As directed	As directed	PRN	PRN	PRN
Cryotherapy	15–20 min 2–3 times daily	15–20 min following exercise	PRN	PRN	PRN
Heat therapy		Apply heat to the thigh muscles before exercise or if dog is stiff but not if inflammation is still present	PRN	PRN	PRN
Massage	5 min 3 times daily prior to starting exercises, massage from toes toward heart	Continue twice daily	Twice daily	Twice daily	Only if desired
PROM	10 repetitions 2 times daily stifle flexion and extension	Continue twice daily 10–20 reps	Continue as before until normal range of motion is achieved	PRN	PRN
Laser therapy	Daily or every other day for first week	Every other day for first week then 2 times weekly	Twice weekly	Twice weekly	Discontinue
Walks	5-min supported leash walk 2–3 times daily	As for first few days	Increase by 5 min each week 2–3 times daily	Increase by 5 min each week Add hills or inclines	15- to 20-min walks 2–3 times daily Add slow jogging
NMES	Quadriceps and biceps femoris 15 min daily	Do daily until dog is using leg well			
Balancing		5 min twice daily on soft foam pad	5 min twice daily	5 min twice daily on balance disc	Replace with hill walks
Cavalettis/ obstacles			5 reps × 6 rails to start	5 min twice daily	5 min twice daily
Sit to stand			Start at 4 weeks postoperative, 5–10 reps twice daily	15–20 reps twice daily	15–20 reps raising rails
Hills				Zigzag low, slow hills 5 min up and down	Increase to 10 min twice daily
Underwater treadmill		10 min 3 times weekly after sutures are removed or if incision is healed	10 min 3 times weekly	15–20 min twice weekly	15–30 min twice weekly until released from rehab
Swimming			2–5 min twice 2–3 times weekly starting at 4–6 weeks	5–15 min several times weekly	10–20 min 3 times weekly

NMES, Neuromuscular electrical stimulation; *PRN,* as needed; *PROM,* passive range of motion.

most common type of meniscal injury. These injuries are associated with a severe traumatic episode that results in multiple ligament injuries. Rupture of the medial meniscocapsular ligament commonly occurs, allowing the entire meniscal body to fold forward.

NOTE Isolated meniscal tears are uncommon; they usually occur in conjunction with cruciate ligament injury.

Meniscal release has been advocated as a means of protecting the medial meniscus following surgical stabilization of the stifle for CCL rupture. This procedure was developed in association with TPLO, which provides active stabilization of the stifle during ambulation but does not prevent cranial drawer in the non-weighted limb. The use of meniscal release is controversial based on its effects on the meniscus and cartilage and its uncertain efficacy. By transecting the meniscus just caudal to the medial collateral ligament or by transecting the meniscotibial ligament, the function of the meniscus is significantly compromised because of the elimination of hoop stresses.

With either midbody release or transection of the meniscotibial ligament, the femoral condyle increases contact with the articular cartilage of the tibial plateau, which contributes to osteoarthritis. Release also impairs the functions of the meniscus in providing stability and congruence. To date, no clinical studies have demonstrated the efficacy of meniscal release in decreasing the incidence of post-TPLO meniscal injury; however, the technique remains in widespread use.

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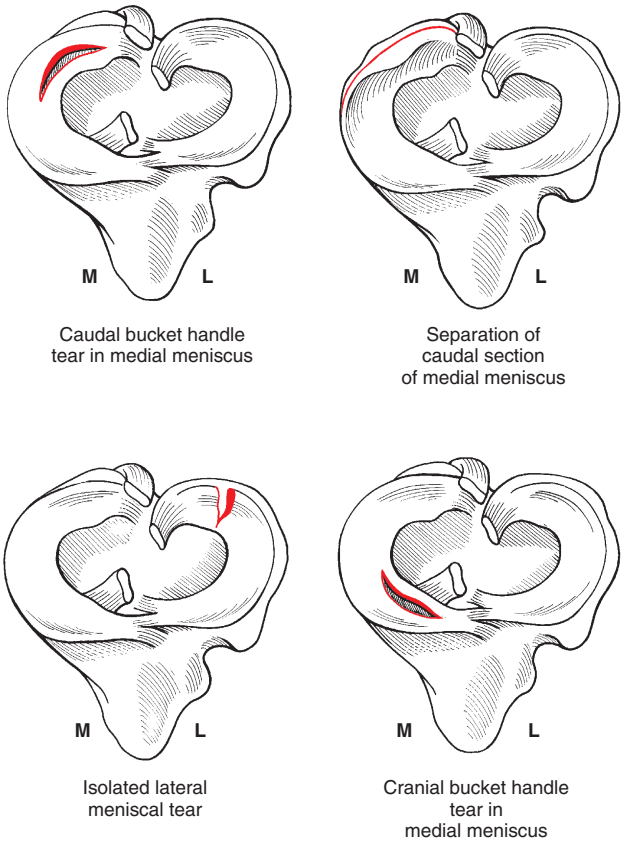


FIG. 34.126 Meniscal tears in dogs. Bucket handle tears occur most often.

DIAGNOSIS

Clinical Presentation

Signalment

Dogs of either gender and any age or breed may be affected. Meniscal tears are rare in cats.

History

Meniscal injury is usually associated with instability of the stifle caused by CCL rupture (see p. 1227). Dogs with meniscal injury and cruciate ligament injury may be in substantially more pain than dogs with cruciate ligament injury alone. If a long-standing lameness suddenly worsens in a dog with CCL injury, tearing of the meniscus should be suspected. The client may report hearing a popping sound when the dog walks, or a popping sound may be noted when the stifle is examined. This is caused by movement of the "free" section of the bucket handle tear.

Physical Examination Findings

Dogs with meniscal injury are frequently in much more pain than dogs with only CCL tears. A meniscal click may be palpable and is most often felt during the later stage of flexion of the joint.[68] Not all patients with meniscal tears have an audible or palpable click. Close inspection of the medial and lateral menisci at surgery provides a definitive diagnosis.

Diagnostic Imaging

Standard craniocaudal and medial-to-lateral radiographs should be taken for diagnostic evaluation of lameness attributed to the stifle joint; however, radiographic findings do not correlate with meniscal injury. Ultrasound of the stifle has been demonstrated in a clinical trial to be effective in the diagnosis of meniscal injury. This technique may prove valuable as a minimally invasive technique in cases in which meniscal injury is not otherwise obvious. MRI has been studied for the evaluation of the stifle joint and meniscal injuries. As MRI requires general anesthesia, arthroscopic evaluation may be a more effective means of diagnosis and providing a means of therapy.[69]

Arthroscopy

Arthroscopy provides a minimally invasive means for assessment of the menisci (see p. 1230). Arthroscopic examination may require creation of a viewing window by shaving of the fat pad, manipulation of the joint by valgus and varus stress, or by positioning of an internal or external joint distractor.

Laboratory Findings

No consistent hematologic abnormalities are seen. Joint fluid analysis may be helpful in the diagnosis of partial cruciate tears; patients with meniscal tears may have cell counts that are slightly higher than those in patients with ligament tears alone.

DIFFERENTIAL DIAGNOSIS

Cruciate ligament injury should be suspected in any animal with a meniscal tear. Sprain of the medial restraints should be differentiated from meniscal injury.

MEDICAL MANAGEMENT

Conservative treatment is not an option if meniscal injury is noted on physical examination because continued back-and-forth sliding of the torn meniscus causes severe pain that will not improve with conservative management and accelerates DJD.

SURGICAL TREATMENT

Methods of treatment include partial meniscectomy, primary repair of peripheral meniscal injuries, and total meniscectomy. A partial meniscectomy involves removal of the torn section of the meniscus. Experimentally, partial meniscectomy carries less morbidity than does a total meniscectomy and is the treatment of choice for bucket handle tears of the medial meniscus. Some human orthopedic surgeons advocate primary repair of a torn meniscal body. However, because of the low morbidity associated with partial meniscectomy and difficulty in suturing meniscal body tears in dogs, primary repair should be reserved for peripheral tears. Tearing of the peripheral meniscocapsular ligaments usually occurs after significant trauma and subsequent injury to both primary and secondary restraints of the stifle joint. The medial meniscus is more commonly involved in conjunction with injury of the medial collateral restraints. Meticulous repair with interrupted sutures using an absorbable suture allows meniscocapsular tissue to heal. Total meniscectomy should be considered only when the peripheral rim of the meniscus is so damaged that primary suturing of the meniscocapsular tissue is not possible.

Meniscal Release

Two techniques are available for meniscal release: midbody transection and transection of the meniscotibial ligament.

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Selection between the two techniques is often based on surgeon preference and surgical approach, although the two approaches do appear to affect the meniscus differently. Midbody release may be performed through an open arthrotomy arthroscopically, or blind. The latter is performed without opening the joint for examination of the meniscus. This approach is not recommended because of the high incidence of meniscal injury in dogs with cruciate ligament rupture and the possibility of meniscal injury in spite of the absence of meniscal click. The menisci should be thoroughly examined in every case of CCL rupture repair. Transection of the meniscotibial ligament is most readily performed arthroscopically.

Preoperative Management

Perioperative antibiotics (see Chapter 9) and preemptive pain management with NSAIDs (see Table 34.3), opiates, or epidural analgesia are indicated for dogs undergoing stifle reconstructive techniques.

Anesthesia

See Table 32.1 for the anesthetic management of animals with orthopedic disease. Opioid epidural administration reduces postoperative discomfort.

Surgical Anatomy

The lateral and medial menisci are two semilunar fibrocartilage discs interposed between the femur and the tibia (Fig. 34.127). They are positioned in the joint with the open side of the C facing the midline and are held in place by cranial meniscotibial ligaments, caudal meniscotibial ligaments, and meniscocapsular ligaments. The lateral meniscus has an additional ligament that inserts into the caudal intercondyloid fossa of the femoral condyle. This ligament and loose meniscocapsular ligaments of the lateral meniscus render the lateral meniscus more mobile than the medial meniscus. Clinically, the lack of mobility of the medial meniscus predisposes it to injury.

Positioning

For open arthrotomy, the animal is placed in lateral recumbency with the affected limb up, or in dorsal recumbency. For arthroscopy, the animal is placed in dorsal recumbency. The limb is prepared for aseptic surgery from the dorsal midline to the tarsal joint. Preparing the limb in a hanging-leg position facilitates manipulation of the limb during surgery.



FIG. 34.127 Appearance of menisci and surrounding soft tissue in a cadaver specimen. Note the C-shaped appearance and tibial insertion of the medial meniscus. The lateral meniscus is larger and inserts into the caudal femur.

SURGICAL TECHNIQUE

Exploratory Arthrotomy

A lateral or medial approach to the stifle may be used (see Figs. 34.113 and 34.114). Visualization of the medial meniscus may be enhanced by a medial arthrotomy (Video 34.13).

Partial Meniscectomy

Partial meniscectomy requires that the torn meniscus be adequately exposed. Exposure is facilitated by suction and by levering the tibial plateau down and forward. *Lever the tibia forward by placing the tip of a small Hohmann retractor behind the caudal edge of the tibial plateau and forcing the body of the retractor against the trochlear groove (Fig. 34.128). When the damaged section of meniscus is visible, remove it with a No. 11 or No. 15 scalpel blade. Incise the most medial attachment of the bucket handle first, then incise the most lateral (and caudal) attachment. After removal of the torn section of meniscus, inspect the remaining meniscus for additional tears.*

Meniscal Release

Midbody Release

Midbody meniscal release is performed just caudal to the medial collateral ligament to permit caudal movement of the caudal portion during tibial translation (Fig. 34.129). *Perform an open arthrotomy or arthroscopy. With an open arthrotomy, lever the tibia forward by placing the tip of a small Hohmann retractor behind the caudal edge of the tibial plateau and forcing the body of the retractor against the trochlear groove (see Fig. 34.141, later).*

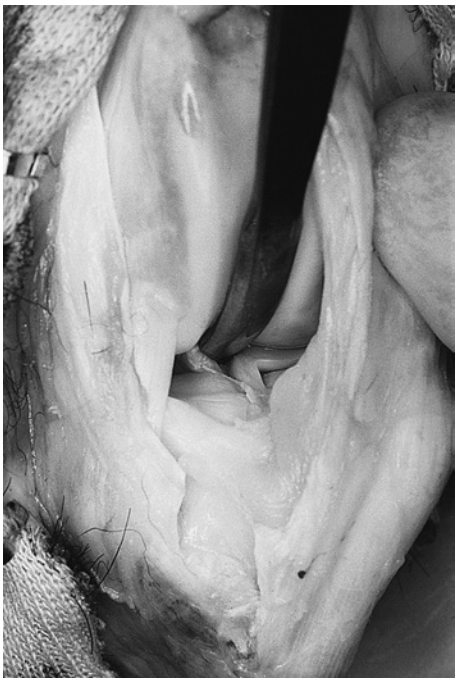


FIG. 34.128 To improve visualization of the caudomedial compartment (where most meniscal tears occur), place a small Hohmann retractor to force the tibia forward and down.

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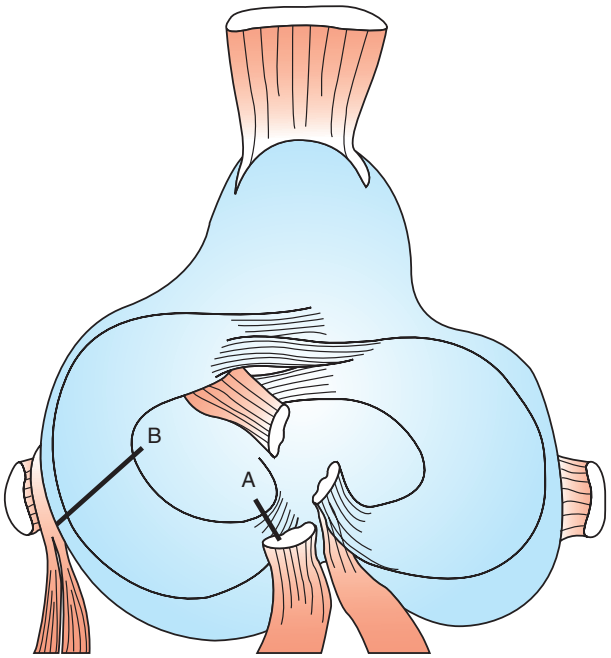


FIG. 34.129 Locations of release of the medial meniscus. Transection of the meniscotibial ligament (*line A*). Transection of the midbody of the meniscus (*line B*).

With arthroscopy, place the limb in valgus stress to expose the medial meniscus. On the medial aspect of the limb, estimate the position of the medial collateral ligament. Insert a needle through the skin and joint caudal to the collateral ligament, and visualize it within the joint. The needle should cross the medial meniscus in the midbody region. *Chase the needle with a No. 11 blade, and transect the meniscus from dorsal to ventral. Examine the transection to verify that it is complete. Close the joint routinely.*

Transection of the Meniscotibial Ligament

Perform an open arthrotomy or arthroscopy. With an open arthrotomy, lever the tibia forward by placing the tip of a small Hohmann retractor behind the caudal edge of the tibial plateau and forcing the body of the retractor against the trochlear groove (see Fig. 34.128). With arthroscopy, place the limb in valgus stress to expose the medial meniscus. Identify the meniscotibial ligament of the medial meniscus. Identify and protect the caudal cruciate ligament. Transect the meniscotibial ligament with a No. 11 blade, No. 12 blade, or hook knife (see Fig. 34.129). Close the joint routinely.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

A small Hohmann retractor is useful for levering the tibia down and forward to allow visualization of the caudal horn of the medial meniscus. A Mosquito Ochsner forceps is used to grasp the meniscus, and a No. 11 or No. 12 blade is used to resect the damaged tissue or perform a meniscal release. A hook knife is useful for transection of the meniscotibial ligament.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative management should follow the recommendations given for reconstruction of the CCL (see p. 1243).

PROGNOSIS

The prognosis for recovery in animals with CCL injury is discussed on p. 1243. Total meniscectomy will promote DJD and should be avoided; however, partial meniscectomy or primary repair of the damaged meniscus lessens the degree of DJD and makes the prognosis for return to normal function more favorable. Dogs with meniscal injury associated with CCL rupture have a poorer long-term outcome than dogs with CCL rupture without meniscal injury.

CAUDAL CRUCIATE LIGAMENT INJURY

DEFINITION

A **caudal cruciate ligament tear** may be complete or partial, with resultant instability causing progressive DJD.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Isolated caudal cruciate ligament tears are rare in small animals because (1) the caudal cruciate ligament is positioned in the joint such that loads that commonly cause ligament injury are directed toward the CCL; (2) the caudal cruciate ligament is stronger than the CCL; and (3) the types of accidents that might cause caudal cruciate ligament rupture are seldom encountered.[70] Caudal cruciate ligament injuries are more commonly associated with severe derangement of the stifle joint. In these patients, combinations of primary joint restraints (CCL, caudal cruciate ligament, medial collateral ligament) and secondary joint restraints (joint capsule, muscle tendon units, meniscocapsular ligaments) are ruptured after a severe traumatic episode, such as an automobile accident.

DIAGNOSIS

Clinical Presentation

Signalment

Dogs or cats of either gender and any breed or age may be affected. Isolated tears are more frequently encountered in large-breed dogs. In cats, tears of the caudal cruciate and medial collateral ligaments commonly occur concurrently.

History

Patients with an isolated caudal cruciate ligament rupture initially have a non–weight-bearing lameness. The lameness progressively improves, but the patient seldom regains athletic status. The animal may have a normal gait at a walk but is lame when undergoing strenuous activity because the caudal cruciate ligament stabilizes the joint primarily when it is flexed. During walking, flexion occurs in the swing phase of the gait; during running and turning, the knee is more flexed in the stance (weight-bearing) phase of the gait.

> **NOTE** Nonathletic dogs usually function normally with caudal cruciate tears.

Physical Examination Findings

Diagnosis of isolated caudal cruciate ligament tears is based on the presence of craniocaudal instability. It is often difficult to differentiate craniocaudal movement caused by CCL rupture

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from that caused by caudal cruciate ligament injury. The following points may help make this differentiation:

- When the joint is held in extension, the degree of palpable instability is less with caudal cruciate ligament tears than with CCL tears.
- With the patient in dorsal recumbency and the limb positioned such that the stifle is flexed and the tibia is parallel to the ground, the tibial tuberosity forms a distinct prominence cranial to the patella. If a caudal cruciate ligament injury is present, the weight of the limb causes a caudal "sag" of the tibia, resulting in loss of the tuberosity prominence.
- When the tibia is moved forward, there is a distinct end point to the cranial movement when the caudal cruciate ligament is ruptured.
- With the tibia in an extended position, there is a distinct caudal subluxation of the tibia when the stifle joint is flexed and internally rotated if the caudal cruciate ligament is torn.

Caudal cruciate ligament tears that are part of a multiple ligament injury are diagnosed by the presence of severe instability (see p. 1252).

Diagnostic Imaging

Radiographs may be helpful in the diagnosis of caudal cruciate ligament injuries. Small bone opacities associated with ligament avulsion may be apparent on the lateral projection just caudal and distal to the femoral condyles. With a lateral radiographic projection, the tibial plateau may be seen to be caudally displaced relative to the femoral condyles.

Laboratory Findings

No consistent laboratory abnormalities are seen. Cytology of joint fluid usually demonstrates mononuclear inflammation typical of osteoarthritis.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include CCL injury (see the previous discussion) and multiple ligament injury.

MEDICAL MANAGEMENT

Long-term evaluation of dogs having reconstruction of isolated caudal cruciate ligament tears shows the prognosis to be good, regardless of the treatment method. Conservative management of isolated caudal cruciate tears (i.e., activity restricted to leash walking for 8 weeks) is an option for smaller dogs or cats and for dogs leading inactive lives.

SURGICAL TREATMENT

Joints with caudal cruciate ligament injury are treated with resection of the remnants of the ligament and stabilized by one of several extracapsular reconstruction techniques: suture stabilization, redirection of the medial collateral ligament, or popliteal tendon tenodesis. Suture stabilization consists of imbrication of the caudomedial joint capsule and placement of a medial or lateral stabilizing suture. Redirection repair uses existing autogenous tissue, such as the medial collateral ligament.

Preoperative Management

These animals should be evaluated for evidence of other ligamentous or bony trauma.

Perioperative antibiotics (see Chapter 9) and preemptive pain management with NSAIDs, opiates, or epidural analgesia (see Chapter 13) are indicated for dogs undergoing stifle reconstructive techniques.

Anesthesia

See Tables 32.1 and 32.2 for the anesthetic management of animals with orthopedic disease. Opioid epidural administration and intraarticular administration of bupivacaine or morphine reduce postoperative discomfort (see p. 980).

Surgical Anatomy

The caudal cruciate ligament originates from the intercondyloid fossa of the craniolateral (inside) surface of the medial condyle (see Fig. 34.111). From the point of origin, the ligament courses distally to insert into the popliteal notch of the tibia.

Positioning

The animal is positioned in lateral recumbency with the affected limb up or in dorsal recumbency.

SURGICAL TECHNIQUE

Exploratory Arthrotomy

Perform arthroscopy or make a standard craniomedial or craniolateral approach to the stifle joint (see p. 1235), and explore internal structures. Resect the remnants of the caudal cruciate ligament.

Suture Stabilization

Drill a hole in the caudomedial corner of the tibial epiphysis. Place a stabilizing suture from the proximal patellar tendon through the predrilled hole (Fig. 34.130). On the lateral side, imbricate the caudal joint capsule, and place a stabilizing suture from the proximal patellar tendon through a predrilled hole in the fibular head. Alternatively, bone anchors may be placed in the medial and lateral aspect of the femur and sutures passed from the bone anchors to the drill holes in the tibial epiphysis and fibular head.

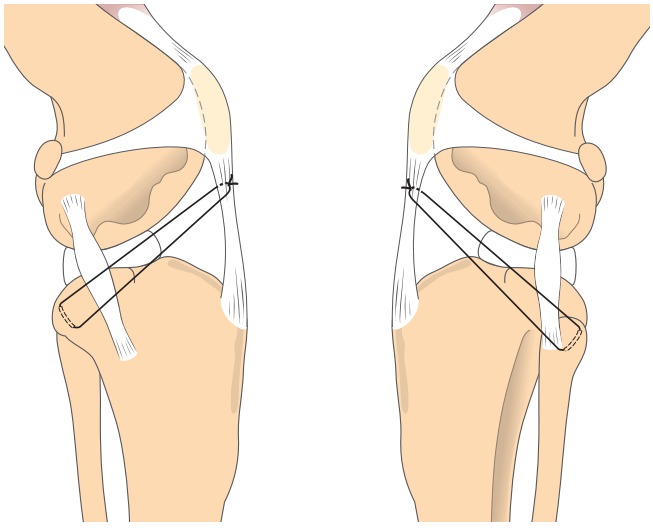


FIG. 34.130 For caudal cruciate ligament tears, extracapsular sutures are placed from the patellar tendon just distal to the patella to the caudal tibia (medially) and fibular head (laterally).

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Use of Autogenous Tissue

Incise through the insertion of the caudal sartorius muscle and medial fascia along the tibial metaphysis. Reflect the muscle and fascia caudally to expose the medial collateral ligament. Free a portion of the body of the ligament with a periosteal elevator and direct the ligament caudally to course in the same sagittal plane as the caudal cruciate ligament. Secure the ligament in this position with a bone screw and spiked washer.

Entrapment (Tenodesis) of the Popliteal Tendon

Make a lateral approach to the stifle joint (see p. 1235), and reflect the fascia lata to isolate the popliteal tendon as it passes beneath the lateral collateral ligament. Entrap the popliteal tendon with a screw and Teflon or polyacetyl washer as it passes caudal and proximal to the fibular head.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

A bone screw and Teflon washer are used to secure the medial collateral ligament or popliteal tendon to bone. Nonabsorbable monofilament and orthopedic suture are preferred for extracapsular stabilization techniques.

POSTOPERATIVE CARE AND ASSESSMENT

Activity should be restricted to specific physical rehabilitation exercises and leash walking for 6 weeks; the animal should be gradually returned to unsupervised activity over a 6-week period (see Chapter 11).

PROGNOSIS

The prognosis is good to excellent for return to normal limb function in most animals after surgery. Clinically and experimentally, DJD does not appear to progress as rapidly after isolated caudal cruciate ligament injury as it does after CCL injury.

COLLATERAL LIGAMENT INJURY

DEFINITION

A **collateral ligament injury** is a complete or partial tear of the medial or lateral collateral ligament.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

As the medial collateral ligament crosses the medial joint surface, it forms a strong attachment to the joint capsule and medial meniscus. This attachment is important in stabilizing the medial meniscus, but it predisposes the caudal body of the meniscus to injury from the medial femoral condyle when CCL rupture is present. The medial and lateral collateral ligaments function in concert to limit varus-valgus motion of the stifle joint. This is most important when the stifle joint is extended and both the medial and lateral collateral ligaments are taut. As the stifle joint flexes, the medial collateral ligament remains tight, but the lateral collateral ligament relaxes to allow internal tibial rotation. This motion permits the foot to turn inward beneath the body during ambulation. As the stifle joint extends, the lateral collateral ligament becomes taut once again to assist in external rotation of the tibia. This motion aligns the foot into proper position for weight bearing.

Isolated medial or lateral collateral ligament tears are rare in small animals. Most injuries that involve the medial or lateral collateral ligaments occur in conjunction with injury to other primary and secondary restraints of the stifle joint. These multiple ligament injuries are often the result of severe trauma directed to the stifle joint and involve injury to a myriad of stifle joint ligaments.

DIAGNOSIS

Clinical Presentation

Signalment

Any age or breed and either gender of dog or cat may be affected.

History

This injury may occur while the animal is exercising (without evidence of trauma) or during a traumatic incident (i.e., vehicular accident), in which the animal has sustained major injuries.

Physical Examination Findings

Diagnosis of collateral ligament injury, whether an isolated ligament tear or part of a complex injury, is based on palpation (Video 34.14). It is important to remember that the stifle joint must be extended to examine for collateral injury. The *valgus stress test* is used to evaluate integrity of the medial collateral ligament. With the patient in lateral recumbency, one hand is used to stabilize the femur while the other hand grasps the distal tibia and applies an upward force (abduction). If the medial joint restraints (medial collateral ligament, joint capsule, peripheral meniscal ligaments) are torn, opening of the medial joint line is apparent (Fig. 34.131). The *varus stress test* is used to evaluate integrity of the lateral collateral ligament. One hand stabilizes the femur while the other hand grasps the distal tibia and applies an inward force (adduction). If the lateral joint restraints are torn, opening of the lateral joint is apparent. Isolated tears show minimal opening, whereas obvious opening occurs with more extensive injuries (lateral collateral ligament, joint capsule, peripheral meniscal ligaments).



FIG. 34.131 Medial collateral ligament injury in a cat. Note the appearance of the stifle when a valgus stress is applied. The foot displaces upward, and the medial joint line opens.

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FIG. 34.132 Stress radiograph of a cat with a medial collateral ligament injury. Note the severity of joint opening when a valgus stress is applied to the joint.

NOTE Be sure to hold the stifle joint in extension when assessing the medial and lateral restraints.

Diagnostic Imaging

Radiographs should be obtained to determine if bone fragments are associated with ligament damage. Craniocaudal and medial-to-lateral radiographs are indicated to confirm the presence or absence of bony avulsions. Stress radiographs are useful for demonstrating an increase in medial or lateral joint space (Fig. 34.132).

Laboratory Findings

Consistent laboratory findings are not seen. Laboratory evaluation depends on the signalment and physical findings in animals that have sustained trauma.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include muscle strains or cranial or caudal cruciate ligament tears and nondisplaced physeal fractures in immature animals.

MEDICAL MANAGEMENT

The decision to use conservative or surgical treatment for isolated collateral ligament injury is based on the degree of injury to the ligament and secondary joint restraints (joint capsule, peripheral meniscal ligaments). This assessment is based on palpation and radiographs. Minimal swelling and only slight opening of the joint space when the joint is placed under stress are indications for conservative treatment, which involves a fiberglass cast applied for 2 weeks and then controlled activity for 6 additional weeks.

SURGICAL TREATMENT

Moderate to severe swelling and significant opening of the joint space when the joint is placed under stress indicate significant injury to the collateral restraints. Surgery is recommended for these patients. Treatment includes reconstruction of the collateral and meniscocapsular ligaments and the joint capsule. Primary repair of the collateral ligament is undertaken if the point of failure is the origin or insertion of the ligament or an intrasubstance tear with large segments of the ligament intact. Occasionally, a small fragment of bone may be present on the end of the ligament that can be incorporated into the repair.

NOTE Be sure to repair all injured ligaments, tendons, and joint capsule.

Preoperative Management

To prevent additional damage to the articular cartilage or menisci, place a modified Robert Jones bandage on the limb (see p. 981) and limit activity to leash walking until definitive surgical treatment can be done. These animals should be evaluated for evidence of other ligamentous or bony trauma. Patients with injuries caused by vehicular accidents should undergo thoracic, cardiovascular, and abdominal evaluation. Perioperative antibiotics and preemptive pain management with NSAIDs, opiates, and epidural analgesia are indicated for animals undergoing stifle reconstructive techniques.

Anesthesia

See Table 32.2 for anesthetic management of animals with orthopedic disease caused by trauma. Opioid epidural administration reduces postoperative discomfort (see p. 980).

Surgical Anatomy

Knowledge of the origin and insertion points of the collateral ligaments is important. The medial collateral ligament originates from the medial femoral epicondyle and courses distally to insert onto the proximal tibial metaphysis (Fig. 34.133). As the ligament crosses the medial joint line, it forms a strong attachment to the joint capsule and medial meniscus. The lateral collateral ligament originates from an oval area on the lateral femoral epicondyle; it then courses distally to insert onto the fibular head. The medial collateral ligament lies deep to the caudal sartorius muscle; the lateral collateral ligament lies deep to the fascia lata. The peroneal (fibular) nerve is a branch of the sciatic nerve. It obliquely crosses the distal aspect to the stifle joint, where it lies superficial to the gastrocnemius muscle and sends an articular branch to the lateral collateral ligament. Care should be taken when dissecting near the lateral collateral ligament to preserve this nerve.

Positioning

To repair a lateral collateral ligament injury, position the patient in lateral recumbency with the affected leg up. For medial collateral ligament injuries, position the animal in dorsal recumbency. If multiple ligament tears are present, the animal can also be positioned in dorsal recumbency to facilitate exposure of both sides of the limb. Suspend the limb and prepare it for aseptic surgery.

SURGICAL TECHNIQUE

Medial Collateral Ligament Injury

Make a medial parapatellar incision. Use a medial approach to expose the medial collateral ligament (see p. 1236). Incise the

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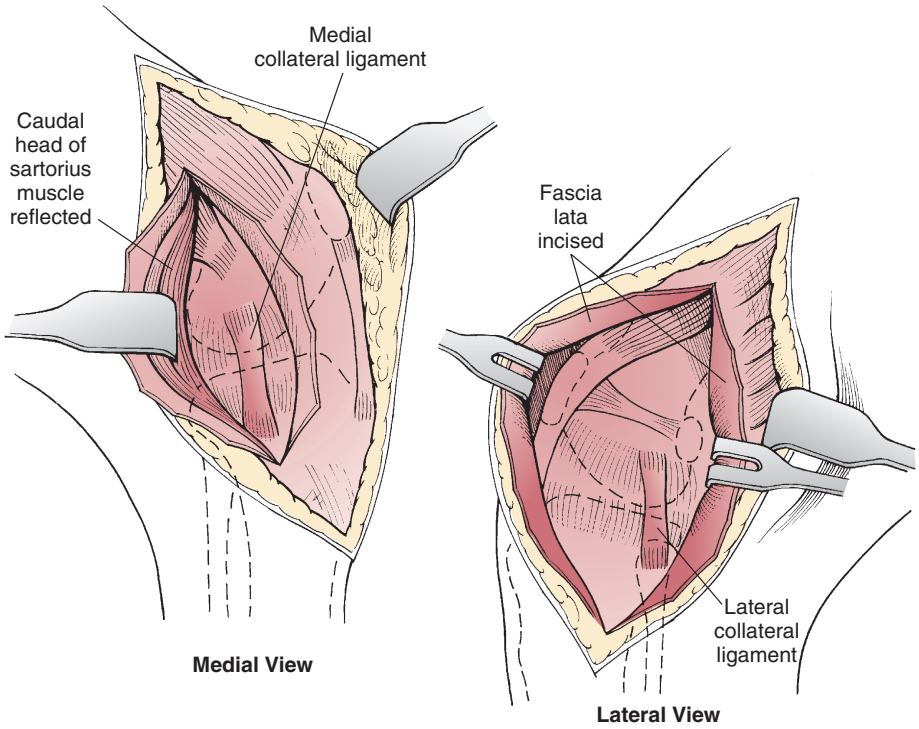


FIG. 34.133 Medial and lateral views of the stifle showing soft tissue structures surrounding the collateral ligaments.

insertion of the caudal head of the sartorius muscle and deep fascia along the craniomedial border of the proximal tibia (Fig. 34.134A). Retract the muscle and fascia caudally to expose the collateral ligament and medial joint capsule. Replace the ligament to its anatomic site, and secure it with a screw and polyacetyl spiked washer (Fig. 34.134B). As an alternative, place a suture anchor at the insertion of the ligament and suture the ligament to the anchor. If the ligament injury is an intrasubstance tear, perform primary repair by suturing the ligament ends; use a locking-loop suture pattern with small, nonabsorbable suture (see p. 1283). Supplement the primary repair with screws or bone anchors and figure-eight support (Fig. 34.134C). After repair of the collateral ligament, carefully reconstruct the meniscocapsular ligaments and joint capsule using interrupted sutures of small, nonabsorbable suture material (polypropylene or nylon).

Lateral Collateral Ligament Injury

Use a craniolateral approach to expose the lateral collateral ligament (see p. 1235). Make a proximal-to-distal parapatellar incision through the fascia lata. Continue the incision distally 4 cm below the tibial crest, parallel to the joint line. Use caution in isolating the peroneal nerve, and protect it carefully during surgery. Reflect the fascia lata caudally to expose the collateral ligament and lateral joint capsule. Repair the ligament as described previously.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Bone screws, polyacetyl spiked washers, suture anchors, and orthopedic suture or heavy, nonabsorbable suture are needed for collateral ligament repair.

POSTOPERATIVE CARE AND ASSESSMENT

The limb is placed in a soft, padded bandage with a lateral splint for 10 to 14 days. Activity should be restricted to specific physical rehabilitation exercises and leash walking for 6 weeks; the animal should be gradually returned to unsupervised activity over a 6-week period (see Chapter 11).

PROGNOSIS

The prognosis for isolated collateral ligament tears is good to excellent. If multiple ligaments are torn, the prognosis is fair.

MULTIPLE LIGAMENT INJURIES (DERANGED STIFLE)

DEFINITIONS

Multiple ligament injuries are injuries in which the cranial or caudal cruciate ligaments and collateral ligaments are damaged simultaneously. **Varus angulation** is an inward rotation of the leg (toward the midline of the body), and **valgus angulation** is an outward rotation (away from the midline of the body). The **primary stifle restraints** are the cruciate and collateral ligaments; the **secondary stifle restraints** are the joint capsule, meniscus, tendon, and muscle.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Multiple ligament injuries are caused by automobile accidents or other major trauma. A common triad of injuries includes

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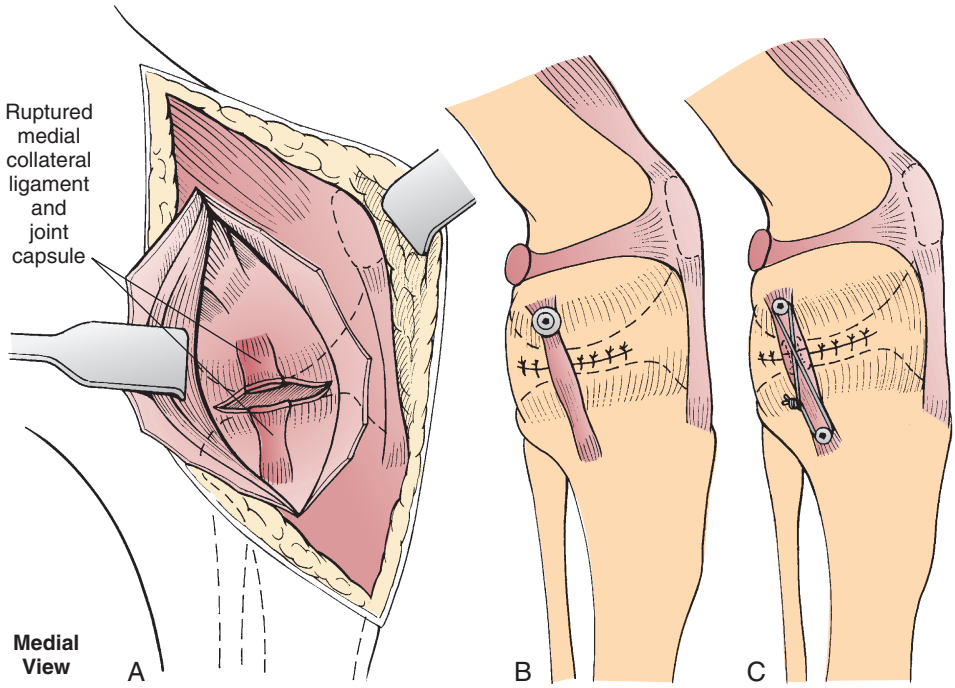


FIG. 34.134 Repair of a medial restraint injury. (A) Incise the insertion of the caudal head of the sartorius muscle and deep fascia along the craniomedial border of the proximal tibia. (B) Replace the collateral ligament in its anatomic site and secure with a screw and polyacetyl spiked washer. As an alternative, use suture anchors. (C) If the ligament injury is an intrasubstance tear, perform primary repair by suturing the ligament ends with a locking-loop suture pattern. Supplement the primary repair with screws and figure-eight support.

cranial and caudal cruciate ligament tears, failure of the primary and secondary medial restraints, and peripheral medial meniscal tears. Varus angulation of the limb with a medial stress indicates lateral injury, whereas valgus angulation of the limb (with a lateral stress applied to the foot) indicates medial injury.

DIAGNOSIS

Clinical Presentation

Signalment

Either gender and any age or breed of dog or cat may be affected.

History

The animal is usually presented for evaluation of an acute, non–weight-bearing lameness. The trauma may or may not have been observed.

Physical Examination Findings

Combined cranial and caudal cruciate tears are characterized by notable craniocaudal movement of the tibia relative to the femur. Concurrent damage to the collateral ligament and joint capsule complexes can usually be determined by palpation. With the leg in extension, a varus stress applied to the distal tibia causes opening of the lateral joint line if the lateral restraints are injured, whereas a valgus stress causes opening of the medial joint line if the medial restraints are injured.

Diagnostic Imaging

Radiographs reveal subluxation of the stifle joint. Careful assessment of both craniocaudal and mediolateral radiographs may show small bone fragments at the origin or insertion of ligaments.

Laboratory Findings

Consistent laboratory abnormalities are not seen.

DIFFERENTIAL DIAGNOSIS

Distal femoral and proximal tibial metaphyseal fractures may show similar clinical signs and should be differentiated from ligament injuries.

MEDICAL MANAGEMENT

Multiple ligament injuries require surgical intervention. Conservative treatment with external coaptation (splints or casts) is not effective in maintaining alignment of the stifle joint and leads to severe DJD.

SURGICAL TREATMENT

Surgical treatment involves careful reconstruction of the cruciate ligaments, the collateral ligaments, and the menisci. The collateral ligament complex is repaired first, followed by reconstruction of the cruciate ligaments. The specific repair and reconstruction techniques used depend on the surgeon's preference and are described in the discussion of each individual ligament. Temporary immobilization using an external fixator can also be performed as a primary surgical method, or for protection of reconstructive surgery.

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Preoperative Management

To prevent damage to the articular surfaces of the joint, a soft, padded bandage with a lateral splint should be applied and activity limited until surgery can be done. Affected animals should be evaluated for evidence of other ligamentous or bony trauma; other fractures are often associated with this injury. Patients with injuries from vehicular accidents should have complete physical examinations, with particular attention to the respiratory and cardiovascular systems. Perioperative antibiotics (see Chapter 9) and preemptive pain management with NSAIDs (see Table 34.3), opiates, or epidural analgesia are indicated for animals undergoing stifle reconstructive techniques.

Anesthesia

See Table 32.2 on p. 978 for the anesthetic management of animals with orthopedic disease caused by trauma. Opioid epidural administration (see p. 980) reduces postoperative discomfort.

Surgical Anatomy

The normal anatomy of the stifle joint is described on p. 1234. With multiple ligament injury, moderate to severe swelling and bruising of soft tissue surrounding the joint are seen. A knowledge of the normal origins and insertions of the collateral ligaments, meniscocapsular ligaments, and ligaments in the joint is required. Torn collateral ligaments are difficult to identify because they are often encased in edematous connective tissue. Menisci are often displaced from their normal positions and folded cranially or caudally (Fig. 34.135).

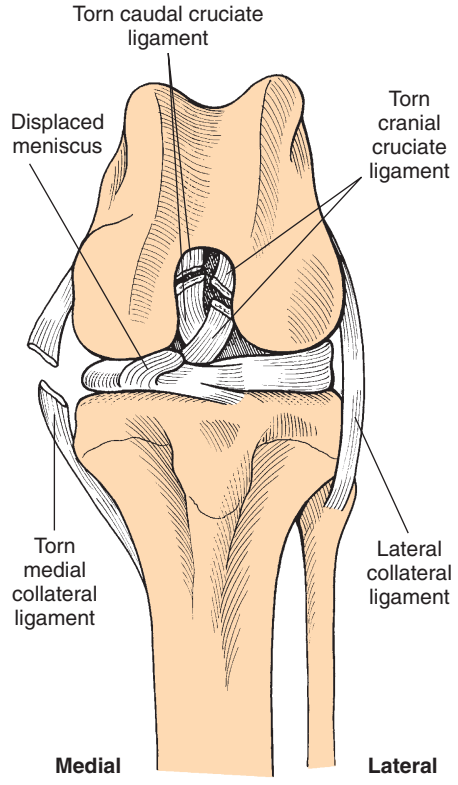


FIG. 34.135 Structures commonly injured with multiple ligament derangement of the stifle joint. Note the loss of the cranial and caudal cruciate ligaments and the disruption of the medial restraints.

Positioning

The patient is positioned in dorsal recumbency with the affected limb up. The leg is prepared from the dorsal midline to the tarsal joint. A hanging-limb preparation facilitates manipulation of the leg during surgery.

SURGICAL TECHNIQUE

Cruciate ligament repair is discussed on p. 1232, collateral ligament repair on p. 1251, and meniscal injuries on p. 1246.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

See requirements for cruciate ligament repair discussed on p. 1243, collateral ligament repair on p. 1252, and meniscal injuries on p. 1248.

POSTOPERATIVE CARE AND ASSESSMENT

After surgery, the limb should be placed in a soft, padded bandage with a lateral splint for 3 weeks. The support bandage should be placed before the patient recovers from anesthesia to allow accurate positioning of the bandage with the leg in extension. The bandage provides comfort in the early postoperative period by immobilizing soft tissue and preventing extension and flexion of the stifle joint. It should be changed weekly, or more often if necessary. Activity should be limited to leash walks or specific physical rehabilitation exercises for the first 8 to 12 weeks postoperatively (see Chapter 11).

PROGNOSIS

The prognosis is good for return to nonathletic performance. Animals with subluxated joints have a better prognosis for return to function than animals with luxated joints and failure of secondary stabilizers. Subsequent to extensive ligament injury, loss of flexion beyond 110 degrees and mild to moderate instability are common after surgery. This instability and loss of normal range of motion limit athletic performance. Severe lameness should be expected in animals that are managed without surgery. Physical therapy is helpful for rehabilitation of the stifle (see Chapter 11). Programs include cryotherapy, passive range of motion, and muscle strengthening exercises.

MEDIAL PATELLAR LUXATION

DEFINITION

Medial patellar luxation is a displacement of the patella from the trochlear sulcus. **Genu varum** is a deformity associated with severe medial patellar luxation, resulting in a "bow legged" appearance.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Medial patellar luxation is a common cause of lameness in small-breed dogs, but it also occurs in large-breed dogs. Most patients with patellar luxation have associated musculoskeletal abnormalities, such as medial displacement of the quadriceps muscle group, lateral torsion of the distal femur, a lateral bowing

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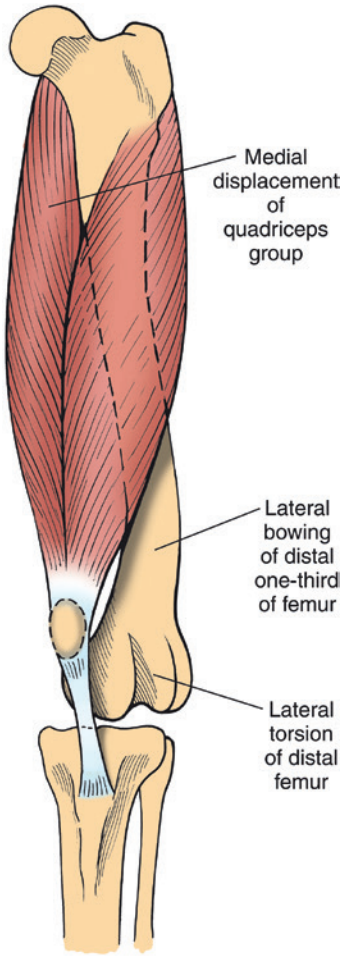


FIG. 34.136 Soft tissue and skeletal abnormalities associated with medial patellar luxation include medial displacement of the quadriceps apparatus, lateral bowing of the distal one-third of the femur, and lateral torsion of the distal femur.

of the distal one-third of the femur, femoral epiphyseal dysplasia, rotational instability of the stifle joint, or tibial deformity (Fig. 34.136).

Medial malalignment of the quadriceps muscles in dogs with medial patellar luxation produces sufficient pressure on the distal femoral physis to retard growth. At the same time, there is less pressure on the lateral aspect of the distal femoral physis, which allows accelerated growth. Decreased length of the medial cortex relative to the increased length of the lateral cortex results in the lateral bowing of the distal femur. Abnormal growth continues as long as the quadriceps is displaced medially and the physes are active. Therefore the degree of lateral bowing depends on the severity of patellar luxation and the patient's age at the onset of luxation.[71] With mild luxations, the quadriceps is rarely displaced medially and has minimal abnormal effect on the distal femoral physis. However, with severe luxations, the quadriceps is medially displaced at all times, and maximal effect on the distal femoral physis results in severe lateral bowing of the distal femur in young patients (Fig. 34.137). Tibial deformities seen with patellar luxations are the result of abnormal forces acting on the proximal and distal physes of the tibia. Tibial deformities described with medial patellar luxation include medial torsion and displacement of the tibial tuberosity, medial bowing



FIG. 34.137 Radiographic appearance of severe skeletal abnormalities associated with medial patellar luxation in a dog.

(varus deformity) of the proximal tibia, and lateral torsion of the distal tibia.[71]

Dogs with medial patellar luxation have abnormal development of the trochlear groove. The degree of abnormality varies from a near-normal trochlea to an absent trochlear groove.[71,72] Articulation of the patella within the trochlear groove exerts a physiologic pressure on the articular cartilage that retards cartilage growth. Continued pressure by the patella is responsible for the development of normal depth of the trochlear groove. If the physiologic pressure exerted by the patella is not present on the trochlear articular cartilage, the trochlea fails to gain proper depth. Immature patients with mild luxations show minimal loss of depth to the trochlear groove because the patella is normally positioned during development. However, immature patients with severe luxations have no trochlear groove because the normal pressure responsible for groove development is not present. The degree of the pathologic skeletal condition associated with patellar luxation varies considerably between the mildest and severest forms; therefore a system for classifying canine patellar luxation has been developed (Box 34.20).

Shortening of the limb because of hip luxation or FHO will cause laxity of the quadriceps mechanism, enabling luxation of the patella in some cases. This usually resolves with treatment of the hip luxation and with time after FHO.

DIAGNOSIS

Clinical Presentation

Signalment

Dogs of any age or breed and either gender may have medial patellar luxations, but small- and toy-breed dogs are most frequently affected. Medial patellar luxations are more common than lateral patellar luxations in large-breed dogs; however, large dogs have a higher percentage of lateral luxations than small dogs.

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BOX 34.20 Grades of Patellar Luxation

Grade I
The patella can be luxated, but spontaneous luxation of the patella during normal joint motion rarely occurs. Manual luxation of the patella may be accomplished during physical examination, but the patella reduces when pressure is released. Flexion and extension of the joint are normal.

Grade II
Angular and torsional deformities of the femur may be present to a mild degree. The patella may be manually displaced with lateral pressure or may luxate with flexion of the stifle joint. The patella remains luxated until the examiner reduces it or it is spontaneously reduced when the animal extends and derotates its tibia.

Grade III
The patella remains luxated medially most of the time but may be manually reduced with the stifle in extension. However, after manual reduction, flexion and extension of the stifle result in reluxation of the patella. There is a medial displacement of the quadriceps muscle group. Abnormalities of the supporting soft tissue of the stifle joint and deformities of the femur and tibia may be present.

Grade IV
There may be an 80- to 90-degree medial rotation of the proximal tibial plateau. The patella is permanently luxated and cannot be manually repositioned. The femoral trochlear groove is shallow or absent, and there is medial displacement of the quadriceps muscle group. Abnormalities of the supporting soft tissue of the stifle joint and deformities of the femur and tibia are notable.

History

Most affected animals have an intermittent weight-bearing lameness. Owners may report that the dog occasionally holds the leg in a flexed position for one or two steps. Dogs with grade IV patellar luxations have severe lameness and gait abnormalities.

Physical Examination Findings

The diagnosis of medial patellar luxation is based on finding or eliciting medial patellar luxation during a physical examination. Physical findings vary and depend on the severity of luxation. Patients with a *grade I luxation* generally show no lameness, and the diagnosis is made as an incidental finding on physical examination. Patients with a *grade II luxation* show occasional "skipping" when walking or running. These patients occasionally stretch the lateral retinacular structures and develop a non–weight-bearing lameness. Lameness in patients with a *grade III luxation* varies from an occasional skip to a weight-bearing lameness. Patients with a *grade IV luxation* walk with the rear quarters in a crouched position because they are unable to extend the stifle joints fully. The patella is hypoplastic and may be found displaced medially alongside the femoral condyle.

Diagnostic Imaging

With a grade III or grade IV luxation, standard craniocaudal and medial-to-lateral radiographs show the patella to be displaced medially, whereas with a grade I or grade II luxation the patella may be within the trochlear sulcus or may be displaced medially (care must be taken to properly position the limb to eliminate artifactual appearing luxations). Full limb radiographs may show varus or valgus deformities and torsion of the tibia and femur. Careful radiographic positioning is critical, as poor positioning results in false-positive limb deformity on radiographs. For more severe cases requiring long bone osteotomy and correction, special views (coronal or skyline view of the femur) or CT aid in determining the specific type and degree of deformity.[71]

BOX 34.21 Basic Techniques for Repair of Medial Patellar Luxation

Medial fascial release
Trochlear wedge or block recession
Tibial crest transposition
Lateral imbrication

Laboratory Findings

Consistent laboratory findings are not seen. Arthrocentesis demonstrates changes compatible with osteoarthritis.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include avascular necrosis of the femoral head, coxofemoral luxation, and CCL rupture. Careful examination of the hip joint is essential because some patients with patellar luxation also have avascular necrosis of the femoral head (see p. 1255) or hip dysplasia (see p. 1209). Shortening of the limb because of hip luxation (see p. 1220) or FHO (see p. 1213) will cause laxity of the quadriceps mechanism, enabling luxation of the patella in some cases. This usually resolves with treatment of the hip luxation and with time after FHO.

MEDICAL MANAGEMENT

Medial patellar luxation may be treated conservatively or surgically.[73] The choice of treatment depends on the clinical history, physical findings, frequency of luxation, and the patient's age. Surgery is seldom warranted in asymptomatic older patients, whereas young animals or those that are lame usually benefit from surgery. Clients should be advised to observe the animal for the development of clinical signs attributable to medial patellar luxation.

SURGICAL TREATMENT

Surgery is advised in symptomatic immature or young adult patients because intermittent patellar luxation may prematurely wear the articular cartilage of the patella.[73–75] Surgery is indicated at any age in patients showing lameness and is strongly advised in those with active growth plates because skeletal deformity may worsen rapidly. The surgical techniques used in actively growing animals should not adversely affect skeletal growth. Owners of dogs with bilateral grade IV patellar luxations should be warned of the likely need for multiple surgeries and probable continued lameness even after successful surgery because of the severity of the underlying long bone abnormalities.[74,75]

Numerous surgical techniques are aimed at restraining the patella within the trochlear groove. Tibial tuberosity transposition, medial restraint release, lateral restraint reinforcement, trochlear groove deepening, femoral osteotomy, tibial osteotomy, antirotational sutures, and transposition of the origin of the rectus femoris have all been advocated for correction of patellar luxation. Generally, a combination of techniques is required to achieve intraoperative stability of the patella (Box 34.21). It is important to understand that the primary abnormality is biomechanical,

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whereby the patella within the straight quadriceps mechanism fails to align with the trochlear groove. Surgeries that involve only deepening of the trochlear groove, capsule and fascial release, and imbrication are more prone to failure, as the patella and trochlear groove have not been permanently realigned.

In most animals, the trochlear groove must be deepened with a trochlear wedge or block recession. Release of the medial retinaculum may be necessary to allow the patella to stabilize in the deepened trochlear groove. Tibial crest transposition must be done to realign the mechanical forces of the extensor mechanism (see p. 1260) unless major corrections of femoral and tibial deformity have been performed. Finally, after the patella is stable, the lateral retinaculum is reinforced with sutures and imbrication of the fibrous joint capsule, placement of a fascia lata graft from the fabella to the parapatellar fibrocartilage, or excision of redundant retinaculum. None of the reinforcement techniques alone is adequate to prevent reluxation permanently. If the mechanical forces pulling the patella out of the trochlear groove have not been neutralized, the reinforced retinaculum eventually stretches.

Osteotomy of the femur is used only in patients with such severe skeletal deformity that maintaining patellar reduction with the previously described techniques is impossible. The deformities usually seen are varus bowing of the distal femur and medial torsional deformity of the proximal tibia. The goal of surgery is to realign the stifle joint in the frontal plane where the transverse axis of the femoral condyles is perpendicular to the longitudinal axis of the femoral diaphysis. This requires accurate preoperative measurement and wedge osteotomy of the femur.[71] Deepening of the trochlear groove, medial restraint release, transposition of the tibial crest, and lateral retinacular reinforcement are also required for success. These techniques require special equipment and training; as such, a trained specialist should perform them.

The quadriceps mechanism is a secondary stabilizer of the stifle joint for cranial translation (cranial drawer). Subsequently, chronic luxation of the patella may lead to increased stress on the CCL and eventual rupture. Combination of CCL rupture and patellar luxation is a relatively common finding in dogs.[76]

Preoperative Management

Perioperative antibiotics (see Chapter 9) and preemptive pain management with NSAIDs, opiates, or epidural analgesia (see Chapter 13) are indicated for dogs undergoing stifle reconstructive techniques.

Anesthesia

See Tables 32.1 and 32.2 for the anesthetic management of animals with orthopedic disease. Opioid epidural administration reduces postoperative discomfort.

Surgical Anatomy

The extensor mechanism of the stifle joint is composed of the quadriceps muscle groups, patella, trochlear groove, straight patellar ligament, and tibial tuberosity (Fig. 34.138). The quadriceps muscle group is formed by the rectus femoris, vastus lateralis, vastus intermedius, and vastus medialis muscles. The vastus medialis and vastus lateralis muscles are fixed to the patella by the medial and lateral parapatellar fibrocartilage. The fibrocartilage rides on the ridges of the femoral trochlea and, along with the medial and lateral retinacula, aids in patellar stability. The medial and lateral retinacula are groups of collagen fibers that course from the fabella to blend with the medial and lateral parapatellar fibrocartilages, respectively. The quadriceps muscle group extends the stifle joint and, along with the entire extensor mechanism, aids in stabilizing the stifle joint. The quadriceps muscle group converges as the patellar tendon on the patella and then continues distally as the straight patellar ligament.

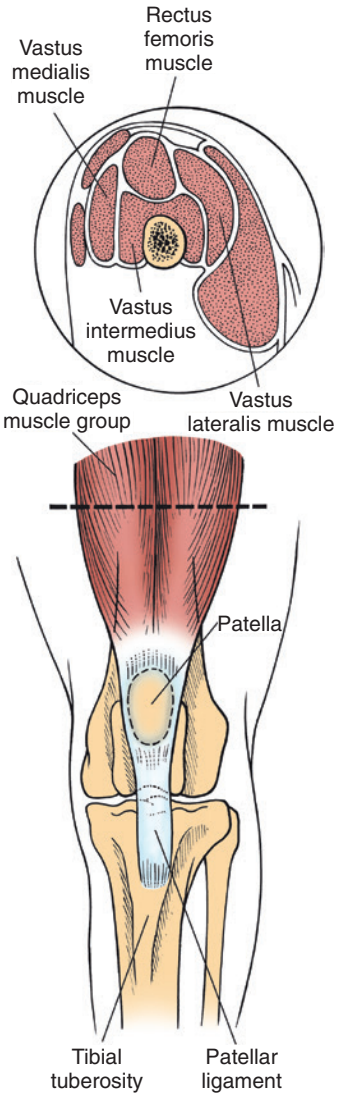


FIG. 34.138 Anatomic diagram of normal soft tissue and skeletal structures associated with the quadriceps extensor mechanism.

The patella is a sesamoid bone embedded in the tendon of the quadriceps muscle. The inner articular surface is smooth and curved so as to fully articulate with the trochlea. The normal gliding articulation of the patella and trochlea is necessary for maintaining the nutritional requirements of the trochlear and patellar articular surfaces. The patella is also an essential component of the functional mechanism of the extensor apparatus. The patella maintains even tension when the stifle is extended and also acts as a fulcrum in a lever arm, increasing the mechanical advantage of the quadriceps muscle group.

The tibial tuberosity is located cranial and distal to the tibial condyles. Its location and prominence are important for the mechanical advantage of the extensor mechanism. The alignment of the quadriceps, patella, trochlea, patellar ligament, and tibial tuberosity must be normal for proper function. Malalignment of one or more of these structures may lead to patellar luxation.

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Special anatomic considerations in patients with medial patellar luxation must be noted. When the patella is positioned medially, the patellar ligament must be identified before making a parapatellar incision to enter the joint. The lateral capsule is stretched and thin, whereas the medial capsule is contracted and thickened. Thickening and contraction of the medial capsule are apparent if medial release is performed. The insertions of the caudal head of the sartorius muscle and the vastus medialis muscle should be identified. The medial ridge of the trochlear sulcus and the ventral surface of the patella may be worn.

Positioning

The animal is positioned in dorsal or lateral recumbency, and the leg is prepared from the dorsal midline to the tarsal joint. Dorsal recumbency allows visualization of unrestrained extensor mechanism deviation and maximum manipulation of the limb to evaluate patellar stability.

SURGICAL TECHNIQUE

Arthrotomy

Make a craniolateral skin incision 4 cm proximal to the patella, and extend the incision 2 cm below the tibial tuberosity. Incise the subcutaneous tissue along the same line. Incise the lateral retinaculum and joint capsule to expose the joint.

Deepening of the Trochlear Groove

Trochlear Wedge Recession

Trochlear wedge recession deepens the trochlear groove to restrain the patella and maintain the integrity of the patellofemoral articulation. In larger patients, an oscillating saw is often used, but in smaller and toy breeds, a fine-toothed, handheld saw or the cutting edge of a No. 20 scalpel blade and mallet may be used to make the cuts in the trochlea. *Cut into the articular cartilage of the trochlea, making a diamond-shaped outline. Be sure that the width of the cut is sufficient at its midpoint to accommodate the width of the patella but preserves the trochlear ridges. Using a surgical blade in very small and young patients, a hand saw in small and medium patients, or a power saw in large patients, remove an osteochondral wedge of bone and cartilage by following the outline previously made (Fig. 34.139A). Make the osteotomy so that the two oblique planes that form the free wedge intersect distally at the intercondylar notch and proximally at the dorsal edge of the trochlear articular cartilage. Remove the osteochondral wedge and deepen the recession in the trochlea by removing additional bone from one or both sides of the newly created femoral groove (Fig. 34.139B).*

If necessary, remodel the free osteochondral wedge with rongeurs to allow the wedge to seat deeply into the new femoral groove. The wedge can also be rotated 180 degrees when it is returned to the femoral groove if doing so aids in heightening the medial ridge. *Replace the free osteochondral wedge when the depth is sufficient to house 50% of the height of the patella (Fig. 34.139C).* The osteochondral wedge remains in place because of the net compressive force of the patella and friction between the cancellous surfaces of the two cut edges.

Trochlear Block Recession

Trochlear block recession deepens the trochlear groove to restrain the patella and maintain the integrity of the patellofemoral articulation. In larger patients, an oscillating saw is often used, but in smaller and toy breeds, a fine-toothed, handheld saw or the cutting edge of a No. 20 scalpel blade and mallet may be used to make the cuts in the trochlea. *Cut into the articular*

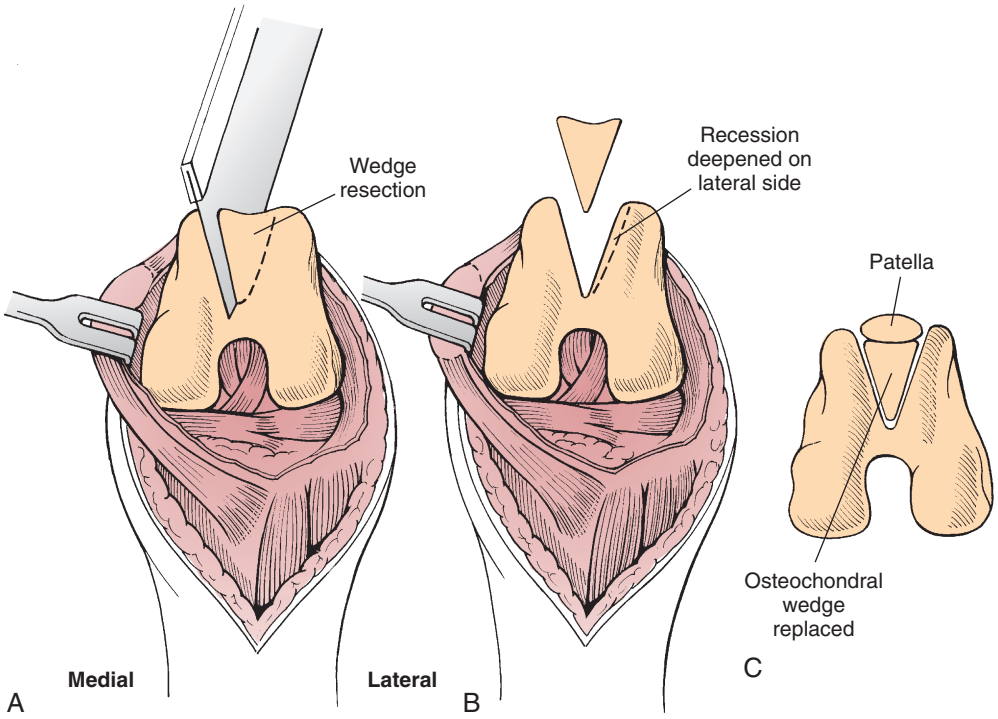


FIG. 34.139 Trochlear wedge recession. (A) Resect an osteochondral wedge from the patellar groove. (B) Remove bone from the sides of the incised groove to deepen the sulcus. (C) Replace the osteochondral wedge.

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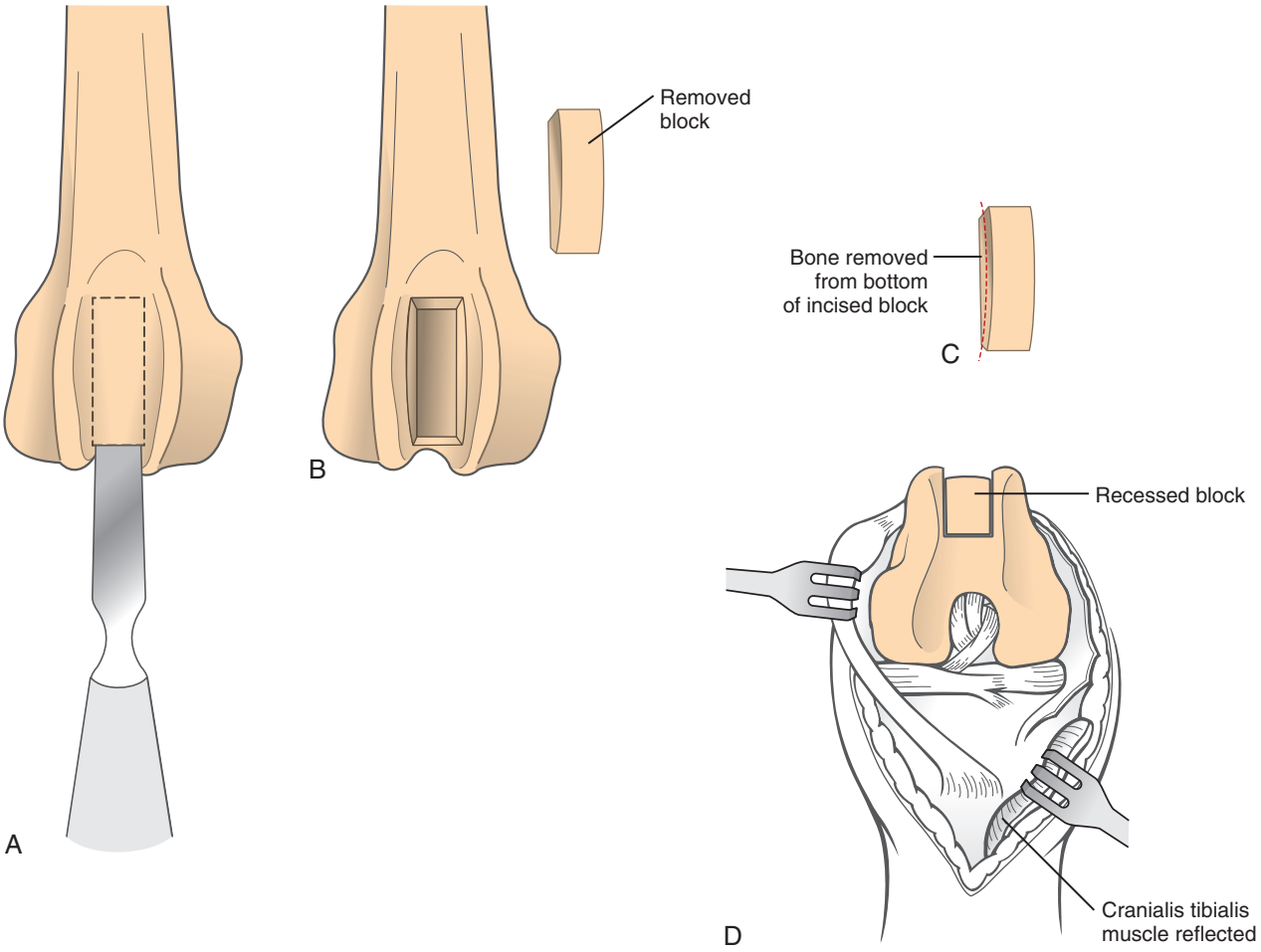


FIG. 34.140 Trochlear block recession. (A) Use a thin saw blade to make two parallel cuts just axial to both trochlear ridges. (B) Use an osteotome from proximal and distal to elevate an osteochondral block from the patellar groove. (C) Remove bone from the bottom of the incised block to deepen the sulcus. (D) Replace the osteochondral block.

cartilage of the trochlea, making a block-shaped outline. Be sure that the width of the cut is sufficient to accommodate the width of the patella but preserves the trochlear ridges. Using a surgical blade in very small and young patients, a hand saw in small and medium patients, or a power saw in large patients, deepen the proximodistal cuts 2 to 6 mm into the bone (Fig. 34.140A). Using an osteotome that is the same width as the osteotomy, elevate the osteochondral segment (Fig. 34.140B). Insert the osteotome from both the proximal and distal extents of the osteotomy, meeting in the middle. Take care to remove an appropriate thickness of bone and to avoid cracking or splitting the osteochondral segment. Deepen the recession in the trochlea by removing additional bone from the base of the groove (Fig. 34.140C). Replace the free osteochondral fragment when the depth is sufficient to house 50% of the height of the patella (Fig. 34.140D). The osteochondral wedge remains in place because of the net compressive force of the patella and friction between the cancellous surfaces of the two cut edges.

NOTE With a medial luxation, it often is best to take more bone from the lateral side of the groove, preserving as much of the medial ridge as possible.

Medial Release

The medial joint capsule is thicker than normal and contracted in patients with a grade III or grade IV medial patellar luxation. In these patients, the medial joint capsule and retinaculum must be released to allow lateral placement of the patella. *Using a scalpel, make a medial parapatellar incision through the medial fascia and joint capsule. Begin the incision at the level of the proximal pole of the patella, and extend it distally to the tibial crest. In more severe cases, the release incision needs to be extended proximally on the medial thigh. If dynamic contraction of the cranial sartorius muscle and vastus medialis muscle directs the patella medially, release the insertions of these muscles at the proximal patella. Redirect the insertions and suture them to the vastus intermedius muscle.*

Place a cruciate or simple interrupted suture that does not close the tissue gap when the patella is in the proper position. Placement of a loose suture will prevent iatrogenic lateral luxation.

Tibial Tuberosity Transposition

Make a lateral parapatellar incision through the fascia lata, and carry the incision distally onto the tibial tuberosity below the joint line. Reflect the cranialis tibialis muscle from the lateral tibial

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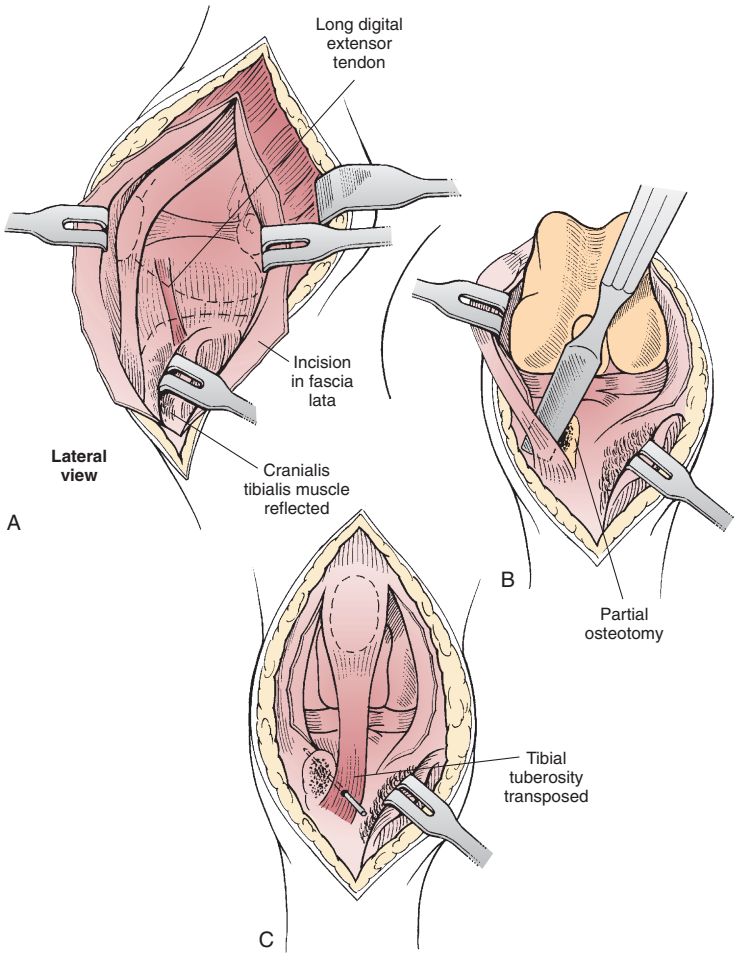


FIG. 34.141 Medial patellar luxations. (A) Transpose the tibial crest laterally. Make a lateral parapatellar incision through the fascia lata and carry the incision distally onto the tibial tuberosity below the joint line. Reflect the cranialis tibialis muscle from the lateral tibial tuberosity and tibial plateau to the level of the long digital extensor tendon. (B) Position an osteotome beneath the patellar ligament and partly ostectomize the tibial crest. Do not transect the distal periosteal attachment. (C) Stabilize the tibial tuberosity in its new location with one or two small K-wires.

tuberosity and tibial plateau to the level of the LDE tendon (Fig. 34.141A). Use sharp dissection to gain access to the deep surface of the patellar tendon for placement of an osteotome, Liston bone cutter, or sagittal saw. Beginning at the level of the patella, make a medial parapatellar incision through the fascia and distally through the periosteum of the tibial tuberosity. Position an osteotome, Liston bone cutter, or sagittal saw beneath the patellar tendon 3 to 5 cm caudal to the cranial point of the tibial tuberosity (Fig. 34.141B). Complete the osteotomy in a proximal to distal direction. Do not transect the distal periosteal attachment. The degree of lateral movement of the tibial tuberosity is subjective, but it is based on the longitudinal realignment of the tuberosity relative to the trochlear groove. *Once the site of relocation is chosen, remove a thin layer of cortical bone with a rasp or osteotome. Lever the tibial tuberosity into position, and stabilize it with one or two small K-wires directed caudally and slightly proximally (Fig. 34.141C). Engage the caudal cortex, but do not exit the pin from the tibia caudally; if the pin protrudes too far from the caudal cortex of the tibia, persistent lameness results. Check the stability of the patella as described previously, and relocate the tuberosity if needed.* Security of the tibial transposition is increased by use of a figure-eight wire added to pins in a tension band wire fashion. The addition of a figure-eight wire is strongly recommended when performing tibial crest transposition in large-breed dogs and should be considered in all but the smallest patients.

Failure of tibial crest transposition results in proximal displacement of the tibial crest and patella. Restoration of normal anatomy following this complication is difficult, and regardless of treatment, permanent disability often results. *Drill a small hole in the cranial cortex of the tibia several millimeters distal to the distal aspect of the osteotomy. Pass orthopedic wire, or nonabsorbable suture in very small patients, through the hole and over the tips of the pins or screw in a figure-eight pattern. Tighten the wires.*

Lateral Imbrication, Tightening, and Reinforcement

For suture imbrication, place a polyester suture through the femorofabellar ligament and lateral parapatellar fibrocartilage (Fig. 34.142). Next, place a series of imbrication sutures through the fibrous joint

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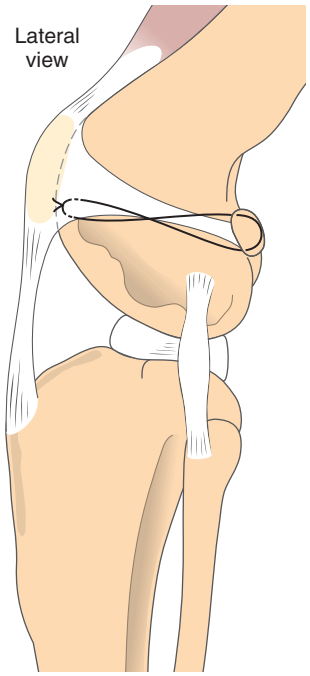


FIG. 34.142 Lateral reinforcement of the retinaculum may be performed by placing a polyester suture through the femoral-fabellar ligament and lateral parapatellar fibrocartilage.

capsule and lateral edge of the patella tendon. With the leg in slight flexion, tie the femoral-fabellar suture and imbrication sutures. If the patella is out of position most of the time, the retinaculum opposite the side of the luxation will be stretched; with medial luxations, there is redundant lateral retinaculum. *Once the patella has been reduced, excise the excess retinaculum and joint capsule, allowing tight closure of the arthrotomy.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

A fine-toothed saw (No. 12 X-acto saw, available at most hobby shops) or sagittal saw are used for trochlear wedge or block recession. An osteotome is needed to perform a block recession. An osteotome and mallet, K-wires or lag screw, orthopedic wire, and a hand chuck or drill are needed to secure the tibial crest when tibial tuberosity transposition is performed.

POSTOPERATIVE CARE AND ASSESSMENT

Activity should be restricted to specific physical rehabilitation exercises and leash walking for 6 to 8 weeks; then the animal should be gradually returned to normal activity over a 6-week period. A sample physical rehabilitation exercise protocol can be found by referring to Table 34.13. Radiographs should be obtained at 6 to 8 weeks to evaluate healing of a tibial crest transposition.

PROGNOSIS

Recurrent luxation after surgery has been reported in up to 50% of joints evaluated.[73–78] However, most are grade I luxations that do not affect clinical function. Most stifle joints function well enough that lameness is not apparent during examination, nor do clients report clinical dysfunction. Most patients with recurrent luxation show reluxation only on physical examination when manual force is used to displace the patella. Correlation of reluxation with the method or methods of surgical correction has not been well defined.[73,77,78] Overall, the prognosis for patients undergoing surgical correction of a grade I to III patellar luxation is excellent for return to normal limb function. DJD progresses despite treatment, but it is not as severe as that associated with chronic CCL rupture. Prognosis for patients with grade IV patellar luxation is guarded. Many joints require multiple surgery, and some patellae cannot be reduced without major corrective osteotomies. Surgical correction of grade IV patellar luxations is not necessarily indicated because function following surgery may be no better than the preoperative function.[75,76]

LATERAL PATELLAR LUXATION

DEFINITIONS

Lateral patellar luxation is an intermittent or permanent displacement of the patella from the trochlear sulcus. **Genu valgum** is a deformity affecting giant-breed dogs associated with lateral patellar luxation, resulting in "knock knees" appearance. **Anteversion** is excessive external rotation of the proximal femur relative to the distal femur. **Coxa valga** is an abnormal increase in the angle formed by the femoral neck and shaft in the frontal plane.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Lateral patellar luxation is seen most often in large-breed dogs, but it does occur in small- and toy-breed dogs. The cause is unknown, but it is thought to be related to shifted force produced by the pull of the quadriceps lateral to the longitudinal axis of the femur and trochlear groove. This abnormally directed lateral force pulls the patella from the trochlear sulcus. Abnormal force placed on the growth plates of immature patients causes skeletal abnormalities that are mirror images of those seen with medial patellar luxation. For further discussion of the pathophysiology of patellar luxation, see p. 1254.

DIAGNOSIS

Clinical Presentation

Signalment

Dogs of either gender and any age or breed may be affected. Lateral patellar luxations are more frequently seen in large breeds of dogs than in small and toy breeds. Medial patellar luxation, however, is more common than lateral luxation in dogs of all sizes. Lateral patellar luxation has not been described in cats.

History

Affected animals are most commonly seen for evaluation of an intermittent, weight-bearing lameness. Owners may report that the animal occasionally holds the leg in a flexed position for one or two steps.

Physical Examination Findings

Physical examination findings vary and depend on the severity of luxation. The diagnosis is determined by finding or eliciting

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TABLE 34.13 Sample Rehabilitation Protocol for Patient With a Patellar Luxation Repair

Treatments/ Modalities	Day 1–3	Day 4–14	2–5 WK	5–8 WK	8+ WK
Pain medications	As directed	As directed	PRN	PRN	PRN
Cryotherapy	10–15 min 3 times daily to the affected stifle First session immediately after surgery	Use after exercise for 15 min	PRN	PRN	PRN
Heat therapy		Apply heat to the muscles before PROM as long as inflammation is not present	PRN	PRN	PRN
Massage	5 min 3 times daily gentle massage	Continue twice daily	Twice daily	Twice daily	Only if desired
PROM	8–10 repetitions 2–3 times daily Limit motion to sagittal plane only	Continue twice daily within comfortable range	All joints affected limb 8–10 reps Sagittal plane only		
Laser therapy	Daily	Every other day for first week then 2 times weekly	Twice weekly	Twice weekly	Discontinue
Walks	5-min supported leash walk 2–3 times daily for toileting only	Increase each walk by 2–3 min each week	Increase by 5 min each week	Increase by 5 min each week Add gentle inclines at week 6	15- to 20-min walks 2–3 times daily Add inclines and trotting as returns to normal
NMES		10 min twice daily to quadriceps and hamstrings of affected leg	10 min twice daily to quadriceps and hamstrings of affected leg	10 min twice daily to quadriceps and hamstrings of affected leg	
Balancing		5 min twice daily on soft foam	5 min twice daily soft foam pad	5–10 min twice daily unstable surface	PRN
Cavalettis (begin at 4 weeks)			Low rails 5–10 reps twice daily	10–15 reps twice daily	10–15 reps twice daily increased height
Sit to stand (begin at week 4)			Start with sling or elevated hind end 5–10 reps twice daily	10–15 reps twice daily	15–20 reps twice daily
Weaves				Slow, 10–15 reps twice daily	15–20 reps twice daily increasing speed
Stairs					Start at 1 flight twice daily working up to 5 flights, adding 1 flight per week at a slow speed
Underwater treadmill			5–10 min daily at a slow speed	15–20 min 2–3 times weekly	15–30 min twice weekly until released from rehab
Swimming (Begin at 6 weeks)				5–10 min daily	10–15 min 3–4 times weekly

Initial work in sagittal plane only and when starting exercises limit initially to one to two per session, gradually building to two to four per session at 5 weeks and beyond. Lameness and pain is an indication to decrease intensity and frequency.
NMES, Neuromuscular electrical stimulation; *PRN,* as needed; *PROM,* passive range of motion.

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lateral luxation of the patella and eliminating other causes of rear limb lameness (see later discussion). Patients with a grade I luxation generally show no lameness, and the diagnosis is made as an incidental finding on physical examination. Patients with a grade II luxation show occasional "skipping" when walking or running. These patients occasionally stretch the medial retinacular structures and appear for the first visit showing a non–weight-bearing lameness. Lameness in patients with a grade III luxation varies from an occasional skip to a weight-bearing lameness. Patients with a grade IV luxation walk with the rear quarters in a crouched position because of inability to extend the stifle joints fully.

Diagnostic Imaging

With a grade III or grade IV luxation, standard craniocaudal and medial-to-lateral radiographs consistently show the patella to be displaced laterally. With a grade I or grade II luxation, the patella may be within the trochlear sulcus when the radiographs are made or may be displaced laterally. Varying degrees of osteoarthrosis may be present. CT aids in determining the specific type and degree of deformity.

Laboratory Findings

Consistent laboratory findings are not seen. Fluid analysis obtained by arthrocentesis demonstrates changes compatible with osteoarthritis.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include hip dysplasia, osteochondritis of the stifle or tarsal joints, panosteitis, hypertrophic osteodystrophy, capital physeal injury, CCL rupture, and muscle strain. It is important to note that many patients with lateral patellar luxation also have evidence of hip dysplasia. Both conditions may contribute to the lameness and require appropriate treatment for the dog to become sound.

MEDICAL MANAGEMENT

Lateral patellar luxation may be treated conservatively or surgically. The choice of treatment depends on the patient's age, the clinical history, and the physical findings. Older patients that are not lame and that have patellar luxation diagnosed as an incidental finding do not require surgical intervention. Rather the client should be instructed to observe for clinical signs associated with patellar luxation.

SURGICAL TREATMENT

The goals and methods of treatment for lateral patellar luxation are similar to those described for medial patellar luxation on p. 1256. The surgical technique used in animals with lateral patellar luxations is similar to that described for osteotomy of the tibial tuberosity in animals with medial patellar luxations (see p. 1258) except that the tuberosity is repositioned and stabilized medially (Fig. 34.143A). Because lateral patellar luxation occurs in large-breed dogs, the use of figure-eight wire with the pins (tension band wire) of the tibial tuberosity is recommended (see Fig. 34.143B). Avulsion of the tibial crest following surgery is difficult to correct due to contracture of the quadriceps and proximal displacement of the patella. Limb salvage is difficult following this complication. The medial retinaculum is reinforced with suture reconstruction, fascial lata transposition, and/or excision of the redundant joint capsule (see Fig. 34.143C). The lateral restraints are released to help neutralize lateral forces acting on the patella. Methods for deepening the trochlear groove are the same as those described for medial patellar luxation. Osteotomies of the femur and tibia may be required to correct severe angular and torsional deformities. Corrective osteotomies are best performed by a specialist with the necessary equipment and training in these complex procedures.

Preoperative Management

Perioperative antibiotics (see Chapter 9) and preemptive pain management with NSAIDs (see Table 34.3), opiates, or epidural analgesia are indicated for dogs undergoing stifle reconstructive techniques.

Anesthesia

See Tables 32.1 and 32.2 for suggested protocols for the anesthetic management of patients with orthopedic disease. Opioid epidural administration reduces postoperative discomfort.

Surgical Anatomy

In patients with lateral patellar luxation, abnormal wear of the lateral trochlear ridge may be evident. The patella and patellar ligament lie lateral to the trochlear sulcus in these patients. The patellar ligament must be identified before making a parapatellar incision to enter the joint. The medial retinaculum appears stretched in these patients, whereas the lateral retinaculum appears contracted. Release of the lateral retinaculum (with incision into the joint) reveals a thickened joint capsule. Rupture of the origin of the LDE tendon is often identified in the lateral pseudo-groove on entry to the joint.

Positioning

The patient is positioned in dorsal recumbency or in lateral recumbency with the affected leg up. The leg should be prepared from the dorsal midline to the tarsal joint. A hanging-leg preparation and dorsal recumbency facilitate manipulation of the leg during surgery.

SURGICAL TECHNIQUE

A description of the surgical technique for correction of patellar luxation was presented earlier and on p. 1258.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Instruments needed for tibial tuberosity transposition are an osteotome and mallet, K-wires or screws, and orthopedic wire to secure the tibial crest transposition, and a hand chuck or drill. In large-breed dogs, creation of the tibial crest osteotomy is simpler with a power sagittal saw. A fine-toothed saw is needed for trochlear wedge recession. An osteotome and saw are needed for trochlear block recession.

POSTOPERATIVE CARE AND ASSESSMENT

Activity should be restricted to specific physical rehabilitation techniques and leash walking for 6 to 8 weeks, at which time radiographs should be obtained to confirm healing of the tibial crest transposition; the animal should be gradually returned to unsupervised activity over a 6-week period. A sample physical

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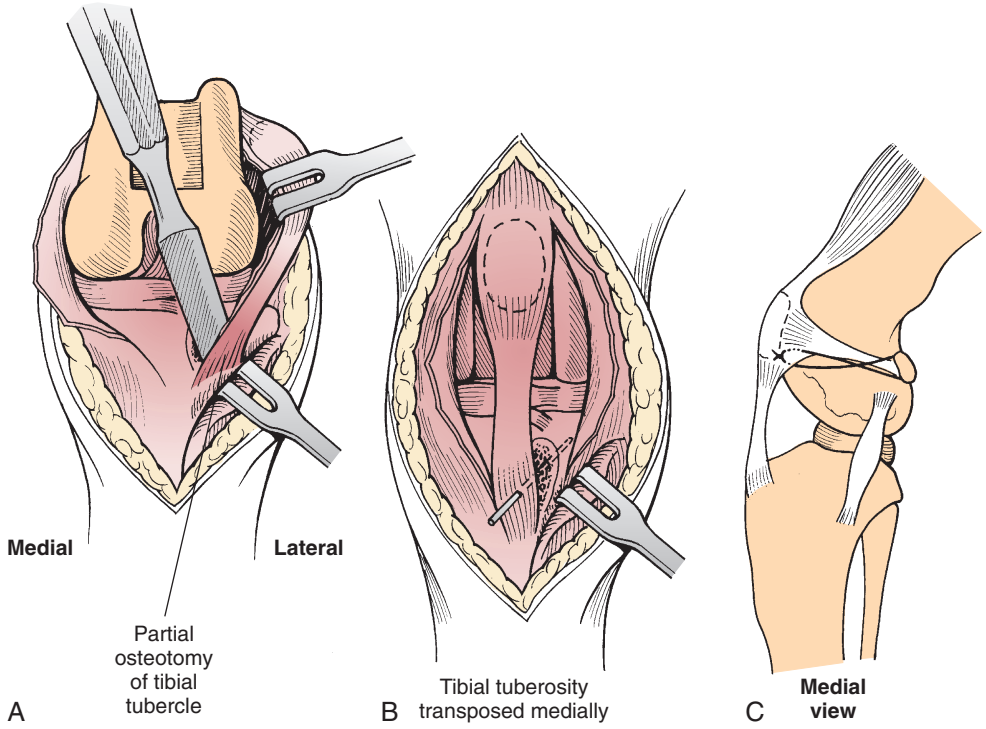


FIG. 34.143 For lateral patellar luxations, transpose the tibial crest medially. (A) Make a parapatellar incision through the fascia lata and carry the incision distally onto the tibial tuberosity below the joint line. Position an osteotome beneath the patellar ligament and partly ostectomize the tibial crest. Do not transect the distal periosteal attachment. (B) Stabilize the tibial tuberosity in its new location with one or two K-wires and a figure-eight wire or a screw. (C) Reinforce the medial retinaculum with suture placed from the fabella to the parapatellar fibrocartilage.

rehabilitation exercise protocol can be found by referring to Table 34.13.

PROGNOSIS

The prognosis is less favorable for large dogs with lateral patellar luxations than for smaller dogs with a medial patellar luxation; however, the prognosis is good for a return to functional activity with grades I to III.[79] The prognosis with grade IV patellar luxation in large-breed dogs is guarded to poor due to the frequent need for multiple surgeries and femoral and/or tibial correction and the severity of the soft tissue deformity and shortening.[79]

OSTEOCHONDRITIS DISSECANS OF THE STIFLE

DEFINITION

OCD or *osteochondrosis* is a disturbance in endochondral ossification that leads to retention of cartilage; it occasionally occurs in the stifle of immature large dogs.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

OCD begins with a failure of endochondral ossification in either the physis or the articular epiphyseal complex, which is responsible for the formation of metaphyseal bone. This condition occurs more commonly in the shoulder, elbow, and hock. The pathogenesis of OCD is discussed on p. 1154. With OCD of the stifle, a piece of cartilage and subchondral bone is usually observed that involves the medial surface of the lateral femoral condyle (most frequently affected) or the medial femoral condyle. The condition is often bilateral.

DIAGNOSIS

Clinical Presentation

Signalment

Affected dogs are usually large (e.g., German shepherds, Great Danes) and young, with the average age at onset of lameness being 5 to 7 months (range, 3 months to 3 years). Males are affected more often than females.

History

Rear limb lameness, which worsens after exercise, may be acute or chronic and mild or severe. Often the onset of the lameness is insidious. Owners frequently complain that the dog is stiff in the morning or after rest, and they are generally concerned that the dog may have hip dysplasia.

Physical Examination Findings

Lameness of one rear limb is usually evident. There may be stifle joint effusion and crepitation, especially if DJD is progressing. In immature dogs, a slight cranial drawer may be noted, especially

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FIG. 34.144 Radiograph of a stifle with osteochondritis dissecans of the lateral femoral condyle. There is a radiolucent defect on the distal surface of the lateral femoral condyle *(arrow)*.

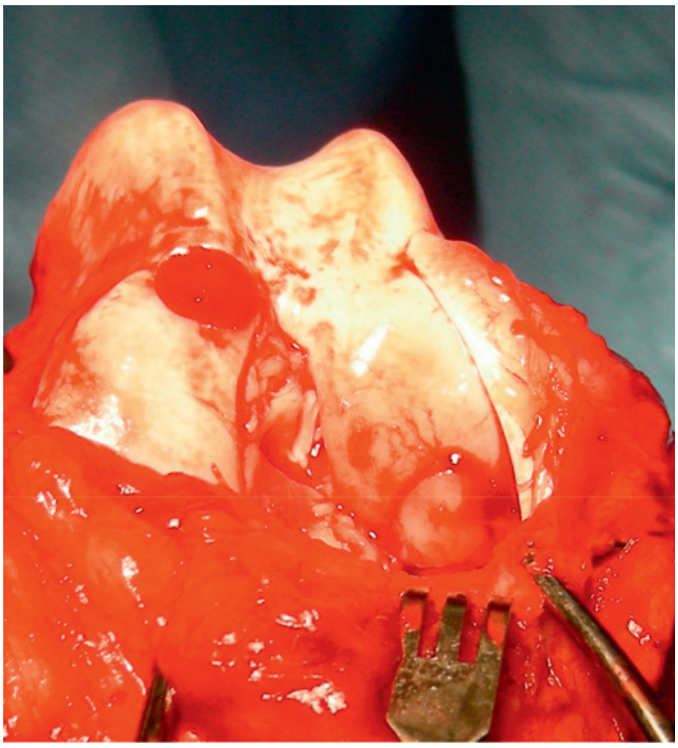


FIG. 34.145 Stifle with osteochondritis dissecans of the lateral femoral condyle treated with autogenous osteochondral plug transfer procedure.

with muscle atrophy. However, when the cranial drawer is tested in animals with stifle OCD, drawer motion should stop abruptly, indicating that the CCL is intact (see p. 1227).

Diagnostic Imaging

Radiographic views should include standard craniocaudal and lateral views of the stifle. Oblique views may be needed to observe the extent of the lesion. Radiographs of both stifles should be obtained to identify bilateral disease. Definitive radiographic diagnosis of OCD is made when a radiolucent concavity is observed on the medial or lateral femoral condyle (Fig. 34.144). More subtle radiographic signs include flattening of the articular surface and subchondral sclerosis.

Arthroscopy

Arthroscopy provides minimally invasive definitive diagnosis of OCD. The site of the lesion is easily visualized following fat pad resection and flap removal; treatment of the underlying subchondral bone is straightforward (see the later discussion).

Laboratory Findings

Results of hematologic and serum biochemical analyses are normal in most affected animals. Results of arthrocentesis may include decreased synovial fluid viscosity, increased fluid volume, and an increased number of mononuclear phagocytic cells (up to 6000–9000 WBC/μL).

DIFFERENTIAL DIAGNOSIS

OCD must be differentiated from other diseases of young growing dogs that affect the rear limb and may cause similar clinical signs of lameness (hip dysplasia and panosteitis). Conditions of LDE tendons (avulsion, partial rupture, mineralization) should be ruled out. CCL rupture is ruled out by physical examination and by the presence of radiographic evidence of OCD.

MEDICAL MANAGEMENT

Medical therapy may be used to treat older dogs with established osteoarthritis. Lame animals should be treated with confinement and administration of NSAIDs (see Table 34.3). After the lameness has subsided, exercise is increased gradually to strengthen the surrounding musculature. Weight control is also important in the management of osteoarthritis.

SURGICAL TREATMENT

Surgical removal of the cartilage flap allows the defect to heal by outgrowth of fibrocartilage from underlying subchondral bone. Surgery may be useful in young animals when the disease is diagnosed before the onset of osteoarthritis. However, surgical management does not usually alter the progression of osteoarthritis.

Many cases of stifle OCD are bilateral, and as the disease occurs most frequently in large- and giant-breed dogs, bilateral arthrotomy may result in significant short-term morbidity. Dogs requiring bilateral surgery are better treated arthroscopically, as the degree of postoperative pain and disability is significantly decreased.

OCD of the stifle may be amenable to treatment with autogenous osteochondral plug transfers to replace the deficient subchondral bone and articular cartilage (Fig. 34.145). Total knee replacement is indicated in cases with severe clinical

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osteoarthritis. These procedures require advanced equipment and training and should be performed by a surgical expert.

Preoperative Management

Perioperative antibiotics (see Chapter 9) and preemptive pain management with NSAIDs (see Table 34.3), opiates, or epidural analgesia are indicated for dogs undergoing stifle exploration (see p. 980).

Anesthesia

See Tables 32.1 and 32.2 for suggested protocols for the anesthetic management of patients with orthopedic disease. Opioid epidural administration reduces postoperative discomfort.

Surgical Anatomy

Surgical anatomy of the stifle is discussed on p. 1233. Either arthroscopy or a standard medial or lateral parapatellar approach can be used to expose the lesion.

Positioning

The dog is positioned in dorsal recumbency with the affected limb suspended for draping. The limb is then released to allow access to the medial or lateral surface of the stifle.

SURGICAL TECHNIQUE

Arthroscopy

Standard arthroscopy portals with fat pad excision provide outstanding visualization and treatment of the lesion with a minimally invasive technique. Dogs requiring bilateral treatment may have less pain and short-term disability when treated arthroscopically than when treated with arthrotomy. *Establish standard craniomedial and craniolateral arthroscopy portals (see pp. 1230–1231). Remove the fat pad as needed to visualize the medial and lateral femoral condyles. If working without a cannula, insert a locking grasper through the instrument portal. Grab the flap and twist it, rolling the grasper toward the bone surface until the flap is separated from the bone. Remove the flap. If working through a cannula system, insert a large aggressive power burr, and shave and suction the flap until gone. After flap removal insert a curette and examine the edges of the lesion for loose cartilage. Make the edges of the lesion 90 degrees to the articular surface and subchondral bone to improve healing. Insert a hand burr and perform mild abrasion arthroplasty or perform microfracture on the lesion. Shut off fluid inflow to the joint, and observe for adequate bleeding from the subchondral bone. Flush and close the joint routinely.*

Arthrotomy

The surgical approach selected depends on the surgeon's preference (see pp. 1235–1236 for the lateral and medial parapatellar approaches). *Regardless of the technique used for exposure, examine the appropriate femoral condyle and remove the flap of cartilage. Perform curettage to remove fragments of cartilage from the edges of the lesion. Curette the underlying bone or perform microfracture on the lesion. Repeatedly flush the joint to remove any small fragments before closure.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

For arthroscopy, a power shaver is required to remove the fat pad. A hand burr or power burr is useful to abrade the subchondral bone. Micropicks and a mallet are necessary for microfracture. For either approach, a bone curette is needed to curette the edges of the lesion.

POSTOPERATIVE CARE AND ASSESSMENT

No bandaging is required following arthroscopic treatment unless there is excessive drainage from the portal sites. Following open arthrotomy, the limb may be bandaged after surgery for 3 to 5 days to provide soft tissue support. Activity should be restricted to specific physical rehabilitation techniques and leash walking for 6 weeks; the animal should be gradually returned to normal activity over a 6-week period (see Chapter 11).

PROGNOSIS

The prognosis depends on the size of the lesion and whether DJD is present. Dogs treated conservatively for OCD of the stifle usually have continued intermittent lameness and progressive osteoarthritis. Those treated surgically for OCD of the stifle may have improved limb function, but lameness may be evident after exercise. Osteoarthritis is usually present even after surgery and requires occasional medical treatment (see p. 1137). Most dogs are functional pets and are only intermittently lame. Treatment of the cartilage defects with autogenous osteochondral transfers may improve the outcome.[80] Total knee replacement may be necessary to manage clinical signs associated with severe osteoarthritis.

TARSUS

LIGAMENT INJURY OF THE TARSUS

DEFINITIONS

Ligament injury to the tarsal joint may result from injury within or surrounding the tarsocrural, proximal intertarsal, distal intertarsal, or tarsometatarsal joints. **Luxation** is a complete separation between one of the above joint surfaces, and **subluxation** is a partial or incomplete separation. The term **varus** denotes an inward deviation of the limb, and **valgus** denotes an outward deviation. **Shearing** or **degloving injuries** result from abrasion, which may cause skin, ligament, and bone loss.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Ligament injuries of the tarsus usually result from severe trauma, such as vehicular accidents. Most injuries are open abrasions with moderate to severe loss of soft tissue or bone or both. Shearing injuries usually involve the tarsocrural joint. They typically occur when the limb is caught beneath a tire and are associated with severe abrasion of the soft tissue and malleoli. Shear injuries can involve the lateral or medial surface of the tarsus, but the medial surface is more commonly injured. Subluxation results from injury of the medial or lateral collateral ligament complex or fracture of the medial or lateral malleolus. Luxation generally results from injury of both the medial and the lateral collateral ligament complexes, fracture of both malleoli, or fracture of one malleolus with injury to the contralateral collateral ligament complex. A number of intertarsal injuries have been recognized as resulting from disruption of various ligament complexes between tarsal bones. Proximal intertarsal

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subluxation, proximal intertarsal luxation, and tarsometatarsal luxation are most common.

DIAGNOSIS

Clinical Presentation

Signalment

Any age or breed and either gender of dog or cat may be affected.

History

Most animals are brought in for evaluation of a non–weight-bearing lameness. Some animals have an associated open wound over the tarsus.

Physical Examination Findings

Complete tarsocrural joint luxation is obvious; the animal is non–weight-bearing and the paw deviates at an unnatural angle. Pain, swelling, and crepitus are present. Subluxations may be more difficult to diagnose, particularly if only one part of the medial or lateral ligament complex is injured. Animals with very unstable subluxations are unable to bear weight, and the paw deviates to the direction opposite the ligamentous damage (i.e., if the subluxation is medial, the paw deviates laterally). If partial tears of the collateral complex are suspected, varus and valgus forces should be applied to the joint while it is extended and flexed. Laxity in extension denotes injury to the long components of the collateral ligament complex, whereas laxity in flexion only denotes injury to the short component of the collateral ligament complex.

NOTE Be sure to place the joint in extension to evaluate medial or lateral restraint injury.

With shearing injuries that cause luxation or subluxation, soft tissue abrasion and bone loss occur. Often, the bone surface and articular cartilage are exposed. With intertarsal luxation and tarsometatarsal subluxation, animals are usually in pain and unable to bear weight on the affected limb, and the involved area is swollen. Abnormal rotation and deviation of the foot are seen.

Diagnostic Imaging

Standard craniocaudal and medial-to-lateral radiographs are often sufficient for complete evaluation (Fig. 34.146). If instability is suspected but not confirmed, craniocaudal and varus-valgus stress films performed with the patient anesthetized are useful (Fig. 34.147).

Laboratory Findings

Consistent laboratory abnormalities are not seen.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include mild sprain, fractures of one or more tarsal bones, OCD of the talus, and arthritis. These conditions can be differentiated from tarsal luxation or subluxation by palpation and radiography.

MEDICAL MANAGEMENT

Medical management is not rewarding with these injuries. Surgery is needed to restore the functional integrity of the joint.



FIG. 34.146 Craniocaudal radiograph of a luxated tarsal joint. Note the fracture of the lateral malleolus and the medial displacement of the tibia.



FIG. 34.147 Varus stress radiograph of the tarsus. Widening of the medial joint space indicates damage to the medial collateral ligament and joint capsule.

SURGICAL TREATMENT

Each patient needs a thorough neurologic and vascular evaluation to determine the feasibility of treatment. With tarsocrural luxation or subluxation, the objectives of treatment are to reestablish joint stability to achieve pain-free weight bearing. Reconstruction of the short and long collateral ligament components is recommended for optimum results. With severe ligament injuries of the tarsus, partial and complete arthrodeses are alternatives to joint reconstruction. With shearing injuries, the objective of management is to achieve a pain-free, functional patient. If the cartilage and bone damage is limited to the malleolus, reconstruction can be attempted. However, if damage to the cartilage and bone is severe, tarsal arthrodesis should be considered.

Proximal intertarsal subluxation with plantar instability occurs when excessive dorsal flexion causes disruption of the plantar ligament complex between the calcaneus and fourth tarsal bones.

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FIG. 34.148 Lateral radiograph of the tarsus of a dog with proximal intertarsal luxation.



FIG. 34.149 Craniocaudal radiograph of a dog with a tarsometatarsal subluxation. Note the fragmentation of the second tarsal bone *(arrow)*.

Treatment involves partial tarsal arthrodesis or selective fusion of the joint surfaces between the calcaneus and fourth tarsal bones.

Proximal intertarsal subluxation can occur with dorsal instability. This injury results from damage to the dorsal ligaments and dorsal joint capsule between the talus and central tarsal bone caused by hyperextension. Treatment in most cases is best accomplished with rigid coaptation. If this is not successful, partial tarsal arthrodesis or selective fusion of the talocentral joint can be performed.

Proximal intertarsal luxation occurs when excessive dorsal flexion causes disruption of the plantar ligament complex both between the calcaneus and fourth tarsal bones and between the talus and the central tarsal bone (Fig. 34.148). Complete luxation is differentiated from subluxation by notable dorsal displacement of the distal tarsus at the proximal intertarsal joint. Treatment is by partial tarsal arthrodesis or selective fusion of the proximal intertarsal joint.

Tarsometatarsal luxation (Fig. 34.149) occurs when excessive dorsal flexion causes disruption of the plantar ligaments and fibrocartilage. The luxation occurs between the distal row of tarsal bones and metatarsal bones. Treatment is best accomplished with partial tarsal arthrodesis or selective fusion of the distal intertarsal joint (tarsometatarsal joint). Occasionally, the luxation occurs laterally between the fourth tarsal bone and adjacent metatarsal bones, with the medial separation occurring between the second and third tarsal bones and the central tarsal bone. In these patients, selective fusion of the joint space between the fourth tarsal and adjacent metatarsal bones is accomplished with a bone plate secured to the fourth tarsal bone and fifth metatarsal bone. Medially a small bone plate is secured to the central tarsal bone proximally and the second tarsal and second metatarsal bones distally.

Preoperative Management

Open wounds should be cleaned and a sterile dressing applied before surgery (see Shearing Injuries). Patients with open wounds should be started on therapeutic antibiotics (see Chapter 9). A bandage with a splint should be applied to support the limb, and the animal should be confined to a small space to prevent further joint damage until definitive surgery can be performed. Preemptive pain management with NSAIDs (see Table 34.3), opiates, or epidural analgesia is indicated for dogs undergoing tarsal reconstruction.

Anesthesia

See Tables 32.1 and 32.2 for selected anesthetic protocols for the management of patients with orthopedic disease. Opioid epidural administration reduces postoperative discomfort. Traumatized animals should be evaluated for other injuries that may affect the anesthetic protocol.

Surgical Anatomy

The tarsus consists of the tibia, fibula, and proximal tarsal, distal tarsal, and metatarsal bones. These bones form the tarsocrural, intertarsal, and tarsometatarsal joints. The tarsocrural joint is formed by the fibula and cochlea of the tibia proximally and the talus and calcaneus distally. The intertarsal joints are formed by articulations between the tarsal bones, and the tarsometatarsal joints are formed by the articulations between the distal tarsal and metatarsal bones. The tarsal joints are supported by a complex arrangement of ligaments. The major ligaments providing medial support of the tarsocrural joint are the long medial collateral ligament, short medial collateral ligament, and calcaneocentral ligament (Fig. 34.150A). Lateral support is provided by the long lateral collateral ligament and short calcaneofibular ligament (see Fig. 34.150B). Knowledge of the anatomic points of origin and insertion of each ligament is important for reconstruction of medial and lateral ligament injuries. A complex network of plantar ligaments, tarsal fibrocartilage, and joint capsule provides most of the stability of the intertarsal and tarsometatarsal joints. Plantar ligaments originate from the calcaneus and attach to the central and fourth tarsal bones before inserting on the metatarsal bones (see Fig. 34.150C).

Positioning

The limb is clipped from the coxofemoral joint to the digits so that the paw can be included in the sterile field. This allows

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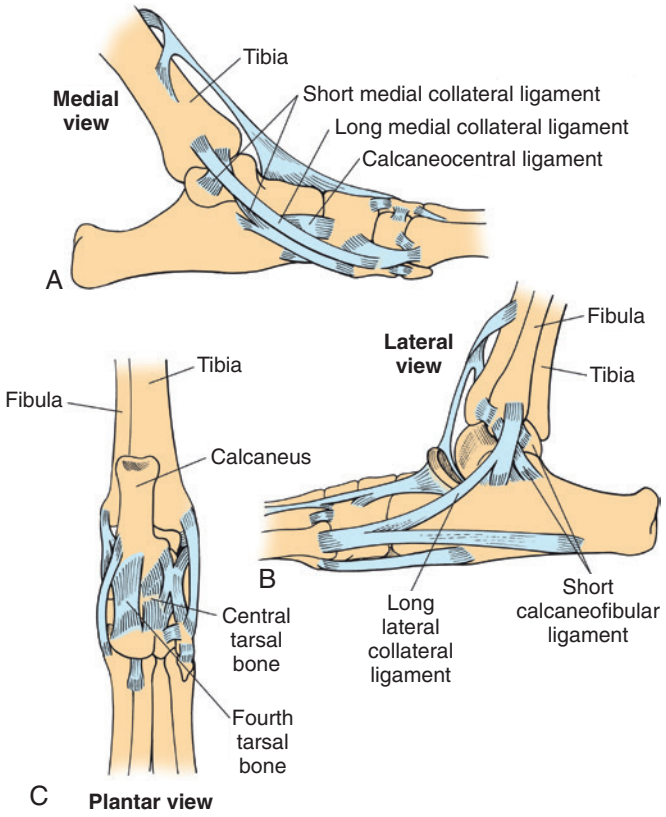


FIG. 34.150 (A) Medial, (B) lateral, and (C) plantar views of ligament structures that provide stability to the tarsal joint.

direct manipulation and visual orientation of the paw during reconstruction. Dorsal recumbency allows exposure of all surfaces of the tarsus. A hanging-leg preparation facilitates manipulation of the limb during surgery (see Chapter 5). The proximal tibia is available for cancellous bone harvest. As an alternative, prepare the proximal humerus for cancellous bone harvest when arthrodesis is performed. For arthroscopy, the patient is placed in dorsal recumbency for a cranial approach to the tibiotarsal joint and in sternal recumbency for a caudal approach.

SURGICAL TECHNIQUE

Tarsocrural Luxation or Subluxation

Depending on which side is injured, make a curved incision centered over the medial or lateral malleolus. Begin the incision 4 cm above the joint line, and continue distally to a point 4 cm below the tarsometatarsal joint line. Incise the subcutaneous tissue and deep fascia along the same line. Once the deep fascia has been incised, remnants of the collateral ligament complex and joint capsule are exposed, and the articular surface is visible. Reduce and align the joint surfaces. Suture the joint capsule and injured ligaments with small, nonabsorbable suture. Protect the repair with nonabsorbable, heavy (No. 2 to No. 5, depending on the animal's size) figure-eight sutures strategically placed to mimic the short and long components of the collateral ligament complex. Drill bone tunnels in the malleolus where the ligament complex originates. Next, drill tunnels where the short and long components of the collateral ligament complex insert. Place two strands of nonabsorbable polyester suture through the predrilled holes in the malleolus. Pass one end of the suture strand through the tunnel drilled to mimic the insertion of the short component, and pass the end of the other strand through the tunnel drilled to mimic the insertion of the long component of the collateral ligament complex. As an alternative, use bone anchors. Place the sutures in a figure-eight pattern and tie them such that the suture placed as the short component is tied with the joint in 90 degrees of flexion (Fig. 34.151). Tie the suture placed to simulate the long component with the joint in a normal standing angle. Protect the sutures with a splint or a rigid or hinged external fixator.

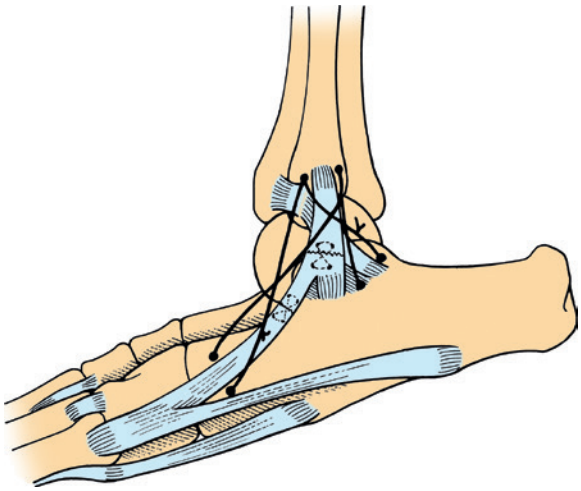


FIG. 34.151 Primary repair of collateral ligaments is supported with heavy suture to simulate the long and short components of the collateral ligament complex.

Shearing Injuries

When the animal is first seen, cover the wound with a sterile dressing and temporarily immobilize the limb with an external splint or external fixator. Once the animal is stable enough to be anesthetized, debride the wound. Liberally flush the wound with 0.05% chlorhexidine. Fill the wound with sterile K-Y Jelly and clip the surrounding area. Transfer the patient to the operating room; debride obvious necrotic tissue, and remove foreign material. If the medial surface is abraded, perform ligament reconstruction by inserting bone screws or bone anchors in the malleolus and talus to mimic the origin and insertion of the medial collateral ligament complex (Fig. 34.152). If the lateral surface is abraded, place bone anchors in the malleolus and calcaneus to mimic the origin and insertion of the lateral collateral ligament complex (Fig. 34.153). Place figure-eight, heavy, nonabsorbable suture or wire between the screws. Tie the suture used to mimic the short component of the ligament complex with the joint at 90 degrees and tie the suture used to mimic the long component of the ligament complex with the tarsus in a normal standing angle. If malleolar fracture is present rather than ligament injury, reduce and stabilize the fracture with a tension band technique. Immobilize the reconstruction for 2 to 4 weeks with a transarticular external skeletal fixator. Place one full pin 6 to 7 cm proximal to the malleolus and one full pin through the metatarsal bones just below the tarsometatarsal joint line. Contour medial

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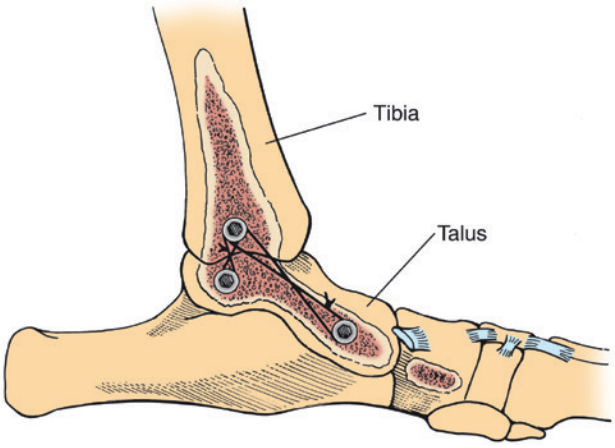


FIG. 34.152 Reconstruction of the medial collateral restraint complex with screws and suture material. Note the proper placement of the proximal and distal talar screws to mimic the insertion points of the short and long components of the restraint complex, respectively.

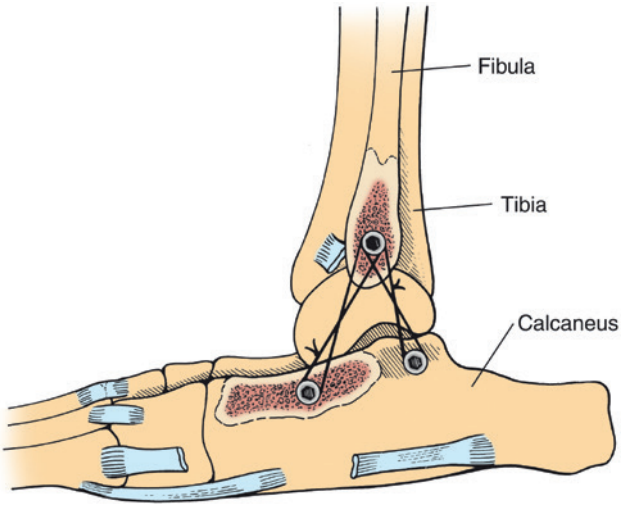


FIG. 34.153 Reconstruction of the lateral collateral restraint complex with screws and suture material. Note the proper placement of the proximal and distal talar screws to mimic the insertion points of the short and long components of the restraint complex, respectively.

and lateral external bars to an angle simulating the standing angle of the tarsal joint. Place a half pin 2 to 3 cm above the malleolus and a second half pin through the central and fourth tarsal bones. Tighten the pin clamps. Alternatively, use a bivalve short (midtibia) cast (see p. 995).

NOTE After placement of the medial and lateral bars, resistance to craniocaudal bending can be improved by adding an additional external bar.

NOTE Conversion from a rigid fixator to a hinged fixator at 2 to 4 weeks postoperatively may aid in rehabilitation.



FIG. 34.154 Craniocaudal radiograph showing stabilization of proximal intertarsal instability with a bone plate placed on the lateral surface of the calcaneus and fifth metatarsal bones.

Partial Tarsal Arthrodesis

Make a lateral incision beginning at the proximal end of the calcaneus and extending distally 3 to 4 cm below the tarsometatarsal joint line. Remove articular cartilage from the joint surfaces and insert a cancellous bone graft. Apply a compression plate to the lateral surface of the calcaneus, fourth tarsal bone, and fifth metatarsal bone (Fig. 34.154).

Tarsometatarsal Luxation

Expose the articular surfaces of the joint through a cranial incision. Reflect the extensor tendons laterally to gain adequate exposure. Remove articular cartilage and insert a cancellous bone graft. Reduce the joint and stabilize with cross pins. Place one pin so that it enters the base of the fifth metatarsal bone, crosses the joint, and is seated in the central tarsal bone. Place the second pin so that it enters the base of the second metatarsal bone, crosses the joint, and is seated in the fourth tarsal bone. If the luxation occurs laterally, between the fourth tarsal bone and adjacent metatarsal bones, with the medial separation occurring between the second and third tarsal bones and central tarsal bone, expose the tarsometatarsal joint with two incisions. Make a lateral incision extending 5 cm proximal and distal to the tarsometatarsal joint line. Make a similar incision medially. Remove articular cartilage from all exposed joint surfaces, and insert a cancellous bone graft. Stabilize the fusion with small bone plates. Laterally, secure a plate to the fourth tarsal and fifth metatarsal bones (Fig. 34.155). Medially, secure a small bone plate to the central tarsal bone proximally and to the second tarsal and second metatarsal bones distally.

Tarsocrural Joint Arthrodesis (Pan Tarsal Arthrodesis)

Tarsocrural joint arthrodesis is indicated for severe injury of the tibial cochlea and condyles of the talus that precludes maintaining a long-term, pain-free articulation. Arthrodesis is also indicated when painful DJD is not responsive to conservative measures, and if repair of an Achilles tendon rupture fails (see p. 1285). In conjunction with tarsocrural joint fusions, the proximal intertarsal and tarsometatarsal joints are also fused. Arthrodesis may be performed with a cranially applied plate, a medially or laterally applied custom plate, or an external fixator. Lateral

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FIG. 34.155 Craniocaudal radiograph showing stabilization of tarsometatarsal instability with a bone plate placed on the lateral surface of the calcaneus and fifth metatarsal bones.

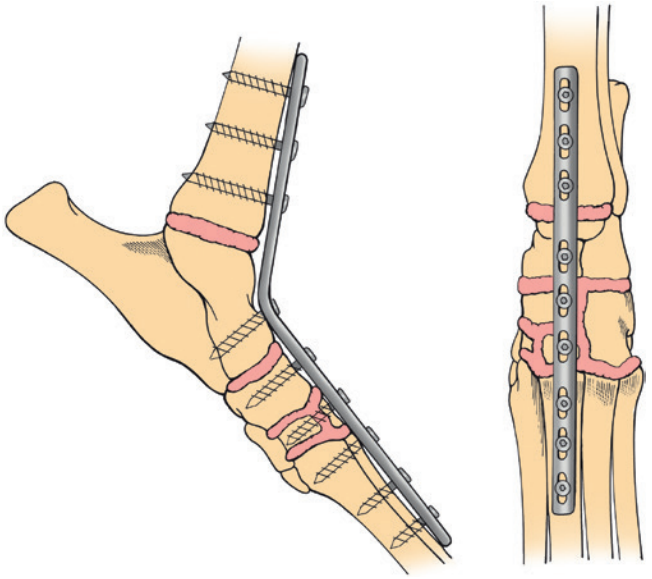


FIG. 34.156 Dorsal placement of a plate for tarsocrural arthrodesis. Because the plate is on the compression surface of the bone, it is subject to failure. If possible, a lengthening plate should be used.

plating requires a specific bone plate. Use of an external fixator for arthrodesis is recommended in cases with moderate to severe soft tissue injury for immediate stabilization. *Determine the normal standing angle of the opposite limb before surgery, and use it to approximate the fusion angle during arthrodesis of the injured limb. For stabilization with a cranial plate or external fixator, make an incision over the cranial surface of the joint. For stabilization with a medial or lateral plate, make an incision over the appropriate surface. Begin the incision over the distal one-third of the tibia, and extend it distally midway down the metatarsal bones. Enter the tarsocrural joint to expose the articular surfaces. With a power saw, remove the articular surface of the distal tibia by cutting perpendicular to the long axis of the bone. Cut the trochlea of the talus to achieve the appropriate fusion angle when the cut surfaces rest flush with each other. Alternatively, remove the cartilage of the tarsocrural joint with a pneumatic burr following the joint contours. Use the normal standing angle of the opposite limb to approximate the fusion angle. Maintain reduction of the cut surfaces with small K-pins, and pack a cancellous bone graft around the joint. Open the joint capsule of the proximal intertarsal and tarsometatarsal joints to expose the articular surfaces. Remove the cartilage with a pneumatic burr and insert a cancellous bone graft. Stabilize the joints with a bone plate applied to the cranial, medial, or lateral surface. Bend a cranial plate to conform to the established fusion angle, and secure it proximally by placing three screws in the distal tibia, two to four screws in the tarsal bones, and three screws in the third metatarsal bone. As an alternative, if a lengthening plate is used to bridge the tarsus, attach the plate to the distal tibia and metatarsus with three or four screws in each bone (Fig. 34.156). For medial or lateral plate application, position the plate so that the bend is centered over the talus and place screws in the talus, central/fourth tarsal bone, metatarsal bone, and tibia. Alternatively, place a type II external fixator by inserting two threaded full pins in the tibia and two full pins in the metatarsal bones, engaging two of the four metatarsal bones.* An additional pin may be inserted in the fourth/central tarsal bone or the talus/calcaneus.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Nonabsorbable suture or wire is generally used for ligament reconstruction. Braided orthopedic suture may be used in closed injuries but should be avoided in open injuries because of an increased risk of infection. Bone screws or bone anchors are used to secure prosthetic ligaments. A power drill, bone plate and screws, and a bone curette and air drill to remove articular cartilage are necessary for arthrodesis and cancellous bone harvest. Alternatively, external fixation pins, bars, and clamps may be used for arthrodesis. External fixation hinges are required for dynamization of the fixator.

POSTOPERATIVE CARE AND ASSESSMENT

After repair of tarsocrural luxation or subluxation, the tarsus should be placed in a normal standing angle and immobilized with rigid external coaptation or a transarticular external skeletal fixator for 3 to 6 weeks. During this time, activity should be limited to leash walking, and the coaptation bandage should be checked frequently. Activity should be restricted to specific physical rehabilitation exercises and leash walking for 6 weeks; then, once the splint or fixator is removed and the surgeon deems it safe, the animal should be gradually returned to unsupervised activity over a 6-week period. Dynamization of the external fixator by use of a hinge apparatus may aid in healing.

If a shearing injury is present, treat the wound as an open wound with daily changes of a sterile adherent dressing and liberal flushing with sterile solution or 0.05% chlorhexidine. Once healthy granulation tissue has formed, apply a nonadherent sterile dressing. If an external fixator is used, follow postoperative care guidelines. After arthrodesis, protect the repair with external coaptation for 6 weeks and restrict activity until radiographic evidence of bone union is seen.

COMPLICATIONS

Complications include infection and implant loosening. Implant removal may be indicated. Continued lameness should be expected in animals with subluxation or with luxation that is not adequately stabilized.

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PROGNOSIS

Adequate limb function can be expected in most patients with tarsal injuries after appropriate surgical intervention. Dogs with shearing injuries treated with prosthetic ligament or external fixation (or both) have limited range of motion and progressive DJD; however, 75% have good to excellent use of the limb.[81] After partial tarsal arthrodesis, most dogs resume normal activity. Implant removal may be indicated if lameness persists after bone healing.[82] Long-term outcome after tarsocrural arthrodesis provides good to excellent results in the majority of patients.[83]

OSTEOCHONDRITIS DISSECANS OF THE TARSUS

DEFINITION

OCD is a disturbance in endochondral ossification that leads to cartilage retention; it occurs in the hocks of immature large-breed dogs.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

OCD begins with a failure of endochondral ossification in either the physis or articular epiphyseal complex, which is responsible for the formation of epiphyseal bone. This condition also commonly occurs in the shoulder, elbow, and stifle. The pathogenesis of OCD is discussed on p. 1154. With OCD of the talus, a large piece of cartilage and subchondral bone is usually observed involving the medial (most frequently affected) or lateral trochlear ridge.[84] Histologically, the flap consists of cartilage; however, subchondral bone trabeculae in the defect are also thickened, and bone marrow fibrosis is noted.

DIAGNOSIS

Clinical Presentation

Signalment

Affected dogs are usually large; Rottweilers are most frequently affected. The average age of onset of lameness is 5 to 7 months, and the condition affects both males and females.

NOTE There is evidence of a hereditary component in the etiology based on the predilection of Rottweilers to develop this condition.

History

Rear limb lameness that worsens after exercise may be acute or chronic. Owners frequently report that the dog is stiff in the morning or after rest.

Physical Examination Findings

Lameness of one rear limb is usually evident. The dog may hyperextend the hocks and have a stiff or stilted gait because of bilateral lameness. Pain may be elicited on hock flexion. Palpation of the hocks should include an evaluation of range of motion. A decrease in the animal's ability to flex the hock indicates secondary DJD. Crepitation during hock flexion and extension, joint effusion, and periarticular swelling may be noted.

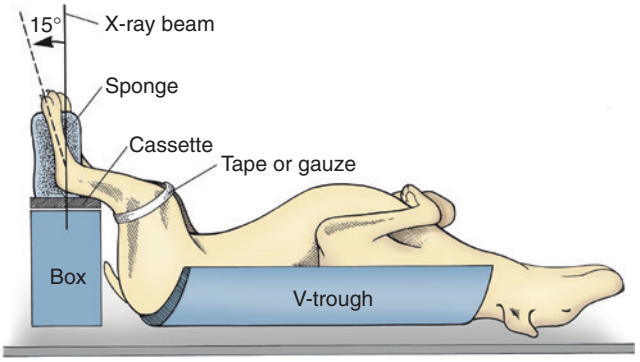


FIG. 34.157 Positioning for a craniocaudal view of the flexed tarsus. This view allows visualization of the lateral trochlear ridge of the talus without superimposition of the calcaneus.

Diagnostic Imaging

Radiographic views of the tarsus should include a standard lateral view of the hock, a flexed lateral view to expose the proximal portion of the talus, a craniocaudal view of the hock (made while the hock is extended to visualize the proximal portion of the trochlear ridges), and a craniocaudal view (made with the hock flexed to visualize the cranial portion of the trochlear ridges and the lateral condylar ridge without superimposition of the calcaneus) (Fig. 34.157). Radiographs of both tarsi should be obtained because bilateral disease is common. Definitive radiographic diagnosis of OCD is made when a radiolucent concavity is observed on the medial or lateral trochlear ridge on a standard craniocaudal (Fig. 34.158A) or a flexed craniocaudal (see Fig. 34.158B) view. Radiographic signs of secondary osteoarthritis are usually seen (see p. 1135). CT can be used to better evaluate the trochlear ridges if OCD is suspected but cannot be confirmed on survey radiography. CT is valuable because it allows evaluation of the trochlear ridges without superimposition of overlying structures.

NOTE Radiograph both tarsi because of the frequency of bilateral disease; however, unless the animal shows clinical signs, surgery of the opposite limb may not be necessary.

Arthroscopy

Arthroscopy provides definitive diagnosis of hock OCD. Arthroscopic approach varies with the location of the lesion (Figs. 34.159 and 34.160).

Laboratory Findings

Results of hematologic and serum biochemical analyses are normal in most affected animals. Results of arthrocentesis may include decreased synovial fluid viscosity, increased fluid volume, and an increased number of mononuclear phagocytic cells (up to 6000–9000 WBC/μL).

DIFFERENTIAL DIAGNOSIS

OCD must be differentiated from other diseases of young growing dogs that affect the rear limbs and may cause similar clinical signs of lameness (i.e., septic arthritis, hip dysplasia and panosteitis, partial cruciate ligament rupture). Decreased range of

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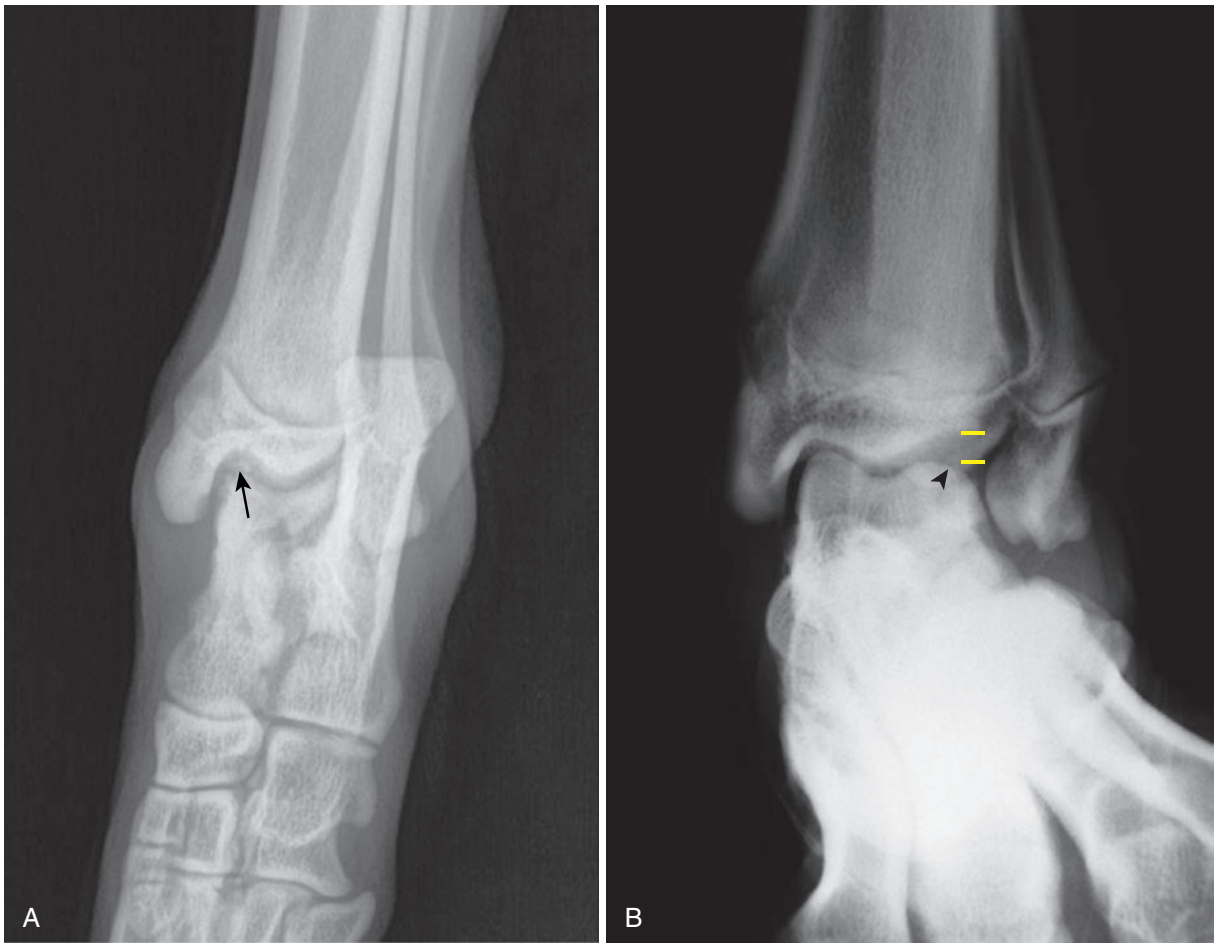


FIG. 34.158 Radiographs of tarsi with osteochondritis dissecans. (A) Standard craniocaudal view with a lesion on the medial trochlear ridge of the talus. A subchondral fissure is visible at the base of the medial trochlear ridge *(arrow)*. (B) Flexed dorsoplantar view with a lesion on the lateral trochlear ridge of the talus. There is a flattening of the lateral trochlear ridge, and the joint space is widened *(arrowhead)*.



FIG. 34.159 Instrumentation for arthroscopy of the hock.



FIG. 34.160 Arthroscopic view of a normal hock joint.

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motion and swelling in the hock usually identify hock disease as a possible cause of lameness.

MEDICAL MANAGEMENT

Medical therapy is generally used to treat older dogs with established osteoarthritis. Therapy should include each of the five principles of management of osteoarthritis. Lame animals are treated with exercise restriction and administration of NSAIDs (see Table 34.3). After the lameness has subsided, a gradual increase in exercise is indicated to strengthen surrounding musculature. Body weight control is critical to minimization of the clinical signs of osteoarthritis.

SURGICAL TREATMENT

Surgical removal of the cartilage flap allows the defect to heal by outgrowth of fibrocartilage from underlying subchondral bone. Surgery may be useful in young animals when the disease is diagnosed before the onset of osteoarthritis and in lame animals in which the condition is unresponsive to medical therapy. However, surgical management does not usually alter the progression of osteoarthritis.

Preoperative Management

Most affected animals are young and otherwise healthy; therefore only a minimal preoperative workup is necessary (i.e., CBC and serum chemistries). Preemptive pain management with NSAIDs (see Table 34.3), opiates, or epidural analgesia is indicated for dogs undergoing tarsus reconstruction.

Anesthesia

See Tables 32.1 and 32.2 for suggested protocols for the anesthetic management of patients with orthopedic disease. Opioid epidural administration reduces postoperative discomfort.

Surgical Anatomy

See Fig. 34.150 for surgical anatomy of the hock. Approaches to the lateral and medial trochlear ridge that do not involve osteotomy of the epicondyle or collateral ligament transection appear to result in less postoperative morbidity and a quicker return to function than do osteotomy approaches.

Positioning

The dog is positioned in dorsal recumbency with the affected limb suspended for draping. The limb is then released to allow access to the medial or lateral surface of the hock.

SURGICAL TECHNIQUE

Arthroscopy

AP Arthroscopy of the hock is more demanding than arthroscopy of other joints because of the small size and complex anatomy. The selection between cranial versus caudal portals and medial versus lateral portals depends on the region of interest. The arthroscope is usually positioned on the same cranial-caudal side as the lesion but on the opposite medial-lateral side as the lesion. If necessary, the arthroscope portals and instrument or egress portals may be switched during the procedure. A 1.9-mm arthroscope is most commonly used. *Insert a 20-gauge needle into the tibiotarsal joint, and infuse the joint with saline or lactated Ringer's solution. Using a No. 15 blade, make a partial thickness incision at the chosen arthroscope site on the same craniocaudal surface, but opposite the mediolateral surface to the lesion. Incise the skin, but do not penetrate the joint capsule. Insert the cannula for a 1.9-mm arthroscope with a blunt obturator into the joint. Be careful not to dislodge the cannula from the joint, as there is limited space between the capsule and the joint surface. Insert a 20-gauge needle mediolaterally opposite the arthroscope for egress (see Fig. 34.159). Explore the joint (see Fig. 34.160). If bone or cartilage fragments are identified, make a full-thickness incision adjacent to the needle to create an instrument portal. Enlarge the portal by inserting a straight Mosquito hemostat and opening the hemostat to dilate the portal. Insert a grasper and remove any fragments and use a curette or hand burr to debride the edge of the lesion and OCD bed until subchondral bleeding is observed. Close the joint routinely with simple interrupted skin sutures.*

Arthrotomy

The surgical approach selected depends on the location of the lesion. *Visualize lesions of the medial trochlear ridge using a dorsomedial and/or plantaromedial surgical approach. Visualize lesions of the lateral trochlear ridge using a dorsolateral and/or plantarolateral surgical approach. Regardless of the technique used for exposure, examine the appropriate trochlear ridge and remove the flap of cartilage. Perform curettage only to remove fragments of cartilage from the edges of the lesion. Repeatedly flush the joint to remove any small fragments before closure.*

Dorsomedial Approach to the Tarsus

Extend the hock and palpate the dorsal portion of the medial trochlear ridge. Make a skin incision starting proximal to the trochlear ridge and extending distally over the trochlear ridge. Incise the subcutaneous tissue along the same line. Identify the tendon of the tibialis cranialis muscle, the saphenous nerve, the cranial tibial artery and vein, and the dorsal branches of the saphenous artery and vein, and retract them laterally. Incise the deep fascia and joint capsule along the midline of the palpable portion of the medial trochlear ridge. Extend the incision proximally into the periosteum of the distal tibia (Fig. 34.161A). Visualize the cranial and distal part of the medial trochlear ridge. Extend the hock to increase the amount of visible trochlear ridge. Identify and remove the cartilage flap. Curette the edges of the lesion and flush the joint. Close the wound by suturing the fascia and joint capsule with absorbable suture in a simple interrupted pattern. Suture the subcutaneous tissue and skin in separate layers.

Plantaromedial Approach to the Tarsus

Flex the hock and palpate the proximal or plantar aspect of the medial trochlear ridge. Incise the skin and subcutaneous tissue caudal to the medial malleolus over the trochlear ridge. Identify the tendon of the long digital flexor muscle and the distal attachment of the caudal tibial tendon and retract them cranially. Identify the tendon of the flexor hallucis longus muscle, the tibial nerve, the plantar branches of the medial saphenous vein and saphenous artery, and the superficial plantar metatarsal vein, and retract them laterally. Incise the deep fascia and joint capsule longitudinally along the midline of the palpable portion of the medial trochlear ridge (see Fig. 34.161B). Identify and remove the cartilage flap. Flex the hock to increase the amount of medial trochlear ridge visible. Curette the edges of the lesion and flush the joint. Close the wound by suturing the fascia and joint capsule with absorbable suture in a simple interrupted pattern. Suture the subcutaneous tissue and skin in separate layers.

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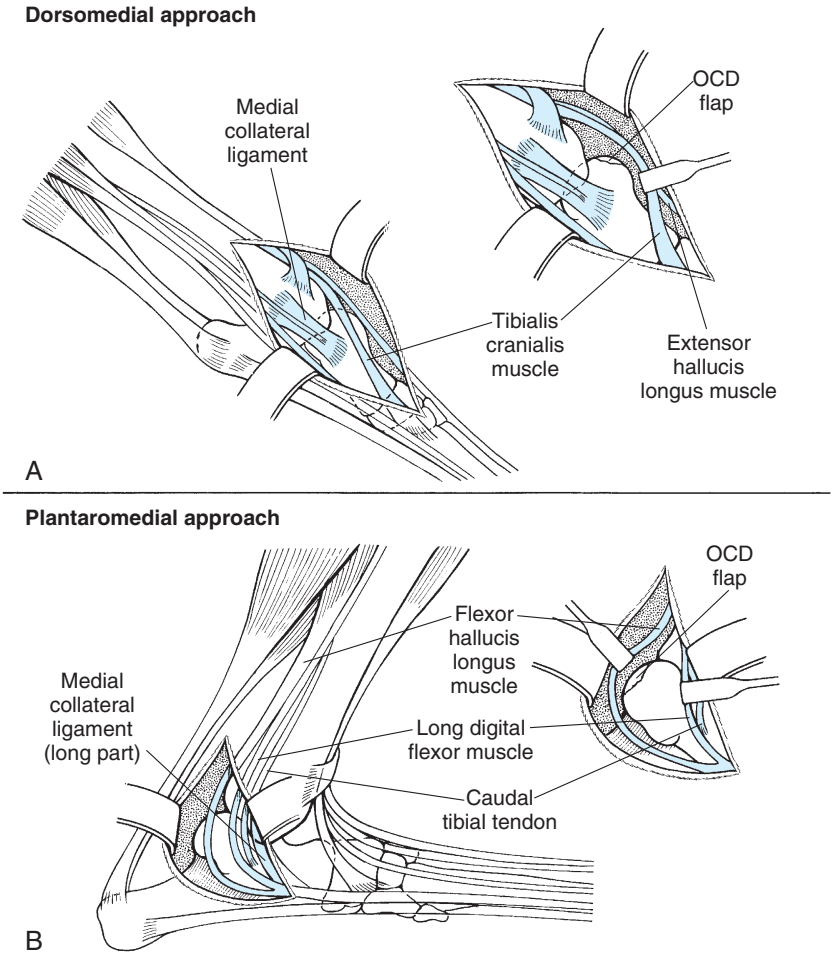


FIG. 34.161 (A) For a dorsomedial approach to the hock, make a skin incision starting proximal to the trochlear ridge and extending distally over it. Incise the subcutaneous tissue along the same line. Identify the tendon of the tibialis cranialis muscle, the saphenous nerve, the cranial tibial artery and vein, and the dorsal branches of the saphenous artery and vein, and retract them laterally. Incise the deep fascia and joint capsule along the midline of the palpable portion of the medial trochlear ridge. Extend the incision proximally into the periosteum of the distal tibia. (B) For a plantaromedial approach to the hock, incise the skin and subcutaneous tissue caudal to the medial malleolus over the trochlear ridge. Identify the tendon of the long digital flexor muscle and the distal attachment of the caudal tibial tendon and retract them cranially. Identify the tendon of the flexor hallucis longus muscle, the tibial nerve, the plantar branches of the medial saphenous vein and saphenous artery, and the superficial plantar metatarsal vein, and retract them laterally. Incise the deep fascia and joint capsule longitudinally along the midline of the palpable portion of the medial trochlear ridge. *OCD,* Osteochondritis dissecans.

Dorsolateral Approach to the Tarsus

Extend the hock and palpate the cranial portion of the lateral trochlear ridge. Make a skin incision starting proximal to the trochlear ridge and extending distally over the trochlear ridge. Incise the subcutaneous tissue along the same line. Identify the tendons of the LDE muscle, the cranial tibial muscle, the extensor hallucis longus muscle, and the dorsal branch of the lateral saphenous vein and the superficial peroneal nerve, and retract them medially. Identify the tendons of the peroneus longus, lateral digital extensor, and peroneus brevis muscles, and retract them in a plantar direction. Incise the deep fascia and joint capsule longitudinally along the midline of the palpable portion of the lateral trochlear ridge (Fig. 34.162A). Visualize the cranial and distal part of the lateral trochlear ridge. Extend the hock to increase the amount of trochlear ridge visible. Identify and remove the cartilage flap. Curette the edges of the lesion and flush the joint. Close the wound by suturing the fascia and joint capsule with absorbable suture in a simple interrupted pattern. Suture the subcutaneous tissue and skin in separate layers.

Plantarolateral Approach to the Tarsus

Flex the hock and palpate the proximal aspect of the lateral trochlear ridge. Incise the skin and subcutaneous tissue plantar to the lateral malleolus over the trochlear ridge. Retract the tendons of the peroneus brevis muscle, the lateral digital extensor muscle, and the peroneus longus muscle dorsally. It is difficult to retract the tendons of the peroneus brevis, lateral digital extensor, and peroneus longus muscles very far because they are firmly embedded in deep fascia

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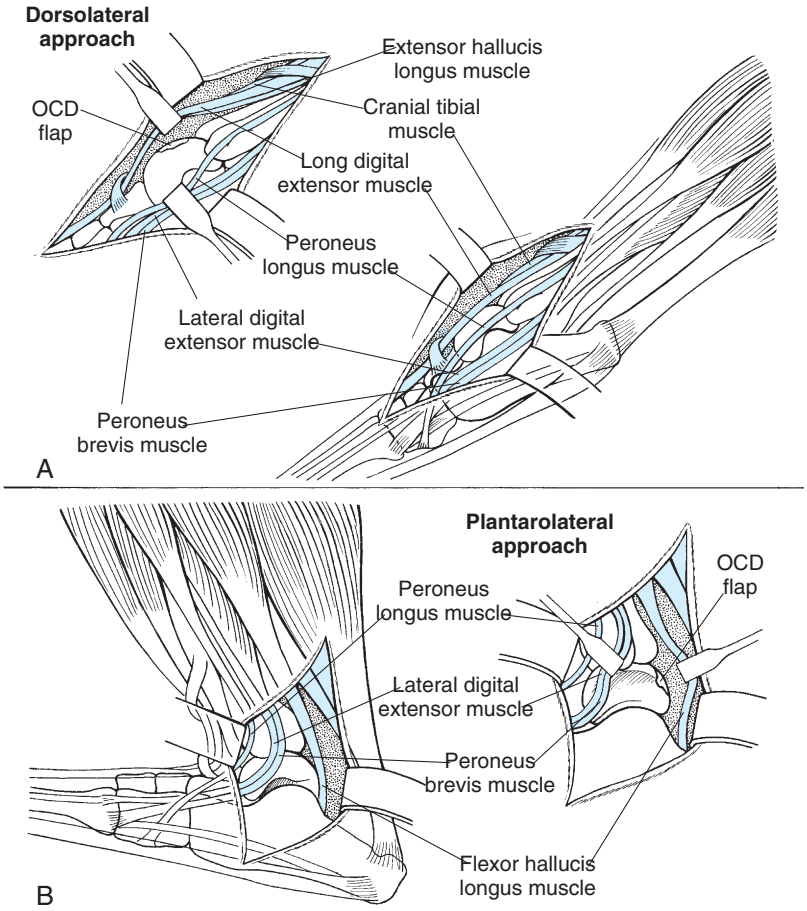


FIG. 34.162 (A) For a dorsolateral approach to the tarsus, make a skin incision starting proximal to the trochlear ridge and extending distally over it. Incise the subcutaneous tissue along the same line. Identify the tendons of the long digital extensor, cranial tibial, and extensor hallucis longus muscles; the dorsal branch of the lateral saphenous vein; and the superficial peroneal nerve; retract them medially. Identify the tendons of the peroneus longus, lateral digital extensor, and peroneus brevis muscles, and retract them in a plantar direction. Incise the deep fascia and joint capsule longitudinally along the midline of the palpable portion of the lateral trochlear ridge. (B) For a plantarolateral approach to the tarsus, make a skin incision starting proximal to the trochlear ridge and extending distally over the trochlear ridge. Incise the subcutaneous tissue along the same line. Identify and retract the tendons of the peroneus brevis, lateral digital extensor, and peroneus longus muscles dorsally. Retract the tendon of the flexor hallucis longus in a medial direction. Incise the deep fascia and joint capsule longitudinally along the midline of the palpable portion of the trochlear ridge.

over the lateral malleolus. *Retract the plantar branch of the lateral saphenous vein and a branch of the caudal cutaneous sural nerve in a plantar direction, and the tendon of the flexor hallucis longus in a medial direction. Incise the deep fascia and joint capsule longitudinally along the midline of the palpable portion of the lateral trochlear ridge (see Fig. 34.162B). Identify and remove the cartilage flap. Flex the hock to increase the amount of lateral trochlear ridge visible. Curette the edges of the lesion and flush the joint. Close the wound by suturing the fascia and joint capsule with absorbable suture in a simple interrupted pattern. Suture the subcutaneous tissue and skin in separate layers.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Ochsner forceps are helpful in grasping the cartilage fragment. A bone curette is needed to curette the edges of the lesion.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative cryo/compressive therapy aids in limiting swelling and pain. The limb should be bandaged after surgery for 3 to 5 days to provide soft tissue support. The animal should be confined to leash walks only for 4 to 6 weeks. The use of professional physical therapy can be very helpful in the management of hock OCD. Physical rehabilitation focuses on maintaining range of motion, strengthening the limb, and encouraging healing with healthy fibrocartilage. No or minimal weight-bearing activity is most effective.

PROGNOSIS

Dogs treated conservatively for OCD of the hock usually have continued intermittent lameness and progressive osteoarthritis.

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Those treated surgically may have improved limb function, but lameness may be evident after exercise.[84] Osteoarthritis is usually present even after surgery and requires medical treatment (see p. 1137); however, most dogs are functional pets and are lame only intermittently. In rare cases, infection of the surgical site may occur after surgery.

NOTE Advise owners that surgical treatment does not appear to alter the progression of osteoarthritis, and affected dogs may require medical therapy after surgery.

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35

Management of Muscle and Tendon Injury or Disease



GENERAL PRINCIPLES AND TECHNIQUE

MUSCLE-TENDON UNIT CONTUSION AND STRAINS

DEFINITIONS

A **contusion** is a bruise of the muscle with varying degrees of hemorrhage and fiber disruption. A **strain** is a longitudinal stretching or tearing of muscle fibers or groups of fibers. Muscle strains tend to occur near a musculotendinous junction and are categorized as grades 1 through 4. Grade 1 strains are disruptions of a limited number of muscle fibers; grade 4 strains are complete ruptures. Contusions and strains result in disruption of the normal architecture of the muscle-tendon unit secondary to interstitial edema, hemorrhage, or overstretching. **Compartment syndrome** is an increase in pressure of a compartmentalized muscle, which is usually due to trauma.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Muscle contusions are caused by external trauma. An external blow disrupts fibril continuity and the vascular compartment, with subsequent hemorrhage into the interstitial space. Muscle strains are caused by overstretching or overuse. These injuries occur but are often difficult to recognize in small animals.

An injured muscle may cause considerable pain during normal body motion. Muscle has an intrinsic ability to heal by regeneration of myofibrils if the sarcolemmal cells survive and the endomysial connective tissue sheath is not destroyed. With mild contusions and strains, cells and the endomysial sheath are not destroyed, and their preservation allows complete healing. However, if the contusion is severe and causes extensive cell death and hemorrhage precluding muscle regeneration, healing occurs with fibrous interposition between muscle ends. Excessive scarring may impede muscle fiber regeneration and interfere with muscle contraction.

Compartment syndrome of muscles occurs when pressure increases within a nonextensible fascial compartment, usually as the result of bleeding in the compartment following trauma. The increased pressure may cause irreversible damage to the muscle and nerves within the compartment. Compartment syndrome is rare in small animals.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, breed, or sex of dog or cat may be affected; however, muscle contusion and strain are diagnosed most commonly in athletic dogs (e.g., racing greyhounds and field trial dogs). They are rare in cats.

History

Contusion and strain injuries often occur during strenuous activity. The animal may be brought to a veterinarian because of a noticeable limp or complete inability to bear weight. The trauma is often unobserved by the client. With mild strains, owners may relate that the animal became reluctant to move 12 to 24 hours after strenuous exercise.

Physical Examination Findings

Clinical signs depend on the severity and chronicity of injury. With mild contusions, the animal may exhibit minimal lameness and the source of pain may be difficult to find on examination. With more severe contusions, pain and swelling are present. Most severe contusions occur in conjunction with fractures, and although the focus is on the fractured bone, injury to the muscle should be evaluated during surgery. Severe muscle strains are recognized by swelling and pain of the affected muscle unit (Fig. 35.1). Chronic muscle strains (e.g., bicipital tenosynovitis, iliopsoas muscle injury) occasionally occur in dogs.

Diagnostic Imaging

Standard craniocaudal and medial-to-lateral radiographs are necessary to rule out bone injury. Ultrasound examination of acute injuries may show no abnormalities or may show disruption of muscle fibers and hematoma formation. Because muscle injuries may be unilateral, comparison with the normal contralateral muscle may help in identification of an abnormality. In chronic stages of a muscle tear, normal muscle fibers may be reformed, or an echogenic scar may be present within the muscle belly. Magnetic resonance imaging (MRI) has been used to diagnose muscle strain in dogs.

Laboratory Findings

Consistent laboratory abnormalities are not found. Creatine kinase may be elevated and myoglobinuria may occur in cases of severe muscle trauma.

DIFFERENTIAL DIAGNOSIS

Muscle contusions and strains must be differentiated from joint sprains, fractures, polymyopathies, and polyarthropathies. Physical examination often differentiates muscle injury from joint sprain. Gentle palpation of muscle contusions causes pain and identifies swelling, whereas pain associated with sprain is elicited on manipulation of involved joints. Arthrocentesis (see p. 1135) helps differentiate joint sprain or arthropathy from muscle injury.

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ABSTRACT

Muscle and tendon injuries in small animals can be challenging to diagnose due to limitations in diagnostic imaging. A knowledge of the most common injuries aids in their identification and treatment.

KEYWORDS

Muscle
Tendon
Contracture
Strain
Tendonopathy
rupture

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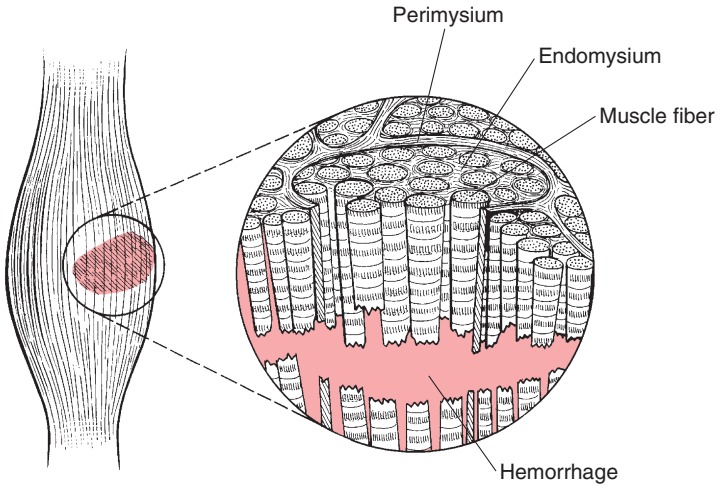


FIG. 35.1 Muscle fibril disruption and interstitial hemorrhage. Blood accumulates between the disrupted fibers.

MEDICAL MANAGEMENT

The primary treatment for muscle contusion and strain is rest. Enforced rest with controlled activity is necessary for at least 3 weeks. If injury is recurrent or severe, longer periods of enforced rest may be necessary. If the muscle is not allowed to heal adequately, repeated injury is likely. Nonsteroidal antiinflammatory drugs (see Table 34.3) can be administered for the first 3 to 4 days, but restricted activity must continue even if lameness and pain disappear.

Physical Rehabilitation

With acute injury (i.e., first 24 hours), cold compresses can be applied to the affected muscle for 15 minutes, three or four times a day. If the injury is more than 24 hours old, topical heat application is recommended. Use care to prevent burning the patient.

NOTE Warn owners that the animal may feel like exercising before it should be allowed, particularly if it has received nonsteroidal antiinflammatory drugs.

SURGICAL TREATMENT

When a severe contusion is recognized during surgical stabilization of a fracture, decompression of the muscle compartment can be achieved through incision of the epimysium (Fig. 35.2). Surgical treatment is necessary only if interstitial fluid accumulation causes sufficient pressure to compromise blood flow (i.e., compartment syndrome). Compartment syndrome has been diagnosed rarely in dogs and has not been reported in cats.

Preoperative Management

Surgical intervention should occur as soon as possible. The animal should be confined before surgery to prevent further damage to the muscle.

Anesthesia

Anesthetic considerations for animals with orthopedic disease are given on p. 976.

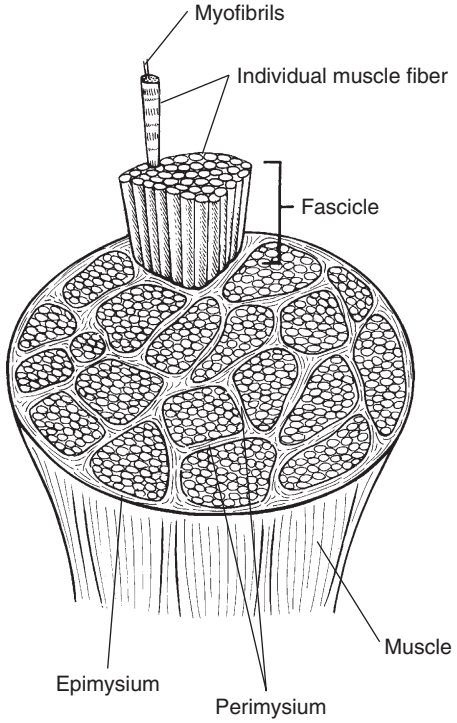


FIG. 35.2 Normal muscle anatomy. Note how the individual muscle fibrils combine to form muscle fibers. Groups of muscle fibers form fascicles, which are surrounded by the perimysium. Groups of fascicles form the muscle, which is covered by a thin fascial layer (epimysium).

Surgical Anatomy

Skeletal muscle is made of long, cylindrical fibers encased within connective tissue sheaths (see Fig. 35.2). Each individual fiber is enclosed within a sheath called the *endomysium*. Each fiber bundle is also enclosed within a sheath *(perimysium)*, as is the entire muscle *(epimysium)*. The connective tissue sheaths house blood vessels and nerve fibers that serve to integrate muscle contraction of individual fibers. Muscles are attached to bone by cordlike

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tendons or flat aponeuroses. The fascial compartment overlying the muscle group appears tight and congested with contusions, and the underlying muscle appears severely bruised and often protrudes from the incision through the fascial compartment.

Positioning

The animal should be positioned so that the affected muscle group can be exposed. A generous area should be clipped and prepared for aseptic surgery to allow the incision to be extended beyond the boundaries of the affected muscle if necessary.

SURGICAL TECHNIQUE

Make an incision through the skin and subcutaneous tissue overlying the muscle to be exposed. Once the muscle group is identified, make an incision through the fascia to decompress the muscle compartment. Suture subcutaneous tissue and skin using standard methods.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Self-retaining retractors are useful for retracting soft tissue from the area of interest.

POSTOPERATIVE CARE AND ASSESSMENT

The wound should be monitored for swelling and/or drainage for 7 to 10 days after surgery. Suture removal can be performed when the skin incision has healed.

PROGNOSIS

Return to normal function should be expected with most contusions and muscle strains; however, repeat injury is likely if adequate rest is not provided.

MUSCLE-TENDON UNIT LACERATION

DEFINITION

Lacerations are tears within the muscle-tendon unit.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Lacerations are usually due to penetration of the muscle-tendon unit by a sharp object. These injuries most commonly involve tendons near the carpometacarpal and tarsometatarsal joints but may involve muscle units elsewhere.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, breed, or sex of dog or cat may be affected.

History

The animal usually has an open wound and a non–weight-bearing lameness.

Physical Examination Findings

Non–weight-bearing lameness is typical after acute penetration of a muscle-tendon unit by a foreign object. The extent of internal damage can be assessed only by visualizing the wound. The wound should be explored after the patient has been stabilized, and this usually requires general anesthesia. Failure to explore the wound can result in lacerations remaining undiagnosed until the patient attempts to bear weight. The greater the time span from injury to repair, the more difficult it will be to appose severed tendon ends.

Dogs with chronic tendon lacerations have lameness exacerbated by exercise. Those with isolated deep digital flexor tendon lacerations have characteristic hyperextension of one digit.

Diagnostic Imaging

Standard craniocaudal and medial-to-lateral radiographs should be taken to determine whether foreign bodies or concurrent fractures are present. Mild swelling may occur in the area of chronic tendon lacerations. Ultrasonography helps localize the site of tendon injury and may differentiate partial from complete tendon rupture. Tendon injuries have a variable sonographic appearance depending on the severity of the trauma and the stage of healing. The normal ultrasound appearance of tendons depends on the incidence of the ultrasound beam. In a sagittal plane, the tendon or ligament will have a parallel, linear fiber alignment pattern that is hyperechoic. Damaged or torn tendons or ligaments will have disruption of the fiber alignment and edema, which appears as hypoechogenicity between fibers. In acute injury, the tendon is often enlarged because of the presence of edema, whereas with chronic injury, thickening of the tendon with focal areas of mineralization may be seen. A complete disruption may be identified by the presence of a hypoechoic band bordered by the retracted hyperechoic tendon ends.

Laboratory Findings

Consistent laboratory abnormalities are not present.

DIFFERENTIAL DIAGNOSIS

Muscle-tendon lacerations must be differentiated from superficial lacerations and muscle strain (see p. 1280).

MEDICAL MANAGEMENT

Lacerations that involve a tendon should be managed with surgery; medical management is not indicated. Minor lacerations of muscle may be treated conservatively.

Physical Rehabilitation

Physical rehabilitation may include cryotherapy and pulsed therapeutic ultrasound. Major lacerations of muscle should be treated with a combination of surgical repair and postoperative physical rehabilitation (see Chapter 11).

SURGICAL TREATMENT

Muscular lacerations require appositional sutures supported with deeper stent sutures. If the laceration goes through the tendon, delicate manipulation and apposition with small-diameter suture are recommended.

Preoperative Management

The wound should be cleaned and a sterile bandage applied until definitive treatment is performed. Exercise should be restricted before surgery.

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Anesthesia

Anesthetic management of animals with orthopedic disease is described on p. 976.

Surgical Anatomy

Tendons are longitudinally oriented bundles of collagen fibers that are surrounded by loose connective tissue sheaths. Blood vessels and nerves also reside within tendon sheaths. The entire tendon is surrounded by the *epitenon,* which is enveloped by an outer connective tissue sheath called the *paratenon*. Tendons crossing joint surfaces are often encased in a tendon sheath to facilitate movement during joint motion.

Positioning

The animal should be positioned so that the tendon and the muscle can be fully exposed. A generous area should be clipped and prepared for aseptic surgery to allow the incision to be extended above and below the injury if necessary.

SURGICAL TECHNIQUE

Muscle Laceration

Thoroughly debride the wound edges to fresh, bleeding muscle (Fig. 35.3). Debride carefully to prevent excess removal of tissue, which makes apposition of the severed ends difficult. Place interrupted sutures in the outer muscle sheath around the circumference of the muscle. Support the appositional sutures with heavy stent sutures placed in a cruciate pattern.

Tendon Laceration

Delicately manipulate and debride the tendon ends. With small, flat tendons, use small-diameter, nonabsorbable material placed in a series as interrupted vertical mattress or cruciate sutures. For larger tendons, select the largest suture diameter that will readily pass through the tendon atraumatically. A locking-loop suture pattern is recommended (Fig. 35.4). *Place each loop of the pattern in a slightly different plane (i.e., near-far, middle-middle, and far-near pattern). Alternatively, use a three-loop pulley or a Bunnell-Mayer, far-near, or near-far suture pattern (see p. 71). Use adjoining fascia to support the tendon appositional sutures.*

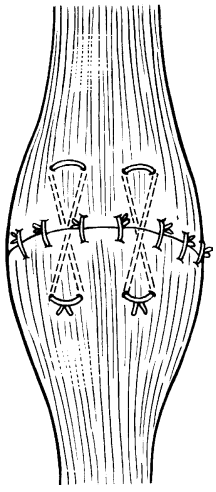


FIG. 35.3 Repair of muscle laceration with appositional sutures supported by tension stent sutures.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Nonabsorbable or absorbable suture material may be used to repair muscle, provided the material maintains its mechanical strength for 3 to 4 weeks. Nonabsorbable suture material is recommended for tendon repair. Swaged-on needles are helpful in limiting tissue trauma during suturing. Self-retaining retractors are useful for holding surrounding tissue away from the working area.

HEALING OF TENDONS AND MUSCLES

Healing of tendons follows a pattern similar to that of other connective tissue. The inflammatory phase is characterized initially by neutrophils and later by mononuclear cells. Tendon injury is rarely isolated but exists in a zone with other wounded tissue. Unsuccessful attempts to isolate tendon healing from surrounding tissue have led to the concept of one wound, one scar. This implies that successful healing is dependent on activation of undifferentiated mesenchymal cells, which migrate into the wound. Mesenchymal cells produce the collagen and matrix that lend strength to the entire wound. The resultant scar unites the tendon ends, which remodel with collagen fibers oriented parallel to lines of stress. Strength is regained through the one-wound, one-scar principle, but function is regained through active and passive postoperative use of the limb.

POSTOPERATIVE CARE

After tendon repair, the limb should be immobilized for 3 weeks with the use of rigid external coaptation, protective orthotics, or external fixation with the joint positioned to release stress on the repaired tendon (see p. 981). When the splint is removed, the limb should be semirigidly immobilized for an additional 3 weeks with a heavy padded bandage or a half cast (i.e., one side of a split cast applied cranially or caudally to the limb). If an orthotics or external fixator was used, it may be dynamized by the use of hinges or resistance bands. Hinges placed at the center of rotation of the joint can be adjusted to increase the range of motion and subsequently the tendon load. Elastic bands placed between the pins above and below the joint to replace the side bars allow partial loading of the tendon. After muscle repair, the limb should be immobilized for 5 days, followed by 4 to 6 weeks of protected activity. The use of platelet-rich plasma (PRP) is

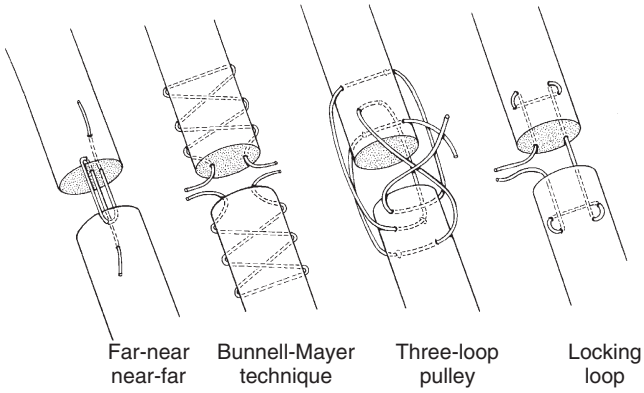


FIG. 35.4 Suture patterns used to appose tendon ends.

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under investigation as an aid in the healing of muscle and tendon in dogs (see Chapter 31).

Physical Rehabilitation

After immobilization for muscle or tendon lacerations, physical rehabilitation is vital to reverse the effects of immobilization on the other joints (see Chapter 11). The animal should be gradually returned to normal activity; premature weight bearing will result in failure of the tendon to heal.

PROGNOSIS

Return to normal function is expected if postoperative recommendations are followed. Failure is generally associated with allowing the animal to exercise before the tendon has completely healed.

MANAGEMENT OF SPECIFIC MUSCLE AND TENDON CONDITIONS

ACHILLES TENDON RUPTURE

DEFINITIONS

Rupture of the muscle-tendon unit is a complete or partial loss of integrity of the muscle-tendon unit caused by extreme overstretching. Muscle ruptures are caused by powerful contraction during forced hyperextension of the muscle-tendon unit.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Achilles tendon ruptures are seen most often in sporting and performance athlete breeds; however, any breed may be affected. The injury may be a partial or complete rupture of the Achilles tendon. Injury of the Achilles mechanism may arise from an acute traumatic episode or from chronic progressive stretching of the tendon. The former often occur secondary to a fall, or a penetrating wound or laceration secondary to automobile trauma, bite wounds, or from contact with a sharp object, such as the edge of a ski. Conversely, chronic injuries are often secondary to overuse that causes chronic stretching and deterioration of the tendon. Chronic injuries more commonly occur in sporting breeds (e.g., field trial dogs and bird hunting dogs) and are often bilateral. Bilateral chronic degeneration of the common calcanean tendons is relatively common in Doberman pinschers, although the cause is unknown.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, breed, or sex of dog or cat may be affected. Athletic dogs are most commonly affected.

As noted previously, Doberman pinschers may have idiopathic bilateral common calcanean tendon degeneration.

History

Affected animals usually exhibit weight-bearing lameness after strenuous activity.

Physical Examination Findings

Tarsal hyperflexion is often noted in animals with Achilles tendon rupture (Fig. 35.5A–B and Video 35.1). The animal is unable to bear weight if the injury occurs secondary to acute trauma, and flaccidity of the Achilles tendon is noted on passive dorsal flexion of the tarsus when the stifle is extended (Video 35.2). If the injury occurs secondary to chronic stretching of the Achilles tendon, the patient will be weight bearing but will walk plantigrade because of hyperflexion of the tarsus. Patients with chronic Achilles tendon injury show varying degrees of tarsal hyperflexion depending on the length of time the injury has been present. If the entire tendon complex is involved, the tarsus and the digits hyperflex; if the tendon of the superficial flexor muscle is not involved, the tarsus will hyperflex and the digits will flex (see Fig. 35.5B). Postural changes associated with palpable swelling of the Achilles tendon confirm the diagnosis. Occasionally, the injury occurs at the myotendinous juncture. Postural changes and careful palpation of the muscle-tendon unit confirm the diagnosis. Doberman pinschers have tarsal hyperextension and swelling of the common calcanean tendon.



FIG. 35.5 (A) Tarsal hyperflexion (plantigrade stance) due to Achilles tendon rupture. (B) Tarsal hyperflexion with digital flexion due to Achilles tendon rupture with intact superficial digital flexor.

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Diagnostic Imaging

Ultrasonography can help determine the location of tendon fiber disruption and may differentiate between partial and complete tears. Ultrasound may also be useful in evaluating healing after surgery. Standard craniocaudal and medial-to-lateral radiographs are indicated to determine whether bone avulsion is present or not.

Laboratory Findings

Consistent laboratory abnormalities are not found.

DIFFERENTIAL DIAGNOSIS

Sciatic nerve injury, tarsal collapse secondary to immune-mediated arthropathy, endocrinopathy, and congenital tarsal hyperflexion must be differentiated from Achilles tendon rupture. Gentle palpation of the muscle-tendon unit reveals loss of continuity and/or an area of swelling with tendon rupture, whereas patients with congenital tarsal hyperflexion lack these findings. When nerve injury is present, neurologic examination will show sciatic reflexes to be absent or decreased.

MEDICAL MANAGEMENT

Surgical repair of completely ruptured tendons is indicated; medical management is not. External coaptation may be tried with partial rupture, but results are usually unsatisfactory. Doberman pinschers with common calcanean tendon degeneration should be treated conservatively with exercise moderation and bandaging or orthotics as necessary to protect the tarsus. Splints are rarely beneficial. Physical rehabilitation may accelerate tendon healing (see Chapter 11).

SURGICAL TREATMENT

Surgical repair involves primary anastomosis of the tendon (tenorrhaphy) or reinsertion of the tendon on the calcaneus. Augmentation of a primary repair using a variety of materials (such as semitendinosus muscle and composite suture) has been described.[1]

Preoperative Management

Activity should be limited until definitive treatment is begun. Open wounds (e.g., laceration of the Achilles tendon) should be cleaned and covered with a sterile dressing.

Anesthesia

Anesthetic management of animals with orthopedic disease is discussed on p. 976.

Surgical Anatomy

The Achilles tendon unit is composed of tendons arising from the gastrocnemius and superficial digital flexor muscles along with a common tendon from the semitendinosus muscle, the gracilis muscle, and the biceps femoris muscle. The common tendon of the gastrocnemius muscle is the major component of the Achilles tendon with acute injury. One can identify each component of the acutely injured Achilles complex and can suture each individually. However, with chronic injury, the tendon ends retract, leaving a void to be filled with fibrous tissue. Identification of each component is not possible. The complex (fibrous scar and Achilles tendon) is treated as a single structure.

Positioning

Position the patient in sternal or lateral recumbency. The limb may be extended beyond the end of the table for sterile preparation and surgery. For Achilles tendon injury, clip and prepare the limb for surgery from proximal to the stifle joint to the phalanges distally.

SURGICAL TECHNIQUE

Achilles Tendon Rupture

Make an incision over the site of injury on the caudolateral surface of the limb. If the injury is acute, identify the three tendons composing the Achilles complex, and suture each tendon separately with an interrupted far-near, near-far pattern (see Fig. 35.4) using nonabsorbable, small-diameter (3-0 to 4-0, depending on the size of the animal) monofilament suture. If the injury is chronic and identification of individual tendon units is not possible; continue surgical dissection to expose the circumference of the thickened fibrous band. Then sequentially remove sections of scar tissue from the center of the mass. Remove enough tissue so that tension is present in the Achilles complex when the stifle joint is in a normal standing position and the tarsus is slightly extended. Be careful to avoid removing too much of the proliferative fibrous tissue. If excess fibrous tissue is excised, apposition of the cut ends will be difficult. *Suture the cut ends with a three-loop pulley pattern (see Fig. 35.4).*

Achilles Tendon Avulsion

Achilles tendon avulsion from the calcaneus is treated by suturing the tendon as described earlier and securing it to the calcaneus by passing the suture through holes drilled through the proximal calcaneus.

Whether the injury is acute or chronic and involves rupture or avulsion of the tendon, support of the tendon anastomosis is critical for a favorable outcome. Support is provided by immobilization of the tarsal joint in slight extension. *Place a raised, threaded transfixation pin through the free end of the calcaneus into the distal tibia. Cut through the pin shaft to leave the pin just below the skin surface. Provide additional support with a fiberglass cast. Alternatively, immobilize the hock in slight extension with external fixators by placing transfixation pins through the distal tibial diaphysis and the calcaneus, connecting them with bilateral external fixator bars.*

FiberLoop Repair

Achilles tendon avulsions or low lacerations may be treated by anastomosis of the distal tendon to the calcaneus with a fiberwire loop pattern to a calcaneal bone tunnel and surgical button. *Make an incision over the site of injury on the caudal surface of the limb. Identify the tendons composing the Achilles complex. Pass a stay suture through the distal aspect of the Achilles tendon. Using a FiberLoop (Arthrex), perform 4 or 5 nonlocking loops (baseball stitch or SpeedWhip [Arthrex]) on the distal aspect of the Achilles tendon. (Fig. 35.-6A) Drill a distal to proximal bone tunnel through the caudal-proximal aspect of the calcaneus. Pass the FiberLoop needle from proximal to distal through the hole and secure the Fiberwire on the distal aspect by suturing over a surgical button (Fig. 35.6B–C). Have the hock joint in full extension when tying the suture over the button. If the injury was a distal laceration, suture the proximal and distal aspects of the Achilles tendon with nonabsorbable suture in an interrupted pattern. Close the surgical wound routinely and place the limb in a bivalved cast with the hock joint in extension. Maintain the cast for 4 weeks then decrease*

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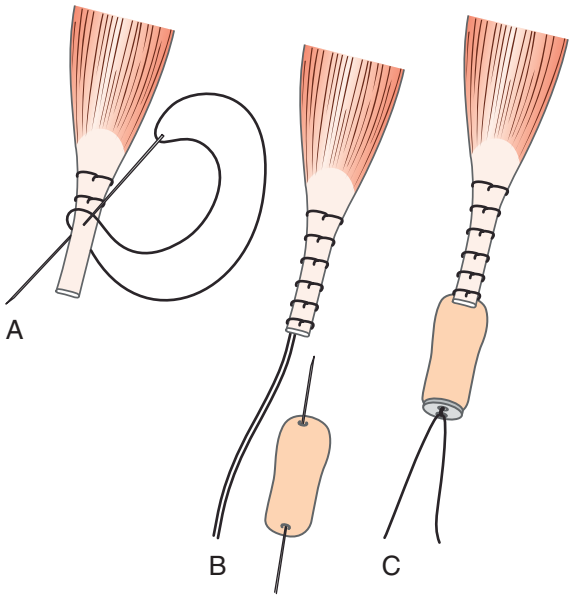


FIG. 35.6 Loop suture technique for repair of Achilles tendon trauma. (A) Secure the loop to the distal aspect of the tendon using the locking loop pattern. (B) Pass the straight needle through a hole in the proximal aspect of the calcaneus. (C) Secure the loop suture on the distal aspect of the calcaneus with a stainless-steel button.



FIG. 35.7 Protective orthotics, initially to limit tarsal flexion and gradually, to allow adjustment to increase range of motion.

to a half cast/splint for 2 weeks. Finally, place the limb in a soft padded bandage for two more weeks. PRP may be injected at the time of surgery to potentially accentuate healing (see Chapter 31). The use of a SpeedWhip technique has been compared to a three-loop pulley for repair of an Achilles tendon rupture in a canine cadaver model.[1] In this study, the three-loop pulley technique provided greater resistance to 3-mm gap formation and greater load to failure. These results are in contradiction to previous studies that showed superior biomechanical properties of SpeedWhip fixation in comparison to alternative fixation methods.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Nonabsorbable suture material is recommended for tendon repair. Swaged-on needles are helpful in limiting tissue trauma during suturing. Self-retaining retractors are useful for holding surrounding tissue away from the working area. A power drill is useful if a bone screw or transfixation pins are to be inserted. The FiberLoop technique requires a swaged-on loop suture or suture passer.

POSTOPERATIVE CARE

The cast and transfixation pin, or external fixators are placed for 3 to 6 weeks, after which both may be removed. The limb should then be supported in a padded bandage to prevent full dorsal flexion of the tarsus. If an external fixator was used, it may be dynamized by the use of hinges or resistance bands. Hinges placed at the center of rotation of the joint can be adjusted to increase the range of motion and subsequently the tendon load. Elastic bands placed between the pins above and below the joint to replace the sidebars allow partial loading of the tendon. Alternatively, protective orthotics can be applied to initially limit tarsal extension for 2 to 3 weeks and then gradually to allow adjustment of increased range of motion over 2 to 10 weeks (Fig. 35.7). Activity should be limited to leash walking for 10 weeks. The use of PRP is under investigation as an aid in the healing of muscle and tendon in dogs (see Chapter 31).

Physical Rehabilitation

After immobilization for muscle or tendon rupture, physical rehabilitation is vital to reverse the effects of immobilization on the joints (see Chapter 11). The animal should be gradually returned to normal activity; premature weight bearing will result in failure of the tendon to heal.

PROGNOSIS

The prognosis for return to strenuous athletic activity is good to excellent. Doberman pinschers with degeneration of the common calcanean tendon often regain some degree of tendon function spontaneously over time. The tendons often remain swollen, but the degree of hock flexion and disability typically improves over several months of conservative therapy.

SUPRASPINATUS TENDINOPATHY

DEFINITION

Supraspinatus tendinopathy refers to mineralized or nonmineralized disease of the insertional tendon of the supraspinatus complex. Mineralization is the accumulation of calcified material within the insertional tendon of the supraspinatus. **Biceps tendon disease** (tendinopathy, tendinitis) is a general term that incorporates various types of injury, including tearing of the tendon and inflammation of the tendon and surrounding synovial sheath.

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GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The cause of mineralization of the supraspinatus tendon is unknown, but it may be associated with aging, overuse, trauma, or hypoxia secondary to hypovascularity of the supraspinatus tendon. Histopathology demonstrates myxomatous degeneration and chondroid metaplasia, which are characteristics of tendon degeneration. Mineralization may also be associated with tearing of tendon fibers. Severe disease may be associated with a mass of mineral and fibrous tissue compressing or deviating the biceps tendon in the bicipital groove. Disease of the biceps and the supraspinatus may occur together, and thickening of the supraspinatus may displace the biceps tendon, resulting in inflammation and pain.[2] In the most severe cases, other diseases of the shoulder (e.g., instability, osteoarthritis) may also be identified; thus shoulder arthroscopy should be performed in dogs with suspected supraspinatus tendinopathy. Radiographic mineralization is a common incidental finding and is not associated with clinical lameness; therefore accurate determination of the cause of the lameness is critical.

DIAGNOSIS

Clinical Presentation

Signalment

Athletic large breed dogs are affected most commonly.

History

Lameness may be mild or severe depending on the severity of the lesion. Most animals have a chronic history of lameness.

Physical Examination Findings

Palpation on the medial aspect of the greater tubercle may cause pain. This response may be magnified by shoulder flexion.

Diagnostic Imaging

Ultrasonography helps evaluate stabilizers of the scapulohumeral joint (i.e., biceps and supraspinatus tendons). In cases of injury to the supraspinatus tendon, ultrasound helps determine the degree of tendon fiber disruption. Mineralization of the tendon appears as hyperechoic foci, which cause distal acoustic shadowing. The most common ultrasound finding in dogs with supraspinatus tendinopathy is enlargement of the tendon of insertion. With radiography, mineralization within the supraspinatus tendon may be seen adjacent to the greater tubercle using a skyline view (Fig. 35.8). Osteophytosis of the intertubercular groove may be apparent and may be due to irritation of the biceps tendon and tendon sheath caused by the enlarged supraspinatus tendon, leading to secondary arthritis. MRI demonstrates enlargement and increased signal in the area of insertion of the supraspinatus tendon.[2]

Laboratory Findings

Consistent laboratory abnormalities are not found.

DIFFERENTIAL DIAGNOSIS

Mineralization of the supraspinatus tendon must be differentiated from other shoulder diseases, including biceps tendinopathies. Differential diagnoses include biceps tenosynovitis, osteochondritis dissecans, shoulder instability, and shoulder osteoarthritis.

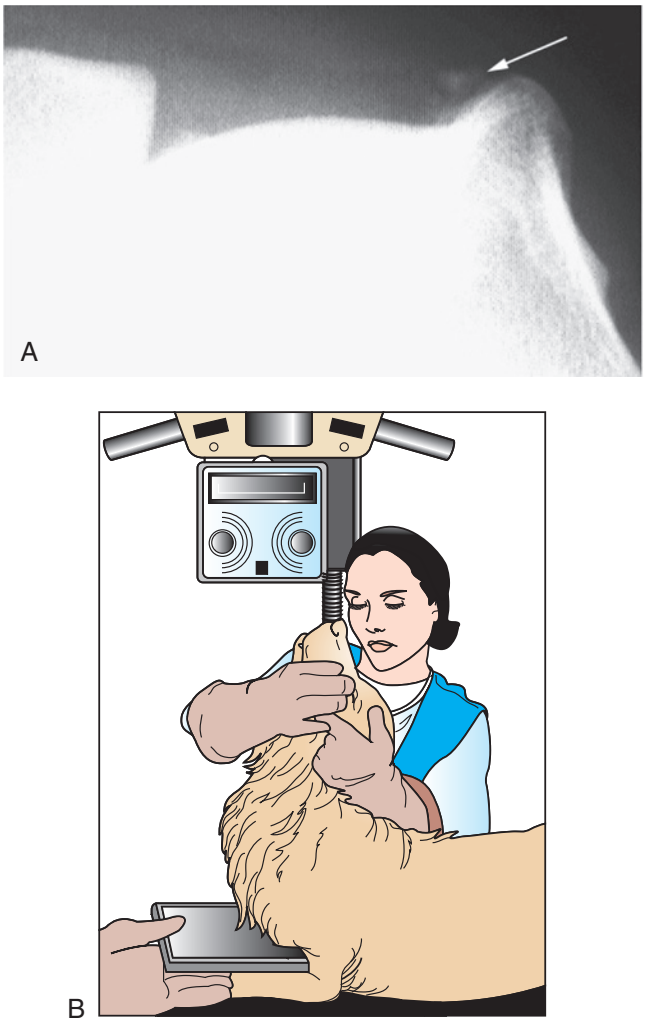


FIG. 35.8 (A) Mineralization of the supraspinatus tendon *(arrow)*. (B) Radiographic positioning is attained with the patient in sternal recumbency and the head extended out of the beam. The cassette is placed above the antebrachium with the humerus nearly vertical. The radiographic beam courses down the bicipital groove.

MEDICAL MANAGEMENT

Medical management of supraspinatus mineralization is often attempted first. Glucocorticoid injections combined with rest are often attempted and may provide temporary relief. Injection of glucocorticoids into the tendon may cause tendon degeneration; therefore multiple injections are not recommended. The use of PRP in the treatment of supraspinatus tendinopathy is currently being investigated (see Chapter 31).

Physical Rehabilitation

Physical rehabilitation for mineralization includes cryotherapy and passive range of motion. Therapeutic ultrasound may aid in eliminating mineral deposits in the muscle (see Chapter 11).

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SURGICAL TREATMENT

Surgery is recommended for confirmed mineralization of the supraspinatus tendon that is unresponsive to medical management. Complete evaluation of the shoulder joint is needed to rule out other associated diseases. Arthroscopy of the shoulder joint is the most efficient means of thoroughly evaluating the joint including the biceps tendon. Surgical treatment involves removal of the mineral from the tendon of insertion of the supraspinatus, resection of diseased tendon, and multiple longitudinal tenotomy. Longitudinal tenotomy may decrease intratendinous pressure, increase vascularization of the critical zone, and induce healing, thus improving or reversing degeneration.

Preoperative Management

Activity should be limited until definitive treatment is initiated.

Anesthesia

Anesthetic management of animals with orthopedic disease is discussed on p. 976.

Surgical Anatomy

The supraspinatus tendon has a broad insertion on the medial and lateral aspects of the greater tubercle. The medial portions are adjacent to the biceps tendon.

Positioning

Position the patient in lateral recumbency. A hanging-leg preparation facilitates manipulation of the limb during surgery. Clip and prepare the limb for surgery from the dorsal midline to the mid-antebrachium.

SURGICAL TECHNIQUE

Perform shoulder arthroscopy to evaluate the remainder of the joint. Perform a craniomedial approach to the shoulder joint and biceps tendon (see Fig. 34.23). Identify and remove the mineralized area or diseased area within the insertional tendon of the supraspinatus. To perform a longitudinal tenotomy, make multiple full-thickness longitudinal incisions in the tendon of insertion with a surgical blade. Close the surgical site routinely.

SURGICAL MATERIALS AND SPECIAL INSTRUMENTS

Gelpi retractors are useful for maintaining exposure to the craniomedial portion of the shoulder joint.

POSTOPERATIVE CARE

Activity should be limited to leash walking for 8 to 10 weeks.

Physical Rehabilitation

Physical rehabilitation can be started immediately after surgery and should include cryotherapy for the first 72 hours, followed by passive flexion and extension of the limb and heat therapy (see Chapter 11). Specific controlled exercises should be used to regain maximum function.

PROGNOSIS

The prognosis for return to normal function is fair to good, depending on the presence of other shoulder diseases.

INFRASPINATUS FIBROTIC CONTRACTURE

DEFINITIONS

Muscle contracture or **fibrosis** may occur when normal muscle-tendon unit architecture is replaced with fibrous tissue, resulting in functional shortening of the muscle or tendon. This shortening may cause abnormal motion in adjacent joints.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Infraspinatus muscle contracture occurs most often in hunting dogs after irreversible muscle fiber injury. The cause is unknown but appears to be a primary muscle disorder rather than a neurologic or immune-mediated disorder. Histologic findings (e.g., degeneration, atrophy, fibroplasia) within damaged areas of the muscle are compatible with severe strain. Disease progression is associated with breakdown of muscle fibers and replacement by fibrous tissue that causes muscle contracture and external rotation and abduction of the limb.

DIAGNOSIS

Clinical Presentation

Signalment

Contracture of the infraspinatus muscle usually occurs in young, adult, sporting breeds.

History

Acute lameness after strenuous activity in the 3 weeks before evaluation for infraspinatus muscle contracture is typical.

Physical Examination Findings

Animals with infraspinatus muscle contracture initially have weight-bearing unilateral forelimb lameness (Video 35.3).[3] Soft tissue swelling around the shoulder joint may be noted. Lameness usually resolves and is absent for 3 to 4 weeks, but then mild lameness and a gait abnormality develop. The characteristic gait abnormality occurs secondary to progressive fibrosis and contracture of the infraspinatus muscle. As the muscle shortens from contracture, external rotation of the shoulder causes elbow abduction and outward rotation of the paw (Fig. 35.9).

Diagnostic Imaging

Ultrasonography helps evaluate stabilizers of the scapulohumeral joint. The normal infraspinatus tendon should show a typical arrangement of parallel, echogenic striae against a hypoechoic background on ultrasound. Acute injury may demonstrate fiber pattern disruption with edema and hemorrhage. Chronic muscle contracture varies in appearance depending on the severity and the stage of the disease. Fibrosis of the muscle is visible as increased echogenicity of the tendon. MRI of infraspinatus contracture demonstrates heterogeneous signal intensity within the muscle and at the musculotendinous junction, with minimal extension into the tendon.[3]

DIFFERENTIAL DIAGNOSIS

Diagnosis of infraspinatus muscle contracture is usually possible from historical and physical examination findings. In the acute phase of injury, inflammation of the infraspinatus tendon must

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FIG. 35.9 Dog with infraspinatus muscle contracture. Note external rotation of the shoulder and internal displacement of the elbow.

be differentiated from other causes of forelimb lameness, particularly those of the shoulder region.

MEDICAL MANAGEMENT

Physical rehabilitation may help prevent contracture if the condition is diagnosed and treated early; however, presentation to a veterinarian at this stage is uncommon, and surgical therapy at the contracted stage is highly successful. Thus surgical treatment is most commonly performed in affected dogs.

Physical Rehabilitation

Physical rehabilitation entails ultrasound therapy combined with stretching and passive range of motion (see Chapter 11).

SURGICAL TREATMENT

Surgical treatment is directed at releasing the fibrotic myotendinous infraspinatus muscle as it crosses the shoulder joint. Surgical treatment (fascial decompression) at the reported early stage (compartment syndrome) has been described in one dog.

Preoperative Management

These animals are otherwise generally healthy, and minimal preoperative care is necessary.

Anesthesia

See p. 976 for anesthetic management of animals with orthopedic disease.

Surgical Anatomy

The infraspinatus muscle-tendon unit is a cuff muscle of the shoulder joint (see Fig. 34.19). The muscle lies next to the scapula, just caudal to the spine. The tendon crosses the joint craniolaterally to insert just distal to the greater tubercle of the proximal humerus. At surgery, the tendon appears thickened and, because of fibrous tissue invasion, has an off-white color rather than the white, glistening appearance typical of normal tendons. The muscle is atrophied and appears pale relative to normal, healthy muscle.

Positioning

Clip the shoulder from the dorsal midline to the elbow, and position the patient in lateral recumbency.

SURGICAL TECHNIQUE

Perform a craniolateral approach to the shoulder joint. Isolate the circumference of the infraspinatus muscle with sharp dissection (see Fig. 34.19). Transect the fibrotic muscle and any fibrous bands restricting movement of the joint. Once the fibrous contracture is incised, the limb will assume a normal position, and a normal range of motion of the shoulder will be possible.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Self-retaining retractors and a periosteal elevator assist tissue retraction and reflection.

POSTOPERATIVE CARE

Special postoperative care is not required after surgery for infraspinatus muscle contracture. These animals usually begin to use the limb within a few days after surgery. The dog should be allowed to fully bear weight as soon as possible. Physical rehabilitation is important to help the animal regain full usage of the limb (see Chapter 11).

PROGNOSIS

The prognosis for infraspinatus muscle contracture is excellent, and return to preinjury function is expected after surgery.

QUADRICEPS CONTRACTURE

DEFINITIONS

Muscle contracture or **fibrosis** *(quadriceps tie-down, fracture disease of the rear limb)* may occur when normal muscle-tendon unit architecture is replaced with fibrous tissue, resulting in functional shortening of the muscle or tendon. This shortening may cause abnormal motion in adjacent joints. **Myopathy** is another term used to describe muscle disease.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Quadriceps muscle contracture usually occurs after distal femoral fracture in young dogs; however, congenital contracture of the quadriceps muscle has been reported. Inadequate fracture stabilization, excessive tissue trauma during surgery, or prolonged limb immobilization with the stifle in extension may singly or in combination contribute to quadriceps contracture. It is often associated with Salter-Harris type I or II fracture in puppies (see p. 984). Quadriceps contracture may occur after splinting in extension for as little as 5 to 7 days. The disease is likely a result of the combination of muscle trauma, rapid bone callus formation, and limb immobilization. Joint stiffness develops because of

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fibrous adhesions between the quadriceps and the fracture callus. With time, adhesions form between the joint capsule and the distal femur, limiting limb use and causing the quadriceps muscle to atrophy. In later stages, the disease also causes bone atrophy, atrophy of cartilage in the stifle, intraarticular fibrosis, and eventual ankylosis of the stifle joint. The cause of congenital quadriceps contracture and the reason why contracture occurs most commonly in young dogs are unknown.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, breed, or sex of dog may develop quadriceps muscle contracture; however, it most commonly occurs in immature patients after distal femoral fracture when inappropriate repair has been done and/or postoperative physical rehabilitation has been inadequate.

History

Animals with quadriceps muscle contracture are usually evaluated for lameness 3 to 5 weeks after sustaining femoral trauma. Frequently, internal reduction and stabilization of a distal femoral fracture or application of an external splint (such as a Thomas splint) for stabilization of a femoral fracture has been performed.

Physical Examination Findings

The stifle joint of animals with quadriceps muscle contracture has a limited range of motion (Fig. 35.10 and Video 35.4). Initially the joint can be fully extended, but it can be flexed only 20 to 30 degrees. Gradually the amount of flexion decreases to less than 10 degrees. Contracture may be such that the stifle joint appears hyperextended. Cranial thigh muscles are generally atrophied and palpated as a thickened cord.

Diagnostic Imaging

Radiography shows evidence of a healing distal femoral fracture. Ultrasound of the normal quadriceps muscle should show a typical echogenic, fibrillar arrangement. Changes in this arrangement vary depending on the type, severity, and stage of trauma.



FIG. 35.10 Dog with quadriceps muscle contracture. Note hyperextension of the femur.

Laboratory Findings

Consistent laboratory abnormalities are not present.

DIFFERENTIAL DIAGNOSIS

The diagnosis of quadriceps contracture can usually be made from historical and physical examination findings.

MEDICAL MANAGEMENT

Prevention of quadriceps contracture should begin within 24 hours of surgery for distal femoral fracture in puppies (see Chapter 11). Therapy should include passive range of motion several times a day, cryotherapy, and massage for edema reduction. Tissue mobilization techniques help prevent fibrosis. Exercises to encourage weight bearing should begin approximately 3 days postoperatively and may include slow leash walking, treadmill walking, and aquatic therapy (see Table 32.7).

Physical Rehabilitation

Physical rehabilitation alone is rarely successful in the management of quadriceps contracture. Forced flexion of the stifle often results in additional fractures to the femur or tibia (see Table 32.9).

SURGICAL TREATMENT

Treatment of quadriceps contracture is aimed at restoring limb function. Release of fibrous thickening and adhesions between joint capsule and femur and between quadriceps muscle and femur is necessary. If a functional range of motion is not achieved after adhesion release, lengthening of the quadriceps muscle-tendon unit is required. Lengthening may be accomplished by a Z-plasty or by release of the origin of each muscle. Recurrence of contracture with resultant loss of stifle joint motion occurs if preventive rehabilitation measures are not taken after surgery. An effective method of maintaining a functional range of stifle motion is to apply a transarticular fixator postoperatively to maintain passive flexion of the stifle joint, while allowing active or passive extension.[4] Alternatively, if sufficient flexion is obtained at surgery, a 90/90 bandage can be used.

Preoperative Management

These animals are otherwise generally healthy, and minimal preoperative care is necessary.

Anesthesia

See p. 976 for anesthetic management of animals with orthopedic disease.

Surgical Anatomy

The quadriceps muscle-tendon unit is composed of the vastus medialis, vastus intermedius, rectus femoris, and vastus lateralis muscles. These muscles all atrophy in patients with quadriceps muscle contracture. Proximally, the rectus femoris muscle palpates as a thickened cord. At surgery, all four muscles of the quadriceps appear small and pale in color. In patients with distal femoral fractures, the atrophied muscles lie superficial to, and are intertwined with, proliferative fibrous tissue. Normal range of motion in the stifle joint is from 180 degrees (full extension) to 30 degrees (full flexion).

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Positioning

Clip the limb from the dorsal midline to the tarsus, and position the patient in lateral recumbency. A hanging-leg preparation facilitates manipulation of the limb during surgery.

SURGICAL TECHNIQUE

Expose the stifle joint and the distal femur through a liberal craniolateral incision (see Fig. 34.113). Elevate and release adhesions between the quadriceps muscle group and the femur with sharp dissection. Release adhesions between the fibrous joint capsule and the femoral condyles. Luxate the patella medially, and flex the joint to its full extent. If a functional range of motion (greater than 40 degrees) is not achieved after the adhesions are released, perform a quadriceps muscle-tendon unit lengthening procedure.

Z-Plasty

Make a longitudinal incision through the center of the muscle-tendon unit beginning 8 to 10 cm proximal to the patella. Extend the incision distally to a point 3 cm proximal to the patella. At the proximal extent of the longitudinal incision, make a transverse incision laterally through the muscle and fibrous tissue. At the distal extent of the longitudinal incision, make a transverse incision medially through the muscle and fibrous tissue. Flex the stifle, and allow the cut edges of the longitudinal incision to slide on each other. When a functional range of flexion is achieved, place interrupted sutures across the longitudinal incision to maintain the desired length of the quadriceps muscle-tendon unit.

Muscle Release

Extend the lateral incision to expose the proximal femur. At the level of the third trochanter, elevate the quadriceps from the medial, lateral, and caudal surfaces of the femur. Incise through the origins of each muscle group to release the quadriceps, and allow distal sliding of the muscle group. Release the vastus intermedius near its point of origin on the ilium. Close the surgical wound using standard methods.

Transarticular Fixator

Insert a half-pin just below the third trochanter of the femur using an end-threaded transfixation pin, and insert a full pin through the tibia 4 cm above the tarsus, using a centrally threaded transfixation pin. Maximally flex the stifle joint, and maintain this position with a heavy rubber band connecting the proximal and distal transfixation pins. Fashion a bandage around the paw, and connect it to the distal transfixation pin to maintain a functional angle of the tarsus.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Self-retaining retractors and a periosteal elevator assist tissue retraction and reflection. Instruments for placement of pins are necessary if a transarticular fixator is placed postoperatively.

POSTOPERATIVE CARE AND ASSESSMENT

After application of a transarticular fixator for quadriceps muscle contracture, pin-to-skin interfaces must be cleaned daily (see p. 1002). Passive flexion and extension of the stifle joint and tarsus should begin as soon as the patient allows limb manipulation (see Table 32.7). Joint movement should be repeated 20 to 30 times and at least three times daily; increased frequency of manipulation improves rehabilitation. The external fixator should be maintained for 3 to 5 weeks. Physical rehabilitation should be continued for an additional 5 weeks after it is removed. If a 90/90 flexion bandage is applied, it should be maintained for 3 weeks. After bandage removal, passive flexion and extension of the joint should be performed as described previously.

PROGNOSIS

The prognosis after surgery for quadriceps muscle contracture is guarded and depends on the degree of degenerative joint changes present and on whether a functional range of motion is obtained at surgery.[4] The prognosis is fair for functional limb use, but contracture may recur following surgery. A normal range of motion is seldom achieved, and most animals are able to flex the stifle only 45 to 90 degrees.

GRACILIS AND SEMITENDINOSUS FIBROTIC MYOPATHY

DEFINITIONS

Muscle contracture or **fibrosis** may occur when normal muscle-tendon unit architecture is replaced with fibrous tissue, resulting in functional shortening of the muscle or tendon. This shortening may cause abnormal motion in adjacent joints. Synonyms include *hamstring contracture, gracilis* or *semitendinosus contracture,* and *gracilis* or *semitendinosus myopathy*. **Myopathy** is a general term used to describe disease of the musculature.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Gracilis or semitendinosus fibrotic myopathy occurs most often in German shepherd dogs or Belgian shepherds. Fibrotic myopathy of the iliopsoas and the sartorius muscles has also been reported.[5,6] The cause of fibrotic myopathy is unknown but is hypothesized to be trauma, immune-mediated disease, or neuropathy. The musculotendinous junction is consistently thickened and fibrotic. A fibrous band is associated with the caudolateral border of the gracilis muscle. The muscle fibers are replaced with dense fibrous connective tissue, causing a nonpainful lameness. Approximately 50% of dogs have bilateral limb involvement.

DIAGNOSIS

Clinical Presentation

Signalment

Gracilis or semitendinosus myopathy occurs in young adult, male German shepherds and in Belgian shepherds. Traumatic gracilis muscle injuries occur in greyhounds.

History

Usually a history of insidious onset of rear limb lameness is characterized by an altered gait associated with gracilis or semitendinosus myopathy.

Physical Examination Findings

Dogs with gracilis or semitendinosus myopathy have a shortened stride, rapid elastic medial rotation of the paw, external rotation

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FIG. 35.11 Circumduction gait due to gracilis or semitendinosus fibrotic myopathy.

of the hock, and internal rotation of the stifle during the mid- to late-swing phase of the stride ("circumduction gait") (Fig. 35.11 and Video 35.5). The lameness is more pronounced at a trot. Affected muscles are palpable as a distinct taut band differentiated by the location of origin. Some dogs are in pain on muscle palpation. Most dogs exhibit a nonpainful lameness. Abduction of the hip and extension of the stifle and hock may be limited.

Diagnostic Imaging

Radiography is not usually helpful in these cases. Ultrasound examination is typically used to determine the extent and severity of the abnormality.

Laboratory Findings

Consistent laboratory abnormalities are not present.

DIFFERENTIAL DIAGNOSIS

The diagnosis of gracilis or semitendinosus myopathy can usually be made from historical and physical examination findings. Sartorius muscle contracture is less common but must be differentiated from them. Differentials include hip dysplasia, cranial cruciate ligament injury, and neurologic disease.

MEDICAL MANAGEMENT

If muscle trauma is identified early (especially in greyhounds), intermittent rest and ice (15 minutes on, 15 minutes off) may minimize permanent muscle damage. Following acute injury, strengthening and range-of-motion exercises should be performed while additional muscle trauma is minimized.

Physical Rehabilitation

Physical rehabilitation alone is rarely successful in the management of chronic contracture of the hamstring or gracilis muscles; it should be combined with surgery.

SURGICAL TREATMENT

Gait abnormalities observed with gracilis or semitendinosus myopathy can be reversed temporarily by resecting the affected muscles; however, the fibrotic band and subsequent lameness usually recur in 2 to 4 months. When surgery is attempted, it must be combined with aggressive, long-term rehabilitation, particularly during the period of fibrous tissue maturation (approximately 3–6 weeks postoperatively).

Preoperative Management

These animals are otherwise generally healthy, and minimal preoperative care is necessary.

Anesthesia

See p. 976 for anesthetic management of animals with orthopedic disease.

Surgical Anatomy

The gracilis muscle originates on the pubic symphysis and inserts along the medial tibial crest, but continues as crural fascia, finally joining with the semitendinosus to contribute to common calcaneal or Achilles tendons, essentially crossing the hip and stifle joints and inserting on the calcaneus. The semitendinosus muscle originates on the tuber ischium and follows a similar course, inserting on the medial portion of the tibia, continuing as crural fascia, and inserting on the tuber calcis. Fibrotic muscle can be palpated before surgery. Affected muscle may be fibrotic throughout its length; more commonly, the distal myotendinous junction of the gracilis muscle is thickened and fibrotic. A thick fibrous band associated with the proximal caudal border of the gracilis may be observed.

Positioning

Clip the rear limb from midline to tarsus, and position the animal in dorsal recumbency. Use a hanging-limb technique to facilitate limb manipulation.

SURGICAL TECHNIQUE

Incise the skin directly over the palpated fibrotic band. Expose and isolate the entire muscle and tendon length with sharp dissection. Resect the entire affected muscle to increase joint range of motion.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Self-retaining retractors and a periosteal elevator assist tissue retraction and reflection.

POSTOPERATIVE CARE

Postoperative physical rehabilitation is critical if significant fibrosis and contracture are to be prevented. Rehabilitation includes passive range of motion, stretching, and aquatherapy (see Chapter 11). Use of a temporary cast to maintain limb extension for 8 to 12 hours per day may help in some animals.

PROGNOSIS

The prognosis for gracilis or semitendinosus myopathy is guarded, with recurrence of fibrosis and restriction of gait occurring within

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4 months in most dogs. Intensive, long-term physical rehabilitation following surgical intervention may improve the prognosis.

SUPERFICIAL DIGITAL FLEXOR TENDON DISPLACEMENT

DEFINITION

Superficial digital flexor tendon displacement (SDFD) occurs when the tendon slides off the calcaneus. The displacement causes lameness of the affected limb.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The superficial digital flexor tendon is the most superficial part of the Achilles tendon and inserts distally on the toes. Tearing of the retinaculum that keeps the tendon over the tuber calcanei allows the tendon to displace medially or laterally. Lateral displacement is more common than medial displacement.[7] Dysplasia of the tuber calcanei leading to a shallow groove may contribute to tendon displacement.

DIAGNOSIS

Clinical Presentation

Signalment

SDFD is rare in dogs and has been reported in one cat. Shelties may be overrepresented for SDFD. No age or size predisposition is known.

History

History may include insidious or acute onset of rear limb lameness.

Physical Examination Findings

Dogs with SDFD have hindlimb lameness and swelling of the tip of the calcaneus. In most cases, it is possible to feel the tendon slide off the tip of the calcaneus (Videos 35.6, 35.7, and 35.8).

Diagnostic Imaging

Radiography is typically not helpful. Ultrasound examination is not necessary for diagnosis but may demonstrate intermittent displacement of the tendon.

Laboratory Findings

Consistent laboratory abnormalities are not present.

DIFFERENTIAL DIAGNOSIS

The diagnosis of SDFD can usually be made from historical and physical examination findings. Injuries of the common calcanean tendon must be differentiated from this condition.

MEDICAL MANAGEMENT

Medical management, including the use of nonsteroidal analgesics, is generally not effective for treatment of SDFD.

Physical Rehabilitation

Physical rehabilitation alone is not successful in the management of SDFD; it should be combined with surgery.

SURGICAL TREATMENT

Surgery involves reduction of the displaced tendon and imbrication of the torn retinaculum.

Preoperative Management

These animals are otherwise generally healthy, and minimal preoperative care is necessary.

Anesthesia

See p. 976 for anesthetic management of animals with orthopedic disease.

Surgical Anatomy

The superficial digital flexor tendon is the most superficial part of the common calcanean tendon. It passes over the tuber calcanei with a bursa between the bone and the flat tendon. A retinaculum holds the tendon in place over the calcaneus.

Positioning

Clip the rear limb from the mid tibia to the foot, and position the animal in sternal recumbency with the affected limb extended over the end of the table.

SURGICAL TECHNIQUE

Incise the skin directly over or slightly medial or lateral to the calcaneus. Expose the superficial digital flexor tendon and the retinaculum. In chronic cases, the retinaculum may be difficult to clearly identify or differentiate from the tendon. *Resect excessive retinacular tissue. Reduce the tendon to its normal position, and suture the retinaculum to hold the tendon in place using simple interrupted nonabsorbable sutures. Close the wound routinely.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Self-retaining retractors assist tissue retraction and reflection.

POSTOPERATIVE CARE

Postoperatively, place the limb in a soft padded bandage with or without a light splint. Maintain the bandage for 1 to 2 weeks. Limit activity for the first 2 weeks; follow this with leash walking for 4 weeks and then a gradual return to normal activity.

PROGNOSIS

The prognosis for SDFD is excellent, and most animals return to normal activity within 2 months.

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36 Other Diseases of Bones and Joints

PANOSTEITIS

DEFINITION

Panosteitis is a disease of young dogs that causes lameness, bone pain, endosteal bone production, and occasionally subperiosteal bone production. Synonyms include *enostosis, eosinophilic panosteitis, juvenile osteomyelitis,* and *osteomyelitis of young German shepherds*.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Panosteitis is an idiopathic entity causing endosteal and periosteal new bone formation. Osseous compartment syndrome due to a protein-rich, high-calorie diet has been proposed as a potential cause. Excessive protein may cause intraosseous edema and secondary increased medullary pressure and ischemia. Although some periosteal new bone is often evident, the predominant change is endosteal bone formation as the marrow is invaded by bone trabeculae. The marrow remains highly cellular with varying degrees of fibrosis, and no evidence of chronic inflammation, acute infection, or malignancy is noted.

DIAGNOSIS

Clinical Presentation

Signalment

Panosteitis predominantly affects male, large-breed dogs. Young dogs (<2 years of age) are most commonly affected; however, older dogs have occasionally been diagnosed with it. German shepherds have a higher risk of developing this disease than other large-breed dogs.

History

The hallmark of panosteitis is shifting leg lameness associated with pain on deep bone palpation. Although initial episodes of panosteitis may present as acute lameness in a single limb, the typical history consists of chronic, intermittent, shifting leg lameness.

Physical Examination

Affected dogs are commonly lame in only a single limb. Firm palpation of affected long bones usually elicits pain.

Diagnostic Imaging

Radiographic signs of panosteitis are progressive. Radiographs of affected limbs are often normal during early stages, and clinical signs may precede radiographic abnormalities by up to 10 days. If clinical signs are consistent with panosteitis but radiographs are normal, radiographs should be repeated in 7 to 10 days. Nuclear scintigraphy is a more sensitive diagnostic test of panosteitis than radiography. The earliest radiographic signs include widening of the nutrient foramen and blurring and accentuation of trabecular patterns (which are often difficult to identify, except in retrospect); these are followed by the appearance of radiopaque, patchy, or mottled bone within medullary canals (Fig. 36.1). Eventually, remodeling of medullary canals occurs, and cortical thickening may remain as the only residual finding.

Laboratory Findings

Consistent laboratory abnormalities are not present.

DIFFERENTIAL DIAGNOSIS

Panosteitis must be differentiated from other orthopedic diseases of large, immature dogs (e.g., osteochondritis dissecans, fragmented coronoid process, ununited anconeal process in forelimbs; hip dysplasia, osteochondritis dissecans of rear limbs). When radiographic evidence of panosteitis and other orthopedic diseases is present concurrently, panosteitis is usually assumed to be causing the acute clinical signs.

> **NOTE** Panosteitis has the best prognosis of all juvenile orthopedic diseases.

MEDICAL MANAGEMENT

The disease is self-limiting; treatment consists of controlling pain. Nonsteroidal antiinflammatory drugs (see Table 34.3) are usually administered during acute episodes of lameness. Exercise restriction is recommended when the animal is lame. Owners should be warned of the likelihood of recurrence, but the long-term prognosis is excellent for complete recovery.

SURGICAL TREATMENT

Surgical treatment of panosteitis is not indicated.

PROGNOSIS

The disease is self-limiting, and most animals eventually have normal function of affected limbs without evidence of pain. However, the disease may continue to affect different limbs,

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ABSTRACT

Several common disease processes affect the musculoskeletal system that are not categorized within trauma or degeneration. Many of these diseases affect young dogs, and it is important that veterinarians be able to recognize and treat them.

KEYWORDS

panosteitis
hypertrophic osteodystrophy
hypertrophic osteopathy
craniomandibular osteopathy
osteosarcoma

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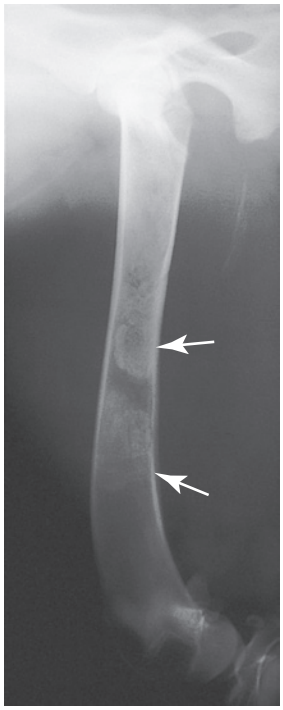


FIG. 36.1 Lateral radiograph of the femur of an immature dog with panosteitis. Note the areas of increased opacity within the medullary canal *(arrows)*.

causing pain and lameness until the dog reaches maturity. Clinical signs rarely persist after maturity.

NOTE Advise owners that panosteitis may recur, but that it usually resolves by the time the dog is 2 years of age.

HYPERTROPHIC OSTEODYSTROPHY

DEFINITION

Hypertrophic osteodystrophy (HOD) is a disease that causes disruption of metaphyseal trabeculae in long bones of young, rapidly growing dogs. Synonyms include *skeletal scurvy, canine scurvy, Moeller-Barlow disease, osteodystrophy types I and II, metaphyseal osteopathy*, and *metaphyseal dysplasia.*

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The cause of HOD is unknown. Proposed causative factors include vitamin C deficiency, oversupplementation of dietary calcium, and infectious organisms. A link to canine distemper virus has been postulated but is not supported by epidemiologic studies. Experimentally, vaccination protocols have been associated with the development of HOD in Weimaraner puppies; however, no specific vaccine has been identified. The pathogenesis is obscure, but an apparent disturbance of metaphyseal blood supply leads to changes in the physis and adjacent metaphyseal bone, causing delayed ossification of the physeal hypertrophic zone. The acute phase lasts approximately 7 to 10 days. Affected animals show signs ranging from mild lameness to anorexia, pyrexia, lethargy, severe lameness, refusal to rise, and generalized weight loss. Clinical signs may wax and wane.

NOTE Affected animals may be very ill, requiring intense supportive care.

Grossly, the metaphyseal regions of long bones are widened with perimetaphyseal soft tissue swelling. A line of separation of metaphyseal trabeculae is present parallel to the growth plate. Histologic microfractures of trabeculae are evident and are surrounded by inflammatory cells and necrosis. Failure of bone deposition on the calcified cartilage lattice of metaphyseal bone is evident.

DIAGNOSIS

Clinical Presentation

Signalment

This disease affects young, rapidly growing, large-breed dogs, and males are affected more often than females. Clinical signs are usually first noted at 3 to 4 months of age; however, they may occur as early as 2 months. Relapses may occur as late as 8 months of age. The highest incidence is in the fall. Weimaraners may be at increased risk.

History

An acute onset of lameness is often reported, and puppies may be so severely affected that they refuse to walk. Inappetence and lethargy are commonly reported by owners. A history of recent diarrhea may precede the onset of lameness.

Physical Examination Findings

Findings on physical examination range from mild lameness to severe lameness affecting all four limbs. More severely affected animals are often unable to stand or walk. Long bone metaphyses are swollen, warm, and painful on palpation. Swelling is often present in all four limbs; however, forelimb swelling may be more obvious, especially in distal radial metaphyses. Severely affected dogs may be depressed, anorexic, and pyrexic (body temperature of up to 106°F [41°C]).

Diagnostic Imaging

Radiographs of affected long bones reveal an irregular radiolucent zone in the metaphysis, parallel and proximal to the physis. This gives the appearance of a double physeal line. Metaphyseal flaring with increased bone opacity occurs because of periosteal proliferation in later stages of the disease. This reaction subsides with time but may leave a permanently widened metaphysis (Fig. 36.2).

Laboratory Findings

Laboratory abnormalities are not usually found. Hypocalcemia has been noted in a few affected dogs, but its significance is unknown. Bacteremia has rarely been reported in association with HOD.

DIFFERENTIAL DIAGNOSIS

This condition should be differentiated from septic arthritis, septic physitis, and panosteitis.

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FIG. 36.2 Radiograph of the distal radius and ulna of a dog with hypertrophic osteodystrophy. A parallel lucency *(arrowheads)* proximal and adjacent to the physis is typical of this disease.

MEDICAL MANAGEMENT

Analgesics should be administered to control pain (see Tables 13.2 and 34.3). Occasionally, severely debilitated animals require fluid support. Glucocorticoids, antibiotics, and vitamin C have been administered to affected dogs but have not proved effective in shortening the course or severity of the disease. Bacterial infection should be ruled out before glucocorticoids are administered.

PROGNOSIS

Most animals recover fully within 7 to 10 days of the onset of clinical signs; however, multiple relapses may occur. Occasionally, severe debilitation or multiple, severe relapses cause owners to request that affected animals be euthanized. Interference with normal physeal development may cause permanent deformity of long bones.

NOTE Warn owners that multiple relapses may occur and that deformity of the long bones may occur.

HYPERTROPHIC OSTEOPATHY

DEFINITION

Hypertrophic osteopathy (HO) is a diffuse periosteal reaction resulting in new bone formation around the metacarpal, metatarsal, and long bones. Synonyms include *pulmonary osteoarthropathy, hypertrophic pulmonary osteoarthropathy,* and *hypertrophic pulmonary osteopathy.*

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

HO can affect all four limbs. It can be a paraneoplastic syndrome (e.g., primary and metastatic lung tumors, esophageal carcinoma, rhabdomyosarcoma of the bladder, anal sac adenocarcinoma, renal transitional cell carcinoma, nephroblastoma), or it may be associated with other diseases (e.g., granulomatous lesions, chronic megaesophagus, patent ductus arteriosus, bacterial endocarditis, heartworm disease). The precise pathophysiology of this disease is unknown. Alterations in pulmonary function have been suggested to increase peripheral blood flow, resulting in connective tissue congestion. The increased peripheral blood flow is thought to be neurally mediated. The periosteum responds by forming new bone on the cortical surfaces of the metacarpal, metatarsal, and long bones. This new bone may be diffuse or nodular. Histologically, the affected area is made of bands of new cortical bone that contain small, fibrous marrow spaces.

DIAGNOSIS

Clinical Presentation

Signalment

Any breed or size of dog may be affected; however, because it is most commonly associated with neoplasia, HO is usually seen in older animals. This condition has rarely been reported in cats.

History

Dogs are usually brought in because of lethargy, reluctance to move, and swelling of the distal extremities. Onset of clinical signs may be acute or gradual.

Physical Examination Findings

Affected limbs are warm and swollen. Because this condition occurs secondary to diseases elsewhere in the body, an effort should be made to identify the underlying causative factors. A thorough physical examination is essential when evaluating affected animals.

Diagnostic Imaging

Radiographs of the limbs reveal a uniform periosteal proliferation, which is seen initially on the phalanges and metacarpal and metatarsal bones (Fig. 36.3). As the disease progresses, periosteal proliferation progresses proximally (i.e., radius/ulna and tibia/fibula). Articular surfaces of long bones are usually spared and appear normal.

Thoracic radiographs are used to identify any underlying pulmonary or mediastinal disease (e.g., primary or metastatic neoplasia, granulomatous lesions, heartworm disease). Abdominal ultrasonography should be performed to identify underlying abdominal disease if thoracic disease is not identified. Advanced imaging modalities (computed tomography [CT], magnetic resonance imaging) may be necessary in some cases to identify the primary lesion.

Laboratory Findings

Results of laboratory analysis usually reflect the underlying disease. Thrombocytosis may occur in some dogs; however, the cause is poorly understood.

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FIG. 36.3 Dorsopalmar view of the forepaw of a dog with hypertrophic osteopathy. Note the periosteal proliferation on the abaxial surfaces of the metacarpal bones and radius and ulna *(arrows).* Note that the carpal joints are spared.

DIFFERENTIAL DIAGNOSIS

This condition should be differentiated from bone neoplasia.

MEDICAL MANAGEMENT

Treatment is aimed at the underlying disease process. Remission of periosteal proliferation may occur after resolution or resection of the primary lesion.

PROGNOSIS

The prognosis depends on the possibility for complete resolution of the underlying disease process. If the primary disease can be resolved, the secondary HO often resolves. Although clinical signs often disappear within 1 to 2 weeks after treatment, bone lesions may take months to remodel.

CRANIOMANDIBULAR OSTEOPATHY

DEFINITION

Craniomandibular osteopathy *(mandibular periostitis, lion's jaw)* is a proliferative bone disease of immature dogs involving the occipital bones, tympanic bullae, and mandibular rami.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The cause of craniomandibular osteopathy is unknown. It is most often recognized in West Highland white terriers, Cairn terriers, and Scottish terriers. A genetic predisposition is suspected in many breeds; in West Highland white terriers, it is thought to have an autosomal recessive inheritance pattern. A similar syndrome (calvarial hyperostotic syndrome) has been reported in bullmastiffs. Craniomandibular osteopathy has been associated with canine leukocyte adhesion deficiency in Irish setters. A link to canine distemper virus has been postulated but is not supported by epidemiologic studies.

Proliferation of new, coarse, trabecular bone occurs adjacent to the mandibular rami, occipital bones, and tympanic bullae in affected animals. This new bone formation causes irregular enlargement of mandibles and tympanic bullae. Existing lamellar bone is resorbed by osteoclasis and is replaced with new bone that expands beyond the periosteal borders. Osteoclastic destruction of the original lamellar bone is accompanied by invasion of inflammatory cells (i.e., neutrophils, lymphocytes, and plasma cells). Normal bone marrow is lost as it is replaced with a vascular fibrous stroma. This proliferative stage of disease occurs when the dog is approximately 5 to 7 months old and is accompanied by intermittent fever, discomfort when eating, and pain when the mouth is forced open. Owners should be warned that multiple relapses may occur; however, bone proliferation decreases as dogs reach maturity and physes close.

DIAGNOSIS

Clinical Presentation

Signalment

Although young West Highland white terriers, Cairn terriers, and Scottish terriers are most commonly affected, this disease has been sporadically reported in other breeds. Clinical signs are usually first noted when the dog is 3 to 8 months of age. Males and females are equally affected. No seasonal distribution has been noted. Calvarial hyperostosis syndrome has typically been considered to be a disease of bullmastiffs.

History

Owners often note that the animal is reluctant to eat, drools, and has trouble chewing food. Pain is often evident when the mouth is opened.

Physical Examination Findings

Affected dogs have bilaterally enlarged mandibles and tympanic bullae. In severe cases, fusion of these structures may occur, preventing the jaws from being fully opened. Pain on opening of the mouth and intermittent fever (body temperature reading of up to 104°F [40°C] for 3–4 days) may be observed.

Diagnostic Imaging

Skull radiographs typically reveal increased irregular bone density of the caudal mandibles; in many dogs the tympanic bullae also appear denser (Fig. 36.4). Whereas approximately one-third of dogs have lesions confined to the mandibles, some animals also have lesions of the base of the skull and/or have a thickened calvarium. As dogs reach maturity, the edges of the new bone become smooth and affected areas shrink.

Laboratory Findings

Specific laboratory abnormalities are not seen. Blood cultures are negative.

DIFFERENTIAL DIAGNOSIS

Craniomandibular osteopathy must be differentiated from infection (i.e., abscesses or osteomyelitis) and primary bone neoplasia.

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FIG. 36.4 Lateral radiograph of the skull of a dog with craniomandibular osteopathy. Note the areas of periosteal proliferation.

MEDICAL MANAGEMENT

Analgesics (see Table 34.3) should be given to control pain until the animal reaches maturity. Severely debilitated animals that cannot open their mouths enough to eat solid foods require oral fluid nourishment or a feeding tube. Although antibiotics and glucocorticoids are often administered during febrile episodes, they will not alter disease progression.

SURGICAL TREATMENT

Surgical therapy is not indicated. Surgical resection of bone bridging the mandible and tympanic bullae in dogs with severely restricted jaw motion has been unsuccessful; however, rostral mandibulectomy (see p. 336) may allow the dog to lap gruel. Affected dogs should not be used for breeding purposes.

PROGNOSIS

The prognosis is guarded until the extent of bone production is known (i.e., at maturity). Excessive bone production, leading to fusion of mandibular and tympanic bullae, can restrict mandibular motion sufficiently to prevent dogs from eating. These animals are often euthanized.

NOTE Warn owners that multiple relapses may occur until maturity.

BONE NEOPLASIA

DEFINITIONS

Primary bone neoplasia arises from cells located within the bone structure. **Metastatic bone tumors** are those that spread to bone from other primary locations. Primary and metastatic tumors may occur in the **appendicular skeleton** (i.e., long bones) or the **axial skeleton** (skull, vertebrae, ribs, and pelvis).

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Primary bone tumors of dogs and cats include osteosarcoma, chondrosarcoma, fibrosarcoma, hemangiosarcoma, giant cell tumor, liposarcoma, periosteal osteosarcoma, periosteal fibrosarcoma, parosteal osteosarcoma, osteoma, multilobular osteoma, multilobular chondroma, osteochondroma, and chondroma (Tables 36.1 and 36.2). Primary tumors of the appendicular skeleton of dogs most commonly arise in the distal radial metaphysis, proximal humerus, proximal or distal femur, and proximal or distal tibia. Benign bone tumors (e.g., osteoma, ossifying fibroma, multilobular osteomas and chondromas, osteochondromas, enchondromas, chondromas) are generally slow growing. Multiple cartilaginous exostosis (MCE, or osteochondromatosis) is a histologically benign disease of cats that occurs after skeletal maturity, in contrast to dogs, in which the disease occurs before growth plate closure (see Table 36.2). Depending on tumor accessibility, complete surgical excision of benign bone tumors is usually curative.

Osteosarcoma is the most common malignant bone tumor in dogs, occurring most often in the metaphyseal region of long bones of large or giant breed dogs.[1–3] Common sites include distal aspect of the radius, proximal aspect of the humerus, distal aspect of the femur, proximal and distal aspect of the tibia, and distal aspect of or diaphyseal region of the ulna.[1–4] In dogs less than 15 kg, tumors are most commonly located in the humerus and femur.[5] A genetic cause of osteosarcoma appears likely based on experimental and clinical studies. Osteosarcoma has been associated with fractures and metallic implants and may also occur within radiation fields following radiation treatment of soft tissue sarcomas.

Osteosarcoma is a locally aggressive tumor, causing bone lysis and/or production. Metastasis is common and usually occurs early in the course of disease. Although fewer than 15% of affected dogs have radiographically detectable thoracic metastases at presentation, 90% die or are euthanized within 1 year of diagnosis because of complications associated with pulmonary metastasis.[4,6] Improved chances for survival are possible with amputation (see p. 1303) or limb-sparing procedures (see p. 1305) combined with chemotherapy.

NOTE Warn owners that despite the fact that there may not be evidence of metastasis at the time of diagnosis, metastases are typically noted within a year.

Osteosarcoma is the most common tumor of the axial skeleton. Common sites include the mandible, maxilla, spine, cranium, ribs, nasal cavity and paranasal sinuses, and pelvis. Because it is the most commonly diagnosed bone tumor of dogs, it is used as the model for evaluation, diagnosis, treatment, and prognosis of bone tumors in this chapter. Although the workup needed to diagnose neoplasia is similar for all bone tumors, treatment and prognosis vary depending on tumor type (see Tables 36.1 and 36.2).

Histologically, osteosarcoma is composed of anaplastic mesenchymal cells that produce osteoid. Histologic subgroups include osteoblastic, fibroblastic, chondroblastic, osteoclastic, poorly

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TABLE 36.1 Malignant Bone Neoplasia in Dogs

Tumor	Incidence	Metastasis	Treatment	Prognosis
Osteosarcoma of the appendicular skeleton	~75% of all bone tumors	High rate of early metastasis to lungs and soft tissue; bone metastasis is a late complication	• Amputation and chemotherapy with carboplatin and/or doxorubicin • Limb sparing in selected cases • Palliative radiation therapy for painful bone lesions	Mean survival time with amputation alone is 12–20 wk, median survival time with limb sparing or amputation plus carboplatin and doxorubicin is approximately 300–400 d
Osteosarcoma of the axial skeleton	Less common than appendicular tumors	Highly metastatic, local recurrence except for mandible, which is slower to metastasize	• Local resection of tumor (i.e., mandible and rib) • Carboplatin, local radiation as adjunct to surgery to reduce local recurrence	Median survival time is ~22 wk, 1-y survival is ~25%, rate of tumor recurrence is ~67%
Fibrosarcoma	<5% of bone tumors	Slower to metastasize than osteosarcoma	• Amputation (doxorubicin may prove beneficial for metastatic disease) • Limb sparing in select cases	Poor prognosis (but complete excision of low-grade tumors without metastases may be curative)
Chondrosarcoma	5%–10% of bone tumors	Slow to metastasize	Amputation (chemotherapy not of proven benefit)	May be good after amputation or resection of lesion
Hemangiosarcoma	<5% of bone tumors	May be multicentric, often involves spleen and right atrium, highly metastatic	• Amputation (chemotherapy not of proven benefit) • Limb sparing in select cases	Poor prognosis because of multiple organ involvement, mean survival time <5 mo
Giant cell tumor	Rare	Metastasis to lymph nodes, lung, and bones	Amputation	Poor prognosis
Liposarcoma	Rare	Metastasis to lung, liver, and lymph nodes	Amputation or local resection	Poor prognosis
Fracture-associated sarcoma	Uncommon, associated with fractures in which healing has been complicated; represents 5% of osteosarcomas	Metastasis occurs in ~15% of cases	• Amputation • Limb-sparing techniques • Chemotherapy	Same as osteosarcoma

TABLE 36.2 Malignant Bone Neoplasia in Cats

Tumor	Incidence	Metastasis	Treatment	Prognosis
Osteosarcoma	Most common primary bone tumor in cats (70%–80%)	Metastasis is uncommon	Amputation, radiation therapy, or excision of skull tumors	Median survival time is 12–50 mo; 12 mo median survival with appendicular osteosarcoma
Fibrosarcoma	Rare, often due to secondary invasion of bone from soft tissue	Incidence is unknown	Amputation	Long disease-free intervals reported (e.g., 10–18 mo)
Chondrosarcoma	4% of bone tumors, reported in the scapula	Incidence is unknown	Amputation	Prognosis guarded
Squamous cell carcinoma	Local bone invasion, occurs in oral cavity, orbit, and digits	See section on oral tumors	Amputation, mandibulectomy, maxillectomy, ± radiation therapy; see section on oral tumors	See section on oral tumors
Multiple cartilaginous exostosis (osteochondromatosis)	Uncommon, usually feline leukemia virus (FeLV) positive, benign physeal lesion in dogs	Multiple sites common; scapula, vertebra, mandible in cats, physeal in dogs	Palliative removal of painful lesions	Guarded in cats (favorable in dogs)

differentiated, and telangiectatic osteosarcoma. An inflammatory component is often present in fracture-associated sarcoma (i.e., a sarcoma that arises in the diaphysis of a long bone at the site of a previous fracture), reflecting altered healing patterns and chronic inflammation associated with these tumors.

Osteosarcoma accounts for the majority of primary bone tumors in cats (approximately 70%–80%). Osteosarcoma occurs more commonly in the hindlimbs of cats and may have a lower rate of metastasis than in dogs. Cats rarely have evidence of metastasis at presentation.

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DIAGNOSIS

Clinical Presentation

Signalment

Large- and giant-breed dogs have the greatest incidence of appendicular bone neoplasia. The median age of dogs with osteosarcoma is 7 years; however, a small, early peak is seen in dogs 18 to 24 months of age.[2–4] Large size is a greater determinant of risk than breed, although greyhounds, Rottweilers, and Great Danes have been shown to be at increased risk. Males may be slightly more commonly affected than females. Primary bone tumors of the axial skeleton are more common than appendicular tumors in small breed dogs[5]; the median age of affected animals is 8 to 9 years. The mean age of osteosarcoma in cats is 8 to 10 years. No apparent breed or sex predilection has been identified in either species for tumors of the axial skeleton.

History

Dogs with primary bone neoplasia affecting the appendicular skeleton are usually examined for lameness and/or localized limb swelling. Pathologic fracture may cause acute, severe lameness. Dogs with primary bone tumors of the axial skeleton usually have pain, reluctance to walk, and visible swelling. Clinical signs may be acute or chronic and progressive. Signs of axial bone neoplasia depend on the location. Neoplasia of the skull may cause difficulty eating and visible swelling, whereas neoplasia of the spine may lead to pain or neurologic deficits.

Primary bone neoplasia in cats may cause deformity and lameness. MCE may result in pain and rapidly progressive swelling. Common sites for MCE include the spine, scapula, and mandible.

Physical Examination Findings

Dogs with appendicular tumors are often lame. The limb may be enlarged and firm. Systemic signs of illness (e.g., fever, anorexia, weight loss) are uncommon in acute stages of the disease. Tumors of the axial skeleton are often palpable as firm swellings. Tumors affecting the vertebral column may cause acute signs of lameness or paralysis. Respiratory abnormalities associated with pulmonary metastasis are rare until late in the disease. Cats with primary appendicular bone neoplasia exhibit lameness and painful firm swelling. Cats with axial neoplasia may exhibit difficulty eating, neurologic deficits, and firm painful swelling.

NOTE Lameness is often the first sign of appendicular bone tumors.

Diagnostic Imaging

Radiographs of affected bones and the thorax should be evaluated. Radiographic signs of osteosarcoma include cortical and trabecular bone lysis, periosteal bone proliferation, and soft tissue swelling (Fig. 36.5). Thoracic radiographs should include a dorsoventral or ventrodorsal view and both lateral recumbent views (i.e., right and left lateral views). These radiographs should be carefully evaluated for evidence of tumor metastasis. Alternatively, contrast-enhanced CT of the thorax can replace survey radiographs; CT is more sensitive than radiography in detecting small metastatic lesions.[4] Although radiographic signs associated with primary bone neoplasia cannot be differentiated from those associated with fungal osteomyelitis, coupling radiographic signs with signalment and history can help determine whether neoplasia should be considered likely. Ultimately, histopathology is necessary for confirmation. If limb-sparing surgery is being considered, magnetic resonance imaging should be used to determine the extent of the disease.

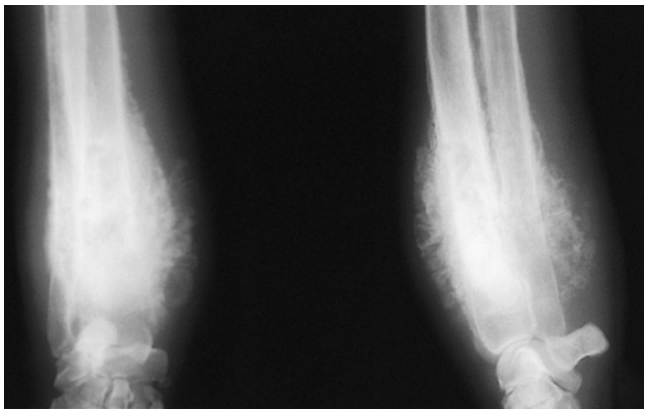


FIG. 36.5 Radiographs of a distal radius of a dog with osteosarcoma. Note the cortical lysis, periosteal proliferation, and soft tissue swelling.

Bone scintigraphy may help identify multifocal bone lesions in dogs with neoplasia. Osteosarcoma rarely affects multiple bones; however, metastatic lesions have been identified on scintigraphy at initial diagnosis. Scintigraphy may also be indicated when other forms of cancer (i.e., multiple myeloma and metastatic bone cancer) are suspected.

NOTE Three-view thoracic radiography or contrast-enhanced CT is essential in evaluating for pulmonary or thoracic wall metastases.

Laboratory Findings

No laboratory abnormalities are consistently found with primary or metastatic bone neoplasia. Fine-needle aspiration and cytology may be suggestive of bone neoplasia in a large percentage of cases. Ultrasound guidance may aid in identifying areas of bone lysis, enabling easier aspiration. Cytology of fine-needle aspirates may provide adequate evidence of primary bone tumor to allow initiation of treatment; however, definitive diagnosis of bone neoplasia usually necessitates histologic evaluation of samples obtained by biopsy or excision. Successful identification of a tumor requires obtaining adequate tissue samples and a pathologist with expertise in interpreting bone samples. Multiple samples should be obtained to increase diagnostic accuracy. Closed trephine (see p. 1302) or Jamshidi bone biopsy (11-gauge × 10 cm; Cardinal Health) from the center of the radiographic lesion is more accurate than biopsy of transitional zones between tumor and normal bone. The latter biopsies are commonly interpreted as reactive bone. Using fluoroscopy or other advanced imaging techniques to choose the biopsy site increases the chances of optimal, diagnostic biopsies. Alternatively, radiographs obtained after biopsy can be used to confirm biopsy location. Biopsies should be obtained in such a way that potentially seeded tumor cells will be removed during subsequent surgery.

Staging

Systemic staging is critical for proper decision making in animals with skeletal neoplasia. Thoracic radiography or CT is indicated.

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Pulmonary nodules must be 6 to 8 mm in diameter to be detected by radiography; CT is more sensitive in the detection of pulmonary disease (see Chapter 14). Bone survey radiography sometimes detects secondary skeletal sites, although this is not routinely performed. Bone scintigraphy may be performed, although it is not readily available and reports on its usefulness are conflicting. Spread to regional lymph nodes is rare, and fine-needle aspiration is not indicated unless an enlarged lymph node is recognized clinically.

DIFFERENTIAL DIAGNOSIS

Suspected primary neoplastic lesions must be differentiated from bacterial osteomyelitis, fungal osteomyelitis, metastatic bone tumor (e.g., prostatic carcinoma), direct extension of soft tissue tumors (e.g., nail bed carcinomas), hypertrophic pulmonary osteopathy, bone infarcts, hypervitaminosis A, periosteal response to trauma, and aneurysmal bone cysts.

MEDICAL MANAGEMENT

Most dogs with skeletal neoplasia ultimately die from metastatic disease, thus extensive research has explored the benefits of chemotherapy to treat micrometastasis. Multiple-modality treatment (e.g., amputation and chemotherapy) has extended the life span of dogs with osteosarcoma.[1,3,6] Numerous protocols are available for administering one or a combination of these drugs. In cases in which tumor resection is impossible, palliative radiation treatment may diminish pain. Readers are referred to an oncology text for additional information regarding medical treatment of osteosarcoma.

SURGICAL TREATMENT

Treatment of appendicular bone tumors involves limb amputation, scapulectomy, acetabulectomy, hemipelvectomy (see p. 1303), or tumor resection combined with limb salvage techniques and chemotherapy (see p. 1305).[1,3,7–9] Limb-sparing techniques are challenging surgical procedures and should be performed only by experienced surgeons with advanced training. Maxillary and mandibular tumors are treated by mandibulectomy (see p. 336) or maxillectomy (see p. 335), plus appropriate chemotherapy or radiation therapy. Spinal tumors can occasionally be treated with en bloc resection, but the procedure is difficult. Tumors of the ribs are treated with en bloc resection.

NOTE Perform fine-needle aspiration or another biopsy technique before amputating a leg because fungal and neoplastic lesions can be radiographically indistinguishable.

NOTE Dogs with severe concurrent orthopedic or neurologic disease may have difficulty ambulating after amputation; they should be evaluated carefully before amputation.

Preoperative Management

A thorough physical examination should be performed to identify concurrent problems that might interfere with anesthesia. Limb amputation involves loss of large quantities of tissue, fluid, electrolytes, and red blood cells. Animals should be well hydrated before surgery, and adequate fluids should be administered during surgery. Broad-spectrum antibiotics should be administered perioperatively, particularly during mandibulectomy, maxillectomy, and limb-sparing techniques (see Chapter 9).

NOTE Perioperative fluid management is essential during limb amputation owing to the large fluid loss associated with the surgery.

Anesthesia

Bone aspirates may be performed under sedation; bone biopsy usually requires general anesthesia. Patients undergoing bone biopsy, amputation, or limb sparing are usually middle-aged to older, and appropriate preanesthetic diagnostics (chemistry panel, complete blood count, thoracic radiographs) should be performed (see p. 976 for suggested anesthetic protocols). In patients undergoing forelimb amputation or limb sparing, analgesia may be provided via a brachial plexus block (see p. 152). Patients undergoing hindlimb amputation should receive an epidural (see p. 153). Patients undergoing midfemoral hindlimb amputation should receive a femoral-sciatic nerve block (see p. 153). Amputation of a front limb or a hindlimb should include direct nerve blocks before nerve transection (see p. 151).

Surgical Anatomy

Surgical anatomy varies with tumor location. Refer to the appropriate bone for an anatomic description.

Positioning

For bone biopsy, forelimb amputation, rear leg amputation, and limb sparing, the animal is usually positioned in lateral recumbency with the affected leg uppermost. A wide area around the proposed biopsy site is clipped and prepped for aseptic surgery. For the other procedures, the leg is prepped from the dorsal and ventral midline to the foot.

SURGICAL TECHNIQUE

Bone Biopsy

A Michele trephine or a Jamshidi bone marrow biopsy needle can be used to obtain a bone biopsy. Michele trephines obtain a larger sample of bone; however, risk of fracture through the biopsy site may be increased (Box 36.1). Jamshidi needles secure a smaller sample that may decrease the risk of pathologic fracture after biopsy. Accurate diagnoses can be obtained with either technique in more than 80% of cases.

NOTE If limb sparing is a goal, take care in locating the biopsy tract in an area that can be easily removed with surgical resection of the tumor. If possible, the same surgeon should perform the biopsy and the limb-sparing procedure (see Video 36.1).

If appropriate, locate the optimal site for biopsy by examining the leg under fluoroscopy while inserting a hypodermic needle into the skin to mark the spot. Once the desired spot for biopsy has been located, make a small skin incision (Video 36.1). For large lesions, make the incision over the center of the lesion. Locate the skin incision so that the biopsy tract that may be seeded with tumor cells during the biopsy can be removed during the definitive treatment

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BOX 36.1 Important Considerations for Bone Biopsy

- Obtain samples from the radiographic center of tumors.
- Obtain multiple samples.
- Take radiographs after biopsy to confirm biopsy site.
- Using Jamshidi needles may reduce the risk of pathologic fracture.
- Have pathologists with expertise in evaluating bone biopsies perform histology.

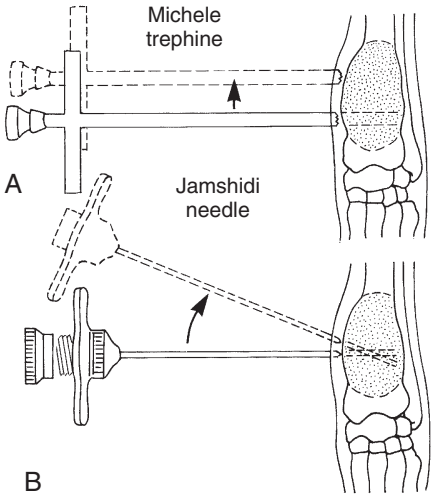


FIG. 36.6 Bone biopsy may be performed with (A) a Michele trephine or (B) a Jamshidi needle.

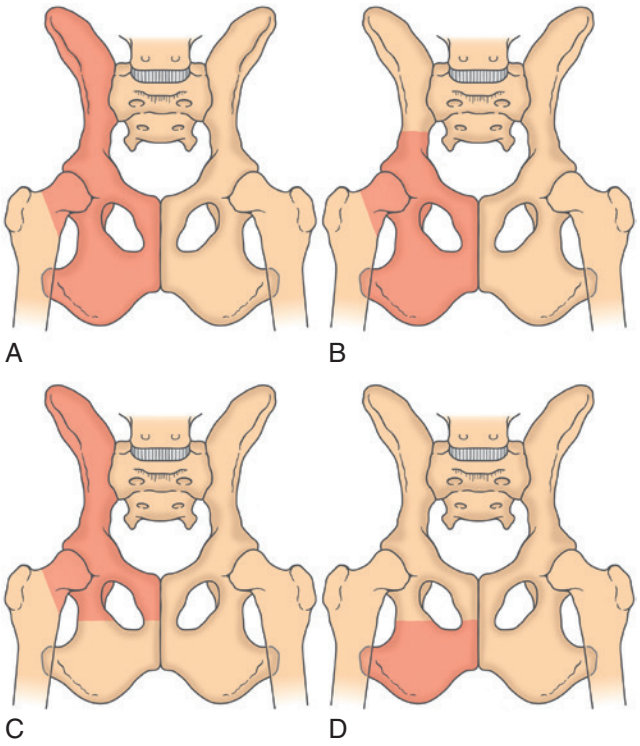


FIG. 36.7 Hemipelvectomy categories. (A) Total hemipelvectomy. (B) Mid-to-caudal partial hemipelvectomy. (C) Mid-to-cranial partial hemipelvectomy. (D) Caudal partial hemipelvectomy.

procedure (position the biopsy tract so that it does not interfere with skin flaps developed to cover the amputated bone end, should amputation be necessary). Push the trephine or the needle through soft tissue to the bone cortex. Remove the stylet, and advance the trephine or cannula through the bone by rotating the instrument back and forth. Once the biopsy instrument has been well seated, gently rock the instrument back and forth to separate the biopsy sample from surrounding bone. Remove the cannula and push the specimen out by inserting the probe into the front opening. Repeat the procedure to obtain multiple specimens (Fig. 36.6).

Amputation

Forelimb amputation may be performed by removing the scapula, or, alternatively, the limb can be removed by disarticulation at the shoulder joint. Scapular removal (forequarter amputation) is often preferred because muscular atrophy around the scapular spine is eliminated.

When tumors affect the femur, the coxofemoral joint should be disarticulated and the entire femur removed. Tumors involving the coxofemoral joint or pelvis require an acetabulectomy or hemipelvectomy. Four variations of hemipelvectomy have been described: total hemipelvectomy (Fig. 36.7A), mid-to-caudal partial hemipelvectomy (Fig. 36.7B), mid-to-cranial partial hemipelvectomy (Fig. 36.7C), and caudal partial hemipelvectomy (Fig. 36.7D).[7,9]

After amputation, the entire tumor should be resubmitted for histologic evaluation to confirm the diagnosis.

Forequarter Amputation

Make a skin incision from the dorsal border of the scapula, over the scapular spine, to the proximal third of the humerus. Continue the skin incision around the forelimb at this level (Fig. 36.8A). Transect the trapezius and omotransversarius muscles at their insertions on the scapular spine. Transect the rhomboideus muscle from its attachment on the dorsal border of the scapula, and retract the scapula laterally to expose its medial surface (Fig. 36.8B). Next, elevate the serratus ventralis muscle from the medial surface of the scapula (Fig. 36.8C). Continue to retract the scapula to expose the brachial plexus and axillary artery and vein. Ligate the axillary artery and vein with a three-clamp and transfixation suture technique (Fig. 36.9). Transect the brachial plexus. Transect the brachiocephalicus muscle, deep and superficial pectoral muscles, and latissimus dorsi muscle near their humeral insertions (Fig. 36.8D–E). Remove the forelimb. To close the wound, approximate the muscle bellies to cover the brachial plexus and vessels, and then suture subcutaneous tissue and skin (Fig. 36.8F).

Coxofemoral Disarticulation

Make a skin incision around the rear limb at the level of the middle third of the femur (Fig. 36.10A). The lateral aspect of the skin incision should extend farther distally than the medial aspect. *On the medial side, open the femoral triangle by incising between the pectineus muscle and the caudal belly of the sartorius muscle to expose and ligate the femoral artery and vein (Fig. 36.10B) using a three-clamp technique. Transect the sartorius, pectineus, gracilis, and adductor muscles approximately 2 cm from the inguinal crease (Fig. 36.10C). Isolate the medial circumflex femoral vessels over the iliopsoas muscle and ligate them. Transect the iliopsoas muscle*

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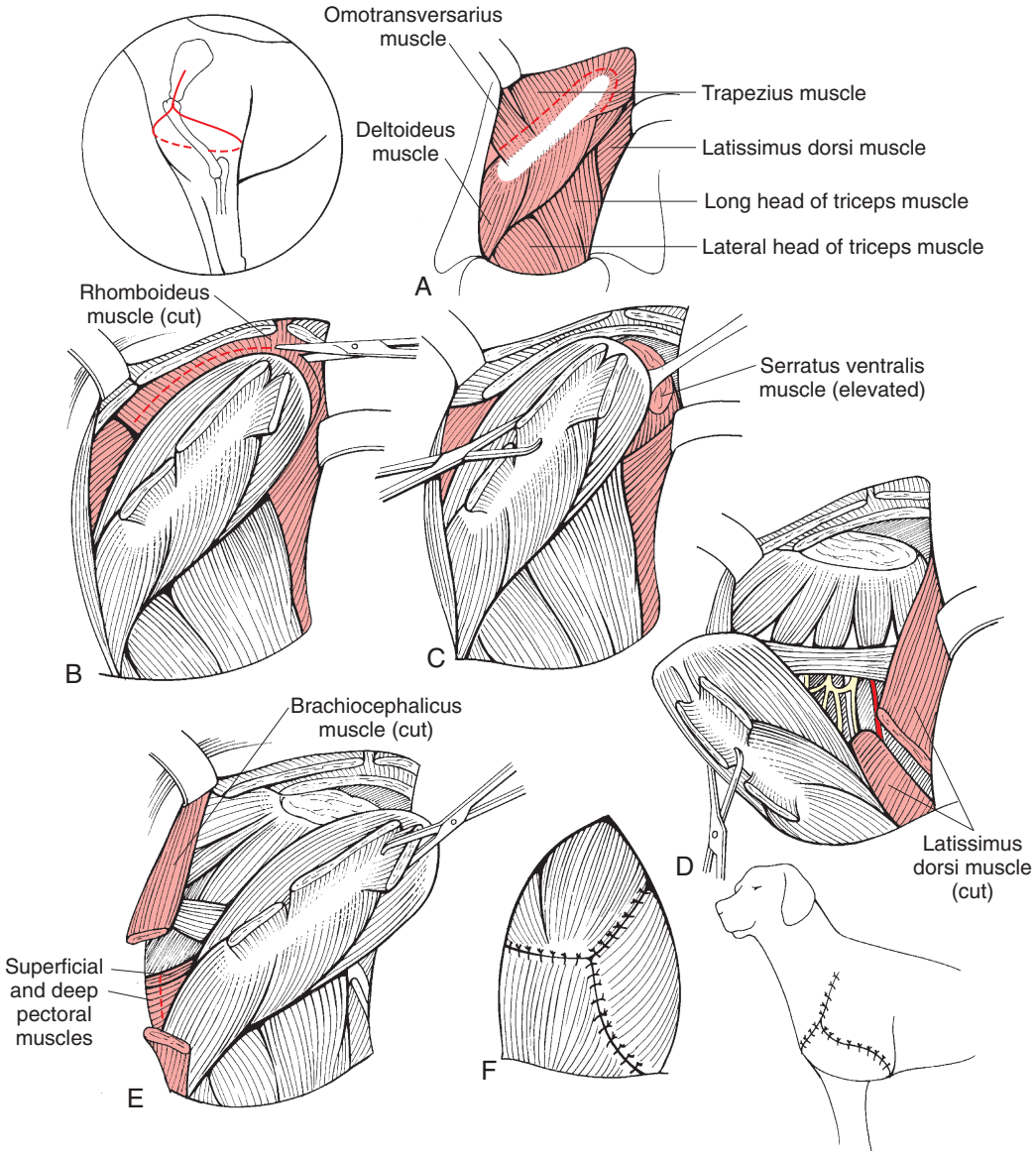


FIG. 36.8 (A) For forequarter amputation, make a skin incision from the dorsal border of the scapula, over the scapular spine, to the proximal third of the humerus. Continue the skin incision around the forelimb at this level. Transect the trapezius and omotransversarius muscles at their insertions on the scapular spine. (B) Transect the rhomboideus muscle from its attachment on the dorsal border of the scapula. (C) Elevate the serratus ventralis muscle from the medial surface of the scapula. (D) Retract the scapula laterally to expose the axillary artery and vein for ligation. Transect the brachial plexus. Transect the latissimus dorsi muscle near its humeral insertion. (E) Transect the brachiocephalicus and deep and superficial pectoral muscles near their humeral insertions, and remove the forelimb. (F) To close the wound, approximate the muscle bellies to cover the brachial plexus and vessels and suture subcutaneous tissue and skin.

at its insertion on the lesser trochanter, and reflect it cranially to expose the joint capsule (Fig. 36.10D). Incise the joint capsule and cut the ligament of the head of the femur (Fig. 36.10E). On the lateral side, transect the biceps femoris muscle and the tensor fascia lata at the midfemoral level, and reflect them proximally to expose the greater trochanter and sciatic nerve (Fig. 36.10F). Sever the sciatic nerve distal to its muscular branches to the semimembranosus, semitendinosus, and biceps femoris muscles. Transect the gluteal muscle insertions close to the greater trochanter (Fig. 36.10G). Transect the semimembranosus and semitendinosus muscles at the level of the proximal third of the femur. Sever the external rotator muscles and the quadratus femoris muscle at their attachments around the trochanteric fossa. Elevate the rectus femoris muscle from its origin on the pelvis. Incise the joint capsule circumferentially and remove the limb. Close the wound by flapping the biceps femoris muscle medially and suturing it to the gracilis and semitendinosus muscles. Flap the tensor fascia lata caudally, and suture it to the sartorius muscle. Suture subcutaneous tissue and skin.

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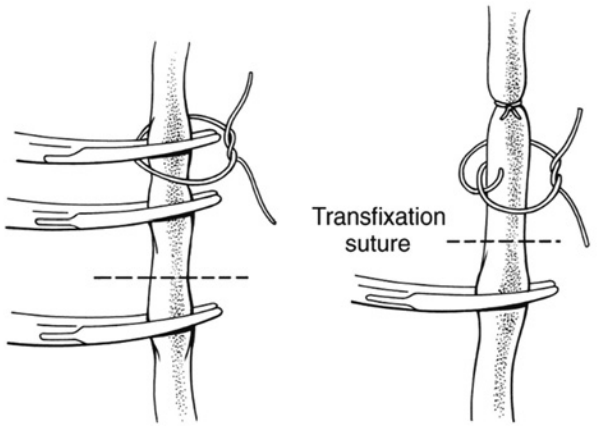


FIG. 36.9 A three-forceps and transfixation suture technique. Place three forceps on the artery and ligate it in the crushed area of the proximal forceps. Place a transfixation ligature distal to the first ligation, and cut the vessel between the middle and distal clamps.

Midfemoral Amputation

Make a skin incision around the rear limb at the level of the distal third of the femur (Fig. 36.11A). The lateral aspect of the skin incision should extend farther distally than the medial aspect. On the medial side, transect the gracilis muscle and the caudal belly of the sartorius at the midfemoral level (Fig. 36.11B). Isolate and ligate the femoral vessels (see Fig. 36.9). Transect the pectineus muscle through its musculotendinous junction (Fig. 36.11C). Transect the cranial belly of the sartorius muscle. Transect the quadriceps muscle proximally to the patella (Fig. 36.11D). Transect the biceps femoris muscle at the same level as the quadriceps muscle. Isolate and cut the sciatic nerve at the level of the third trochanter. Transect the caudal muscles, including the semimembranosus, semitendinosus, and adductor muscles at the midfemoral level (Fig. 36.11E). Elevate the insertion of the adductor muscle from the linea aspera of the femur (Fig. 36.11F). Cut the femur at the junction of the proximal and middle thirds of the diaphysis, and remove the limb. Close the wound by flapping the quadriceps muscle caudally to cover the femoral stump and suturing it to the adductor muscle. Flap the biceps muscle medially, and suture it to the gracilis and semitendinosus muscles. Muscles should be apposed to completely protect the distal end of the femur. *Suture subcutaneous tissue and skin.*

Acetabulectomy

Make a skin incision around the rear limb at the level of the middle third of the femur (see Fig. 36.10A). Extend the lateral aspect of the skin incision farther distally than the medial aspect. On the medial side, open the femoral triangle by incising between the pectineus muscle and the caudal belly of the sartorius muscle to expose and ligate the femoral artery and vein (see Fig. 36.10B) using a three-clamp technique. Transect the sartorius, pectineus, gracilis, and adductor muscles approximately 2 cm from the inguinal crease (see Fig. 36.10C). Isolate the medial circumflex femoral vessels over the iliopsoas muscle, and ligate them. Transect the iliopsoas muscle at its insertion on the lesser trochanter, and reflect it cranially to expose the joint capsule (see Fig. 36.10D). Incise the joint capsule and cut the ligament of the head of the femur (see Fig. 36.10E). On the lateral side, transect the biceps femoris muscle and the tensor fascia lata at the midfemoral level, and reflect them proximally to expose the greater trochanter and sciatic nerve (see Fig. 36.10F). Sever the sciatic nerve distal to its muscular branches to the semimembranosus, semitendinosus, and biceps femoris muscles. Transect the gluteal muscle insertions close to the greater trochanter (see Fig. 36.10G). Transect the semimembranosus and semitendinosus muscles at the level of the proximal third of the femur. Sever the external rotator muscles and the quadratus femoris muscle at their attachments around the trochanteric fossa. Elevate the rectus femoris muscle from its origin on the pelvis. Using a sagittal saw, cut through the ilial shaft, ischium, and pubis to remove the acetabulum and limb. Close the wound by flapping the biceps femoris muscle medially and suturing it to the gracilis and semitendinosus muscles. Flap the tensor fascia lata caudally, and suture it to the sartorius muscle. Suture subcutaneous tissue and skin.

Limb-Sparing Techniques

Some dogs with preexisting orthopedic and neurologic disease do not ambulate adequately after amputation. Additionally, some owners will not permit amputation. Limb-sparing techniques that involve en bloc resection of the tumor and replacement with a bone or metal substitute can be used in selected cases. Numerous limb-sparing techniques (e.g., cortical allograft, cortical allograft with polymethylmethacrylate, cortical ulnar autograft with microvascular transfer, rollover cortical ulnar autograft, intraoperative radiation, bone transport, stainless steel prosthetic bone substitute) have been described. The most suitable candidates for limb sparing are dogs with osteosarcoma of the distal radius that affects less than 50% of the bone. Limb sparing is considered an advanced surgical technique and should be performed only by experienced surgeons with specific training in the procedure.

Cortical Allograft with Carpal Arthrodesis

AP *Position the dog in lateral recumbency. Dissect around the pseudocapsule of the tumor. Osteotomize the bone 3 to 5 cm proximal to the radiographic margin of the tumor. Take a biopsy specimen from the proximal margin of resection to check for tumor. Transect the extensor carpi radialis muscle and remove it with the tumor, along with any other muscles or tendons that are involved.* The distal margin of resection is the joint surface.

Incise the joint capsule, and dissect the tumor free. Remove the articular cartilage of the carpal bones in preparation for carpal arthrodesis. Replace the resected bone with a cortical allograft stabilized with a long, dynamic compression plate (see p. 993). Filling the graft with polymethylmethacrylate decreases the incidence of implant loosening and allograft fracture, but controversial results have been reported regarding possible delays in graft incorporation. *Make sure that the plate is of sufficient length that at least four screws can be positioned in the proximal radius and three positioned distal to the graft. Harvest autogenous cancellous bone (or harvest the graft before dissecting the tumor to prevent contamination of the donor site), and place it at the host-graft interface and at the arthrodesis site (Fig. 36.12). If desired, insert a closed suction drain adjacent to the graft before closing the wound. Close subcutaneous tissue and skin routinely.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

A Michele trephine or a Jamshidi needle is needed for a bone biopsy. An osteotome and mallet, oscillating saw, or Gigli wire is used to sever the bone when a midhumeral or midfemoral amputation is performed. Nonabsorbable (polypropylene or

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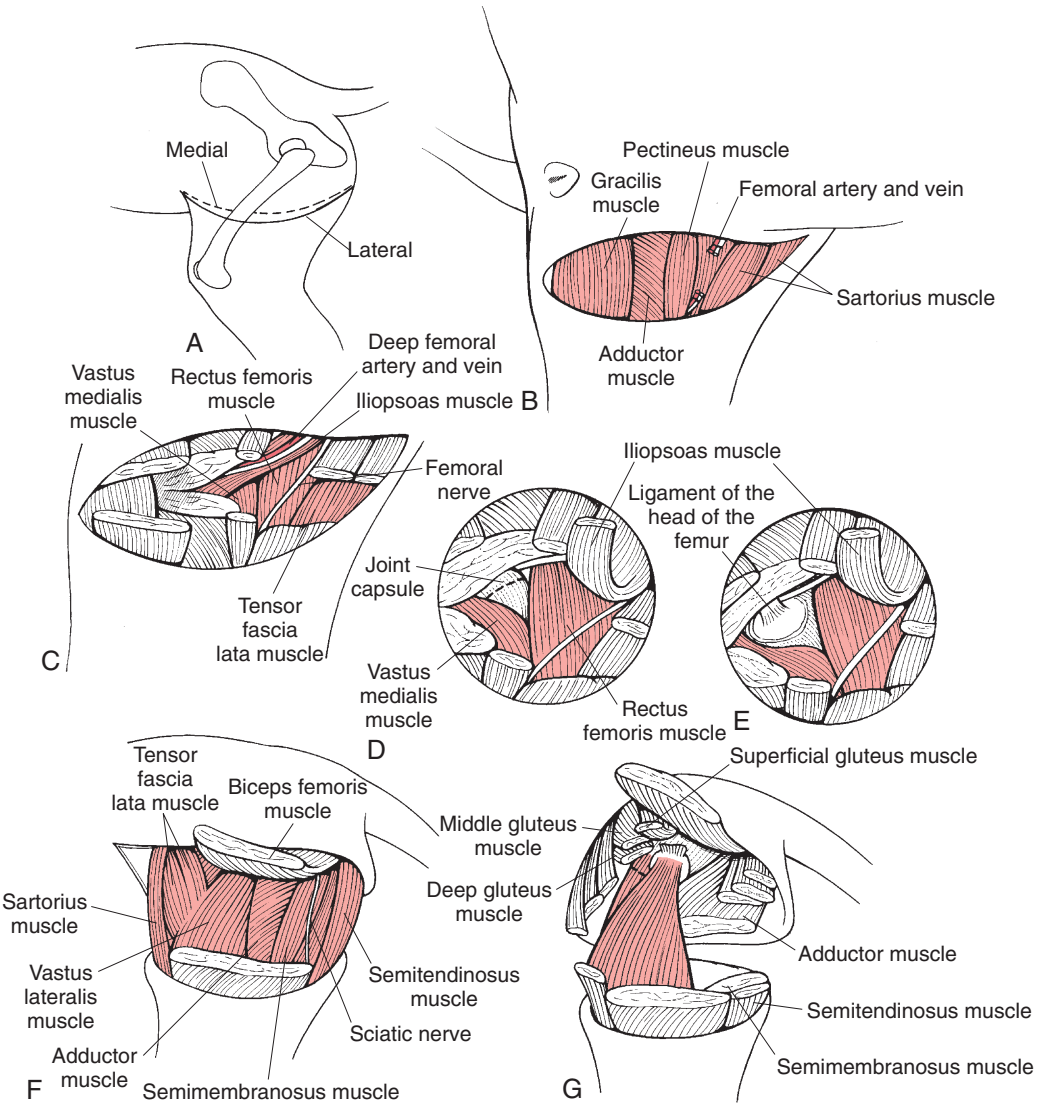


FIG. 36.10 (A) For coxofemoral disarticulation, make a skin incision around the rear limb at the level of the middle third of the femur. (B) On the medial side, open the femoral triangle by incising between the pectineus muscle and the caudal belly of the sartorius muscle to expose and ligate the deep femoral artery and vein. (C) Transect the sartorius, pectineus, gracilis, and adductor muscles approximately 2 cm from the inguinal crease. (D–E) Transect the iliopsoas muscle at its insertion on the lesser trochanter, and reflect it cranially to expose the joint capsule. Incise the joint capsule and cut the ligament of the head of the femur. (F) On the lateral side, transect the biceps femoris muscle and the tensor fascia lata at the midfemoral level. (G) Sever the sciatic nerve distal to its muscular branches to the semimembranosus, semitendinosus, and biceps femoris muscles. Transect the gluteal muscle insertions close to the greater trochanter. Transect the semimembranosus and semitendinosus muscles at the level of the proximal third of the femur. Sever the external rotator muscles and the quadratus femoris muscle at their attachments around the trochanteric fossa. Elevate the rectus femoris muscle from its origin on the pelvis. Remove the limb.

nylon) or strong synthetic absorbable (polydioxanone or polyglyconate) suture should be used for vessel ligation during amputation. Plating equipment and an allograft are needed for limb sparing.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative care after biopsy is minimal, but most dogs are in pain after the biopsy, probably because of subperiosteal hemorrhage. Pressure bandages may be needed if bleeding is excessive. After amputation, the surgical site should be observed for swelling, redness, and/or discharge. If hemorrhage or seroma formation is noted, pressure may be applied to the surgical site by applying a circumferential bandage around the thorax or pelvis. Cryotherapy aids in decreasing inflammation and pain. Mobility should be encouraged after surgery so that the animal can learn to walk on three legs; however, some animals require initial support to prevent falling, especially on slick surfaces. Most

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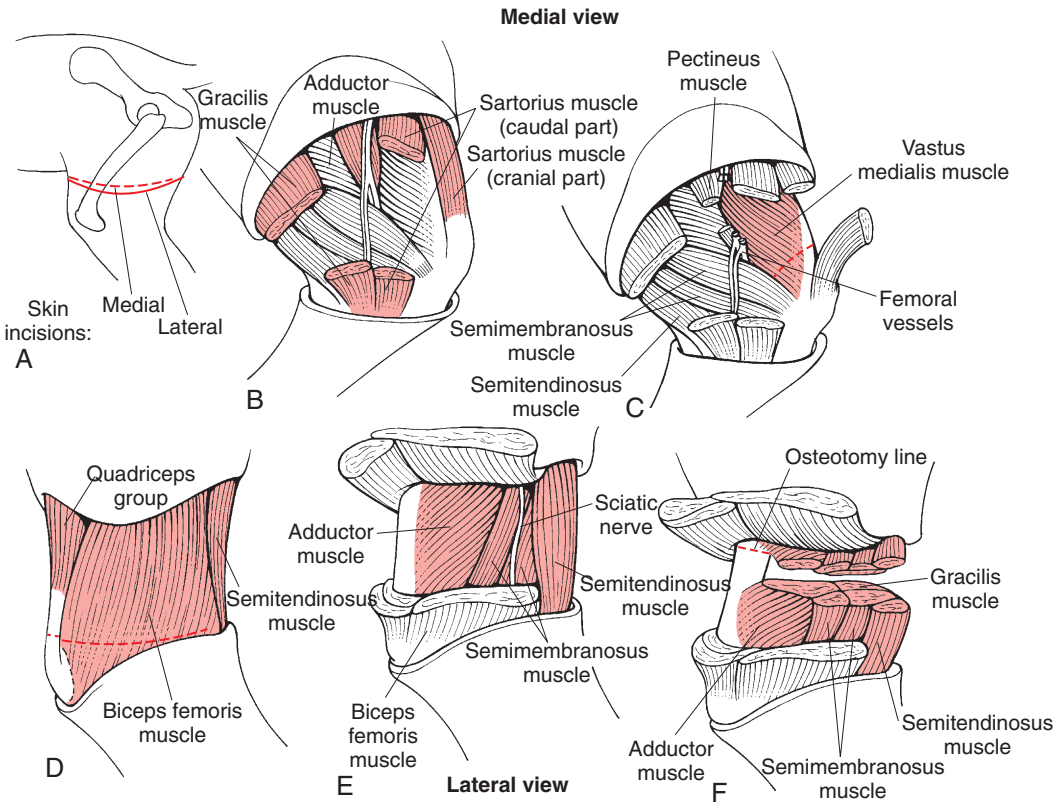


FIG. 36.11 (A) For midfemoral amputation, make a skin incision around the rear limb at the level of the distal third of the femur. (B) On the medial side, transect the gracilis muscle and the caudal belly of the sartorius at the midfemoral level. (C) Isolate and ligate the femoral vessels. Transect the pectineus muscle through its musculotendinous junction. Transect the cranial belly of the sartorius muscle. (D) Transect the quadriceps muscle proximally to the patella. (E) Transect the biceps femoris muscle at the same level as the quadriceps muscle. Isolate and cut the sciatic nerve at the level of the third trochanter. Transect the semimembranosus, semitendinosus, and adductor muscles at midfemoral level. (F) Elevate the insertion of the adductor muscle from the linea aspera of the femur. Cut the femur at the junction of the proximal and middle thirds of the diaphysis and remove the limb.

animals learn to ambulate on three limbs within 4 weeks (many by 1 week after surgery); however, some dogs may require coaxing and encouragement. Pain relief is often evident after amputation of limbs with large neoplastic lesions. Potential complications of amputation include seroma formation, bleeding, infection, and suture line dehiscence.

NOTE Many owners are reluctant to accept amputation and must be advised of the animal's ability to adapt to walking on three legs. Keeping a video of a dog with an amputation to show owners considering this procedure may be helpful (Video 36.2).

Postoperative care after limb sparing includes care of the closed suction drainage system (if used) and removal of the drain when drainage subsides (usually 1 day after surgery). The leg should be supported in a padded bandage to control postoperative swelling. The incision must be protected from self-mutilation with bandages and/or an Elizabethan collar. Decreased exercise is recommended for 3 to 4 weeks; however, controlled exercise or physical therapy may be necessary to prevent flexure contracture of the digits. Analgesics should be continued postoperatively for 10 to 14 days (see Tables 13.2 and 34.3).

PROGNOSIS

Dogs with appendicular osteosarcoma have a poor prognosis owing to the aggressive biological behavior of this tumor. Some factors that have been found to negatively influence prognosis include age, histologic grade, and lymph node metastases.[2] The overall median survival time from the time of diagnosis for dogs undergoing amputation alone is 5 months, but adjuvant chemotherapy may increase the overall median survival time to approximately 1 year.[2] Euthanasia is typically requested by owners when pulmonary metastases cause depression, anorexia, and/or respiratory distress.

Limb-sparing procedures in conjunction with chemotherapy generally results in good or adequate limb function in most dogs. Median survival times with limb sparing have been reported to be compatible to amputation when combined with adjunct chemotherapy.[3] Complications associated with traditional limb sparing vary dramatically according to the study cited and include local tumor recurrence, implant complications, and infection.

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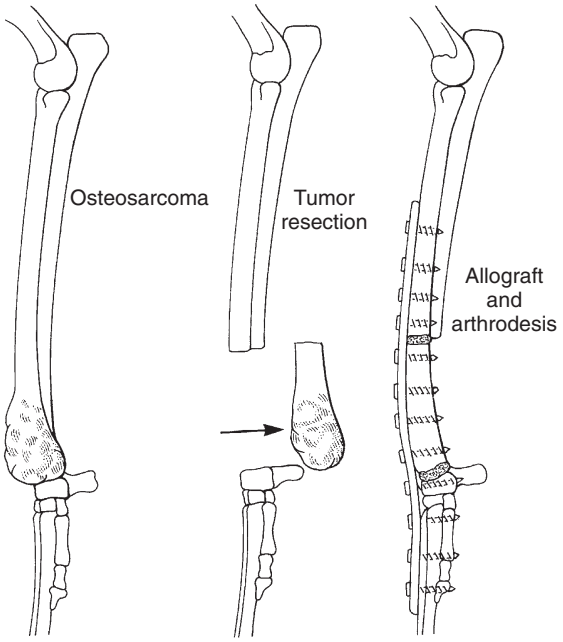


FIG. 36.12 A limb-sparing procedure for distal radial osteosarcoma. Resect the affected bone and soft tissue. Replace the bone with a cortical allograft, and stabilize it with a compression plate. Perform an arthrodesis of the carpus concurrently.

Interestingly, improved survival has been noted in dogs treated with limb sparing when the surgical site became infected.

In contrast to osteosarcoma, dogs with appendicular chondrosarcoma can be treated effectively with amputation alone. Survival time is significantly associated with histologic grade.

Cats with appendicular osteosarcoma often survive for 2 years or longer after amputation. Adjuvant chemotherapy has not been effective in the management of osteosarcoma in cats. The prognosis for cats diagnosed with MCE is poor based on the tendency of these lesions to recur following surgical excision.

JOINT NEOPLASIA

DEFINITIONS

Primary joint neoplasms are tumors that arise from synovial linings of diarthrodial joints, tendon sheaths, and/or bursae. The tumors of practical importance are those arising from synoviblastic tissue, called **synovial sarcomas** *(malignant synovioma, synovial cell sarcoma),* **histiocytic sarcomas,** and **synovial myxomas**.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Synovial cell sarcomas are rare tumors arising from synovioblastic mesenchyme in deep connective tissue around joints. Histiocytic sarcomas likely arise from macrophage/monocyte cell lines. Synovial myxomas arise from fibroblasts in myxomatous tissue. Joints above the carpus and tarsus are more commonly affected than distal joints. The biological behavior of these tumors ranges from slow growth to aggressive invasion of adjacent tissue. Metastasis to regional lymph nodes, lungs, bone, and other locations may occur.

DIAGNOSIS

Clinical Presentation

Signalment

Synovial sarcomas occur most commonly in large, middle-aged dogs; however, a breed predisposition has not been identified. Bernese mountain dogs, flat-coated retrievers, and golden retrievers may be affected more commonly with histiocytic sarcomas.

History

Affected dogs and cats are usually lame. A mass near a joint may occasionally be noticed by owners. Masses may grow slowly for a period; rapid growth may then occur.

Physical Examination Findings

Masses vary in size but are usually firm with some fluctuant areas. The degree of lameness appears to correlate with the amount of bone involvement.

Diagnostic Imaging

Radiographs of involved joints are needed to evaluate the extent of bone and soft tissue involvement. Lobulated soft tissue masses may be present in the joint region. Bone changes (e.g., lysis of subchondral bone and cortex, new bone production) are often noted on either side of the joint (Fig. 36.13). This contrasts with the appearance of primary bone tumors, which seldom appear to affect the bones on either side of the joint or to "cross a joint." Thoracic radiographs or contrast-enhanced CT should be performed to evaluate for the presence of pulmonary metastasis.

Laboratory Findings

Consistent laboratory abnormalities are not present.

DIFFERENTIAL DIAGNOSIS

Neoplastic joint lesions must be differentiated from synovial cysts, which are well-circumscribed masses attached to the joint capsule, tendon sheath, or bursa. Synovial cell sarcomas must be differentiated from cruciate disease, fungal and infectious joint diseases (e.g., septic arthritis, erosive arthritis, villonodular synovitis), and other tumors (e.g., fibrosarcoma, rhabdomyosarcoma, liposarcoma, hemangiopericytoma, malignant fibrous histiocytomas, giant cell tumors of soft tissue, primary bone tumors). A definitive diagnosis requires a biopsy of the lesion; however, excisional biopsy (limb amputation) rather than incisional biopsy may be warranted if massive bone destruction precludes therapy other than amputation. Fine-needle aspiration can reveal malignant cells but will not allow visualization of tumor architecture necessary for definitive diagnosis of tumor type in many cases.

SURGICAL TREATMENT

Amputation is the recommended treatment for these tumors (see p. 1303). Local excision is not recommended because of the high recurrence rate. Concurrent chemotherapy may be beneficial.

POSTOPERATIVE CARE AND ASSESSMENT

See p. 1306 for recommendations regarding the postoperative care of animals after amputation. Periodic examination of affected

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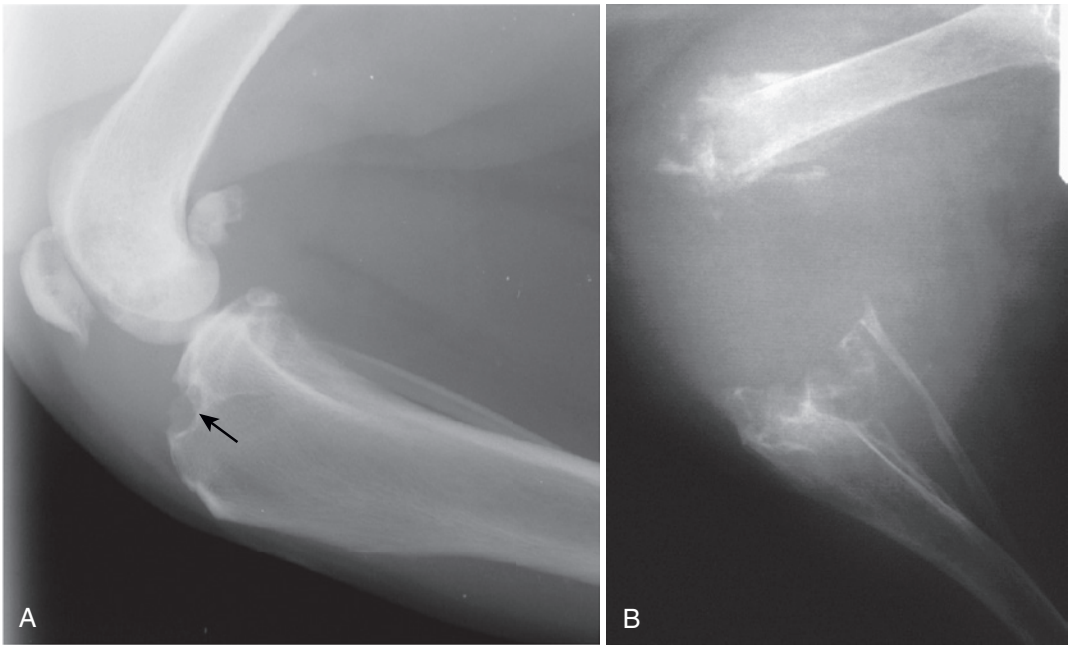


FIG. 36.13 Lateral radiograph of a dog with synovial cell sarcoma of the stifle joint. Note the (A) subtle lysis of the proximal tibia *(arrow)* and soft tissue opacity in the stifle and (B) extensive lysis of the distal femur and proximal tibia. Because both sides of the joint are affected, a soft tissue tumor should be suspected.

animals is recommended for early detection of local recurrence or metastasis.

PROGNOSIS

The prognosis varies with type of tumor. Although these tumors have historically been considered to be slow-growing tumors that metastasize late in the course of disease, a 2010 study suggested that metastasis is common after amputation.[10] The average survival time for dogs with synovial cell sarcoma is 32 months, 5 months with histiocytic sarcoma, and 30 months with synovial myxoma.[10,11] Histiocytic sarcoma has the highest rate of metastasis, whereas synovial myxoma has the lowest. Dogs undergoing surgical tumor excision or amputation have a significantly higher survivability than those receiving no treatment.

OSTEOMYELITIS

DEFINITIONS

Osteomyelitis is an inflammation of bone, although most commonly the term is used to describe inflammation due to infection. Acute **osteomyelitis** is characterized by systemic illness, pain, and soft tissue swelling without visible radiographic alterations in bone. **Chronic osteomyelitis** exists when acute and systemic clinical signs have subsided, but infection manifested by draining sinuses, recurrent cellulitis, abscess formation, and progressive destructive and proliferative osseous changes is present. A **sequestrum** is a piece of devitalized bone that has become separated from surrounding tissue. **Glycocalyx** (biofilm) is a combination of bacterial slime and host cellular debris that shrouds bacterial colonies and facilitates bacterial adhesion.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Most bone infections in dogs and cats are bacterial in origin. Many of these are monomicrobial infections with β-lactamase–producing *Staphylococcus pseudintermedius* predominating. Polymicrobial infections also occur and may harbor mixtures of *Streptococcus* spp., *Proteus* spp., *Escherichia coli, Klebsiella* spp., and *Pseudomonas* spp. Anaerobic bacteria are an important cause of osteomyelitis; they are present in more than two-thirds of bone infections. Anaerobes may exist alone as the sole causative agent, or more commonly as single organisms in polymicrobial infection. Anaerobes isolated from bone infections include *Actinomyces* spp., *Clostridium* spp., *Peptostreptococcus* spp., *Bacteroides* spp., and *Fusobacterium* spp. Characteristics of anaerobic infections include fetid odor, sequestration of bone fragments, and evidence of bacteria with differing morphology on gram-stained smears.

NOTE Treatment failure may result from lack of identification and inappropriate treatment of anaerobic bacteria.

Bacterial osteomyelitis is often classified as hematogenous or posttraumatic; however, the former is rarely reported in dogs. Fracture-associated osteomyelitis has been reported to occur most commonly in the radius/ulna and femur. Bone is normally resistant to infection; thus predisposing factors are required for osteomyelitis to occur. Factors associated with osteomyelitis include bone necrosis or sequestration, fracture instability, ischemia, implants or other foreign materials, and alteration of the systemic or local immune response. A common source of bacterial inoculation is surgical site contamination during open

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fracture reduction. Although the type and quantity of bacteria inoculated are important factors in the development of bone infection, bacteria alone do not necessarily cause osteomyelitis. Other important factors in the pathogenesis of posttraumatic osteomyelitis are (1) extent of soft tissue damage and alteration of blood supply, (2) formation of a biofilm (glycocalyx), and (3) stability of fracture repair. Tissue damage may be caused by the injury or may occur during the surgical procedure. Damaged soft tissue and devitalized bone serve as excellent culture media for bacteria. Bacterial proliferation is also potentiated by foreign materials in the wound (e.g., synthetic suture materials or implants). Glycocalyx is a combination of bacterial slime and host cellular debris that shrouds bacterial colonies and facilitates bacterial adhesion. The biofilm also protects bacteria from phagocytosis, host antibodies, and antibiotic effectiveness. Osteomyelitis is worsened by fracture instability. Continual motion impairs revascularization of spaces between fractured bone ends, which in turn prevents host defense mechanisms from protecting the area.

Mycotic bone infections are acquired through hematogenous dissemination of inhaled spores. Offending organisms are endemic in some geographic locations and include *Blastomyces dermatitidis, Coccidioides immitis,* and, less commonly, *Histoplasma capsulatum, Cryptococcus neoformans,* and *Aspergillus* spp. Although viral osteomyelitis is considered uncommon, newer evidence suggests that some canine bone diseases may be viral in origin. Sequences of ribonucleic acid (RNA) homologous with canine distemper viral RNA have been detected in osteoblasts of dogs with metaphyseal osteopathy (HO). Other causes of osteomyelitis include parasites, foreign bodies, and corrosion of metallic implants. Diagnosis of osteomyelitis is often suspected on the basis of history, clinical signs, and radiographic findings.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, breed, or sex of dog or cat may be affected.

History

Historic findings may include recent open reduction and stabilization of a fracture, bite wounds, open traumatic wounds, or habitation in an endemic fungal region; however, fungal osteomyelitis must never be eliminated simply because the patient does not live in an endemic area.

Physical Examination Findings

Clinical features of osteomyelitis vary depending on the stage of the disease. The initial response of bone to infection is inflammation; soft tissue in the area can become hot, reddened, swollen, and painful (Fig. 36.14). The animal is often pyrexic, depressed, and partially or totally anorexic. Distinguishing between acute osteomyelitis and inflammation associated with surgical intervention is frequently difficult. Persistence of an elevated temperature for longer than 48 hours postoperatively or a neutrophilic left shift increases the likelihood that infection is present rather than just surgically induced trauma. However, lack of either of these findings does not exclude infection. Animals with chronic osteomyelitis are usually brought in for evaluation of draining tracts and/or lameness. Pyrexia, anorexia, and other clinical signs associated with systemic disease are frequently lacking.



FIG. 36.14 A dog with femoral osteomyelitis. Note the draining tract on the caudolateral aspect of the thigh.

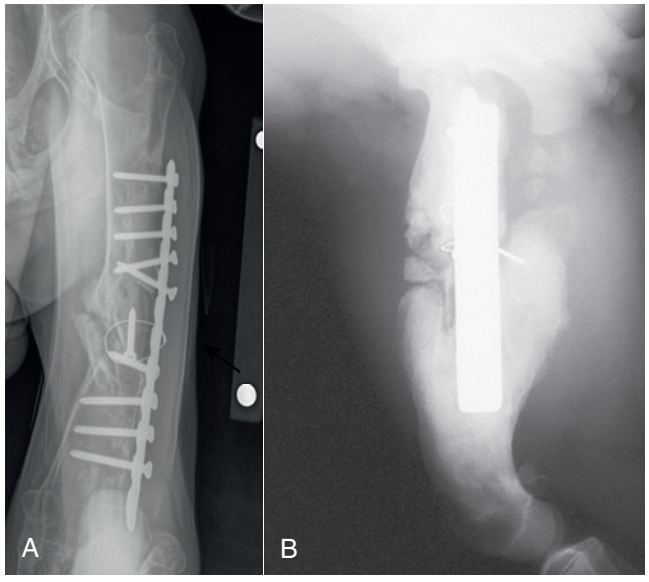


FIG. 36.15 Radiograph of a dog with (A) acute and (B) chronic osteomyelitis following open reduction and stabilization of a femoral fracture with cerclage wires. Extensive periosteal reaction and sequestrum formation are present.

Diagnostic Imaging

Specific radiographic findings vary depending on the stage of disease, site of infection, and pathogenicity of infective organisms. Soft tissue swelling is the first sign of acute osteomyelitis and may be observed as early as 24 hours postinfection. Radiographic signs may lag behind the clinical presentation. Early radiographic changes include periosteal proliferation with deposition of new bone in a lamellar pattern oriented perpendicular to the long axis of the bone. Lamellated periosteal reactions are typically associated with osteomyelitis, whereas solid periosteal new bone formation is not typically associated with infection (Fig. 36.15). As the infection progresses, lysis of the medullary cavity becomes apparent. Sequestra may develop if bone has been devitalized, and sclerosis and lysis become interspersed throughout cortical and medullary bone at the site of infection as *involucra* (new bone formation around a sequestrum) form. Sequestered bone appears more radiographically opaque than surrounding bone.

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Laboratory Findings

With acute osteomyelitis, systemic evidence of infection is often present, which is typically indicated by an elevated white blood cell count and neutrophilia with or without a left shift. Laboratory analysis in dogs with chronic osteomyelitis is usually normal.

Microbiological culturing is definitive for bacterial osteomyelitis and is essential in determining the organism's in vitro susceptibility to antimicrobial drugs. Sample specimens for culture should not be collected from draining tracts. Organisms cultured from draining tracts correlate with pathogens isolated at surgery in fewer than half of patients. Preferably, aerobic and anaerobic cultures should be obtained from bone collected during surgical intervention. Alternatively, collection of culture samples via fine-needle aspiration of material directly surrounding the involved bones may sometimes be useful if adequate material is obtained for culture. Fungal cultures and cytologic or histologic evaluation of biopsies may be diagnostic of fungal osteomyelitis. Serology may be helpful in diagnosing blastomycosis and coccidioidomycosis. Cryptococcosis is best diagnosed by detecting antigenemia. Urine excretion of antigens may diagnose blastomycosis and histoplasmosis. Fungal hyphae may often be found in urine sediment cytology in dogs with systemic aspergillosis, and there is an enzyme-linked immunosorbent assay or ELISA for *Aspergillus antigenemia* that appears useful. Serology for antibodies against histoplasmosis or systemic aspergillosis is of dubious sensitivity and specificity.

NOTE Collect samples for bacterial culture from deep fine-needle aspirates. Do not culture draining tracts.

DIFFERENTIAL DIAGNOSIS

Osteomyelitis must be differentiated from bone neoplasia. Radiographic changes of osteomyelitis are similar to those following surgery in which periosteal trauma has been excessive. Hematogenous metaphyseal osteomyelitis must be differentiated from HOD.

MEDICAL MANAGEMENT

Medical management consisting of antibiotic therapy and application of warm packs may be effective in patients with hematogenous osteomyelitis or in those with postoperative osteomyelitis, although infection can rarely be cleared as long as implants remain in place. Medical therapy is reasonable when the affected area exhibits signs of inflammation, but sequestra, necrotic tissue, or pockets of exudate make medical therapy unlikely to be successful by itself. Additionally, if the patient has postoperative osteomyelitis, implant and bone stability must be present for medical management to be effective. After bone healing, implant removal is usually necessary to resolve the infection. Appropriate antibiotic therapy is determined by culture and sensitivity and is continued for a period of at least 28 days.

SURGICAL TREATMENT

If sequestra or pockets of exudate are present, drainage and debridement of necrotic tissue are necessary. The fracture must be stable, and appropriate antimicrobial therapy should be instituted on the basis of culture and susceptibility testing. Treatment of chronic osteomyelitis entails maintenance or provision of fracture stability, removal of loose implants and sequestered bone, cancellous bone grafting of bony deficits, and appropriate antimicrobial therapy. Implanted bactericidal carriers (polymethylmethacrylate or a biodegradable polymer) have been used extensively in humans and in horses, but their use in small animals has been limited.

Preoperative Management

In patients with acute osteomyelitis, antibiotic therapy should be initiated immediately with bactericidal agents that have a broad spectrum of activity against aerobic and anaerobic bacteria (e.g., clindamycin plus enrofloxacin). Definitive antimicrobial therapy is determined through culture and susceptibility testing. Antimicrobial therapy of animals with chronic osteomyelitis should be based on culture and sensitivity testing of the offending organisms at surgery. Perioperative antibiotics should not be administered to these patients until intraoperative cultures have been obtained.

Anesthesia

Most animals with osteomyelitis are otherwise healthy, and a variety of anesthetic regimens can be used (see p. 976). If preoperative bloodwork suggests organ dysfunction (i.e., hepatic or renal), refer to Chapters 20 and 24, respectively, for specific anesthetic recommendations.

Surgical Anatomy

Refer to Chapter 33 for anatomy of the bone involved.

Positioning

Refer to Chapter 33 for exposure of the bone involved.

SURGICAL TECHNIQUE

Acute Osteomyelitis

Open infected wounds and debride necrotic tissue. If a fracture is present, stabilize bone fragments with an appropriate implant system (external skeletal fixators are usually preferred).

NOTE Stabilization of fractures is the key to successful treatment of osteomyelitis. Bone union will occur in the presence of infection if fragments are stable.

If previous surgery was performed, and if the fracture remains stable, leave the original implants in place. If the implants have loosened, choose another fixation system to provide rigid fixation. Establish drainage by treating the surgical site as an open wound. Irrigate the wound with 0.05% chlorhexidine, and then pack it with sterile gauze soaked with 0.05% chlorhexidine. Cover wounds with a sterile outer dressing that will absorb drainage products accumulated between bandage changes. Once the infection has been eliminated, suture the wound. Alternatively, close the wound over a continuous suction drain leaving the drain in place for 1 to 3 days depending on the amount and consistency of drainage.

Chronic Osteomyelitis

Determine the degree of fracture stability by palpation and radiographic assessment. If fractures and original implants are stable,

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leave them in place. However, if implants are loose and fractures have not healed, remove the loose implants and rigidly stabilize the fracture. Identify and remove sequestered bone through radiographic imaging and surgery. At surgery, recognize sequestered bone as that having a yellowish discoloration with no soft tissue attachments. Do not try to stabilize sequestered bone fragments; instead, remove them and place an autogenous cancellous bone graft in areas devoid of bone. Establish drainage as described previously.

Antibiotic-impregnated polymethylmethacrylate beads may be considered for treating chronic infections, especially those associated with cortical allografts used for limb sparing. Bead strings are constructed using a bead mold in which the cement mixture is pressed onto stainless steel or nylon sutures. The strings are secondarily sterilized with ethylene oxide before implanting. The slow elution of the antibiotic can produce wound fluid concentrations up to 200 times those obtained with systemic antibiotics, exceeding the minimum inhibitory concentration for up to 80 days without toxic effects. The beads are usually removed after treatment.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Aerobic and anaerobic culturettes should be available. Other instruments needed include those necessary for insertion of the selected implant, bone curettes, self-retaining retractors, and a selection of absorbable suture material. Nonabsorbable suture material should generally be avoided in infected tissue. Jackson-Pratt drains are useful for continuous suction drainage.

POSTOPERATIVE CARE AND ASSESSMENT

In patients with acute osteomyelitis, antibiotic therapy should be continued for at least 3 to 4 weeks. With chronic osteomyelitis, antibiotics should be administered for at least 4 to 6 weeks. If the wound is managed in an open fashion, the area should be irrigated with 0.05% dilute chlorhexidine twice daily and packed with a chlorhexidine-soaked sponge. Using umbilical tape secured to the skin on either side of the incision that can be tied over the gauze packing facilitates frequent changing of the packing materials. The limb should be bandaged until the wound is closed to decrease the likelihood of iatrogenic infection. Continuous closed suction drains are maintained for 1 to 4 days depending on the volume and consistency of the drainage. If a fracture is present, postoperative care is dictated by the fracture configuration and the stabilization procedure used. Generally, activity should be restricted to leash walks until the fracture has healed. Affected animals should be observed daily for signs of recurring fever, pain, swelling, and/or draining tracts and analgesic provided as appropriate (see Tables 13.2 and 34.3).

PROGNOSIS

If all bone sequestra are removed and fractures are adequately stabilized, the prognosis for resolving infection and returning the patient to normal activity is good. Removal of all implants following bone union is usually necessary to completely resolve infection.

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37

Neurodiagnostic Overview for the Small Animal Surgeon

As the complexity of neurosurgical cases seen by surgeons and neurologists has increased, so has the complexity of diagnostic procedures and treatments. Many of the diagnostic procedures discussed in this chapter will be performed and/or interpreted primarily by neurologists and radiologists. Neurodiagnostic procedures include, but are not limited to, radiography, contrast radiography, ultrasonography, computed tomography (CT), magnetic resonance imaging (MRI), electrodiagnostic procedures (e.g., electromyography, nerve conduction studies), cerebrospinal fluid (CSF) examination, stereotactic brain biopsy, and nerve/muscle biopsy. Whereas a superficial understanding of more commonly performed electrodiagnostic procedures is more than sufficient for a surgeon's purposes, the same cannot be said about advanced imaging. In particular, it is imperative for the small animal surgeon who intends to maintain a foothold in neurosurgery to have at least a working knowledge of basic MRI physics and some experience in reading magnetic resonance images (see Chapter 14). Contrast radiography is almost completely outdated, with most private specialty and academic specialty hospitals preferentially using MRI for both brain and spinal cord disorders. The purpose of this chapter is to provide small animal surgeons with the fundamental principles of various neurodiagnostic modalities. The modalities of CT and MRI are not discussed in this chapter; they are covered in detail in Chapter 14.

RADIOGRAPHY AND CONTRAST RADIOGRAPHY

Indications

As noted previously, the roles of radiography and contrast radiography in modern veterinary neuroimaging have diminished tremendously coincident with ready availability of MRI. High-quality radiographs of the skull and vertebrae typically require sedation or general anesthesia and are generally low yield in comparison with CT and MRI. Despite this, certain disorders that cause bone lysis and/or production (e.g., skull tumors, diskospondylitis) will usually be apparent on survey radiographs (Fig. 37.1). In addition, survey radiographs are typically indicated in spinal trauma patients to evaluate for any obvious fractures or luxations (Fig. 37.2).

The most common contrast radiographic procedure performed in veterinary neurology is myelography, and although its use has almost disappeared in recent years, it is generally less costly and may be more readily available than CT or MRI in certain situations (e.g., emergencies). Myelography is a procedure in which spinal radiographs are obtained following injection of a radiopaque contrast agent into the subarachnoid space (Box 37.1). When used it may assist in the diagnosis of certain myelopathies where survey radiographs are normal or inconclusive despite neurologic evidence of myelopathy. Myelography can help estimate the location, extent, and severity of spinal lesions. The ability to easily and rapidly visualize the entire spinal cord is an advantage of myelography over CT and MRI. Myelography can be performed via a cisternal or lumbar tap. Cerebrospinal fluid should be collected before contrast injection because the contrast agent will change the composition of the CSF and may prevent accurate analysis for at least 3 to 5 days. Lumbar myelography often results in better image quality, even for lesions in the cervical region, and is safer than cisternal myelography. Techniques and dosages for myelography are provided in Box 37.1.

Complications and Contraindications

Despite the positive aspects of myelography, it is an invasive procedure associated with a low level of inherent risk (Box 37.2). Postmyelographic seizures have been reported to occur in up to 20% of dogs; this adverse event is more likely to occur in dogs larger than 20 kg and is more likely after cisternal versus lumbar contrast injections. The likelihood of postmyelographic seizure activity increases with increasing total volume of contrast agent injected (not the dose on a mL/kg basis). Male Doberman pinschers with caudal cervical spondylomyelopathy (i.e., "wobblers" syndrome) may also be particularly predisposed to postmyelographic seizure activity. Most patients that seizure will do so only once or twice in the 24 hours following the procedure, and seizures usually cease with intravenous (IV) diazepam injection (see Box 37.1). Administering a single IV dose of levetiracetam (20 mg/kg over 5 minutes) during anesthetic recovery appears to reduce the incidence of postmyelographic seizures. Maintaining slight elevation of the patient's head during and after the procedure (until the patient is awake) and ensuring hydration with IV fluids during and 24 hours after myelography will limit the occurrence and severity of postmyelographic seizures.

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ABSTRACT

It is important for neurosurgeons to be familiar with a number of diagnostic modalities used in clinical neurology, including computed tomography (CT) and magnetic resonance imaging (MRI). Radiography is still used as a screening tool for several neurologic disorders; however, the use of contrast radiography has been declining with the increasing availability of MRI and CT. Nonetheless, it is still warranted in certain situations. Surgeons should be familiar with the basics of electrodiagnostic testing primarily so that they will have an understanding of what tests may be indicated for certain neurologic conditions. Familiarity with techniques for cerebrospinal fluid collection and the ability to interpret the results are also important.

KEYWORDS

Radiography
contrast radiography
myelography
electrodiagnostics
cerebrospinal fluid analysis

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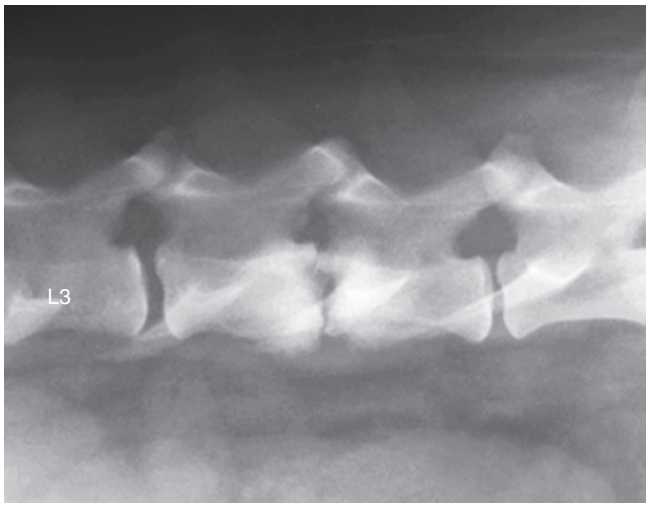


FIG. 37.1 Lateral thoracolumbar radiograph of a dog with advanced diskospondylitis.



FIG. 37.2 Lateral cervical radiograph of a dog with a comminuted cervical vertebral fracture.

BOX 37.1 Techniques and Dosages for Myelography

- Obtain cerebrospinal fluid for analysis before injecting contrast.
- Use a nonionic, iodinated, water-soluble contrast agent (e.g., iohexol, iopamidol).
- Administer a test injection (e.g., 0.5–1.0 mL, depending on patient size) initially to ensure that the contrast is in the subarachnoid space, before administering the remainder of the calculated contrast dose.
- For a regional study (e.g., cervical myelogram with cisternal contrast injection), inject 0.3 mL/kg body weight.
- For full studies (e.g., cervical myelogram with lumbar injection), inject 0.45 mL/kg body weight.
- Inject the contrast slowly—approximately 2–3 mL/min.
- Maintain slight elevation of the patient's head during and after the procedure (until the patient is awake), and ensure hydration with IV fluids during and for 24 hours after myelography.
- If seizures occur, administer IV diazepam injection (0.2–0.4 mg/kg).
- Administration of a single IV dose of levetiracetam (20 mg/kg over 5 min) during anesthetic recovery may reduce the incidence of postmyelographic seizures.
- Observe patient closely for the first 24 h following the procedure.

BOX 37.2 Contraindications and Potential Complications of Myelography

- Contraindicated in patients with:
  - Suspected inflammatory central nervous system disease
  - Elevated intracranial pressure
  - Historical or clinical indication of an underlying encephalopathy
- Potential complications
  - Seizures
  - Parenchymal damage from insertion of the needle
  - Transient neurologic worsening
  - Permanent neurologic worsening due to inadvertent contrast injection into the parenchyma or the central canal of the spinal cord

NOTE Whereas large-breed dogs with cervical lesions that have large volumes of contrast injected into the cerebellomedullary cistern may be particularly at risk for postmyelographic seizures, you should closely observe all dogs undergoing myelography (e.g., in an intensive care unit) for the first 24 hours following the procedure.

Other potential complications associated with myelography include parenchymal damage from insertion of the needle. Although this is rare, it may occur, especially in the cervical region. Worsened neurologic status post myelogram is usually caused by transient chemical myelitis secondary to contrast injection. The risk for this may be higher in patients with preexisting inflammatory disease or chronic spinal cord compression (e.g., chronic type II disk disease). The risk of transient neurologic worsening appears to be highest in dogs with caudal cervical spondylomyelopathy. These dogs typically regain premyelogram neurologic status within 72 hours. Inadvertent contrast injection into the parenchyma or the central canal of the spinal cord may cause worsened neurologic status. In most cases, patients recover from this iatrogenic trauma, but permanent deficits may remain in a small number of animals.

NOTE Myelography is contraindicated for patients with suspected inflammatory central nervous system (CNS) disease because it may cause worsened neurologic status. Myelography is also contraindicated in patients with possible elevated intracranial pressure (ICP). Because cervical hyperesthesia is occasionally associated with forebrain lesions, historical or clinical indication of an underlying encephalopathy should prompt consideration of an alternative imaging modality (e.g., CT, MRI).

Anatomy and Evaluation of Studies

Four basic myelographic patterns have been identified: normal, extradural, intradural/extramedullary, and intramedullary (Fig. 37.3). Normally, the contrast columns parallel each other and conform to the vertebral canal, except at the cauda equina region where the subarachnoid space tapers. The spinal cord ends at approximately the L6 vertebral region in most dogs and at the first sacral vertebral region in most cats, although much variation has been noted between breeds.

The spinal cord is normally wider at the cervical and lumbosacral intumescences. The ventral subarachnoid space is often

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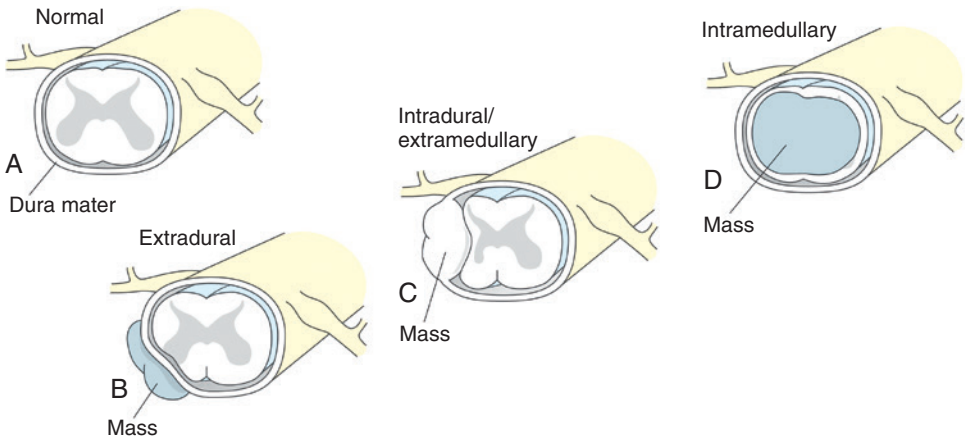


FIG. 37.3 Schematic illustrations demonstrating typical myelographic patterns for (A) normal, (B) extradural, (C) intradural/extramedullary, and (D) intramedullary lesions.

less prominent than the dorsal subarachnoid space in the thoracolumbar region in dogs. The dorsal subarachnoid space in the atlantoaxial region is often wider than the remainder of the spinal cord. The cervical spinal cord region in cats often appears wider on myelography compared to dogs. A normal myelographic pattern may be found in patients with degenerative myelopathy, fibrocartilaginous embolic myelopathy, and inflammatory myelopathies.

Extradural Pattern

Intervertebral disk extrusion/protrusion is the most common cause of an extradural myelographic pattern. Other causes of extradural patterns include vertebral fracture/luxation, congenital vertebral anomalies, hypertrophied soft tissue structures (e.g., interarcuate ligament, synovial membranes), extradural hemorrhage, vertebral neoplasia, and soft tissue neoplasia (e.g., feline lymphosarcoma). The nature of an extradural compression is best appreciated when viewed tangential to the direction of the cord deviation. For example, if a disk extrusion is compressing the cord from ventral to dorsal with no lateralizing component, the myelographic pattern as viewed from a ventrodorsal view (parallel with direction of compression) could be misinterpreted as intramedullary. With intervertebral disk extrusions, which are often ventrolateral (i.e., ventral but somewhat lateralized), it is usually helpful to obtain oblique views in addition to standard dorsal and ventral views to ascertain the correct side of disk extrusion for purposes of surgical planning. The accuracy of correctly identifying the side of disk extrusion is thought to be higher for oblique versus ventral views, but the accuracy is best when both views are obtained. Combining myelography with CT (CT/myelogram) is much more likely to provide accurate localization of disk extrusions than either CT or myelography alone.[1] Although CT alone is more likely to provide the correct side of a lateralized disk extrusion/protrusion than myelography alone, the author prefers to combine the two modalities if MRI is not an option. In dogs with more than one calcified disk extrusion/protrusion on CT imaging, it is common for only one site to be the active extrusion site; the others being old and inactive protrusions. In these situations, myelographic contrast best demonstrates the extent of spinal cord swelling (delineating which disk site is the active extrusion), whereas the CT provides better evidence (than myelography) of lateralization of the extrusion. MRI was superior to myelography for correctly identifying recurrent disk extrusions in dogs in one study.[2]

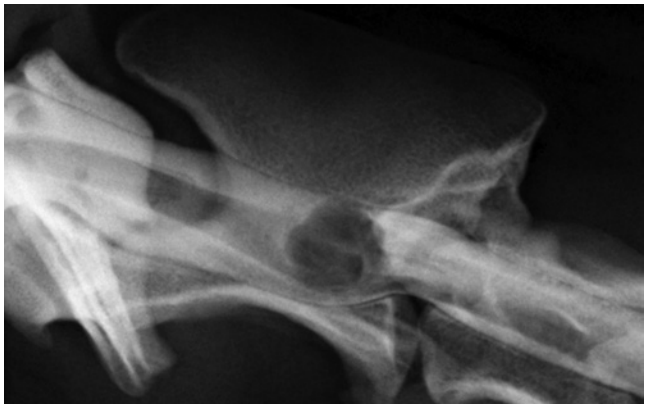


FIG. 37.4 Lateral myelographic appearance of a thoracolumbar lesion with a "golf tee sign," indicative of an intradural/extramedullary lesion.

Intradural/Extramedullary

An intradural/extramedullary pattern is produced when a lesion within the subarachnoid space (intradural) is not invading the parenchyma of the cord (extramedullary). As the contrast flows around the obstructive lesion, it may be outlined, appearing as a "filling defect." Sometimes, the filling defect is incompletely outlined and resembles a golf tee, hence the term *golf tee sign* (Fig. 37.4). Intradural/extramedullary patterns are most often associated with neoplasia, primarily meningiomas and nerve sheath tumors. Intradural hemorrhage may rarely lead to this myelographic pattern. Intradural/extramedullary lesions may produce enough spinal cord swelling that contrast is excluded from the region of the mass. In such cases, the myelographic pattern may appear to be intramedullary; a CT is often performed through the abnormal region because contrast is better visualized on CT images.

Intramedullary

An intramedullary pattern is typically associated with spinal cord edema, expansile parenchymal masses, or intraparenchymal

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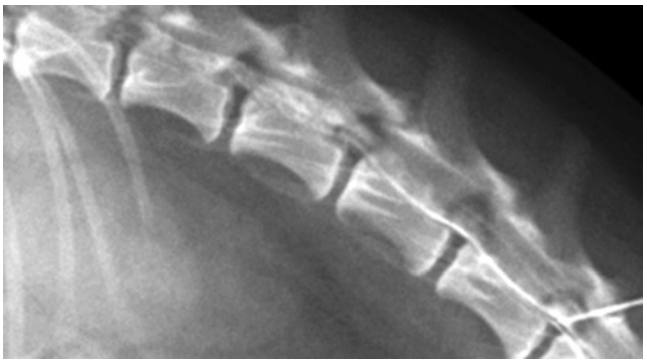


FIG. 37.5 Lateral myelographic view of a dog with myelomalacia. Note the mixing of the contrast agent with the spinal cord parenchyma.

hemorrhage. Differential diagnoses include fibrocartilaginous emboli, neoplasia (e.g., astrocytoma, lymphosarcoma), inflammatory disorders (e.g., granulomatous meningoencephalitis in dogs, feline infectious peritonitis [FIP] in cats), and trauma (e.g., hemorrhage, edema). In addition to apparent spinal cord swelling on myelographic images, contrast leakage into the spinal cord parenchyma may be appreciated in cases of spinal cord myelomalacia (Fig. 37.5).

Epidurography and diskography are infrequently performed contrast radiographic procedures used to evaluate the cauda equina region. *Epidurography* involves injection of contrast agent into the epidural space at the L7-S1 space, the sacrocaudal junction, or between coccygeal (caudal) vertebrae. *Diskography* entails injection of contrast agent directly into the L7-S1 disk, something that can be done only if the disk is degenerate. A combination diskogram/epidurogram can also be performed.

To perform a combination diskogram/epidurogram, inject the disk first, take radiographs, then withdraw the needle from the disk so that the needle tip is in the epidural space. Inject contrast again and obtain additional radiographs.

ELECTRODIAGNOSTICS

Electrodiagnostic examinations take advantage of the body's electrical properties to help characterize neurologic disorders. These tests require specialized instrumentation and require that individuals be trained in performing and interpreting the tests. Electrodiagnostic testing in dogs and cats has declined substantially, coincident with advances in neuroimaging. Despite the plethora of electrodiagnostic procedures available, only a handful of these tests are done on a regular basis. In clinical practice, the most frequently performed electrodiagnostic procedures are electromyography (EMG) and motor nerve conduction velocity (MNCV). Muscle/nerve biopsy is often performed after these procedures (see Chapter 44). The most common site for muscle/nerve biopsy is the region of the common peroneal nerve. Two main categories of electrical activity are measured in clinical neurology: spontaneous and evoked. Spontaneous potentials are electrical signals that are produced by the body in the absence of an externally applied stimulus. Evoked potentials are electrical impulses caused by an externally applied stimulus.

Electromyography

Electromyography is the recording of spontaneous electrical activity from muscle. *Perform this test with the patient under general anesthesia. Use a concentric needle electrode. Insert the needle electrode into muscle tissue and record muscle activity. Reposition the needle several times to sample different areas of the muscle belly and evaluate multiple muscles. Evaluate both the sound and the appearance of spontaneous muscle activity during the study (Table 37.1).*

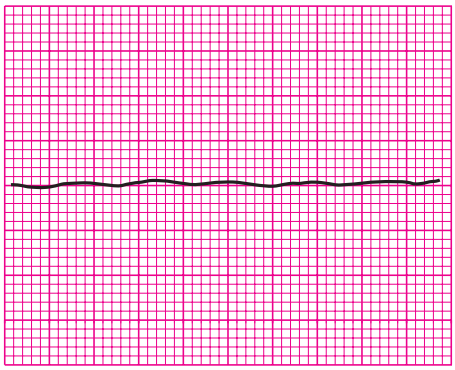


FIG. 37.6 Normal silent electromyographic tracing from a dog.

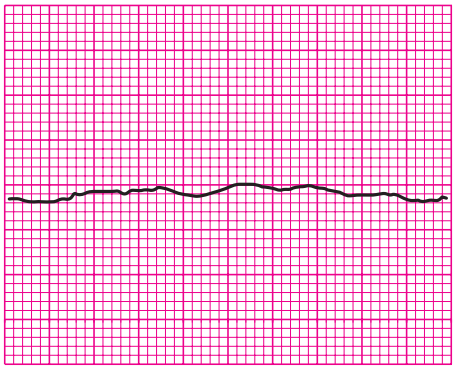


FIG. 37.7 End plate potentials recorded from a normal dog.

Abnormal muscle activity from EMG evaluation is sensitive but not specific; muscle fibers often become hyperexcitable with both denervation (due to neuropathies) and more direct damage (myopathies). Therefore abnormal EMG activity confirms the presence of a neuropathic or myopathic process but is not specific for either. Electromyographic abnormalities may not be detectable for 5 to 7 days following denervation. Not all myopathies or neuropathies are characterized by abnormal EMG activity. Muscle tissue is generally silent on EMG evaluation (Fig. 37.6) of the anesthetized patient. Small deviations from baseline (monophasic potentials) are occasionally recorded from muscles, especially near motor points (sites where major nerve trunks connect with muscle bellies). This normal activity is called *end plate noise* or *end plate potentials* and reflects small depolarizations (miniature end plate potentials) at neuromuscular junctions (Fig. 37.7). End plate noise sounds similar to small waves breaking at the seashore or the sound heard when one listens to a sea shell. Immediately after insertion of the needle electrode into a muscle belly, a short burst of electrical activity (i.e., insertional activity) typically occurs. This is due to mechanical irritation and damage to muscle fibers by the needle and is normal unless it lasts longer than 1 to 2 seconds after the needle stops moving. Replacement of muscle tissue by fat or connective tissue in chronic denervation or myopathies may lead to the absence of insertional activity.

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TABLE 37.1 Abnormal Electromyographic Activity

Terminology	Sound	Interpretation	Clinical Implications
End plate noise or end plate potentials	Small deviations from baseline (monophasic potentials). Sound like waves on the beach.	Normal; reflects small depolarizations (miniature end plate potentials) at neuromuscular junctions. More prominent at motor points—where a nerve enters a muscle group.	If absent, may be due to specific location of needle (not near a motor point). If absent near motor points, may reflect replacement of muscle tissue by fat or connective tissue because of chronic neuropathy or myopathy.
Insertional activity	Transient deviations from baseline. Positive or negative cluster of high-frequency spikes with a crisp static sound—much like the sound of a hand moving over a fully inflated balloon. Also referred to as *injury potentials.*	Normal unless it lasts longer than 1 to 2 sec after the needle stops moving. This activity is due to muscle fibers mechanically stimulated or damaged by needle insertion.	Absence of this activity may occur with replacement of muscle tissue by fat or connective tissue in chronic denervation or myopathies.
Fibrillation potentials	When single, popping noises; when occurring as a train or continuous run, like eggs or bacon frying or a heavy rain falling on a tin roof	Abnormal—occurs with denervation or myopathies	Severe or chronic neuropathy or myopathy
Positive sharp waves	When occur in a train or burst, may sound like a racecar	Abnormal—occurs with denervation or myopathies	Neuropathy or myopathy
Complex repetitive discharges	Revving motorcycle engines or airplanes flying	Abnormal—occurs with denervation or myopathies	May be from bared muscle spindles; often associated with chronicity
Myotonic discharges	"Dive-bomber" sound. Unlike complex repetitive discharges, these have both waxing and waning components.	Typically recorded following needle electrode insertion or repositioning	Myotonia (congenital or due to hyperadrenocorticism)

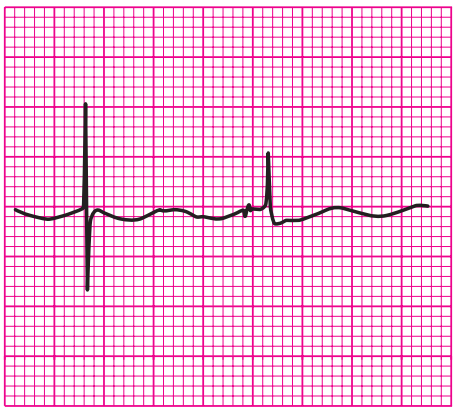


FIG. 37.8 Fibrillation potentials.

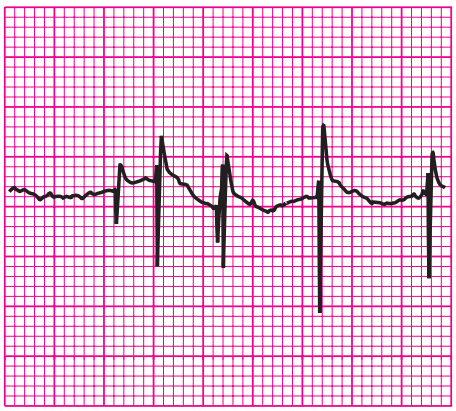


FIG. 37.9 Positive sharp waves.

In addition to prolonged insertional activity, abnormal EMG activity includes fibrillation potentials, positive sharp waves, and complex repetitive discharges. In general, all of these abnormal potentials indicate neuropathy or myopathy but are not specific for either. Fibrillation potentials are biphasic or triphasic spikes of short duration that are thought to arise from individual muscle fibers (Fig. 37.8). They sound like popping noises. When occurring as a train or a continuous run, the sound is like that of eggs or bacon frying, or a heavy rain falling on a tin roof. Fibrillation potentials are believed to represent severe or chronic disease, compared with positive sharp waves. Positive sharp waves often occur concurrently with fibrillation potentials. These potentials are of longer duration than fibrillation potentials and appear to be monophasic (Fig. 37.9). Positive sharp waves are believed to originate from individual muscle fibers, but a conduction block in the sarcolemma leads to the more prolonged potential. When positive sharp waves occur in a train or burst, it sounds like a race car driving past. Complex repetitive discharges or bizarre high-frequency potentials are synonymous catch-all terms applied to polyphasic potentials that do not appear to be fibrillation potentials or positive sharp waves. These potentials are thought to arise from bared muscle spindles and are often associated with chronicity. They tend to have constant amplitude and frequency (i.e., do not wax and wane). The sounds of these potentials are varied and have been described as revving motorcycle engines and airplanes flying. Myotonic discharges are often described as a distinct entity, rather than a subcategory of complex repetitive discharges. These are high-frequency, biphasic or

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triphasic repetitive discharges that wax and wane, producing a "dive-bomber" sound. They are typically recorded following needle electrode insertion or repositioning. Although not specific for any disorder, these discharges are most often associated with myotonia (congenital or due to hyperadrenocorticism).

Electroencephalography

Electroencephalography (EEG) refers to the recording of spontaneous electrical activity of the cerebral cortex and the interpretation of these recordings. Historically, EEG had many practical applications, including its use in localizing seizure foci. In modern veterinary neurology, the clinical utility of EEG examination is low. The likelihood of EEG examination contributing substantially to the management of a patient with an established generalized seizure disorder is negligible. EEG is fairly inaccurate for localizing focal brain abnormalities, and it provides no structural information. EEG may be helpful in cases of focal seizure disorders in which the diagnosis of the condition as a seizure disorder is sometimes equivocal. EEG examination may also be useful as a determinant of brain death in comatose patients who have been resuscitated following cardiac arrest. A high degree of subjectivity is involved in the interpretation of abnormal EEG recordings. In general, frequencies and amplitudes that appear inadequate or excessive for the physiologic conditions under which they are measured (e.g., high-voltage, slow-frequency activity in an awake patient) are indicative of brain dysfunction. Spike and spike-wave activity (Fig. 37.10) are indications of a seizure disorder.

Brainstem Auditory Evoked Response

Brainstem auditory evoked response (BAER) testing utilizes the auditory pathway for evaluation of hearing and brainstem disorders. The patient is administered auditory stimuli in the form of clicks delivered via specialized ear plugs. The resultant evoked response is measured via subcutaneous scalp electrodes arranged in specific patterns. The normal BAER consists of four or five waves that are time-locked to the sound stimulus (Fig. 37.11). These waves appear within 10 ms of the sound delivery. The BAER is not appreciably affected by sedation or anesthesia, so it can be performed on awake, sedated, or anesthetized patients. The waves of the BAER correspond to sequentially stimulated neuronal groups and white matter tracts associated with the auditory pathway. These waves represent a caudal-to-rostral chain of propagated depolarization, starting from the cochlear portion of cranial nerve VIII and ending at the caudal colliculus (midbrain) and medial geniculate (thalamus) region. The appearance and latency (time between sound stimulus and appearance of the wave) of each wave following the first wave are dependent on the integrity of neural tissue caudal to the site of generation of the specific wave, as well as on the tissue constituting the generator site for that specific wave. With congenital hearing disorders, the BAER is typically a flat line because no wave I and therefore no subsequent waves are present. Interwave intervals and left-right comparisons can be used to discern whether a brainstem lesion is present in patients with intact hearing. With brain death (e.g., cardiac arrest), the BAER may be a flat line or may have one or two initial waves, with the remaining waves being absent. Because the inner ears are very susceptible to hypoxemia, an EEG should be performed in an arrest patient with a flat BAER study to discern hypoxia-related deafness versus brain death.

Motor Nerve Conduction Velocity

MNCV studies are performed primarily in animals suspected of having neuropathies. For MNCV measurement, an electrical stimulus is applied to a nerve with subcutaneous electrodes, and the resultant depolarization of a muscle supplied by that nerve is recorded with a recording electrode. The depolarizing event is a large biphasic or triphasic muscle potential formed by action potentials of many muscle fibers from many motor units. It is typically referred to as a *compound muscle action potential (CMAP)* or an *M wave*. The latency or the time from the stimulus to onset of the M wave is measured by the computer. A minimum of two sites of a nerve must be stimulated to calculate an MNCV. The distance between the two stimulation sites (in meters) is divided by the difference in latency from stimulus artifact to M wave appearance for the two sites (in seconds) to arrive at MNCV in m/sec. The M wave is the result of orthodromic (proximal to distal) propagation of nerve depolarization.

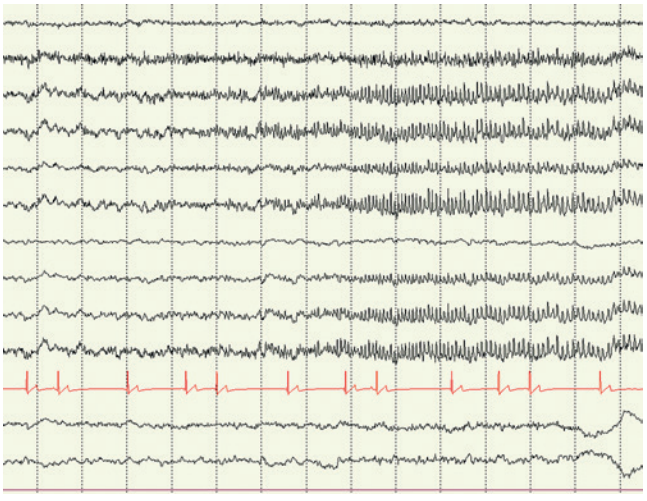


FIG. 37.10 Spike and spike-wave activity from an electroencephalographic recording of a patient with idiopathic epilepsy. (Courtesy Dr. Dan Fletcher.)

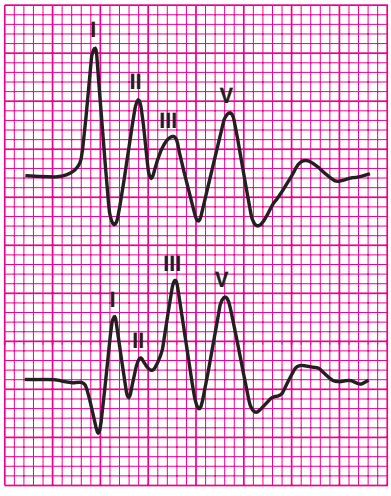


FIG. 37.11 Normal brainstem auditory evoked response recording from a cat.

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When a nerve is artificially stimulated, as in MNCV testing, it also depolarizes distally to proximally. Measurement of F waves takes advantage of this phenomenon. The depolarization that travels proximally in a stimulated nerve will cause the lower motor neurons of that nerve to depolarize. This depolarization will cause a second orthodromic (proximal-to-distal) impulse propagation and a smaller and more delayed compound muscle action potential called the *F wave.* This test has limited utility in veterinary medicine, but it can be used to assess the integrity of ventral roots in disorders like polyradiculoneuritis or brachial plexus injuries.

General anesthesia is required for MNCV measurement. If the patient has clinical evidence of a generalized disorder (e.g., suspect polyneuropathy), the sciatic nerve and its branches (i.e., peroneal or tibial nerves) may be preferable test sites. After a nerve is severed, axons distal to the severed area will continue to conduct normally for up to 4 days. The nerve is stimulated at two or three sites, and the MNCV is calculated after the distance from stimulating to recording electrodes is manually measured (Fig. 37.12). This measurement is performed using a tape measure and is the most likely source of error in this test (Box 37.3). Characteristics of MNCV are provided in Box 37.3. Small-amplitude M waves or polyphasic M waves are often indicative of neuropathy but may result from myopathies.

Sensory Nerve Conduction Velocity

Sensory nerve conduction velocity (SNCV) is less commonly performed in dogs and cats than MNCV measurement. This test is typically performed by stimulating a distal cutaneous nerve branch and measuring compound action potentials over proximal sites on the parent nerve. The technique is similar to that used for MNCV, but the depolarization events of interest are directed proximally, and compound action potentials are from axonal depolarization and therefore are of much smaller magnitude than the CMAPs or M waves recorded in MNCV studies. SNCV recording is primarily used to evaluate patients with suspected neuropathies, especially if MNCV evaluation is normal or equivocal. It is generally thought that SNCV is more sensitive to early neuropathic processes compared with MNCV.

BOX 37.3 Considerations for Motor Nerve Conduction Velocity (MNCV) Measurement in Dogs and Cats

- Using a tape measure for nerve length estimate is a potential source of error.
- Normal MNCV
  - In older patients: at least 50 m/s
  - In young to middle-aged patients: usually >60 m/s
- Decreased body temperature causes MNCV to decrease by 1.8 m/s for each degree Celsius below normal.
- Proximal nerve segments normally conduct faster than distal nerve segments.
- Demyelination is more likely to affect MNCV than is axonal loss.
- Small-amplitude or polyphasic M waves often indicate neuropathy (may result from myopathies also).

Repetitive Nerve Stimulation

The **repetitive nerve stimulation** (RNS) test measures successive CMAPs (M waves) induced by repetitive stimulation of the nerve that supplies the muscle from which the potentials are recorded. With stimulation rates of five or fewer per second, the sequential M waves should be of equal amplitude and area as the first. A decremental response of 10% or greater indicates a problem with neuromuscular transmission. Although not specific for the disease, a decrementing RNS is usually indicative of myasthenia gravis (MG) (Fig. 37.13). General anesthesia is required for RNS in dogs and cats. Stimulating the peroneal nerve at the knee or hock level and recording from a digital muscle are recommended.

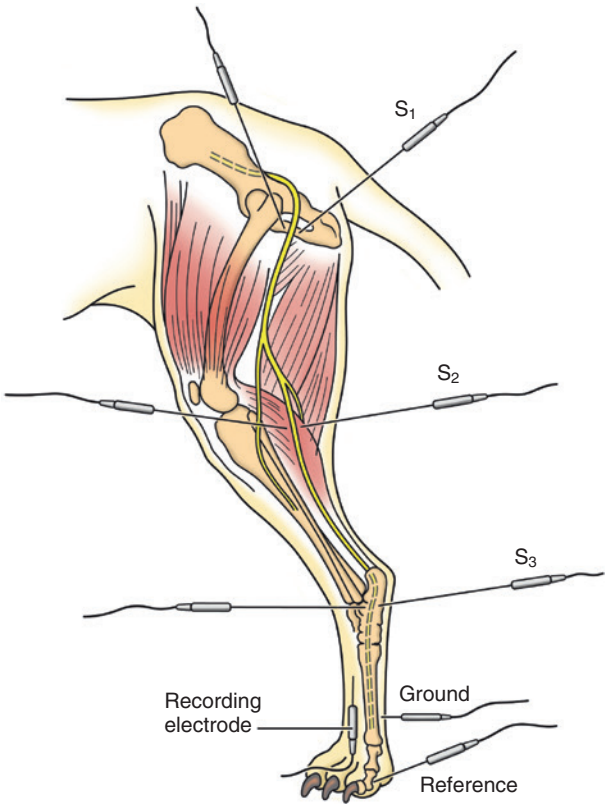


FIG. 37.12 Schematic illustration of the typical setup for motor nerve conduction velocity measurement of the sciatic and peroneal nerves.

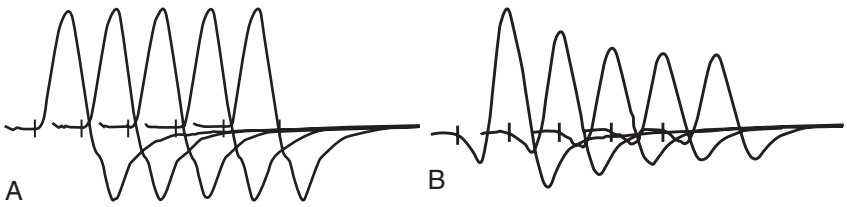


FIG. 37.13 (A) Normal repetitive nerve stimulation (RNS) result compared with (B) a decrementing RNS recording. The latter recording is from a myasthenic dog.

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The setup is the same as for MNCV, but only one stimulation site is necessary for RNS. In cases of focal MG, the limb RNS is sometimes normal. In suspect cases of focal MG with normal limb RNS, a decremental response in facial musculature (e.g., orbicularis oculi muscle) following facial nerve stimulation is often seen.

CEREBROSPINAL FLUID ANALYSIS

CSF should be collected from patients before contrast radiographic procedures and often after a CT or MRI examination. The cell count and protein level of the CSF can be thought of as the CNS analog of the complete blood count (CBC) and serum protein level for the systemic circulation, respectively. Abnormal CBC and serum protein results often assist in the diagnosis of systemic illness when viewed in the context of other laboratory abnormalities, as well as historic complaints and clinical findings. These abnormalities are typically not indicative of any specific disease when viewed as isolated test results. Similarly, results of CSF analysis often contribute to a diagnosis, but rarely by themselves provide a specific diagnosis. CSF analysis is very sensitive in that it is often abnormal in patients with neurologic disease; however, it is highly nonspecific in most cases. Collection and evaluation of CSF are indicated for encephalopathies and myelopathies for which imaging suggests an inflammatory process. For other disorders of the brain and spine (e.g., neoplasia), CSF evaluation may be helpful; however, the potential benefits of CSF collection and evaluation in such cases should be weighed against the potential risk to the patient and additional expense. In most cases of brain neoplasia, CSF collection does not represent a substantial risk, but tentorial herniation is possible, especially if the ICP is very high. Furthermore, CSF collection is not likely to add substantially to the diagnosis. If surgical removal of a well-delineated brain or spinal mass is planned, CSF evaluation is not likely to alter that plan.

To perform a CSF tap, collect 1 to 1.5 mL (approximately 10 drops) of CSF in a sterile glass tube, preferably without ethylenediaminetetraacetic acid (EDTA) (e.g., red-top tube). Collect both a red-top sample (for protein and cell concentrations and culture) and a purple-top (EDTA) sample (for cellular morphology) for analysis. Generally, you can remove 1 mL per 5 kg body weight of CSF safely at one time for analysis. EDTA may falsely elevate protein concentrations and falsely lower cell concentrations in small samples. Because EDTA is bactericidal, it may interfere with CSF culture results in cases of CNS bacterial infection; however, EDTA may help preserve cellular morphology.

CSF is most commonly obtained from the cerebellomedullary cistern (cisternal tap). CSF collected from this site may be more representative of lesions involving the brain than CSF collected from a lumbar puncture. The widely held belief that lumbar punctures are safer than cisternal punctures in cases of increased ICP is not substantiated clinically. However, if cerebellar herniation is obvious on sagittal MRI with compromise of the cerebellomedullary cistern, a lumbar puncture is preferred. Anatomic landmarks useful in performing cisternal CSF taps include the external occipital protuberance, the cranial aspect of the dorsal spine of the axis (C2 cervical vertebra), and the transverse processes ("wings") of the atlas (C1 cervical vertebra).

Cisternal Puncture

Use a noncollapsing endotracheal tube to avoid occluding airflow during the procedure (avoid red rubber endotracheal tubes because these tubes are more prone to complete kinking with neck flexion when compared with polyvinyl chloride [PVC] endotracheal tubes). Shave and aseptically prepare the skin in the region of the tap. Place the patient in lateral recumbency and have an assistant flex the neck. Keep the animal's nose parallel with the table. Have the assistant "tuck in" the animal's chin, and push the external occipital protuberance toward the individual performing the tap (Fig. 37.14). Place a form of support under the neck (e.g., rolled-up paper towel) to help keep the spine of the axis and the external occipital protuberance inline.

While wearing sterile gloves, insert a 22-gauge spinal needle with a stylet (20-gauge in larger patients) on the midline, directing it toward the occipitoatlantal space. Estimate the proper location for needle insertion by locating the cranial aspect of the C2 spine with an index finger, then press firmly with the fingertip as the finger is simultaneously advanced cranially. In most patients, you

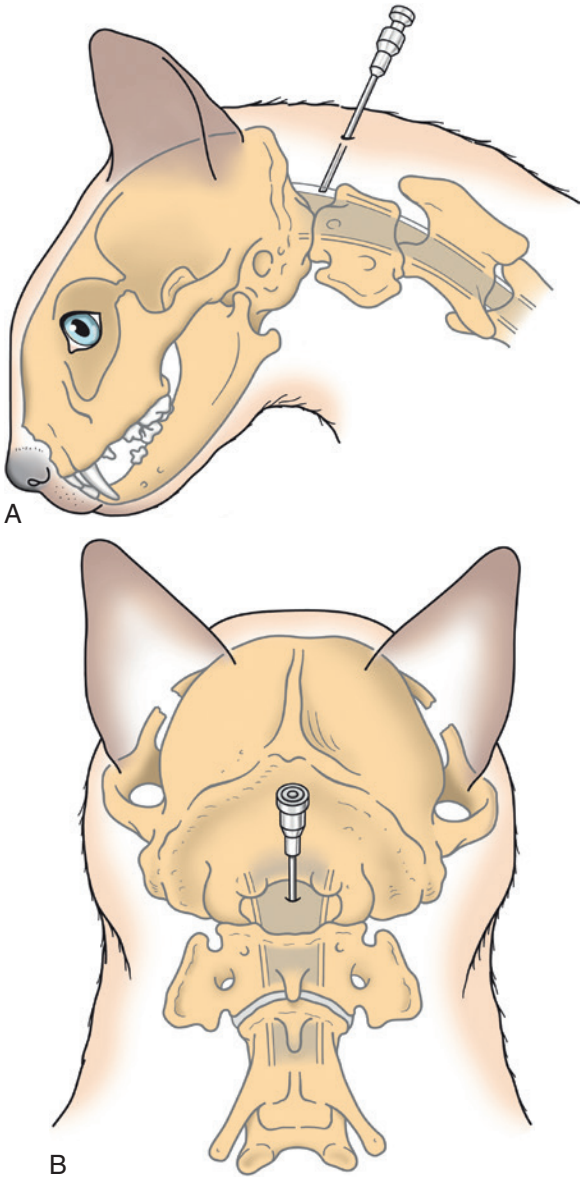


FIG. 37.14 Anatomic landmarks for cerebellomedullary cisternal cerebrospinal fluid collection in the cat. (A) Lateral view. (B) Dorsoventral view.

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will palpate a ridge or divot approximately one-third of the distance between the cranial aspect of the C2 spine and the external occipital protuberance. This ridge is the cranial aspect of the arch of C1.

Insert the needle just cranial to the ridge to allow entry into the occipitoatlantal space. Alternatively, draw an imaginary line across the cranial limits of the wings of C1 and a perpendicular line from the external occipital protuberance caudally. Insert the needle at the intersection of these lines. Puncture the skin first, then use the index finger and thumb of one hand (left hand for a right-handed person) to stabilize the needle against the skin surface as the other hand slowly advances the spinal needle. After every few millimeters of advancement, remove the stylet to observe for CSF flow. Typically, you will feel the needle pass through fibrous tissue planes, producing a "popping" sensation; however, this is not consistent. *If the needle abuts bone, redirect the tip slightly cranial or caudal into the dorsal subarachnoid space.* If the needle tip is pushed through the cord and lacerates the basilar artery, severe complications can ensue.

Lumbar Puncture

Lumbar puncture for CSF collection (lumbar tap) is usually performed at the L4-L5 space in large dogs or at the L5-L6 space in smaller dogs and cats (Fig. 37.15). Lumbar CSF may be more representative of lesions involving the thoracolumbar spinal cord than CSF from a cisternal puncture. *Place the patient in lateral recumbency and shave and aseptically prepare the skin at the puncture site for CSF collection. Advance the patient's pelvic limbs cranially to open up the interarcuate space. Face the ventral aspect of the patient, and bend over the patient to insert the spinal needle. Insert the spinal needle just lateral to midline, adjacent to the caudodorsal limit of a spinous process (L6 for L5-L6 puncture; L5 for L4-L5 puncture). Insert the needle at a 30- to 60-degree angle from an imaginary line drawn perpendicular to the long axis of the spine.* After the interarcuate space is entered, the needle will pass through the dorsal dura mater. Often at this point, a twitch of the pelvic limbs and/or tail will be noted. *Advance the needle to the floor of the vertebral canal, and withdraw the stylet. Allow CSF to drip into a collection tube.*

Although the spinal needle penetrates the spinal cord during a lumbar CSF tap, this does not appear to cause any clinical problems.

A variety of tests can be performed on CSF; the most clinically useful of these is cell count with differential and protein concentration (Box 37.4). For some diseases (e.g., canine distemper virus, FIP [coronavirus] CNS infection in cats), amplification of genetic material via polymerase chain reaction may be indicated. Ideally, cell counts should be performed within 30 minutes of CSF collection; however, recent evidence indicates that reliable cell counts may be obtained up to 48 hours later if the CSF is preserved through the addition of autologous serum.

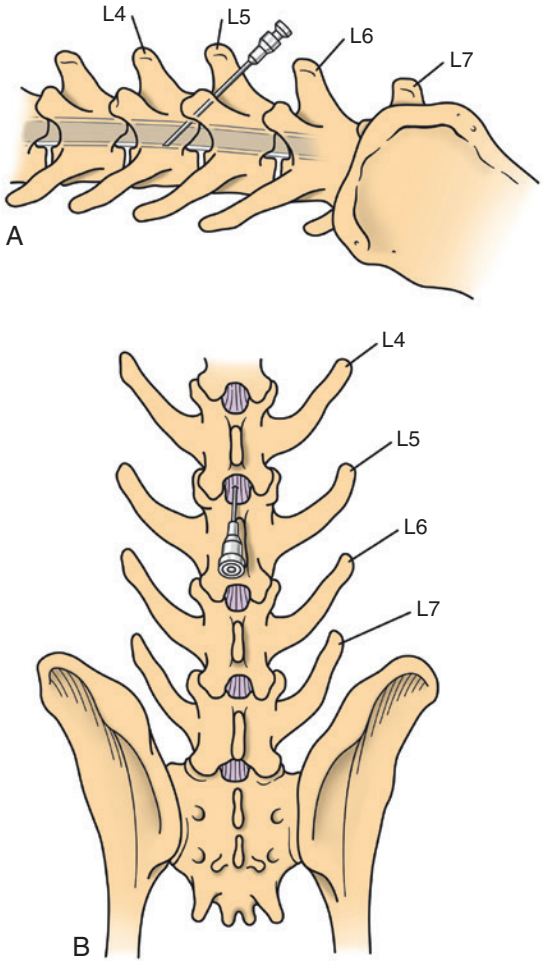


FIG. 37.15 Anatomic landmarks for obtaining cerebrospinal fluid via lumbar puncture in the dog. (A) Lateral view. (B) Dorsoventral view.

NOTE Prior treatment may alter the expected results of CSF analysis, especially in patients with inflammatory disease treated with corticosteroids.

Normal CSF is clear and colorless and has the consistency of water. Prior hemorrhage (occurring a minimum of 10 hours before CSF collection) in the CSF may result in a yellow tinge, referred to as *xanthochromia*. This discoloration can persist for 2 to 4 weeks following hemorrhage into the subarachnoid space

BOX 37.4 Interpretation Guidelines for Canine and Feline Cerebrospinal Fluid

Normal Nucleated Cell Count
<5 cells/μL

Normal Protein Concentration
<27 mg/dL cisternal tap
<45 mg/dL lumbar tap

Effect of RBC Contamination on Nucleated Cell Count
Dogs: 500 RBCs/μL = 1 WBC/μL
Cats: 100 RBCs/μL = 1 WBC/μL

Effect of RBC Contamination on Protein Concentration
1200 RBCs/μL = 1 mg/dL protein

Effects of RBCs and WBCs on CSF Turbidity
Turbidity from WBCs: >200 WBCs/μL
Turbidity from RBCs: >400 RBCs/μL

CSF, Cerebrospinal fluid; *RBC,* red blood cell; *WBC,* white blood cell.

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but is usually resolved by 4 to 8 days. Other potential causes for xanthochromia include severe icterus and markedly elevated CSF protein levels.

Gross blood contamination may be iatrogenic or may result from ongoing hemorrhage in the subarachnoid space. Iatrogenic hemorrhage is more common with lumbar taps than with cisternal taps. Although iatrogenic hemorrhage interferes with interpretation of CSF results, the extent to which it does so is controversial. It has been suggested that each 500 to 700 red blood cells (RBCs)/mL in a hemorrhagic CSF tap may account for one white blood cell (WBC)/mL in dogs, and each 100 RBCs/mL may account for one WBC/mL in cats (see Box 37.4). However, it has also been demonstrated that RBC counts in CSF as high as 15,000/mL can occur with minimal elevation of the WBC count. The effect of hemorrhage on CSF protein levels is typically low; approximately 1200 RBCs/mL is needed to increase the protein concentration by 1 mg/dL.

Increased turbidity of CSF is usually due to an elevated number of cells (more than 200 WBCs/mL, more than 400 RBCs/mL) and is occasionally due to increased protein levels (see Box 37.4). Elevated protein levels in CSF will cause the fluid to be more viscous. CSF that clots is rare and is caused by a markedly increased protein concentration.

Although the actual number may vary with the laboratory used, fewer than five nucleated cells per mL of CSF are usually present (see Box 37.4). In normal dogs and cats, lumbar CSF typically has fewer WBCs/mL than cisternal CSF. The distribution should predominantly consist of mononuclear cells with only occasional neutrophils.

Although each laboratory will establish normal ranges, normal protein concentration for cisternal CSF is less than 27 mg/dL in dogs and cats (see Box 37.4). Normal protein levels will be higher when the CSF is collected from a lumbar puncture (approximately twice that of cisternal CSF, or <45 mg/dL).

Generally, the greater the meningeal or ependymal involvement, the greater the number of WBCs expected in the CSF. Deep-seated parenchymal lesions may be associated with mildly increased or normal cell counts, often with elevated protein levels. Increased nucleated cell counts in the CSF are referred to as *pleocytosis*. A normal cell count with an elevated protein level is often referred to as *albuminocytologic dissociation*.

A neutrophilic pleocytosis is often associated with bacterial infection and corticosteroid-responsive (aseptic) meningitis. Other diseases in which neutrophils may predominate in CSF include some viral encephalitides (e.g., acute canine distemper infection, FIP meningoencephalitis in cats), fungal infections, meningiomas, and fibrocartilaginous embolic myelopathy. Neutrophils associated with infectious disease (e.g., bacterial meningoencephalitis) are more likely to degenerate than those that occur in noninfectious (e.g., corticosteroid-responsive meningitis) disorders.

Mononuclear cell pleocytosis refers to a predominance of lymphocytes or macrophages in the CSF. This is the most common pleocytosis encountered and is usually associated with granulomatous meningoencephalitis in dogs (see p. 1453). The necrotizing encephalitides (i.e., pug/Maltese encephalitis, Yorkshire terrier encephalitis) are usually characterized by primarily lymphocytic pleocytosis. Lymphosarcoma involving the CNS may also be associated with a lymphocytic pleocytosis. A predominantly mononuclear pleocytosis can be caused by fungal, viral (e.g., canine distemper virus), protozoal, rickettsial, and chronic bacterial infections.

Eosinophilic pleocytosis is rare. It has been associated with aberrant parasite migration in the CNS, rabies virus, and cryptococcal, protozoal, and protothecal infections. A rare idiopathic condition called *eosinophilic meningoencephalitis* is characterized by a substantial proportion of eosinophils in the CSF.

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38 The Neurologic Examination and Relevant Neuroanatomy

Performing and interpreting a neurologic examination in dogs and cats requires a working knowledge of functional and dysfunctional neuroanatomy; an in-depth understanding of neuroanatomy is not necessary for proficiency in localizing their neurologic lesions. A neurologic examination is performed in a systematic fashion and can, in most cases, be done within 10 to 15 minutes. The two important questions to be answered by the neurologic examination are (1) Does the patient have neurologic disease? and (2) If so, where is the lesion? A number of principles should be followed when localizing lesions in small animals. Although fairly specific neurologic localization should be attempted, all lesions will be in one of three general locations: brain, spinal cord, or peripheral nervous system. The anatomic subdivisions within each of these locations are provided in Box 38.1. The order chosen for the various tests performed during the neurologic examination is not important, but the order of the examination should be consistent so that nothing is forgotten. Manipulations that may result in a painful patient response should be done at the end of the neurologic examination. The neurologic examination may need to be modified in certain situations (e.g., hemiwalking would not be advised in a suspected thoracolumbar fracture/luxation patient). Performing abbreviated neurologic examinations or "quick looks" should be avoided; the responsibility for incorrect localization of a lesion will fall upon the examiner, and having performed an incomplete neurologic examination will not be a viable excuse for an error. Ambulatory patients should be allowed to navigate the examination room before the "hands on" part of the neurologic examination. This allows the surgeon to evaluate for circling behavior, visual deficits (e.g., bumping into objects), and gait abnormalities. Palpation of the entire animal is recommended to evaluate for areas of muscle atrophy, particularly in thick-haired patients in which atrophy is less visible. The components of a standard neurologic examination include evaluation of mental status, cranial nerves, attitude/posture, gait, postural reactions (proprioception), spinal reflexes, and nociception (normal and abnormal pain responses).

MENTAL STATUS

A patient's mental status reflects the integrity of the cerebrum, as well as parts of the brainstem (ascending reticular activating system). Anatomically, the diencephalon is the rostralmost aspect of the brainstem. Because diencephalic lesions cause clinical signs that are often indistinguishable from cerebral lesions, the term *forebrain* (cerebrum/diencephalon) is often used clinically to distinguish this part of the brain from the remainder of the brainstem. The major subdivisions of the brain are displayed graphically in Fig. 38.1. In dogs and cats with altered mentation, it is sometimes necessary to describe both the quality and the content of mental status. The quality of mentation refers to the level of alertness; this ranges from a normal level of alertness to coma. Between these two extremes are obtundation and stupor (Table 38.1). *Obtunded* animals tend to appear depressed, listless, and disinterested in spontaneous activity. Although these patients often appear drowsy, they are easily aroused with a minor stimulus (vocal cues, other noises). Unlike other forms of depression (e.g., those due to metabolic disease), obtunded patients are often described as appearing "out of it." *Stupor* describes a dog or cat that is not conscious but can be aroused with a strong stimulus (e.g., toe pinch). *Coma* refers to a state of unconsciousness that persists even after the application of a strong stimulus. Although not absolute, stupor and coma are often indicative of brainstem lesions (i.e., midbrain through medulla), whereas obtunded mental status is often associated with forebrain disease (i.e., cerebrum/diencephalon). Some patients, particularly those with forebrain dysfunction, will display an alert (but abnormal) quality or level of mental status with abnormal content of consciousness (e.g., dementia associated with cognitive dysfunction). Behavioral abnormalities are separate from mental status changes, although a fine line may be present between these two assessments. Subtle behavioral changes may not be apparent to the examiner; in such situations, the owner's assessment of their pet's behavioral status is crucial.

CRANIAL NERVES

The twelve cranial nerves and their associated functions are summarized in Table 38.2. The locations of these nerves in relation to the brain subdivisions from which they originate are shown in Fig. 38.2. Cranial nerve examination is straightforward and easily performed. It is customary and appropriate for the clinician to think of each cranial nerve in terms of its region or origin in the brain because dysfunction of a cranial nerve may represent damage to the neurons in a nucleus comprising that nerve (brain lesion) or to the nerve itself. It should be kept in mind that these nerves must travel a variable distance to their respective foramina, so there are clinical situations in which a specific cranial nerve is compromised at some distance from its associated brain subdivision (e.g., cavernous sinus syndrome). The cranial nerves and their respective brain origins are as follows: cranial nerves (CNs) I and II—forebrain (cerebrum/diencephalon); CNs III and IV—midbrain (mesencephalon); CN V (motor only)—pons (ventral metencephalon); and CNs VI through XII—medulla (myelencephalon). The sensory nucleus and the tract of CN V is very extensive and is found from the midbrain rostrally to the

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ABSTRACT

All small animal surgeons should be proficient at performing and interpreting the results of a neurologic examination. Although neuroanatomy may appear to be a daunting obstacle to overcome for many clinicians, the working knowledge of functional neuroanatomy required for clinical proficiency in consistently accurate neurolocalization is not overwhelming. This chapter provides a concise and logical review of the necessary components of the neurologic examination for small animal patients, along with the relevant neuroanatomy associated with these components.

KEYWORDS

Neurologic examination
cranial nerves
proprioception
spinal reflexes

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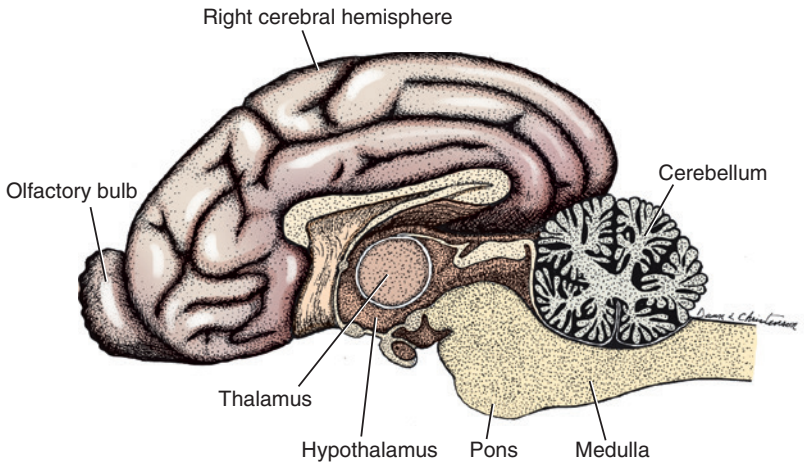


FIG. 38.1 Schematic midsagittal illustration of the brain, depicting major anatomic subdivisions.

TABLE 38.1 Altered Mental Status

Mental Status	State of Animal	Response to Stimuli	Lesion Site
Obtundation	Depressed, listless, disinterested in spontaneous activity	Can be aroused with minor stimulus	Typically forebrain
Stuporous	Not conscious but arousable	Can be aroused with strong stimulus	Typically brainstem or diffuse forebrain
Comatose	Unconscious	Unconsciousness persists even after the application of a strong stimulus	Brainstem or diffuse forebrain

BOX 38.1 Anatomic Subdivisions of the Nervous System

Brain
Forebrain (cerebrum/diencephalon)
Brainstem (midbrain through medulla)
Cerebellum

Spinal Cord
C1-C5 segments
C6-T2 segments
T3-L3 segments
L4-caudal segments

Peripheral Nervous System
Nerves
Muscles
Neuromuscular junctions

first few cervical spinal cord segments caudally. CNs IX, X, and XI act in combination to perform the same basic functions (laryngeal, pharyngeal, and esophageal innervation), so they can be thought of collectively as one cranial nerve.

CN I or the *olfactory nerve* is routinely ignored because it is difficult to assess the modality of smell. CN II or the *optic nerve* conveys the sensory modality of vision. Visual ability can be assessed in a number of ways. Probably the most common test performed for evaluation of vision is the menace response. This is a learned response (should be present by 3 months of age in dogs and cats) and therefore is not a reflex. When a menacing gesture is made toward the patient with the examiner's hand, the appropriate response is for the patient to blink (Fig. 38.3 and Video 38.1). The afferent part of this response is conducted by CN II, and the efferent part (the blink) is accomplished via the facial nerve (CN VII). Visual following is somewhat objective, especially with cats. In this test, objects (such as cotton balls or toys) are thrown or dropped in front of the patient; the normal expectation, if vision is intact, is for the patient's eyes and head to follow the objects. CN III or the *oculomotor nerve* has two major components: somatic motor and parasympathetic. The somatic motor portion of CN III innervates most of the extraocular musculature: dorsal rectus, medial rectus, ventral rectus, ventral oblique, and levator palpebrae superioris muscles. The parasympathetic component of CN III is responsible for pupillary constriction. The visual and pupillary constriction pathways are illustrated in Fig. 38.4. Damage to the oculomotor nucleus or CN III may lead to ventrolateral strabismus and/or a dilated pupil that is variably (depending on extent of the damage) responsive to light. Evaluation of the parasympathetic aspect of CN III function/dysfunction is done by shining a bright light into the eye and observing for pupillary constriction. Both pupils should constrict when a light is shone into one eye, with the eye being stimulated (direct) having stronger constriction than the opposite (indirect) eye. In this reflex, called the *pupillary light reflex*, the afferent information is carried via CN II, and the efferent response is mediated via CN III. It is important to use a strong light source when attempting to elicit a pupillary light reflex in a patient. Dogs and cats in a hospital surrounding often have high sympathetic tone, and using a weak light source on these patients may not be sufficient to effect pupillary constriction. The trochlear nucleus and its associated nerve, the *trochlear nerve* (CN IV), are responsible for innervation of the dorsal oblique muscle of the eye. This muscle prevents the outward (lateral) rotation of the dorsal aspect of the globe. Isolated deficits of this nucleus and nerve are rarely

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TABLE 38.2 Cranial Nerves and Their Associated Functions

Cranial Nerve (CN)	Function/Innervation
CN I	Olfaction
CN II	Vision
CN III	• Somatic motor to most of the extraocular muscles (dorsal, medial, ventral rectus; ventral oblique; levator palpebrae superioris) • Parasympathetic innervation to pupil (pupillary light response)
CN IV	Somatic motor to dorsal oblique muscle of the eye
CN V	• Somatic motor to muscles of mastication • Somatic motor to tensor tympani muscle • Sensory to most of face
CN VI	Somatic motor to lateral rectus and retractor bulbi muscles (extraocular)
CN VII	• Somatic motor to muscles of facial expression • Somatic motor to stapedius muscle • Parasympathetic innervation to salivary glands (mandibular, sublingual)[a] and lacrimal, palatine, and nasal glands[b] • Sensory to inner pinna • Sensory (mechanoreception, thermal) and taste to rostral two-thirds of tongue (chorda tympani nerve)[c]
CN VIII	Vestibular function and hearing
CN IX-XI	• Somatic motor for laryngeal and pharyngeal function (nucleus ambiguus) • Parasympathetic innervation to salivary glands (parotid and zygomatic—CN IX)[d] • Parasympathetic innervation of viscera (CN X) • Sensory innervation of pharynx (CN IX and X) • Sensory and taste to caudal one-third of tongue (CN IX)
CN XII	Somatic motor to extrinsic and intrinsic tongue muscles

[a]Postganglionic axon in CN V, mandibular branch (after mandibular and sublingual ganglia).
[b]Postganglionic axon in CN V, maxillary branch (after pterygopalatine ganglion).
[c]Chorda tympani nerve joins lingual branch of mandibular branch of CN V near middle ear.
[d]Postganglionic axon in CN V, mandibular branch (after otic ganglion).

encountered in small animals; this CN is typically ignored in the cranial nerve examination of dogs and cats.

The motor nucleus of CN V in the pons and its associated efferent axons in CN V, the *trigeminal nerve,* are responsible for motor innervation of the muscles of mastication. Clinical evaluation of this CN is based primarily on visual and/or palpable evidence of masticatory muscle atrophy (Fig. 38.5). With bilateral dysfunction, the patient may not be able to close the mouth effectively. As mentioned, the sensory nucleus and tract of CN V is quite extensive. This is evaluated by checking for sensation around the face (Fig. 38.6 and Video 38.2). In addition, performing the corneal reflex evaluates the integrity of CNs V (sensory), VI, and VII (Fig. 38.7 and Video 38.3). The surface of the cornea is innervated by the ophthalmic branch of CN V. When the cornea is lightly touched with a moistened cotton applicator, the normal reflex is a blink (CN VII) and retraction of the globe (CN VI). CN VI, the *abducent nerve,* is derived from the abducent nucleus in the rostral medulla and provides somatic motor innervation to the retractor bulbi and lateral rectus muscles. Damage to this cranial nerve will impair the ability of the eyeball to retract (as in the corneal reflex) as well as to move laterally (when checking for physiologic nystagmus). CN VII, the *facial nerve,* innervates the muscles of facial expression and is derived from the facial nucleus in the medulla. This nerve is evaluated clinically by assessing for facial symmetry and performing the blink reflex, corneal reflex, and trigeminofacial reflex (Fig. 38.8). This latter reflex involves stimulating the maxillary region (CN V) and observing for a blink (CN VII). The facial nerve also supplies sensory innervation to the inner pinna, so this region can be lightly stimulated (e.g., hemostat tips) and the patient observed for a behavioral response.

CN VIII is the *vestibulocochlear nerve,* which carries the sensory modalities of hearing and balance. It is difficult to accurately test hearing during a neurologic examination; animals with normal hearing and normal mentation will turn to acknowledge loud or sudden noises. If clinically indicated, hearing is evaluated using an electrodiagnostic procedure called a *brainstem auditory evoked response test* (see p. 1318). The integrity of the vestibular system can be evaluated in several ways. A patient with normal vestibular function should not display a head tilt, ataxia, strabismus, or spontaneous (resting) nystagmus. Turning a patient's head to the side should induce a normal physiologic nystagmus directed horizontally with a fast phase in the direction of the head rotation. Dogs and cats are moved in various positions in an effort to elicit abnormal (positional) strabismus and nystagmus, both of which would indicate an abnormality with the vestibular system. Abnormalities of the vestibular system may be due to damage to the peripheral portion (inner ear) or the central portion (medulla and/or cerebellum) of this system (Fig. 38.9).

CNs IX *(glossopharyngeal nerve),* X *(vagus nerve),* and XI *(accessory nerve)* are considered together because they act as a group to innervate striated muscles of the larynx, pharynx, and esophagus. The cell bodies of these CNs are located in the nucleus ambiguus found in the ventrolateral medulla (Fig. 38.10). Laryngeal function cannot be evaluated in the awake patient, but dysfunction can be inferred from inspiratory stridor in dogs with bilateral laryngeal dysfunction. The ability to swallow normally can be deduced by performing the swallow or gag reflex. This test can be performed in one of two ways (Video 38.4). The first involves direct stimulation of the pharyngeal mucosa with a cotton applicator or a gloved finger (Fig. 38.11). The disadvantage of this method is that it is applicable only to

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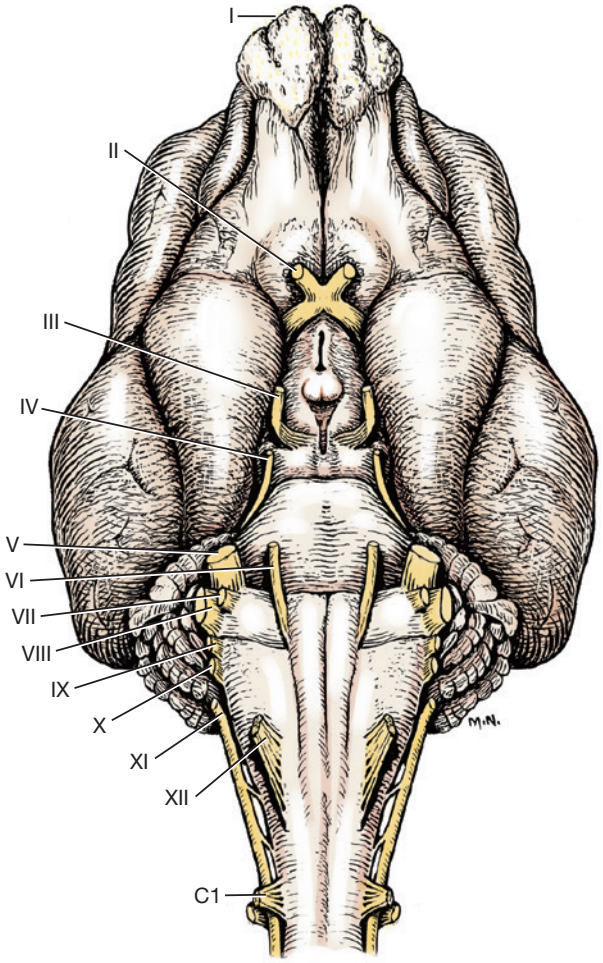


FIG. 38.2 Ventral aspect of the feline brain, showing relative anatomic positions of the cranial nerves.

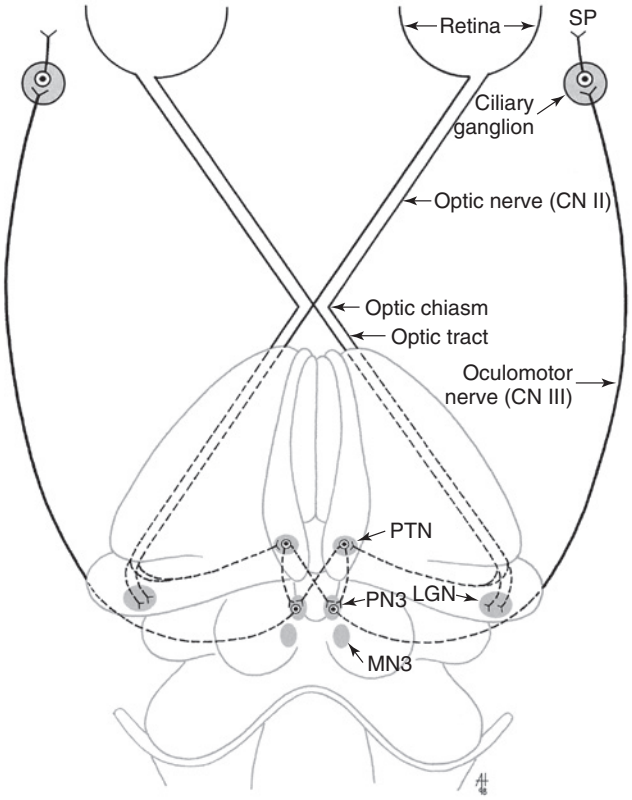


FIG. 38.4 Neuroanatomic pathways for vision and papillary constriction. *LGN,* Lateral geniculate nucleus; *MN3,* motor nucleus of cranial nerve III; *PN3,* parasympathetic nucleus of cranial nerve III; *PTN,* pretectal nucleus; *SP,* sphincter pupillae muscle. (Modified from Dewey CW. *A Practical Guide to Canine and Feline Neurology*. 2nd ed. Hoboken, NJ: Wiley-Blackwell; 2008.)



FIG. 38.3 The menace response is elicited by making a threatening gesture at the eye, which should induce a blink.

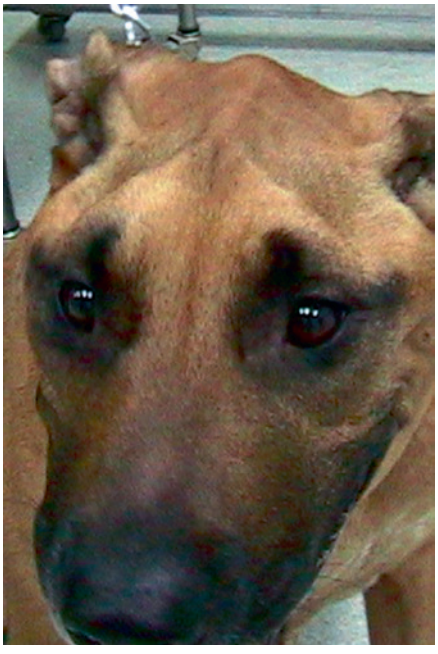


FIG. 38.5 Dog with severe masticatory muscle atrophy.

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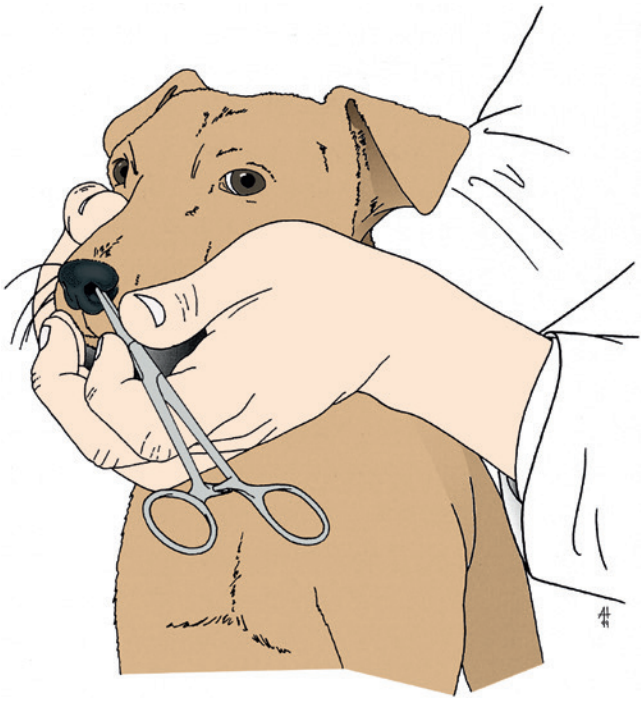


FIG. 38.6 The sensory portion of the trigeminal nerve (cranial nerve [CN] V) is tested by stimulating the nasal mucosa with a blunt instrument. Normal patients will pull their head away. (Modified from Dewey CW. *A Practical Guide to Canine and Feline Neurology*. 2nd ed. Hoboken, NJ: Wiley-Blackwell; 2008.)



FIG. 38.7 The corneal reflex is tested by touching the cornea with a cotton-tipped applicator moistened with saline. The normal response is retraction of the globe. (Modified from Dewey CW. *A Practical Guide to Canine and Feline Neurology*. 2nd ed. Hoboken, NJ: Wiley-Blackwell; 2008.)



FIG. 38.8 The palpebral or blink reflex is elicited by touching the skin medial or lateral to the eye. This will normally induce a blink.



FIG. 38.9 Dog with a positional ventrolateral strabismus. The patient was tentatively diagnosed with granulomatous meningoencephalomyelitis (see p. 1453).

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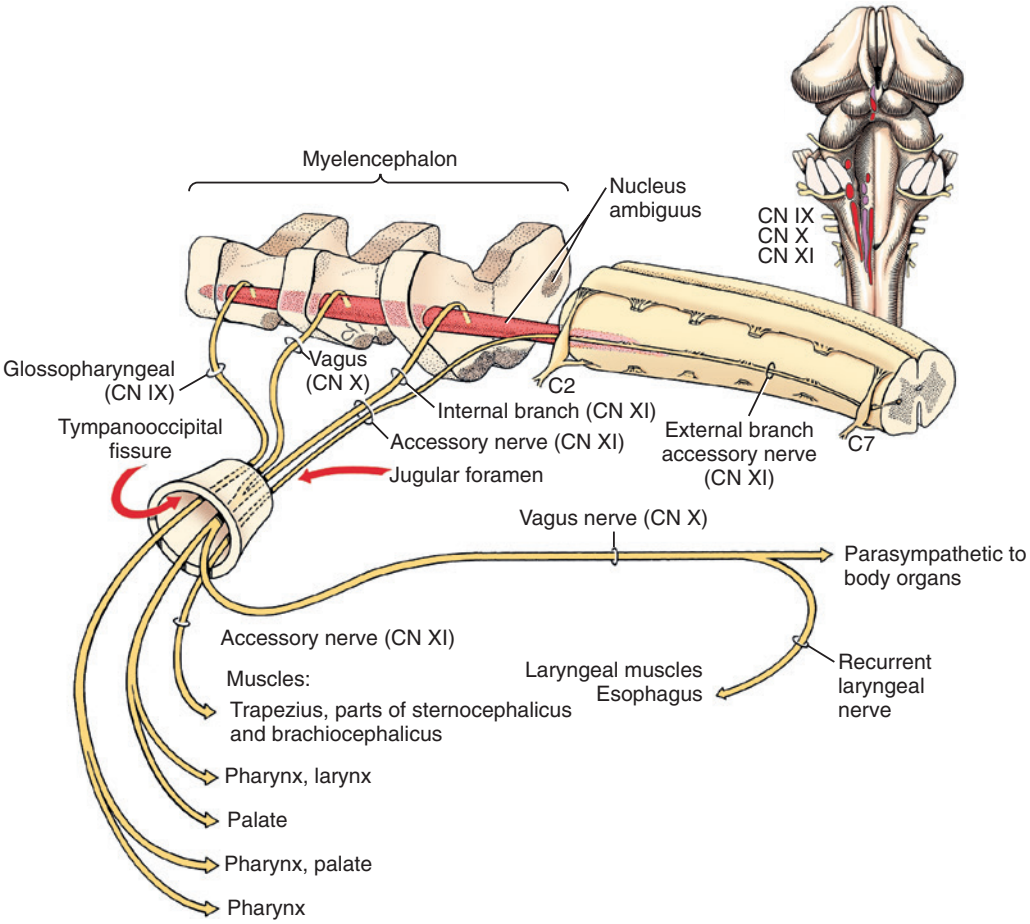


FIG. 38.10 Schematic illustration of the nucleus ambiguus and its innervations of the striated muscle of the larynx, pharynx, and esophagus. (From DeLahunta A. *Veterinary Neuroanatomy and Clinical Neurology*. 3rd ed. St. Louis: Saunders; 2009.)

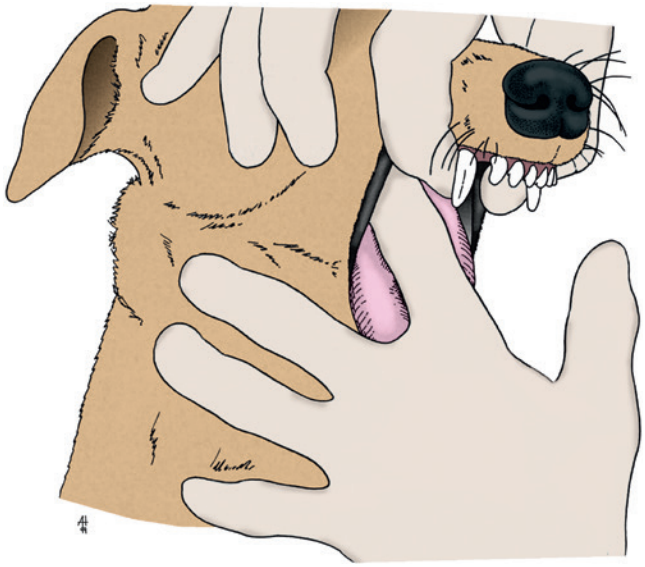


FIG. 38.11 The gag reflex is tested by touching the pharynx, which results in elevation of the palate and contraction of the pharyngeal muscles. (Modified from Dewey CW. *A Practical Guide to Canine and Feline Neurology*. 2nd ed. Hoboken, NJ: Wiley-Blackwell; 2008.)

very cooperative patients. The advantage is that the strength of the gag reflex can be directly felt (if a gloved finger is used). The other, safer (for the examiner) method, is to palpate the pharyngeal musculature externally, craniodorsal to the larynx (Fig. 38.12), and observe for a swallow from the patient. CN XII, the *hypoglossal nerve*, innervates the tongue musculature. This nerve originates from neuronal cell bodies in the hypoglossal nucleus located in the caudal medulla (Fig. 38.13). Tongue function can be evaluated by watching the patient drink water or lick his or her nose (dogs and cats often do this after the swallow/gag reflex, cats often lick their nose and swallow if the nose is rubbed by the examiner). In cooperative patients, the tongue can be grasped and directly observed for any atrophy (Fig. 38.14 and Video 38.5).

ATTITUDE POSTURE

Attitude refers to the position of the patient's eyes and head in relation to the body, whereas *posture* denotes the position of the body with respect to gravity. Abnormal attitudes are often manifested as a head tilt or a head turn. A head tilt, in which one ear is lower than the other, is a sign of unilateral vestibular dysfunction (Fig. 38.15). A head turn, in which the nose may be directed right or left but the ears are in the same horizontal plane (Fig. 38.16), is consistent with a forebrain lesion or cervical

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FIG. 38.12 External palpation of the pharyngeal musculature to elicit swallowing reflex.

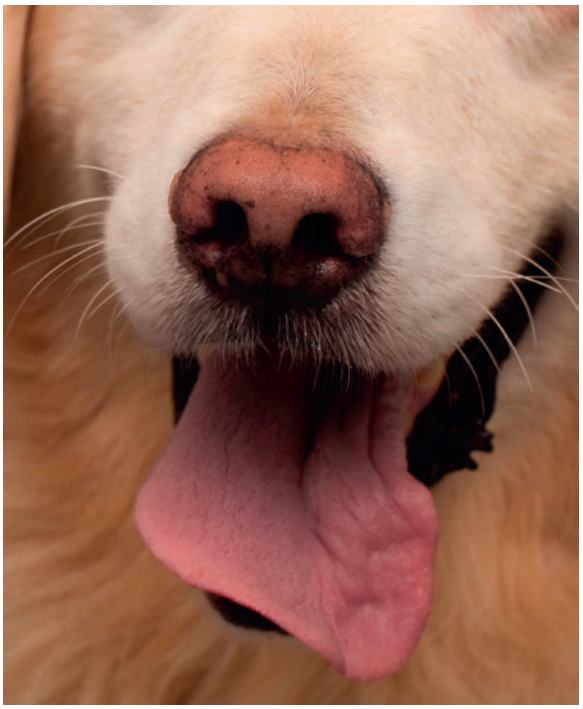


FIG. 38.14 Dog with unilateral atrophy of the tongue.

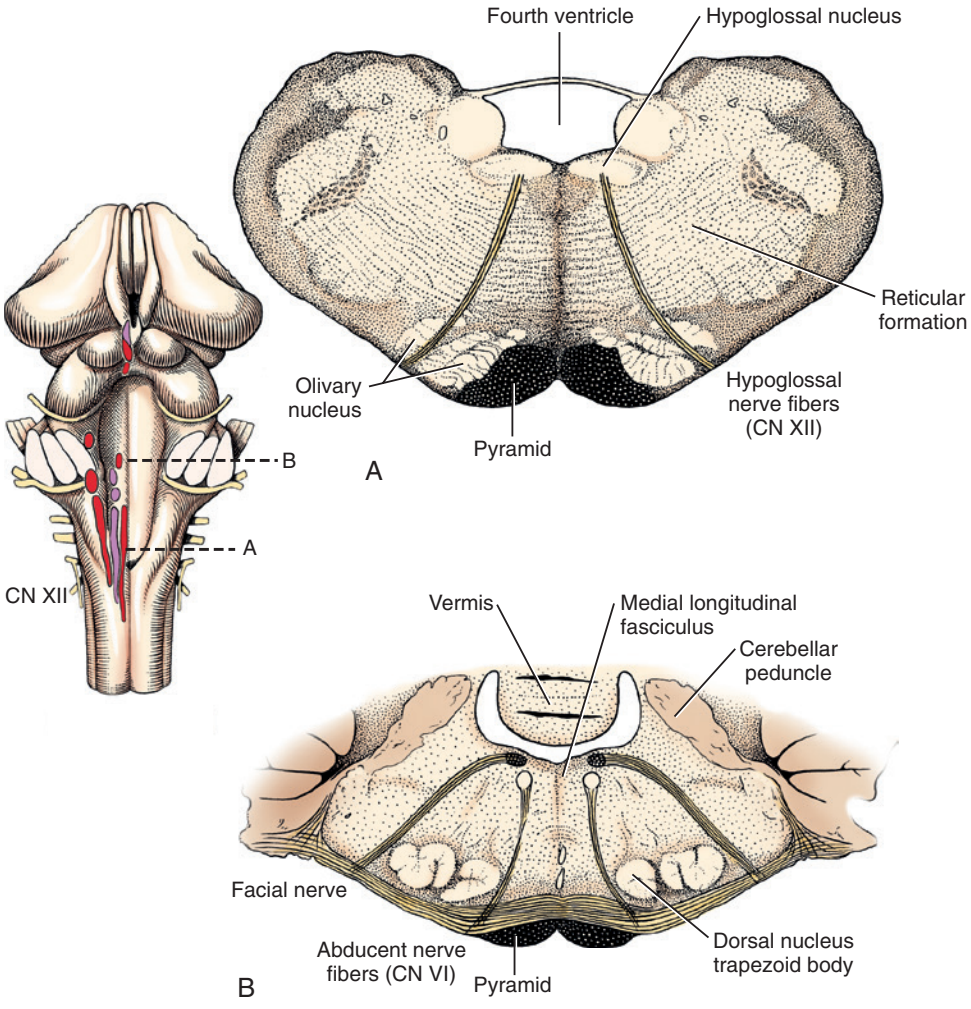


FIG. 38.13 Transverse section through the brainstem at the level of the hypoglossal nucleus and nerve in the caudal medulla. (From DeLahunta A. *Veterinary Neuroanatomy and Clinical Neurology*. 3rd ed. St. Louis: Saunders; 2009.)

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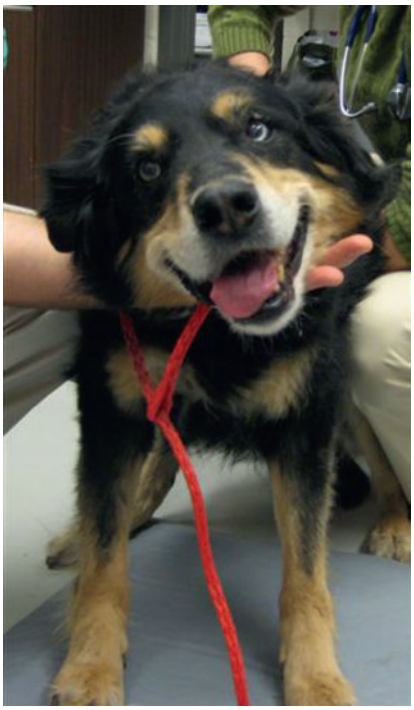


FIG. 38.15 Dog with a head tilt.



FIG. 38.16 Dog with a head turn.



FIG. 38.17 Dog with a wide-based pelvic limb stance due to cerebellar dysfunction. (Courtesy Dr. Joan Coates.)

syringomyelia (this curving of the spine is referred to as *scoliosis* or *torticollis*).

Abnormal postures are numerous and include a wide-based stance with the pelvic limbs or all four limbs (Fig. 38.17), which suggests abnormal proprioception; overflexion of the tarsal joints ("dropped" hocks) when standing, suggesting tibial nerve dysfunction; and holding up one limb ("root signature") as the result of spinal nerve irritation, among others. Some classic postures described in the literature often cause confusion, probably because they are often discussed as if they would be difficult to discern from each other. The most common of these encountered clinically is the Schiff-Sherrington posture. This posture is characterized by rigid extension of the thoracic limbs (with preservation of thoracic limb function) accompanied by pelvic limb paresis or plegia. Increased extensor tone to the thoracic limbs is due to the interruption of a group of cells in the lumbar gray matter called *border cells*, or of those cells' cranially directed axonal processes (Fig. 38.18). These cells project their axons cranially to tonically inhibit the lower motor neurons (LMNs) of thoracic limb extensor muscles. When the border cells or their axons are disrupted as the result of a spinal cord lesion caudal to the cervical intumescence region, the thoracic limb extensors are "released" from this tonic inhibition. The thoracic limbs have excessive extensor tone (especially when the patient is in lateral recumbency), but there are no associated thoracic limb neurologic deficits. Most patients with Schiff-Sherrington posture have T3-L3 myelopathies, probably because this is a very common neuroanatomic presentation, and most are nonambulatory in the pelvic limbs. This posture is an anatomic phenomenon without prognostic significance. Dogs and cats with lower lumbar spinal cord lesions can also display this posture as the result of interruption of the border cells directly (they are located in the dorsolateral ventral gray matter from L1–L7 spinal cord segments). As there is no brain involvement with this phenomenon, it should not be confused with other postures that display thoracic limb hyperextension (i.e., decerebrate and decerebellate rigidity).

Decerebrate rigidity (Fig. 38.19) is a posture caused by a severe brainstem lesion; it is characterized by rigid extension of all four limbs and often includes *opisthotonus* (dorsiflexion of the head and neck). Because of the location of the causative lesion, dogs

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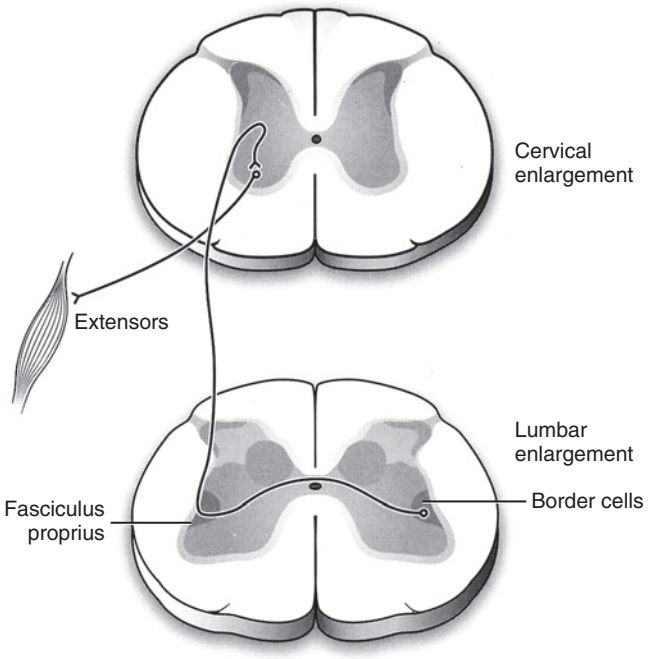


FIG. 38.18 The ascending inhibitory pathway to thoracic limb lower motor neurons, interruption of which leads to the Schiff-Sherrington phenomenon. (Modified from Burke MJ, Colter SB. A practical review of canine and feline spinal cord anatomy. *Prog Vet Neurol.* 1990;1:358–370.)



FIG. 38.19 Cat exhibiting decerebrate rigidity caused by a brainstem lesion.

and cats with decerebrate rigidity typically have severe disturbances of consciousness (i.e., stupor or coma). Decerebellate rigidity is less commonly encountered and is due to acute cerebellar damage. This posture is characterized by rigid extension of the thoracic limbs and flexion of the pelvic limbs. With isolated cerebellar involvement, there should be no impairment of consciousness. Because of the anatomic proximity of the cerebellum and brainstem, it is more common to encounter patients with lesions impairing both (e.g., cerebellomedullary angle lesions) than to encounter patients with purely cerebellar dysfunction. These patients may display a posture with characteristic features of both decerebrate and decerebellate rigidity. In such scenarios, it is important to realize that there is no clinical utility in expecting a patient to adhere to a classic posture when the neuroanatomic localization has already been made.

GAIT

Generation of gait in dogs and cats is primarily dependent on brainstem centers. Unlike in primates, the forebrain has minimal input on their gait generation, but it does provide purposefulness of movement. The main regions of the brainstem and their associated tracts thought to be important for gait generation (Fig. 38.20) are the red nucleus and rubrospinal tract, the pontine reticular formation and the pontine reticulospinal tract, and the medullary reticular formation and the medullary reticulospinal tract. For a coordinated gait to be sustained, proprioceptive information regarding limb position is constantly delivered to these gait-generating nuclei during motion (primarily via the cerebellum, which receives this information via spinocerebellar tracts). In addition, a balanced and coordinated gait requires normal function of the peripheral and central vestibular systems. Observation of an animal's gait often reveals a wealth of information regarding lesion localization. In addition to looking for directionality to the patient's gait (e.g., circling in one direction), other abnormalities of the gait itself, such as lameness, toe-dragging, ataxia, and paresis/plegia, may be noticed. In most cases, lameness is due to orthopedic disease. However, some neurologic disorders manifest as lameness, especially early in the disease process. These include irritation of a nerve root due to disk extrusion, resulting in lameness or holding up a limb (i.e., "root signature," which is most common in thoracic limbs), lameness of one thoracic limb due to a malignant nerve sheath tumor, and unilateral or bilateral pelvic limb lameness due to degenerative lumbosacral stenosis and compression of the cauda equina nerves.

Ataxia invariably points to neurologic disease and is often characterized as sensory ataxia, vestibular ataxia, or cerebellar ataxia (Box 38.2). Sensory ataxia is due to interference with ascending spinal cord proprioceptive pathways and is manifested by a swaying gait. The patient with sensory ataxia may also drag his or her toes. An overall clumsy nature to the gait is apparent, and the patient may actually fall when turning. Vestibular ataxia and cerebellar ataxia may be difficult to differentiate in some patients because the vestibular system is closely associated with the cerebellum. However, if such a patient has other clear evidence of central vestibular dysfunction, such differentiation is superfluous. With unilateral peripheral or central vestibular lesions, animals tend to display a head tilt and to lean or fall toward the side of the head tilt. These patients tend to display general ataxia of all four limbs, but the direction to which the patient falls or leans is consistent. Dogs and cats with bilateral peripheral vestibular dysfunction may display a very characteristic gait, characterized by wide, side-to-side head excursions while ambulating. Cerebellar ataxia also affects all four limbs. In addition to a clumsy gait, evidence of hypermetria or exaggerated limb protraction is often noted while ambulating. This "high-stepping" gait is characteristic of cerebellar dysfunction. When stationary, some of these patients may display head tremors, especially if offered a treat or some other enticement (intention tremor), and/or a whole-body sway from side to side (titubation). Dogs and cats with cerebellar lesions may also display obvious signs of vestibular dysfunction (e.g., strabismus, nystagmus, head tilt). The cerebellum is intimately tied to the vestibular system, and

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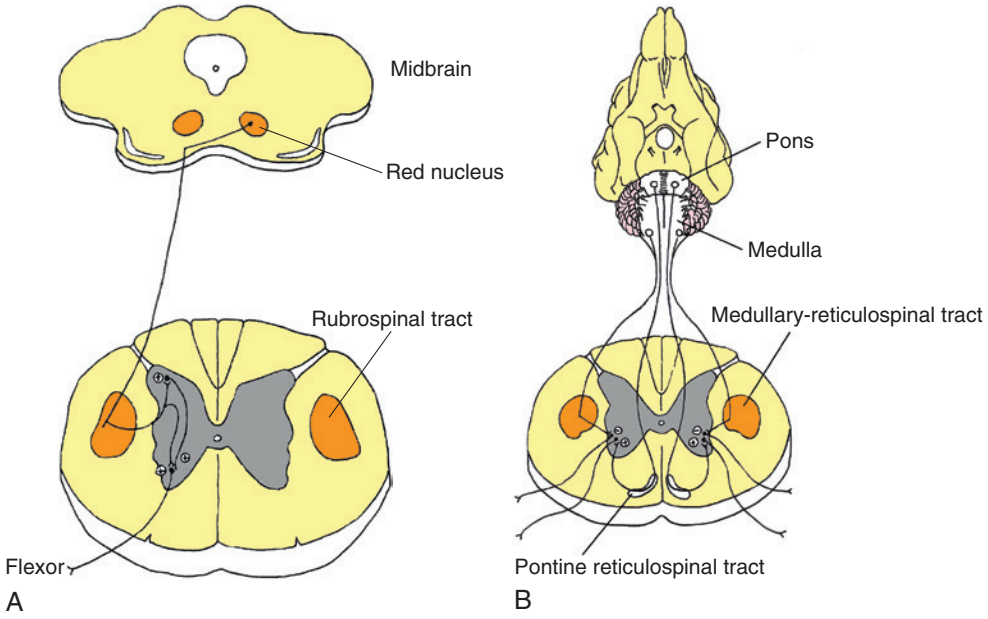


FIG. 38.20 Important gait-generating brainstem nuclei and tracts in dogs and cats. (A) The red nucleus and rubrospinal tract. (B) The pontine and medullary reticulospinal tracts. (Modified from Burke MJ, Colter SB. A practical review of canine and feline spinal cord anatomy. *Prog Vet Neurol.* 1990;1:358–370.)

BOX 38.2 Characterization of Ataxia in Dogs and Cats

Sensory Ataxia

- Due to interference with ascending spinal cord proprioceptive pathways
- Manifested as a swaying gait
- Toe dragging may be evident
- Clumsy gait
- May fall when turning

Vestibular Ataxia

- Head tilt often present
- Leaning or falling to one side (side of head tilt, if present)
- General ataxia of all four limbs
- Wide, side-to-side head excursions with bilateral peripheral vestibular dysfunction

Cerebellar Ataxia

- Hypermetria often evident, in addition to clumsy gait
- Head tremors and/or whole-body sway (titubation) when stationary
- Often have signs of vestibular dysfunction

the vestibular nuclei in the medulla are directly ventral to the cerebellum. It is very common to observe animals with gaits suggestive of cerebellovestibular dysfunction.

Paresis denotes a partial loss of voluntary movement, whereas *plegia* (or paralysis) means a complete loss of voluntary motion. The terms paresis and plegia do not by themselves connote anything regarding muscle tone; nonetheless, clinicians tend to confuse muscle flaccidity with paresis. LMN paresis/plegia is typified by muscle flaccidity and poor to absent spinal reflexes. Patients have sustained damage to the LMNs and/or their axons that directly innervate the muscles responsible for walking (e.g., quadriceps muscles). Upper motor neuron (UMN) paresis/plegia is typified by normal or exaggerated muscle tone and spinal reflexes. These animals have sustained damage to the UMN white matter tracts descending from the brainstem that "tell" the LMNs what to do during gait generation. In either situation, paresis or plegia refers only to a decreased ability or inability to ambulate. In addition, the ability to perceive pain (i.e., nociception) is not implied in the description of gait. This is a separate part of the neurologic examination. Prefixes are added to the terms paresis or plegia to fully describe a patient's gait. *Paraparesis* and *paraplegia* are terms used to describe animals with pelvic limb weakness and lack of voluntary motor ability, respectively. For animals with all four limbs involved, the terms *tetraparesis* and *tetraplegia* are used (some prefer *quadriparesis* and *quadriplegia*, respectively). When one limb is involved (e.g., brachial plexus injury), the terms *monoparesis* and *monoplegia* are used. The full description of gait in a paretic patient includes a subjective assessment of whether a patient can walk unassisted. This is sometimes open to interpretation. The author considers a patient that cannot walk more than a few steps without falling over to be nonambulatory. Such a patient would be described as nonambulatory paraparetic.

POSTURAL REACTIONS

Postural reactions evaluate the pathways involved in gait-proprioceptive and motor pathways. General proprioception is conceptually divided into conscious and unconscious proprioception and refers to position sense, or the ability for an animal to judge where his or her body parts are in space and in relation to the rest of the body. Although an oversimplification, *conscious proprioception* is conveyed by the dorsal column/medial lemniscus pathways (i.e., fasciculus gracilis, fasciculus cuneatus, spinomedullary tracts) (Fig. 38.21) and represents position sense that is

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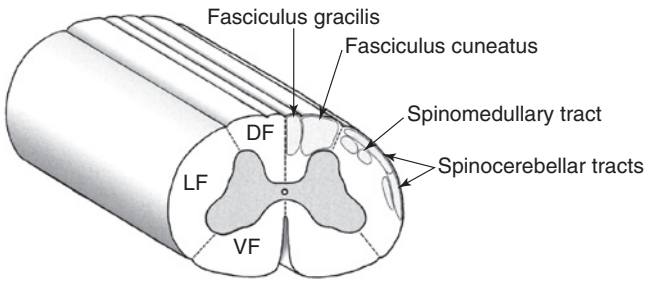


FIG. 38.21 Ascending proprioceptive pathways in the spinal cord. *DF,* Dorsal funiculus; *LF,* lateral funiculus; *VF,* ventral funiculus. (Modified from Dewey CW. *A Practical Guide to Canine and Feline Neurology*. 2nd ed. Hoboken, NJ: Wiley-Blackwell; 2008.)

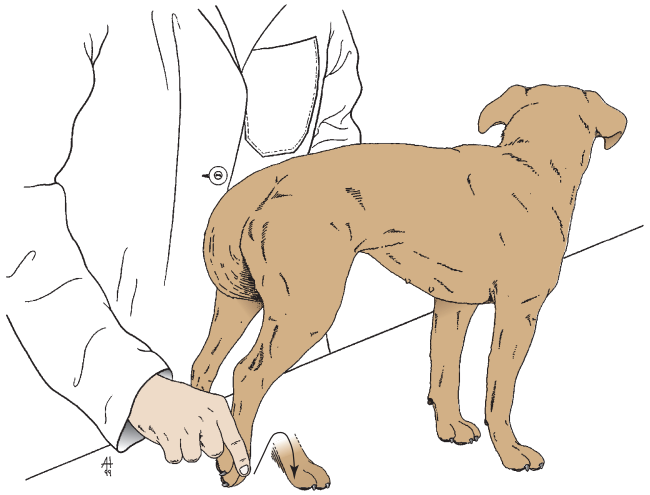


FIG. 38.22 Proprioceptive positioning is evaluated with the patient supported in a standing position. The dorsal surface of the paw is placed on the floor. The patient should immediately replace the foot to a normal position. (Modified from Dewey CW. *A Practical Guide to Canine and Feline Neurology*. 2nd ed. Hoboken, NJ: Wiley-Blackwell; 2008.)

perceived at the cerebral level; *unconscious proprioception* is conveyed mainly by spinocerebellar pathways (i.e., dorsal and ventral spinocerebellar tracts, cuneocerebellar tract, rostral spinocerebellar tract) and represents position sense that is processed by the cerebellum, without conscious involvement. Conscious proprioceptive pathways are represented primarily in the contralateral cerebral cortex, whereas unconscious pathways are essentially ipsilateral. Conscious proprioception is best evaluated with the animal in a standing position, whereas unconscious proprioception is best evaluated with the animal moving. Isolated conscious proprioceptive deficits with normal gait (i.e., normal unconscious proprioception) is a hallmark of forebrain disease, whereas isolated unconscious proprioceptive deficits with normal proprioceptive positioning responses (i.e., normal conscious proprioception) are characteristic of cerebellar disease. In most patients with brainstem and spinal cord lesions, a combination of conscious and unconscious proprioception (general proprioception) compromise is expected.

A number of postural reaction tests can be performed while the neurologic examination is conducted. These various tests include proprioceptive positioning (Fig. 38.22), tactile placing (Fig. 38.23), hopping (Fig. 38.24), hemiwalking (Fig. 38.25), and



FIG. 38.23 Tactile placing is tested by covering the patient's eyes and moving the patient toward the edge of the table. Normal patients will place their paws on the table as soon as they contact the table edge. (Modified from Dewey CW. *A Practical Guide to Canine and Feline Neurology*. 2nd ed. Hoboken, NJ: Wiley-Blackwell; 2008.)

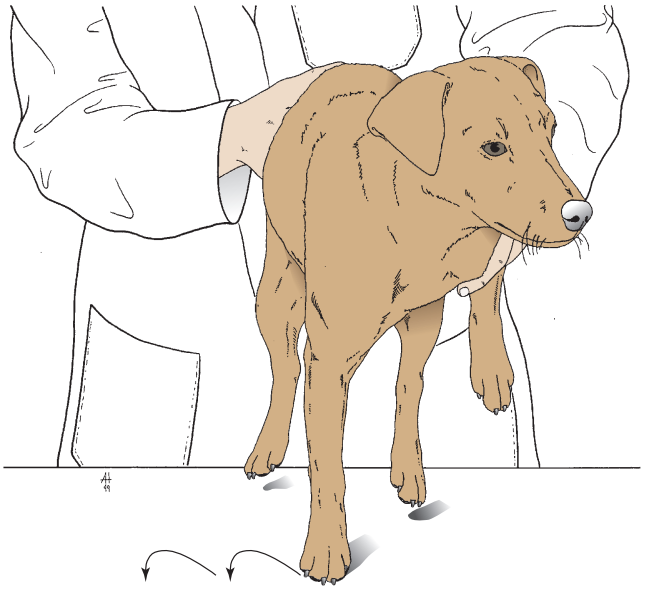


FIG. 38.24 The hopping response is tested by holding the patient in a way that most of the weight is borne on one limb. The patient is moved laterally. Normal animals will hop briskly and symmetrically (hopping is performed as well bilaterally). (Modified from Dewey CW. *A Practical Guide to Canine and Feline Neurology*. 2nd ed. Hoboken, NJ: Wiley-Blackwell; 2008.)

wheelbarrowing (Fig. 38.26). Because these tests all evaluate proprioception, it is not necessary to perform all of them on each patient. For each of these tests, it is important to evaluate for asymmetry, as well as for obvious deficiencies in their execution by the patient. *Proprioceptive placing* (Video 38.6) entails supporting the patient (to avoid stimulation of the vestibular system) while placing one paw at a time over the

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FIG. 38.25 Hemiwalking is tested by lifting the limbs on one side and moving the patient laterally. Normal animals will hop briskly and symmetrically. (Modified from Dewey CW. *A Practical Guide to Canine and Feline Neurology*. 2nd ed. Hoboken, NJ: Wiley-Blackwell; 2008.)

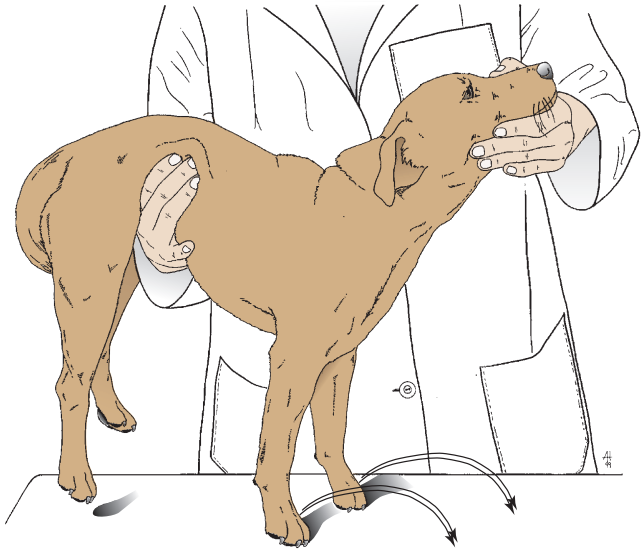


FIG. 38.26 Wheelbarrowing is tested by supporting the patient under the abdomen so that the pelvic limbs do not touch the ground and then moving the patient forward. Normal patients will walk briskly without knuckling and without evidence of hypermetria. (Modified from Dewey CW. *A Practical Guide to Canine and Feline Neurology*. 2nd ed. Hoboken, NJ: Wiley-Blackwell; 2008.)

dorsal surface; the normal reaction to this maneuver is rapid replacement of the paw to a normal position. Cats and small breed dogs often resist this technique, tending to rapidly withdraw the limb every time the examiner attempts to grasp the paw; in these patients, tactile placing and hopping may be more informative. *Tactile placing* is performed by covering the patient's eyes and moving his or her paws to the edge of a table; the normal response is rapid placement of the paw on top of the table. This technique involves picking up the pet, so it is generally performed on small dogs and cats. *Hopping* is performed by holding the animal so that most of his or her weight is borne on the limb being evaluated. The patient is then moved laterally. The normal response is a brisk hopping motion to the side to which the animal is moved. *Hemiwalking* involves supporting one side of the patient and having the patient walk laterally with the thoracic and pelvic limbs of one side. The thoracic limb is typically a bit faster than the pelvic limb, but a quick and organized hopping motion is expected. Normal animals have difficulty hopping and hemiwalking in a medially oriented direction, so these tests should be performed only with the patient being directed laterally. *Wheelbarrowing* is performed by supporting the patient under the abdomen so that the pelvic limbs are non–weight-bearing and moving the patient forward with the head in an extended position. This test is most useful in patients with subtle thoracic limb proprioceptive deficits, most notably those with caudal cervical myelopathy. In this scenario, many such patients will appear to "float" (a very protracted swing phase of the gait) with the thoracic limbs once the ability to compensate visually is removed.

SPINAL REFLEXES

Spinal reflexes are used to assess the integrity of sensory and motor components of the particular reflex, in addition to the influence of descending UMN pathways. Two types of spinal reflexes are evaluated during the neurologic examination: *withdrawal reflex* and *tendon (stretch) reflex*. The former is a polysynaptic reflex elicited by the application of a noxious stimulus to a digit. The latter is essentially a monosynaptic reflex elicited by tapping a taut tendon or ligament with the muscle of interest in extension. The author has not found numeric grading of reflexes to be of clinical value; rather, referring to reflex strength as absent, weak (present but diminished in comparison with normal), normal, or exaggerated has greater clinical utility. *Clonus* refers to a repetitive flexion and extension of a joint following a single stimulus and is an indication of chronic UMN disease. Absent to weak spinal reflexes are indicative of LMN disease, whereas normal to exaggerated spinal reflexes are consistent with UMN disease. The finding of normal to exaggerated spinal reflexes must be evaluated in the context of the remainder of neurologic examination findings. Excited patients often display exaggerated patellar reflexes; in the absence of any other neurologic deficits, this should be regarded as normal. Lack of a patellar reflex in one or both pelvic limbs in older dogs (>10 years old) is a common age-related phenomenon; in the absence of any other neurologic deficits, this finding has little to no clinical significance.

Thoracic limb reflexes routinely tested include the withdrawal reflex, the biceps reflex, and the triceps reflex. As has been mentioned, the withdrawal reflex is a polysynaptic reflex for which the normal response is flexion of all joints of the limb in response to a mild noxious stimulus (Video 38.7). The *biceps reflex* is a stretch reflex that evaluates the integrity of the musculocutaneous nerve and the C6–C8 spinal cord segments. This is performed with the patient in lateral recumbency and the thoracic limb of interest fully extended and pulled backward (Fig. 38.27). The index finger is placed on the tendon of insertion of the biceps muscle, and the finger is tapped with a pleximeter.

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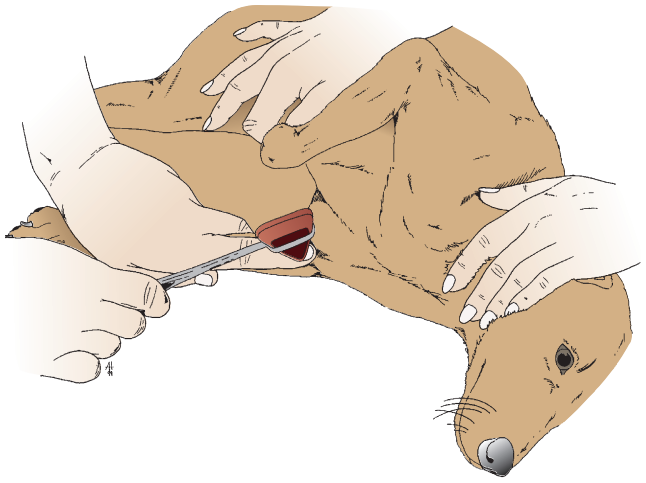


FIG. 38.27 The biceps reflex is elicited by striking the examiner's finger placed over the biceps tendon just proximal to the elbow. (Modified from Dewey CW. *A Practical Guide to Canine and Feline Neurology*. 2nd ed. Hoboken, NJ: Wiley-Blackwell; 2008.)

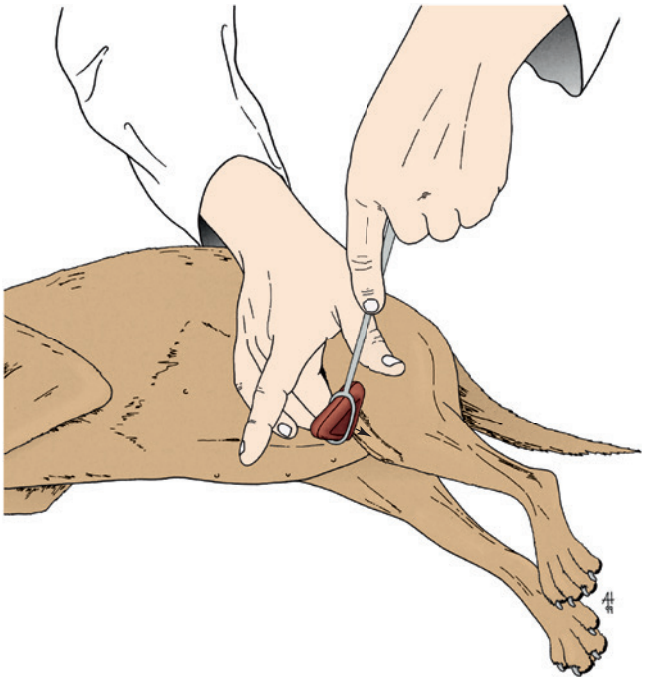


FIG. 38.29 The patellar reflex is elicited by percussing the patellar ligament between the patella and the tibial tuberosity. (Modified from Dewey CW. *A Practical Guide to Canine and Feline Neurology*. 2nd ed. Hoboken, NJ: Wiley-Blackwell; 2008.)

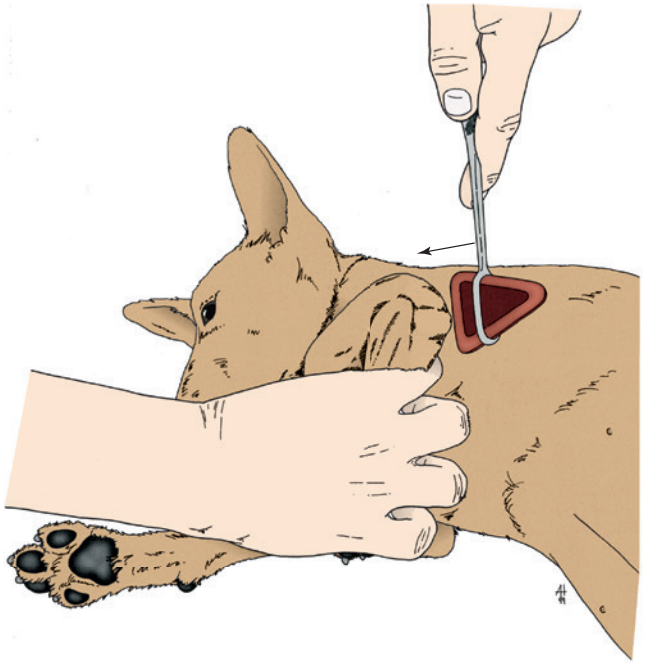


FIG. 38.28 The triceps reflex is elicited by percussing the triceps tendon just proximal to the olecranon. (Modified from Dewey CW. *A Practical Guide to Canine and Feline Neurology*. 2nd ed. Hoboken, NJ: Wiley-Blackwell; 2008.)

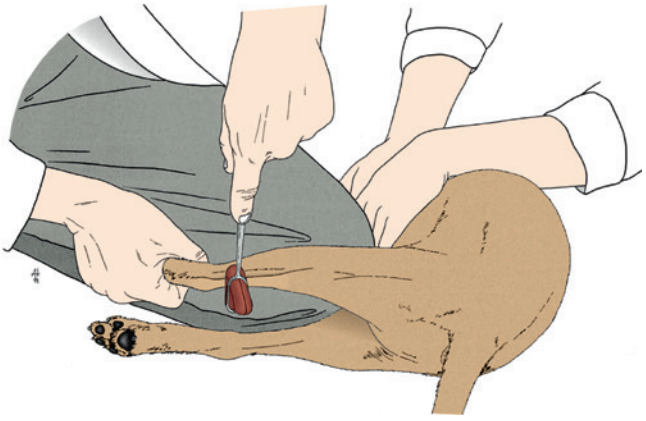


FIG. 38.30 The gastrocnemius reflex is elicited by percussing the gastrocnemius tendon just proximal to the calcaneus. (Modified from Dewey CW. *A Practical Guide to Canine and Feline Neurology*. 2nd ed. Hoboken, NJ: Wiley-Blackwell; 2008.)

The normal response is contraction of the muscle, which can be appreciated along the length of the brachium. The *triceps reflex* tests the integrity of the radial nerve and spinal cord segments C7–T1. This reflex is often difficult to appreciate in dogs and cats, so its absence should not be overinterpreted. The shoulder is flexed, and the triceps tendon is struck medially with a pleximeter (Fig. 38.28). The normal response is contraction of the entire triceps muscle mass (Video 38.8).

Pelvic limb reflexes typically assessed include the withdrawal reflex, the patellar reflex, and the gastrocnemius reflex. As with the thoracic limb, the *pelvic limb withdrawal reflex* is a polysynaptic reflex in which all joints of the pelvic limb flex in response to a mild noxious stimulus. The *patellar reflex* is a tendon reflex in which the patellar ligament is tapped (with the stifle joint in some degree of flexion) and extension of the lower limb (crus) is observed (Fig. 38.29 and Video 38.9). The patellar reflex assesses the integrity of the femoral nerve and the L4–L6 spinal cord segments. The *gastrocnemius reflex* (Fig. 38.30) is performed by tapping the gastrocnemius tendon with the stifle extended and the hock flexed. A normal response is contraction of the caudal

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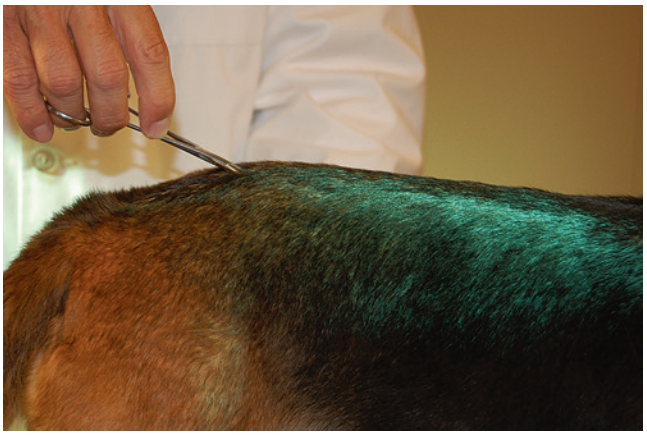


FIG. 38.31 The panniculus or cutaneous trunci reflex is evaluated by lightly pinching the skin just lateral to the spine, starting over the lumbosacral region and proceeding cranially. The normal response is a bilateral contraction of the cutaneous trunci muscle, resulting in a twitch of the skin over the thorax and abdomen.



FIG. 38.32 Gently stroking or pinching the perineum tests the perineal reflex. The normal response consists of flexion of the tail and contraction of the external anal sphincter.

thigh musculature (Video 38.10). This tendon reflex assesses the integrity of the sciatic nerve and the L6–S2 spinal cord segments.

Other spinal reflexes routinely assessed are the cutaneous trunci reflex and the perineal reflex. The *cutaneous trunci (panniculus) reflex* evaluates the integrity of the lateral thoracic nerve, the C8-T1 spinal cord segments, and the sensory input to the spinal cord (dorsal nerve roots) in the dermatomes being stimulated. The skin is pinched lightly with hemostats over the dorsum (Fig. 38.31), and the normal response is contraction of the cutaneous trunci musculature, manifested as skin twitch over the thorax and abdomen (Video 38.11). The contraction is bilateral. An obvious cutoff point in this reflex along the spine in patients with spinal cord disease supports a lesion in the spinal cord one to four segments cranial to the cutoff (spinal cord segments are typically cranial to their associated spinal nerves). This reflex usually cannot be elicited in the lumbosacral and cervical regions and is often difficult to appreciate in normal cats. The *perineal (anal) reflex* assesses the integrity of the sacral spinal cord (S1–S3) segments and various branches of the pudendal nerve. The skin around the perineum is lightly touched, eliciting contraction of the anal sphincter (Fig. 38.32 and Video 38.12). This stimulation also typically elicits tail flexion, which evaluates the integrity of the coccygeal (caudal) spinal cord segments and associated nerves.

The *crossed extensor reflex* is an abnormal reflex that indicates the presence of a UMN lesion located cranial to the spinal cord segments responsible for the reflex of interest. With the patient in lateral recumbency, a digit of one limb is stimulated (e.g., finger pinch) to elicit a withdrawal reflex. A positive crossed extensor reflex is observed if the opposite limb extends as the other flexes. This represents an UMN release phenomenon. This action should be normally inhibited when the patient is in lateral recumbency. *Spinal shock* refers to a transient lack of reflex activity caudal to a severe spinal cord lesion; this phenomenon of hypotonia and hyporeflexia with lesions that should cause UMN signs is uncommon and short-lived in dogs and cats compared with humans. A form of this phenomenon may frequently be observed in dogs with spinal cord infarction (fibrocartilaginous embolic myelopathy) in the T3–L3 spinal cord region. These

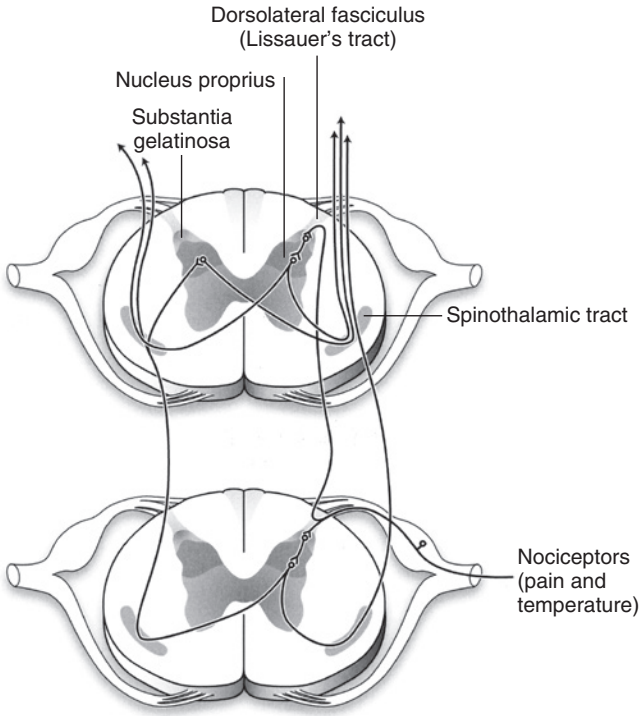


FIG. 38.33 Schematic illustration of the classic spinothalamic tract for nociception in the dog and cat. (Modified from Burke MJ, Colter SB. A practical review of canine and feline spinal cord anatomy. *Prog Vet Neurol.* 1990;1:358–370.)

patients typically exhibit UMN signs with the exception of poor withdrawal reflexes; the withdrawal reflex returns to normal within 72 hours.

PAIN SENSATION (NOCICEPTION)

The modality of nociception or pain perception is transmitted from the periphery to the brain via a number of spinal cord pathways. The classic, contralateral spinothalamic pathway (Fig. 38.33) of people is not considered to be as contralateral (more of a multisynaptic bilateral pathway) in dogs and cats;

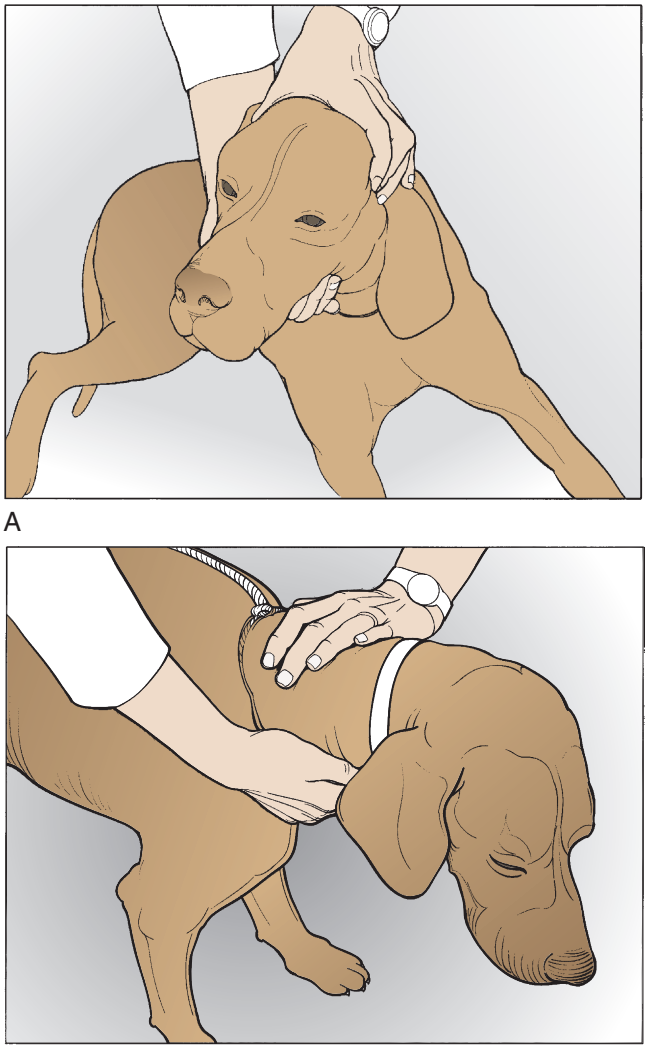
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FIG. 38.34 Testing for deep pain perception is performed by using a hemostat to pinch the digit. A conscious response, such as crying or turning the head, indicates perception of deep pain. (Modified from Dewey CW. *A Practical Guide to Canine and Feline Neurology*. 2nd ed. Hoboken, NJ: Wiley-Blackwell; 2008.)

however, there are numerous dogs and cats with hemineglect syndrome in which the patient fails to acknowledge noxious stimuli applied to the side of the body opposite to the side of a cerebral lesion. Other pain-transmitting pathways considered important in dogs and cats include the spinocervicothalamic, spinoreticular, spinomesencephalic, and propriospinal pathways. For the veterinarian, two areas of nociceptive function/dysfunction are of interest: (1) the presence or absence of normal pain perception ability and (2) the presence or absence of excessive pain perception (e.g., hyperesthesia, paresthesia). It is important to realize that this aspect of the neurologic examination involves the examiner's interpretation of the patient's *perception* of pain. Perception infers that there should be a behavioral response to whatever stimulation the examiner applies. Reflex activity may well be elicited by the application of a noxious stimulus, but this is *not* what is being assessed at this point in the neurologic examination. If a patient's spinal cord is physically or physiologically transected at the level of T13–L1, pinching the digits of the pelvic limbs will elicit a withdrawal reflex because all necessary components of that reflex are still intact. However, nociceptive information will not traverse the lesion site and will not reach the cerebrum, so there will be no conscious reaction (e.g., attempting to bite, looking back at the examiner, vocalization).

Superficial pain refers to sharp, well-localizable, "fast" pain that is conveyed in the spinal cord primarily by larger, more rapidly conducting axons (e.g., type A-delta). *Deep pain* refers to dull, less-localizable, "slow" pain that is conveyed by smaller, less rapidly conducting axons (e.g., type C). These categories do not represent separate pathways, and the distinction between the degrees of stimulus needed to achieve an obvious behavioral response qualifying as a superficial or deep pain response is subjective and arbitrary. In practical terms, if a patient exhibits a behavioral response to a light skin pinch on the digit with your fingertips, both superficial and deep pain sensation can be judged to be intact. The lack of such a response when a digit is clamped with an instrument such as a hemostat (Fig. 38.34) suggests a more profound injury to the nociceptive–transmitting spinal cord pathways and typically a less favorable prognosis.



A

B

FIG. 38.35 Palpation of the (A) head and (B) neck for evidence of hyperesthesia. (Modified from Dewey CW. *A Practical Guide to Canine and Feline Neurology*. 2nd ed. Hoboken, NJ: Wiley-Blackwell; 2008.)

The entire spinal region and head should be palpated for areas of excessive pain response. For the thoracic and lumbar spine, this is done by pushing on either side of the dorsal spinous processes with thumb and forefinger. For the cervical region, palpating the ventral aspect of the vertebrae is the most sensitive method for finding areas of hyperesthesia. Evidence of head pain is typically elicited in patients with intracranial space-occupying or inflammatory diseases when the head is lightly squeezed above the zygomatic arch region (Fig. 38.35).

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39

Surgery of the Brain



GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Creating a defect in the skull to access the brain is a **craniotomy.** Standard craniotomies typically performed in dogs and cats include **lateral** or **rostrotentorial craniotomy, transfrontal craniotomy,** and **suboccipital craniotomy. Intracranial pressure (ICP)** is the pressure exerted by tissues and fluid within the cranial vault and is normally in the range of 5 to 12 mm Hg in dogs and cats. **Cerebral perfusion pressure (CPP)** represents the forward (i.e., arterial to venous) driving pressure that is the main determinant of brain blood flow and hence brain oxygenation and nutrient support. **Autoregulation** refers to the intrinsic ability of the brain to maintain a constant ICP within the mean arterial pressure (MAP) extremes of 50 to 150 mm Hg. However, significant variability has been noted between individuals, and these numbers serve only as a guideline. **Pressure autoregulation** links brain vascular tone with systemic blood pressure (when MAP increases, vasoconstriction in brain blood vessels prevents an ICP increase and vice versa), whereas **chemical autoregulation** refers to the direct responsiveness of brain vasculature to the partial pressure of carbon dioxide ($PaCO_2$) in arterial blood (hypercapnia leads to brain vascular dilation and vice versa). **Compliance** is a change in volume per unit change in pressure within the intracranial compartment and reflects the ability of that compartment to accommodate excess volume (e.g., mass, hematoma) by shifting fluids within the compartment.

GENERAL CONSIDERATIONS

The three main types of craniotomy are sometimes combined to provide increased exposure to parts of the brain for various conditions; they may be modified when necessary. For example, bilateral rostrotentorial craniotomies may be required at times; this may also involve removing the top of the skull. Increased ventral exposure may be needed in some cases during a rostrotentorial craniotomy; this may require resection of part of the zygomatic arch and/or the coronoid process of the mandible. When working in the caudal fossa region, it may be necessary to extend a suboccipital craniotomy laterally by sacrificing one of the transverse sinuses. Sometimes more room is needed caudally in this approach, which is achieved by partial or complete removal of the dorsal arch of the atlas (C1). Ventral approaches to the brain are seldom performed in dogs and cats and therefore will not be discussed in this chapter. The relationship between ICP and CPP and MAP is as follows: CPP = MAP – ICP. Compliance decreases as intracranial volume increases, as is shown in Fig. 39.1. Once an intracranial volume is reached that is close to the limit of the compliance ability of the intracranial compartment, even small increases in intracranial volume (e.g., brain edema, brain vasodilation from anesthesia) will lead to large increases in ICP.

Preoperative Management

The specifics of preoperative management for the brain surgery patient depend to a large degree on the underlying disease process. With increased availability of magnetic resonance imaging (MRI) in dogs and cats, the repertoire of surgical brain disorders in these species has expanded (see also Chapter 14). Patients with disorders typified by slow-growing mass lesions (e.g., brain tumors, intracranial arachnoid cysts [IACs]) will often benefit from low doses of oral prednisolone (e.g., 0.5 mg/kg every 12 hours) in the time period before surgery is scheduled and for some time afterward. Prednisolone alleviates edema surrounding the lesion and typically improves the patient's neurologic status dramatically within 24 to 48 hours. Glucocorticoids should not be given as an emergency treatment for acute brain injury (particularly the "high-dose methylprednisolone" protocol) because they have repeatedly failed to demonstrate any statistically significant benefit in terms of central nervous system (CNS) trauma, and they have the potential for serious adverse gastrointestinal effects.

Adequate fluid support must be provided before anesthesia is given. Overzealous intravenous (IV) fluid administration is thought to exacerbate brain edema, especially in trauma patients; however, fluid restriction is strictly contraindicated. The response of the vasculature to hypotension is vasodilation. In a closed vault such as the cranium, the result is an increase in space taken up by the vasculature. The patient with brain pathology may have additional elevations in ICP caused by this vasodilatory response to hypotension. Conversely, marked hypertension causes an increase in cerebral blood volume and promotes cerebral edema. Therefore the goal is to keep blood pressure within the normal range, avoiding hypotension and marked hypertension. Patients that present with an increased ICP often have systemic hypertension: an indication that efforts to reduce ICP are necessary. Normal saline is the fluid of choice for brain surgery. Because of the blood-brain barrier, osmotic pressure, not oncotic pressure, is the driving force for movement of water out of the vasculature and into the interstitial and intracellular fluid compartments of the brain. In other words, sodium is of vital importance. To minimize brain edema, serum tonicity should be maintained. This is best done by using normal saline and rechecking serum sodium levels when diuretics such as mannitol are used. In acute brain injury patients, hypertonic saline and/or colloid solutions (e.g., hetastarch) are usually advised in combination with normal saline to support normal blood pressure while minimizing the likelihood of further brain edema (see Table 4.5).

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ABSTRACT

Indications for brain surgery include congenital disorders, tumors, and trauma. Neurosurgeons must understand anesthetic considerations relevant to the patient undergoing brain surgery, as well as related concepts of intracranial pressure dynamics. Knowing typical magnetic resonance imaging findings for the various brain disorders encountered by small animal surgeons is vital. Surgeons must also be familiar with approaches to specific brain regions and surgical instrumentation used for brain surgery. Medical management and prognosis of encephalopathies, including antiseizure drug options, chemotherapy, and radiation therapy protocols are of key importance.

KEYWORDS

Brain surgery
intracranial pressure
craniotomy
decompression

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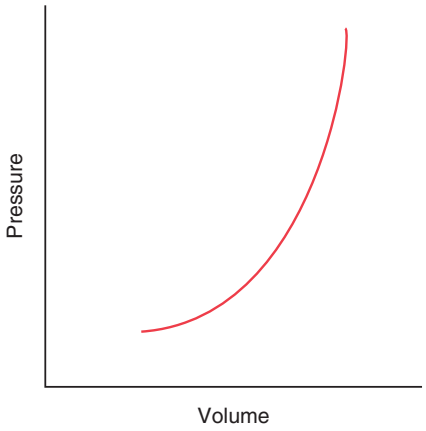


FIG. 39.1 Intracranial pressure versus volume curve.

Depending on the specific disease process, it may be necessary to provide supplemental oxygen to the brain surgery patient before and after surgery. This is often required for victims of severe brain injury. Patients who are conscious and are not obviously deteriorating neurologically should be administered supplemental oxygen via face mask, nasal oxygen catheter, or transtracheal oxygen catheter (see p. 29). Face masks tend to stress dogs and cats and should be used only temporarily, until another form of oxygen (O_2) delivery can be instituted (e.g., nasal O_2). Use of an O_2 cage is generally ineffective for administering supplemental O_2 to severely brain-injured patients because most of these patients require frequent or constant monitoring. Oxygen cages do not allow for concomitant close patient observation (requires opening the cage door) and maintenance of a high-oxygen environment. With nasal and transtracheal O_2 catheters, an inspired oxygen concentration of 40% is provided with flow rates of 100 and 50 mL/kg per minute, respectively. In cases of traumatic brain injury, care must be taken to avoid placing nasal O_2 catheters farther than the level of the medial canthus (to avoid entering the cranial vault through a fracture site). Inadvertent jugular vein compression, which can cause increased ICP, should be avoided while transtracheal O_2 catheters are placed. High flow rates with nasal O_2 catheters may induce sneezing, which can raise ICP. Patients who are losing or have lost consciousness should be intubated and ventilated. In patients with oscillating levels of consciousness or airway obstruction secondary to trauma, a tracheostomy tube may be indicated for assisted ventilation.

IV mannitol (0.5–1.0 g/kg over 10–20 minutes) is the drug of choice for decreasing ICP in brain surgery patients. The most clinically important and immediate (within a few minutes) beneficial effect of mannitol is a decrease in ICP due to decreased blood viscosity and reflex vasoconstriction of brain arterioles. If mannitol is given too rapidly, it causes vasodilation with subsequent increases in cerebral blood volume and ICP and a corresponding decrease in systemic blood pressure. To avoid these blood pressure changes, it is recommended that mannitol be given over 10 to 20 minutes. Over the next 15 to 30 minutes after mannitol administration, fluid is osmotically drawn from extracellular spaces throughout the body, including the brain. Other effects of mannitol include scavenging free radical species and reducing cerebrospinal fluid (CSF) production. Beneficial effects of mannitol last between 2 and 5 hours. Concerns regarding the potential of mannitol to exacerbate preexisting hemorrhage or to cause a reverse osmotic shift are unfounded.

TABLE 39.1 Antiseizure Medications and Oral Dosages for Dogs and Cats

	DOSAGE	
Drug	**Dogs**	**Cats**
Phenobarbital	3–5 mg/kg q12h	2.5 mg/kg q12h
Potassium bromide	35 mg/kg divided, q12h	Not recommended
Gabapentin	10 mg/kg q8h	10 mg/kg q8h
Felbamate	15 mg/kg q8h	Unknown
Zonisamide	5 mg/kg q12h if not on phenobarbital; 8–10 mg/kg q12h if receiving phenobarbital	10 mg/kg q24h
Levetiracetam	20 mg/kg q8h	20 mg/kg q8h
Pregabalin[a]	2–4 mg/kg q12h	1–2 mg/kg q12h
Topiramate[a]	5–10 mg/kg q8–12h	Unknown
Imepitoin	10–30 mg/kg q12h	Unknown

[a]It is recommended to start both pregabalin and topiramate at a low initial dose (e.g., 2 mg/kg q12h) for the first week or two (to avoid sedation).

Antiseizure drugs should be administered to dogs and cats with brain disorders characterized by recurrent seizure activity. A number of alternatives to phenobarbital (PB) and potassium bromide are available for managing seizures in dogs and cats (Table 39.1). Most of these drugs are generic and inexpensive. *Zonisamide* is particularly advantageous to use in dogs with brain tumors. Phenobarbital has the tendency to cause profound sedation in dogs with brain tumors, even at low doses; this effect is sufficiently severe that PB is not recommended in these dogs. Zonisamide does not typically cause sedation and is both effective and inexpensive. Other potentially useful antiseizure drugs for dogs include gabapentin, levetiracetam, felbamate, and pregabalin. *Pregabalin,* the "next generation" of gabapentin, has a longer half-life of elimination than its predecessor and is considered to be more effective. Both pregabalin and gabapentin can cause sedation, although this effect is typically mild. *Levetiracetam* is an extremely useful drug in dogs and cats with brain tumors, particularly when the IV form of the drug is used for short-term management. Levetiracetam seldom has side effects, even at high doses. When side effects occur, sedation and ataxia are most likely. A single bolus injection (20 mg/kg over several minutes) of levetiracetam results in therapeutic plasma levels of the drug for 8 to 12 hours. Levetiracetam does not undergo hepatic metabolism. Oral levetiracetam is a useful drug in both dogs and cats, although a "honeymoon effect" is often seen in dogs with this drug, in which it becomes apparently less effective over time. A potential reason for this phenomenon with levetiracetam may be that the elimination of this drug is accelerated considerably when dogs are concurrently receiving PB. *Felbamate* is an effective and safe antiseizure drug for dogs that does not cause sedation; however, it is expensive, often difficult to acquire, and therefore is used infrequently. *Topiramate* has been shown to be an effective add-on antiseizure drug in one study of refractory canine epilepsy.[1] Reported side effects of topiramate are sedation and ataxia. Topiramate is relatively expensive, and therefore not used frequently by the author. A new antiseizure drug, *imepitoin,* is available in Europe for use in dogs. In one study, imepitoin was

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comparable in efficacy with PB in epileptic dogs.[1] Imepitoin does not cause elevations of hepatic enzymes. Reported side effects of imepitoin include sedation, polyuria and polydipsia, increased appetite, and mild, transient hyperactivity; side effects were less likely with imepitoin compared with PB in one clinical trial.[2,3] For cats, PB, levetiracetam, zonisamide, gabapentin, and pregabalin are useful antiseizure drugs. PB is very effective in cats and usually does not produce side effects commonly encountered in dogs, although it can lead to sedation at high plasma concentrations. Oral diazepam and bromide are contraindicated in cats.

In any patient with suspected increased ICP, care should be taken not to compress the jugular veins which will further increase ICP. Additionally, when these patients are recumbent or are being prepared for surgery, the head should be slightly elevated (approximately 30 degrees) to encourage venous drainage from the head and limit venous congestion.

In animals undergoing brain surgery, IV antibiotics (e.g., cefazolin 22 mg/kg) should be administered perioperatively (30 minutes before surgery is begun and every 90–120 minutes thereafter during surgery) and should be discontinued after surgery or within 24 hours. Some neurosurgeons prefer to continue them orally (e.g., cefadroxil 22 mg/kg every 8 hours) for 10 to 14 days postoperatively.

Anesthetic Considerations

Premedication in patients with increased ICP should be avoided. Decreased respirations and hypercapnia may increase ICP and can be lethal. Patients without an increased ICP can be given midazolam or diazepam. Glucocorticoids and anticonvulsants should be continued until surgery.

The induction period needs to be highly controlled in these patients. Barbiturates and propofol are the induction agents of choice (Table 39.2). Alfaxalone is a synthetic neuroactive steroid that acts via gamma-aminobutyric acid type A receptors in the CNS; it can be used for induction and/or maintenance of anesthesia (see Chapter 12). Alfaxalone has been shown to have similar cardiorespiratory effects and quality of anesthesia as propofol. Although the effects of alfaxalone on brain hemodynamics have not been fully investigated in dogs and cats, the predecessor of this drug was shown to have favorable effects on ICP, cerebral metabolic rate (CMR), and cerebral blood flow (CBF).[4,5] A smooth induction and intubation should be performed, with every effort made to avoid blood pressure extremes and prolonged apnea. Hypertension and tachycardia occurring during intubation may have to be treated by administering esmolol or by deepening the anesthetic with additional boluses of propofol, thiopental, or alfaxalone. Hypotension should be quickly treated with vasopressors (e.g., ephedrine or phenylephrine) but not with fluid boluses (see Table 39.2). The airway needs to be secured very carefully because access to the head will be restricted during surgery. Positioning of the animal with its head elevated is beneficial for patients with increased ICP. Take care to avoid occluding the jugular veins when positioning the head to avoid causing an elevation in ICP.

Anesthetic agents influence CBF, cerebral metabolism, CO_2 reactivity, and cerebral blood pressure autoregulation. Barbiturates, propofol, isoflurane, and sevoflurane all decrease cerebral metabolism. Although these anesthetic agents also decrease CBF, it is the decrease in CMR that provides neuroprotection. Barbiturates have been extensively studied in neuroanesthesia and have been used primarily because of their hypnotic effect, decrease in CMR, and anticonvulsant activity. Even though they cause dose-dependent decreases in CBF, barbiturates cause a corresponding decrease in CMR and cerebral oxygen requirements uniformly throughout the brain. Combined with their apparent ability to increase CSF absorption, barbiturates generally cause a decrease in ICP. Propofol acts very similarly, with decreases to CBF, CMR, and ICP. It also has significant anticonvulsant properties. However, systemic vasodilation and subsequent hypotension with propofol administration should be anticipated and treated. Although etomidate decreases ICP, it is avoided in neurosurgery patients because it causes increased seizure activity and thus an increased CMR. Inhalant anesthetics cause a dose-dependent decrease in CMR, as well as an increase in cerebral perfusion. This is especially desirable during periods of hypotension. Isoflurane is the inhalant anesthetic agent of choice because of its ability to increase CSF absorption and to minimally increase ICP (see Table 39.2). Inhalant anesthetics, particularly halothane and desflurane, should be used cautiously in animals with increased ICP.

Particular attention should be paid to $PaCO_2$ in anesthetized brain surgery patients because hypercapnia can lead to severe increases in ICP and decreased CPP. Most anesthetic drugs used clinically have the potential to cause hypoventilation and resultant hypercapnia. Arterial blood gas measurement is the best way to monitor $PaCO_2$ levels. End-tidal CO_2 ($EtCO_2$) measurement is a useful monitoring tool but tends to underestimate true $PaCO_2$ levels. In patients that have normal pulmonary function, $EtCO_2$ is approximately 5 mm Hg greater than $PaCO_2$. In younger animals, the number is lower, and in animals younger than 1 year, $EtCO_2$ may be equal to $PaCO_2$. These gradients cannot be unilaterally assumed with all patients because of alterations in pulmonary function, especially in anesthetized patients, in which atelectasis and ventilation/perfusion mismatching occur. Nevertheless, $EtCO_2$ can serve as a guideline and, when used with arterial blood gases during neurosurgery, is an important part of monitoring. Venous CO_2 concentration ($PvCO_2$) is also helpful and is usually less than 5 mm Hg greater than $PaCO_2$. However, in patients with perfusion deficits, peripheral $PvCO_2$ levels can be significantly higher than arterial values and should be interpreted cautiously. Historically, it has been recommended to keep $PaCO_2$ levels in the range of 25 to 35 mm Hg to prevent excessive brain vasodilation. Recent evidence suggests that $PaCO_2$ less than 30 mm Hg may lead to excessive vasoconstriction with subsequent impairment of CPP. Excessive hyperventilation may be deleterious to patients whose ICP elevation is not caused by hypercarbia-induced dilation of brain vasculature. Mild hyperventilation to keep $PaCO_2$ between 30 and 35 mm Hg is currently used in human neurosurgical patients. Indiscriminate use of hyperventilation to decrease ICP should be avoided, especially in trauma patients. However, it may be warranted for short periods in emergency cases in which the brain stem is herniating, until more definitive therapy can be initiated.

In general, anesthetic agents considered preferable for use in brain surgery patients include benzodiazepines (e.g., diazepam, midazolam), barbiturates (e.g., pentobarbital), propofol, opioids, isoflurane, and sevoflurane (see Table 39.2). As mentioned previously, it is believed that alfaxalone also has favorable effects on brain hemodynamics, although this is less well-established than it is with other drugs. Opioids have little to no effect on ICP, and pentobarbital, propofol, and benzodiazepines decrease ICP with some neuroprotective properties. The potential adverse effects of opioids on ICP are minimal to nonexistent if appropriate doses are used. The concern is depression and inadequate respirations

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TABLE 39.2 Anesthetic Considerations and Drugs for Dogs and Cats Undergoing Brain Surgery

Preoperative Considerations

Associated conditions	• May be young with other congenital defects • If traumatic brain injury, may have evidence of other trauma • If older patient, may have other comorbidities
Bloodwork	HCT Electrolytes, especially sodium Serum creatinine ALT TP Blood glucose Urinalysis Blood gas, if available
Physical examination	Varies according to cause May be depressed and/or painful
Other diagnostics	Blood pressure ECG Radiographs (thoracic ± abdominal) CT or MRI

Premedications	• Oxygen with flow-by or face mask
	• Avoid sedatives in depressed patients or when increased ICP is known or suspected
	• Prednisolone (0.25–1 mg/kg PO, IV) when indicated
	• Administer GI protectant of choice
	• In patients that have minimal depression, give:
	  • Midazolam (0.2 mg/kg IV, IM), *or*
	  • Diazepam (0.2 mg/kg IV)
	• Before induction, avoid all respiratory depressants, including but not limited to opioids. Avoid acepromazine, xylazine, medetomidine, and dexmedetomidine in depressed, hypotensive, or elderly patients.

Intraoperative Considerations

Induction	Preoxygenate for 3 minutes with face mask or flow-by oxygen then give: • Thiopental (if available, see p. 133; 6–10 mg/kg IV bolus), *or* • Propofol (2–8 mg/kg, IV bolus) • Alfaxalone (2–5 mg/kg, IV bolus)

Maintenance	• Isoflurane preferred, *plus*
	  • Fentanyl (2–10 µg/kg IV PRN in dogs; 14 µg/kg in cats) for short-term pain relief, *plus* PRN
	  • Fentanyl CRI (1–5 µg/kg IV loading dose, then 2–30 µg/kg/h IV), *or*
	  • Hydromorphone[a] (0.05–0.2 mg/kg IV PRN in dogs; 0.05–0.1 mg/kg IV PRN in cats), *or*
	  • Oxymorphone (0.05–0.2 mg/kg IV), *or*
	  • Morphine[b] (0.1–1 mg/kg IV PRN in dogs; 0.05–0.2 mg/kg IV PRN in cats), *or*
	  • Buprenorphine[c] (0.005–0.02 mg/kg IV PRN)
	• If hypotensive (to keep MAP 60–80 mm Hg), give phenylephrine, ephedrine, norepinephrine, or dopamine (see Box 19.6)
	• Position patient with head elevated if possible but avoid jugular vein occlusion
	• Mannitol (0.5–1 g/kg IV over 10–20 min) if needed for cerebral edema
	• Hypertonic saline (4 mL/kg IV) if needed for brain edema
	• Ventilation parameters:
	  • Controlled or assisted ventilation is necessary
	  • SpO_2 >95%
	  • $EtCO_2$ 35–40 mm Hg
	  • $PaCO_2$ 30–35 mm Hg
	  • Tidal volumes 7–10 mL/kg
	  • Respiratory rates 10–20
	  • Peak airway pressures <20 mm Hg
	  • Avoid prolonged inspiratory times
	  • Avoid positive end-expiratory pressure

Fluid needs	• 7–10 mL/kg/h to replace evaporative losses plus 3 × EBL. • Consider colloids for EBL replacement in 1:1 ratio (see Table 4.5)
Monitoring	• BP • ECG • Respiratory rate • SpO_2 • $EtCO_2$ • Temperature • Arterial line • U/O (urinary catheter is needed if diuretics are used) • Repeat sodium levels if mannitol is given

Continued

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TABLE 39.2 Anesthetic Considerations and Drugs for Dogs and Cats Undergoing Brain Surgery—cont'd

Postoperative Considerations

Analgesia	• Fentanyl CRI (1–10 µg/kg IV loading dose, then 2–20 µg/kg/h IV), *or* • Hydromorphone CRI (0.025–0.1 mg/kg/h IV in dogs), *or* • Hydromorphone[a] (0.05–0.2 mg/kg IV, IM q3–4h in dogs; 0.05–0.1 mg/kg IV q3–4h in cats), *or* • Oxymorphone (0.05–0.2 mg/kg IV, IM q3–4 h), *or* • Morphine[b] (0.1–1 mg/kg IV or 0.1–2 mg/kg IM q1–4h in dogs; 0.05–0.2 mg/kg IV or 0.1–0.5 mg/kg IM q1–4h in cats), *or* • Buprenorphine[c] (0.005–0.02 mg/kg IV, IM q4–8h or 0.01–0.02 mg/kg OTM q6–12h in cats)
Monitoring	SpO2 Blood pressure ECG HR Respiratory rate Temperature U/O
Bloodwork	HCT TP Electrolytes, especially sodium, if mannitol was given Blood gas Blood glucose
Estimated pain score	Mild to moderate

[a]Monitor for hyperthermia in cats.
[b]Give slowly to prevent histamine release.
[c]Buprenorphine is a better analgesic than morphine in cats.
ALT, Alanine aminotransferase; *BP,* blood pressure; *CRI,* constant-rate infusion; *CT,* computed tomography; *EBL,* estimated blood loss; *ECG,* electrocardiogram; *$EtCO_2$,* end-tidal CO_2; *GI,* gastrointestinal; *HCT,* hematocrit; *HR,* heart rate; *ICP,* intracranial pressure; *IM,* intramuscular; *IV,* intravenous; *MAP,* mean arterial pressure; *MRI,* magnetic resonance imaging; *OTM,* oral transmucosal; *$PaCO_2$,* partial pressure of carbon dioxide; *PO,* orally; *PRN,* as needed; *SpO_2,* hemoglobin saturation with oxygen; *TP,* total protein; *U/O,* urine output.

causing elevations in $PaCO_2$. Therefore it is recommended that opioids not be used in the preoperative period but rather during surgery, when ventilation is controlled, and during the postoperative period. Poorly controlled pain is much more likely to increase ICP than is judicious use of opioids postoperatively. Additionally, continuous-rate infusions of opiates (e.g., fentanyl, morphine) begun during surgery and continued into the postoperative period are less likely to lead to respiratory depression with a subsequent ICP increase due to hypercapnia (see p. 132).

Because of known negative effects on brain autoregulatory mechanisms and tendencies to increase ICP, anesthetic drugs to be avoided include ketamine, halothane, and desflurane. Ketamine is a controversial drug that was initially considered contraindicated in patients with increased ICP. Traditionally it was thought to always increase CBF; however, it has been shown NOT to increase CBF or ICP in mechanically ventilated animals. In fact, mechanically ventilated, traumatic brain injury human patients have a decreased ICP. Ketamine may also have a neuroprotective effect because of the antagonism of *N*-methyl-D-aspartate receptors, but this is unproven. Nevertheless, giving ketamine to already intubated and ventilated patients is not the same as inducing with ketamine. There are better choices that do not carry the risk of marked hypertension at induction, hence ketamine is not recommended.

The alpha-2 agonists dexmedetomidine, medetomidine, and xylazine have some neuroprotective qualities. Both dexmedetomidine and medetomidine decrease ICP in patients already anesthetized with an inhalant anesthetic. Unfortunately, their variable effects on blood pressure make them less appropriate as induction drugs. However, if used in low doses in a balanced anesthesia protocol, they may reduce the hypertension followed by hypotension seen in these patients (see Table 39.2).

Traditionally, acepromazine has been thought to potentiate seizure activity in patients predisposed to seizures. Clinical evidence contradicts this historical dogma. Very low doses of acepromazine (0.01–0.02 mg/kg IV) are often helpful in relieving what appears to be postoperative dementia in some brain tumors and brain trauma patients. As with other drugs, acepromazine at higher doses can cause hypotension, which can have deleterious effects on ICP and CPP. Furthermore, patients with intracranial masses are often older patients, and acepromazine may have long-lasting effects.

STANDARD SURGICAL APPROACHES TO THE BRAIN

Important vascular structures to avoid during brain surgery are the dorsal sagittal sinus and the transverse sinuses (Fig. 39.2). Laceration of these structures can cause life-threatening hemorrhage. Ligation or obstruction of the dorsal sagittal sinus (particularly the caudal two-thirds of the sinus), the confluens sinuum (the midline juncture of the paired transverse sinuses), or both transverse sinuses will probably cause fatal brain swelling. During the suboccipital approach, it is common to experience hemorrhage from occipital emissary veins at the ventrolateral limits of the supraoccipital bone during periosteal muscle elevation; this hemorrhage is controlled by pushing bone wax into the mastoid foramen through which this vessel traverses.

For all standard approaches, the author prefers a midline incision. Unless brain exposure is very large (e.g., removal of the top portion of the calvarium) or the craniotomy procedure is likely to lead to an obviously abnormal cosmetic appearance postoperatively (e.g., removal of the zygomatic process of the frontal bone), the craniotomy defect is not repaired. If repair of the skull defect is considered necessary, titanium mesh and polymethylmethacrylate (PMMA) can be used (Fig. 39.3). The PMMA is placed on either side of the mesh; the mesh

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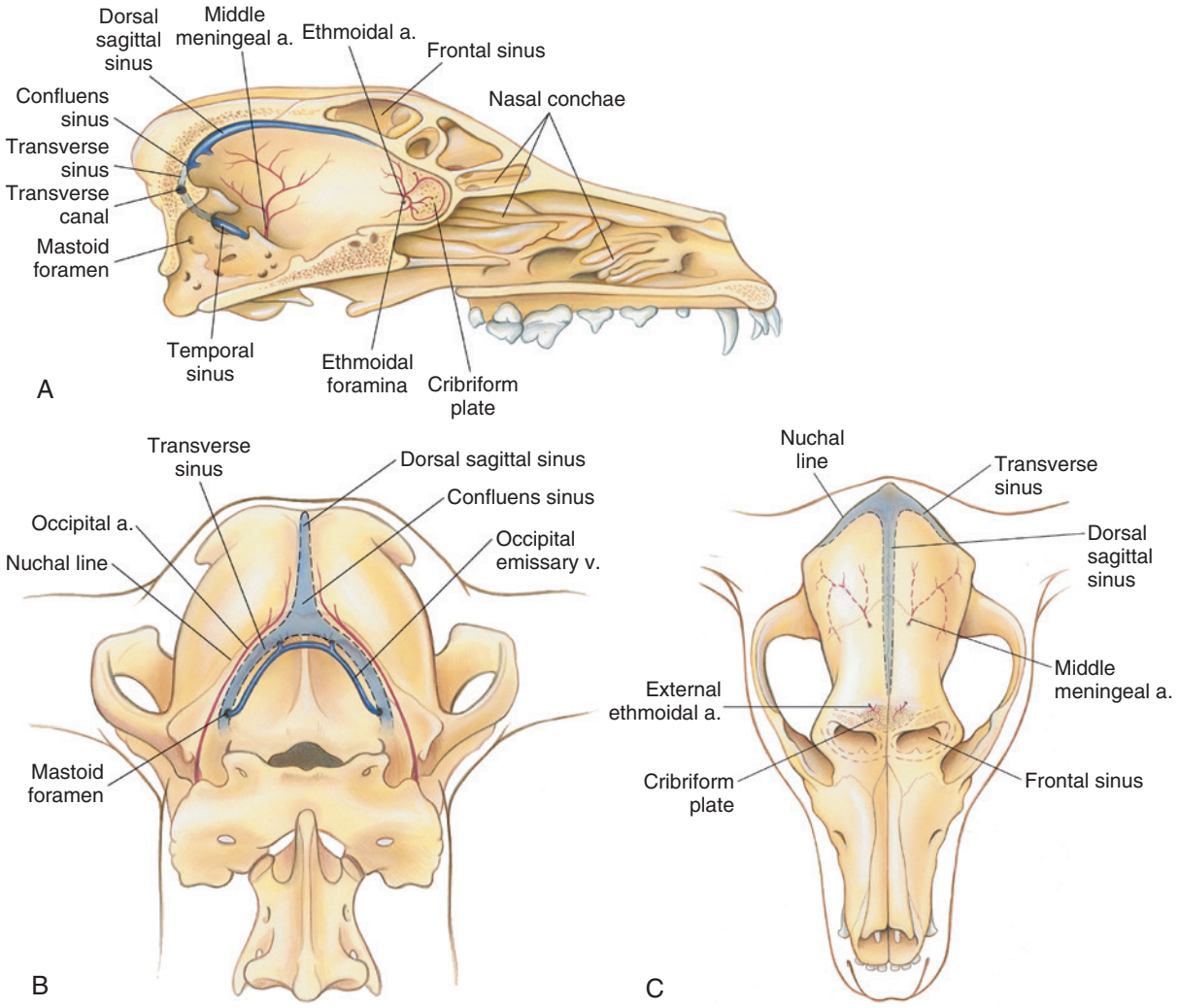


FIG. 39.2 Schematic illustration of the canine skull, showing important surgical landmarks and vascular structures. (A) Mid-sagittal view. (B) Caudal view. (C) Dorsal view.

keeps the PMMA from sagging into the craniotomy site during hardening. Titanium rather than steel is used so that repeat MRI is possible if necessary. Postoperative CSF leakage is a clinical problem encountered in human brain surgery; however, this phenomenon does not occur in dogs and cats following craniotomy. Replacing resected dura/arachnoid with autologous or synthetic dural substitute is generally not necessary in dogs and cats; however, the author repairs dural defects if the craniotomy necessitates invasion of the frontal sinus area. A layer of Gelfoam can be placed over the exposed brain before the time of closure.

Once the dura/arachnoid is opened and the brain is exposed, it is important for the surgeon to be efficient in his/her use of time. Although surgery is "not a race," prolonged brain tissue exposure appears to be associated with increased postoperative morbidity, even when appropriate measures are taken to keep the brain moist during surgery (e.g., moistened lint-free cottonoid sponges placed on the brain surface).

Rostrotentorial (Lateral) Craniotomy

Shave the skin and aseptically prepare the area from the level of the medial canthi rostrally to the cranial aspect of the second cervical vertebra caudally; the lateral limits of the prepared region are the zygomatic arches. Shave and aseptically prepare any aspect of the ears that may be in the surgical area. Make a midline incision from approximately the level of the lateral canthi to several centimeters past the external occipital protuberance. Sharply incise the cutaneous coli muscles and transect the cervicoscutularis muscle near midline, leaving approximately a centimeter for reattachment during closing. Sharply incise the temporalis fascia with a No. 11 blade, leaving at least several millimeters attached to the skull for reattachment purposes. Reflect the temporalis musculature ventrally by elevating it off the side of the skull with Freer periosteal elevators (cats and small dogs) or Army-Navy osteotomes (large dogs). Create an oval outline of the intended defect with a high-speed air drill. Deepen the edges of this outline until a thin layer of periosteum is left. The ventral aspect of the defect does not have to be drilled full thickness. *Once the dorsal and lateral aspects of the craniotomy defect outline are completely drilled, use a periosteal elevator to lever the bone flap off the skull after positioning the elevator in the dorsal aspect of the defect (Fig. 39.4). Once the bone flap is removed, further enlarge the craniotomy defect using rongeurs. After removal of the bone flap, use bipolar cautery to stop hemorrhage from bleeding meningeal vessels.*

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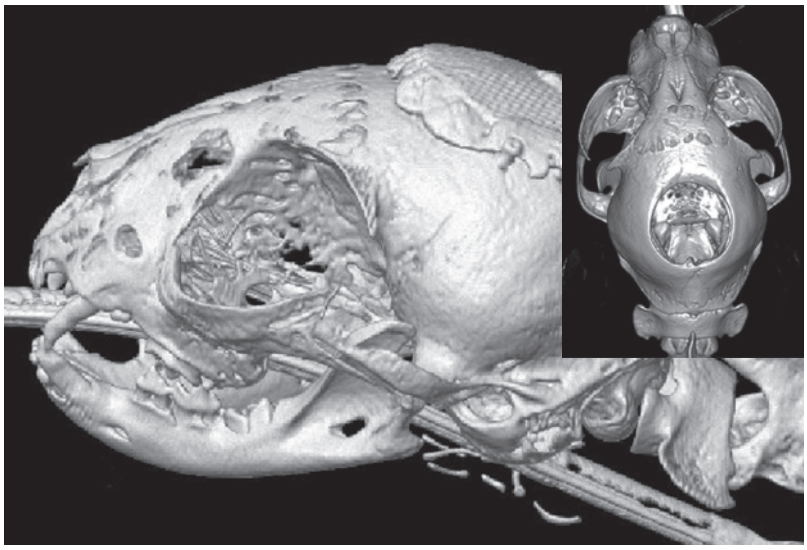


FIG. 39.3 Postoperative three-dimensional computed tomography–reconstructed image of a cat with a meningoencephalocele that had a skull defect *(inset)* repaired with a titanium/polymethylmethacrylate plate. (From Dewey CW, Brewer DM, Cautela MA, et al. Surgical treatment of a meningoencephalocele in a cat. *Vet Surg.* 2011;40:473.)

Transfrontal Craniotomy

The most common reason for performing a transfrontal craniotomy is to remove canine meningiomas in the olfactory bulb region of the brain. Several variations of this approach provide excellent access to the olfactory bulb area and the rostral frontal lobe (prefrontal area) of the cerebrum—areas that are difficult or impossible to access via a standard rostrotentorial craniotomy. As with other approaches, this transfrontal craniotomy approach can be expanded caudally, on either side of the skull, should increased exposure or further brain decompression be required.

Shave the skin and aseptically prepare it from the level of the infraorbital foramina rostrally to the level of the caudal aspect of the zygomatic arches dorsally. Extend the lateral limits of the prepared skin area to the level of the zygomatic arches and include the skin around the eye. Make a midline incision rostrally from the level of the medial canthi to the level of the bregma. Using a combination of sharp dissection and Freer elevators, separate the underlying subcutaneous tissues and frontalis musculature from the underlying bone. Remove the outer bone plate of the frontal sinus to expose the frontal sinus, the ethmoturbinates, and the internal table of the frontal bone.

This bone can be removed in a variety of sizes and shapes with the use of a high-speed air drill or an oscillating saw blade. A triangular shape is often chosen for a unilateral approach, or a diamond pattern for a bilateral approach (Fig. 39.5). Because of the limited exposure (most olfactory meningiomas occupy the majority of the olfactory bulb region bilaterally) provided by the unilateral approach, the bilateral approach is typically performed. *If the bone plate is to be replaced, use the saw blade or a small drill bit when removing the bone.*

Suboccipital Craniotomy (Foramen Magnum Decompression)

The most common indication for a suboccipital craniotomy in dogs is to relieve compression at the craniocervical junction in cases of Chiari-like malformation (CLM). In this scenario, the craniotomy is combined with a dorsal laminectomy of the atlas (C1) and is referred to as a *foramen magnum decompression* (FMD). For other purposes (e.g., brain tumor removal), the suboccipital craniotomy is typically performed without including the C1 dorsal laminectomy. If more exposure is required (e.g., brain tumor removal from the caudal fossa), the suboccipital craniotomy can be extended rostrally on one side by sacrificing one transverse sinus. *For any version of this procedure, place the dog in sternal recumbency with the neck ventroflexed. Shave and aseptically prepare the dorsal aspect of the head and neck from the level of the bregma to the level of the third or fourth cervical vertebra, with a width approximately equal to the width of the atlas. Make a dorsal midline incision extending from approximately 1 cm rostral to the external occipital protuberance cranially to the middle of the second cervical vertebra caudally. Separate the superficial dorsal cervical musculature (Fig. 39.6A) at the median raphe, exposing the underlying biventer cervicis muscles. Separate the paired biventer cervicis muscles on midline, exposing the rectus capitis dorsalis muscles (Fig. 39.6B). Remove the caudal aspects of the rectus capitis dorsalis muscles from the cranial half of C2 using sharp dissection and periosteal elevation, and split these muscles on the midline. Sharply incise the cranial aspects of the rectus capitis dorsalis muscles from the nuchal crest, exposing the caudal portion of the occiput and the arch of the atlas. Control hemorrhage with bipolar electrocautery. Use a high-speed air drill with a 3- to 4-mm diameter round drill bit and Lempert rongeurs to remove a portion of the occiput and the dorsal aspect of the first cervical vertebra (Fig. 39.6C–D).* Approximately 75% of the length of the dorsal arch of C1 is routinely removed. *In cases in which there appears to be dorsal constriction at C1–C2 (see Chapter 40), remove the entire arch of C1 as well as part of the dorsal lamina of C2.*

HEALING OF THE BRAIN

Damaged brain tissue heals via glial proliferation (glial "scar"). A connective tissue covering ("neo dura") forms over incised or

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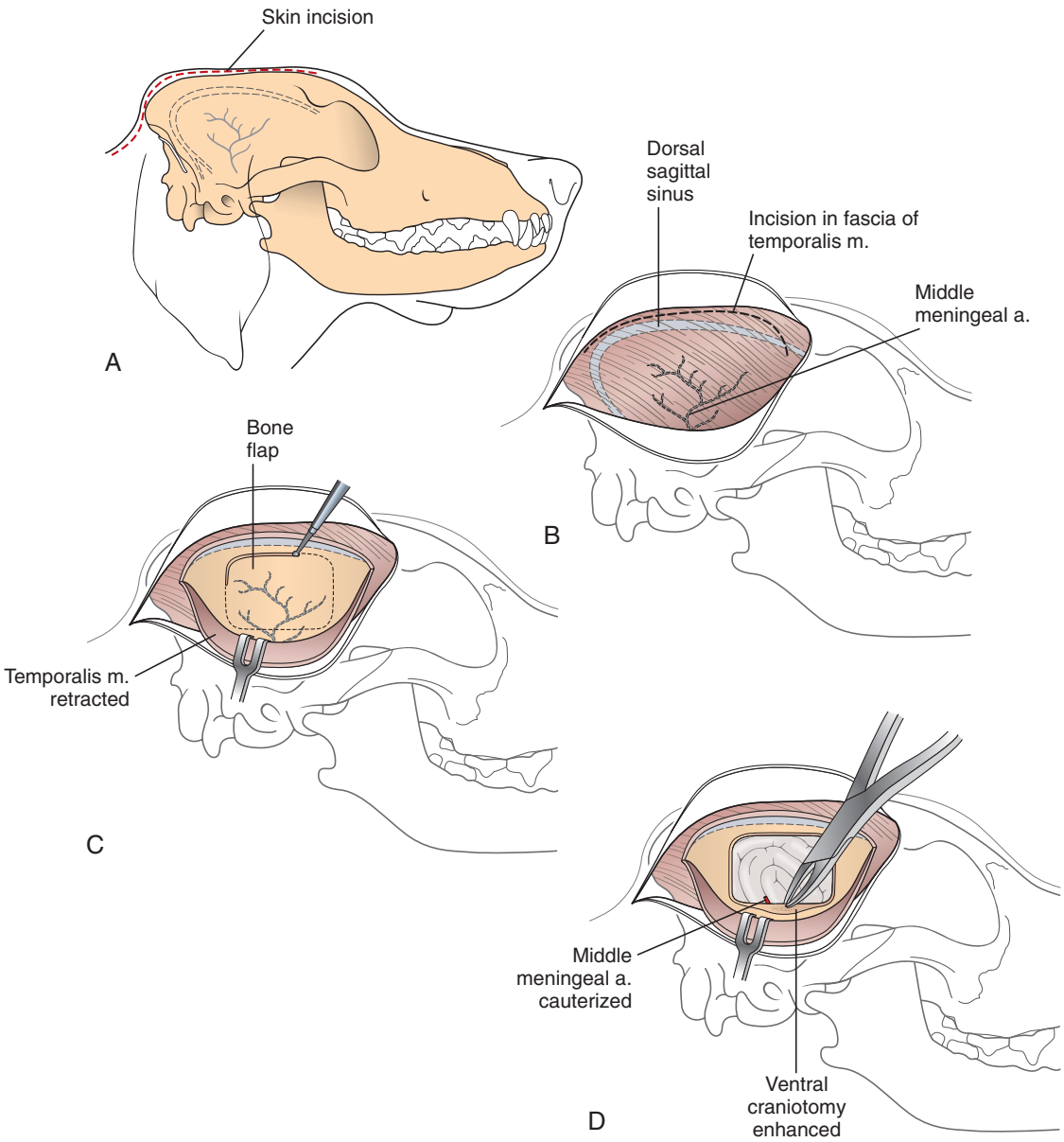


FIG. 39.4 Using a high-speed air drill, create an oval outline for the intended craniotomy defect as shown. The dorsal and lateral aspects of this defect need to be drilled completely through. If a groove is present in the ventral aspect, it will break off when the bone flap is levered off the skull.

resected dura/arachnoid within 2 months of surgery. Surrounding musculature will adhere to the outer surface of this covering, but CSF typically separates the brain from the inner surface of this membrane. If a bone flap is replaced or a synthetic covering (e.g., PMMA, titanium mesh/PMMA) is applied to the craniotomy defect, the neo dura may be adherent to the bone or synthetic flap if the patient undergoes reoperation for tumor recurrence.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Instruments essential for performing brain surgery include a high-speed air drill, rongeurs of several styles (e.g., Lempert, Kerrison, Ruskin; see Figs. 7.17 and 7.18), microsurgical instrumentation (e.g., several sizes of probes, strabismus and tenotomy scissors, nontoothed Bishop-Harmon forceps, lens loops [see Fig. 16.3]), and several sizes of Frazier suction tips. Small needle holders (e.g., Castroviejo) and 5-0 to 6-0 suture material (polypropylene or polydioxanone) are needed for meningeal suturing. Monopolar cautery is needed for hemorrhage during the surgical approach. Bipolar cautery is required for dealing with meningeal, tumor, and brain parenchymal hemorrhage. Gelfoam and Surgicel are also helpful in controlling meningeal and parenchymal hemorrhage. Bone wax is used to control hemorrhage from cancellous bone and transverse

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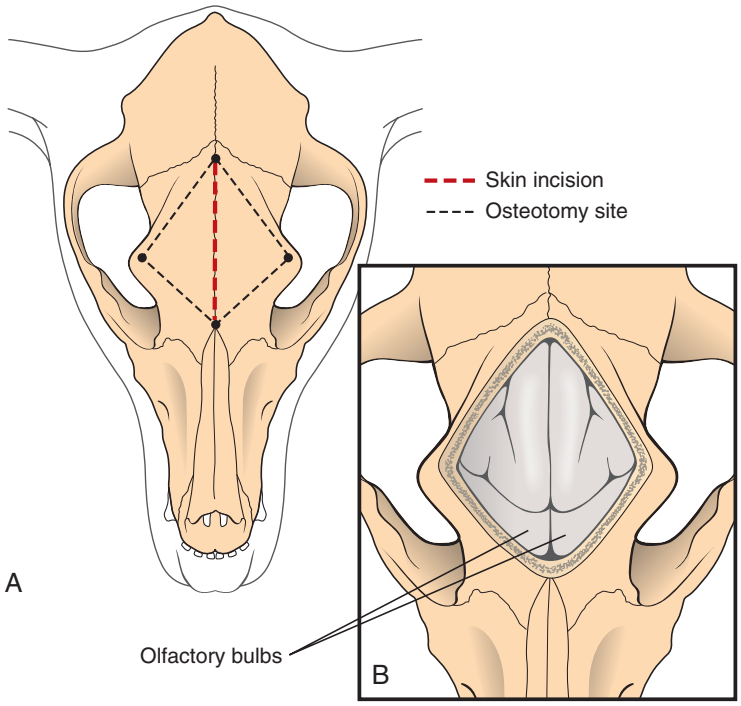


FIG. 39.5 For modified bilateral transfrontal craniotomy: (A) make a midline incision from the caudal edge of the nasal bones at the level of the medial canthus of the eye caudally to the caudal extent of the frontal sinus. (B) Remove a diamond-shaped bone flap using an oscillating bone saw. Then remove the internal table of the frontal bone and a portion of the ethmoturbinates using a rongeur.

sinuses (if transected). Lint-free cottonoid sponges are used to keep the brain moist while it is exposed. Magnification is often useful and is typically provided by optical telescopes. An additional light source (e.g., headlamp) may be needed for some procedures. A Cavitron ultrasonic aspirator (CUSA) is useful for resecting infiltrative masses or working near blood vessels; the high cost of this equipment may limit its use, however. Intraoperative ultrasound is useful both in locating intraaxial lesions and in judging the completeness of their removal before the time of closure.

POSTOPERATIVE CARE AND ASSESSMENT

All postoperative craniotomy patients should be recovered in an intensive care unit and monitored for 3 to 5 days postoperatively. The cage should be padded (e.g., with bubble wrap) to avoid injury to the surgical site. Because postoperative brain surgery patients may have subtle pharyngeal dysfunction, nothing is given per os for the first 24 hours. After this time, small "meatballs" of canned food are offered several times a day, in addition to water. For patients on oral prednisolone therapy, this therapy is gradually tapered over 5 to 7 days. Antibiotics are continued according to the specifics of the disease process and clinician preference. Anticonvulsant drugs and analgesics (see Table 39.1 and Chapter 13) should be administered as needed. Serial packed cell volume (PCV) measurements should be made for the initial 24 hours after surgery and blood transfused as necessary, especially in cats. Oxygenation and ventilation status should be assessed via blood gas analysis, depending on the specific case. Supplemental oxygen should be provided if necessary.

For patients that are unable or unwilling to voluntarily ambulate, frequent turning (e.g., every 4 hours) is essential to avoid pulmonary atelectasis and subsequent pneumonia. The patient's neurologic status should be assessed several times per day, and any deterioration addressed medically (e.g., mannitol) and/or surgically (reimaging and reoperation). Suspected pneumonia (typically older, large dogs following brain tumor removal) should be treated aggressively (e.g., nebulization/coupage, broad-spectrum IV antibiotics) in a timely manner. Following hospital discharge, recheck examinations focus on the rate of neurologic recovery and on wound healing.

COMPLICATIONS

Potential intraoperative and immediate postoperative complications of brain surgery include brain swelling, hemorrhage, and cardiopulmonary dysfunction (e.g., brainstem surgery). Postoperative complications include anemia (primarily cats following meningioma removal), pneumonia (mainly large, older dogs after brain tumor removal), seizures, wound infection, and tumor recurrence.

SPECIAL AGE CONSIDERATIONS

In older dogs and cats, the dura is often adhered to the endosteum of the calvaria, so it may be stuck to the bone flap as the flap is removed. Sometimes the tumor (meningioma) is also attached to the bone flap/dura in these patients. Older dogs, especially of larger breeds, are at risk for postoperative pneumonia following brain tumor removal.

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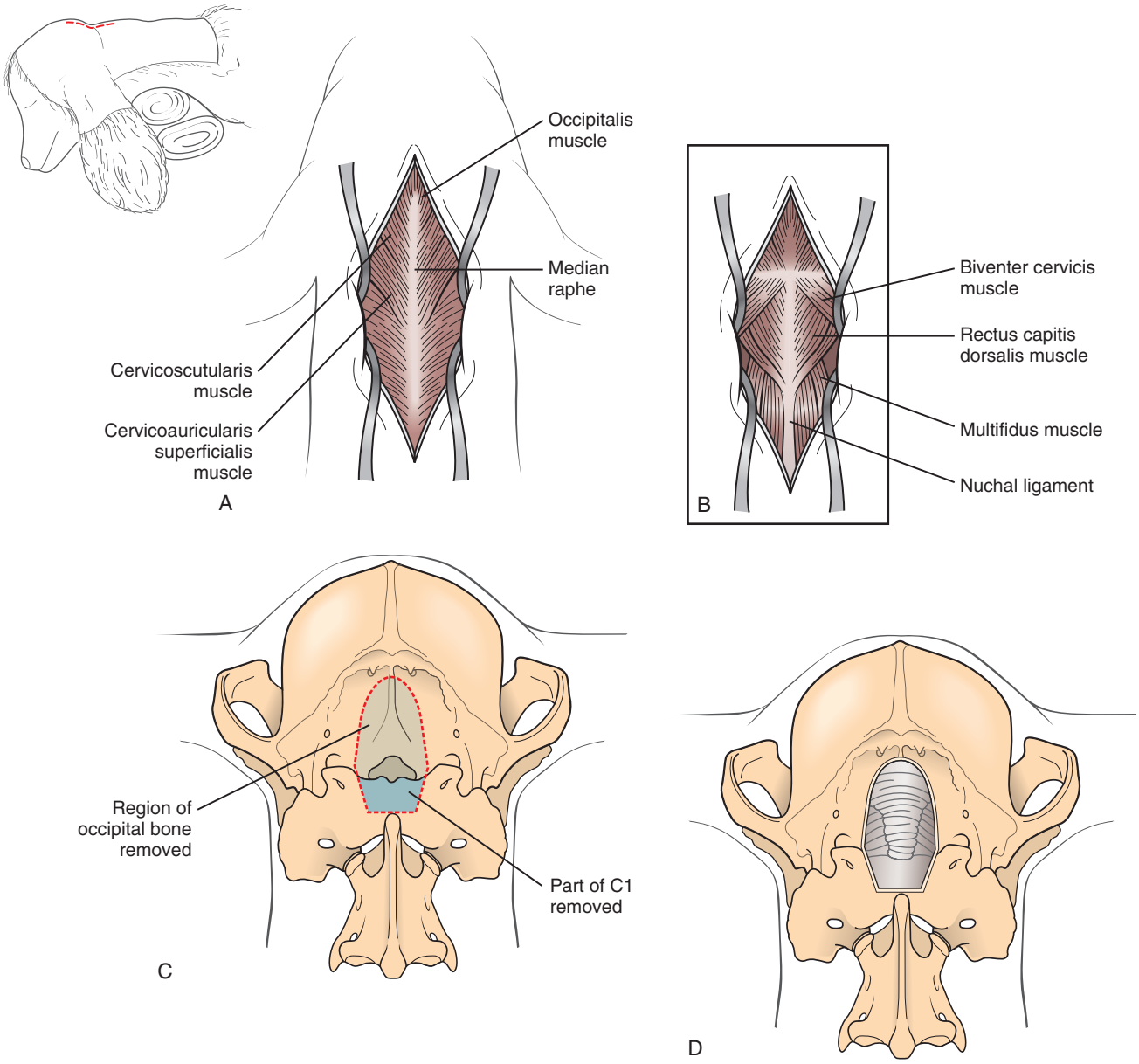


FIG. 39.6 (A) Initial surgical approach for foramen magnum decompression (FMD), suboccipital approach. (B) Illustration of deeper structures encountered during FMD. (C) Bone exposure during FMD approach and (D) completed FMD approach.

SPECIFIC DISEASES

CONGENITAL HYDROCEPHALUS

DEFINITION

Congenital hydrocephalus is an abnormal accumulation of CSF within the ventricular system (primarily lateral ventricles) of the brain that can lead to clinical signs of encephalopathy.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The pathophysiology of CNS damage associated with hydrocephalus is complex and involves destruction of the ependymal lining of the ventricles, white matter damage by interstitial fluid accumulation, and eventual neuronal injury in the cerebral cortex.

Conventional theories regarding the pathophysiology of hydrocephalus contend that ventriculomegaly results from obstruction of CSF flow within the ventricular system (e.g., mesencephalic aqueduct stenosis) and/or insufficient absorption of CSF into the venous system at the arachnoid villi level. These theories are based on the "bulk flow" concept of CSF, in which most CSF is presumed to be absorbed by the arachnoid villi. A more recently proposed theory, called the *hydrodynamic theory*, maintains that hydrocephalus develops as the result of abnormal (reduced) intracranial compliance and the resultant effect of this compliance defect on brain capillaries. In the normal animal, individual brain capillaries remain open during the entire cardiac

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cycle (systole and diastole). This is important because much of CSF absorption actually occurs at the capillary level, rather than at the arachnoid villi. Hydrocephalic patients are believed to have poor intracranial compliance as an underlying disorder that leads to hydrocephalus. The increased capillary pulse pressure caused by decreased compliance leads to a pulsatile transmantle pressure gradient directed from the cerebral tissue toward the lateral ventricles. These abnormal capillary pulsations can occur within normal mean ICP limits. Rebound pressure from the recurring gradient, as well as hyperdynamic CSF flow in the mesencephalic aqueduct, leads to ventricular enlargement over time (increased intraventricular pressure). The end result is that hydrocephalus may develop within the confines of normal ICP (i.e., normal pressure hydrocephalus). Hydrocephalus, especially if progressive, can cause neurologic dysfunction from compression and stretching of brain parenchyma, as well as from brain ischemia and interstitial edema.



FIG. 39.7 Puppy with congenital hydrocephalus exhibiting bilateral ventrolateral strabismus.

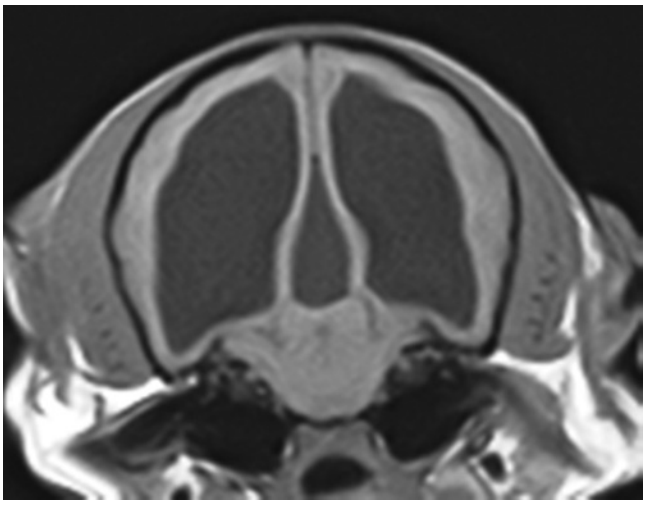


FIG. 39.8 T2-weighted axial brain image of a dog with congenital hydrocephalus.

DIAGNOSIS

Clinical Presentation

Signalment

Congenital hydrocephalus is most commonly reported in dogs, especially small breeds (e.g., Chihuahua, Yorkshire terrier, Maltese, Boston terrier, English bulldog, toy/miniature poodle, Lhasa apso, Pomeranian, Pekingese).

History

Affected animals typically have a history of clinical signs of neurologic dysfunction, including obtunded mentation (see p. 1323), behavior abnormalities, circling, pacing, restlessness, and seizure activity. Seizures are much less commonly associated with congenital hydrocephalus than are behavioral abnormalities and abnormal mentation. Some hydrocephalic patients may also exhibit vestibular and/or cerebellar dysfunction. Many animals, especially of predisposed breeds (see earlier), may have "hydrocephalus" based on lateral ventricular enlargement appreciated on advanced imaging, yet have no discernible neurologic dysfunction. It is important to distinguish ventriculomegaly, which is considered a variant of normal for a number of breeds, from clinical hydrocephalus (see Diagnostic Imaging).

Physical Examination Findings

Common physical characteristics of hydrocephalic patients include a large, dome-shaped head, open fontanelles or larger calvarial defects, and bilateral ventrolateral strabismus (Fig. 39.7). The strabismus may be due to orbital skull malformation, rather than to vestibular dysfunction, and has been referred to as the *setting sun sign*. It is important to realize that small-breed dogs often have open fontanelles with no attendant clinical signs of neurologic dysfunction. Concurrent congenital abnormalities of the brain (e.g., IAC, Dandy-Walker syndrome, CLM) occasionally occur in hydrocephalic dogs, accounting for cerebellovestibular dysfunction.

Diagnostic Imaging

Diagnosis of congenital hydrocephalus is based on a combination of characteristic clinical features, demonstration of ventriculomegaly, and the absence of other causes of encephalopathy. Ultrasonography (through open fontanelles or calvarial defects) and advanced imaging techniques (i.e., computed tomography [CT] and MRI) (Fig. 39.8) have supplanted more invasive methods of documenting ventriculomegaly (e.g., contrast ventriculography). A vast majority of congenital hydrocephalus cases involve primarily dilation of the lateral ventricles (internal hydrocephalus). Occasionally, the bulk of CSF accumulation is within the subarachnoid space (external hydrocephalus), with the brain displaced axially (Fig. 39.9). MRI is the preferred imaging modality for the diagnosis of hydrocephalus. In addition to providing superior anatomic detail, there are certain MRI characteristics that can help differentiate clinically relevant hydrocephalus from asymptomatic ventriculomegaly. In a recent retrospective study, certain MRI characteristics were helpful in distinguishing clinically relevant internal hydrocephalus from asymptomatic ventriculomegaly.[6] Electroencephalography has been used historically to assist in the diagnosis of congenital hydrocephalus; affected patients typically exhibit slow-frequency, high-voltage activity. However, these electroencephalography findings are relatively nonspecific and seldom contribute to the diagnosis of congenital hydrocephalus.

Laboratory Findings

Results of complete blood count, serum biochemical profile analysis, and CSF analysis are typically normal in affected animals.

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FIG. 39.9 T2-weighted axial brain image of a cat with external hydrocephalus.

DIFFERENTIAL DIAGNOSIS

Diseases that may lead to clinical signs of encephalopathy in young dogs and cats should be considered as differentials. Additionally, the possibility of secondary hydrocephalus (i.e., obstructive hydrocephalus due to a brain tumor) needs to be ruled out. Degenerative diseases (e.g., lysosomal storage diseases), anomalous conditions (e.g., IAC), metabolic problems (e.g., hepatic encephalopathy, mitochondrial encephalopathy/organic aciduria), inflammatory/infectious disease (e.g., granulomatous meningoencephalomyelitis [GME], necrotizing encephalitis, canine distemper virus infection), and toxin exposure should be considered in these patients. In most cases, signalment and physical characteristics (e.g., dome-shaped head, bilateral ventrolateral strabismus) are fairly specific for a diagnosis of congenital hydrocephalus.

MEDICAL MANAGEMENT

Medical therapy may be effective in some patients, whereas others require surgical shunting procedures for long-term control of clinical signs. Medical treatment of congenital hydrocephalus is directed at reduction of CSF production. In the author's opinion, medical therapy for patients with obvious neurologic signs attributable to hydrocephalus is usually ineffective, especially in the long term. Oral prednisolone, at an initial dosage of 0.25 to 0.5 mg/kg every 12 hours, may decrease CSF production (Table 39.3). Prednisolone should be reduced over several weeks to the lowest possible dosage required to control clinical signs. Furosemide, a loop diuretic, decreases CSF production via inhibition of the sodium/potassium cotransport system (see Table 39.3). The diuretic acetazolamide is a carbonic anhydrase inhibitor (carbonic anhydrase is a necessary enzyme for CSF production) that is typically dosed at 10 mg/kg body weight, orally every 6 to 8 hours. Omeprazole, a proton pump inhibitor, has been shown to decrease CSF production in dogs, although the clinical effectiveness of this drug has not been documented. For all of these drugs, it is recommended that the dose be tapered to the lowest amount needed to control clinical signs of disease to avoid serious side effects. Anticonvulsant drugs should be administered if the patient seizures. As mentioned, medical therapy for congenital hydrocephalus may provide some level of disease palliation in mild cases but often fails in the long term. When treatment decisions are made, the potential side effects of long-term corticosteroid and/or diuretic therapy should be considered, along with the questionable efficacy of medical therapy for congenital hydrocephalus. Electrolyte depletion (especially potassium) and dehydration are concerns when diuretics are used for prolonged periods, particularly when combined with glucocorticoids.

TABLE 39.3 Drugs for Treatment of Congenital Hydrocephalus

Drug	Dosage
Prednisolone	0.25–0.5 mg/kg PO q12h
Furosemide	0.5–4.0 mg/kg PO q12–24h
Acetazolamide	10 mg/kg PO q6–8h
Omeprazole	10 mg q24h (dogs <20 kg); 20 mg q24h (dogs >20 kg) PO

PO, Orally.

SURGICAL TREATMENT (Videos 39.1 and 39.2)

The goal of surgical treatment of hydrocephalus is to continually divert excessive CSF from the ventricles of the brain to the peritoneal cavity. A number of shunts are available, all of which have the same basic design: a rostral end for placement into the lateral ventricle, a one-way valve, and a distal end for placement into the peritoneal cavity (Fig. 39.10).

Preoperative Management

See p. 1338 for preoperative management of the brain surgery patient.

Anesthesia

See p. 1340 for anesthetic management of the brain surgery patient.

Surgical Anatomy

See Chapter 38 and p. 1342 for surgical anatomy of the skull and brain. Patients with congenital hydrocephalus may have sections of skull missing, in addition to the classic midline open fontanelles. Familiarity with the orientation of the abdominal musculature in the flank area is essential for performance of a grid approach to the peritoneal cavity. At the level of the flank incision, the external abdominal oblique muscle is directed caudoventrally, the internal abdominal oblique muscle is oriented in a caudodorsal direction, and the transversus abdominis runs in a dorsoventral direction, essentially parallel to the last rib.

Positioning

The front end of the patient is placed in sternal recumbency, with the head in a neutral angle. The pelvic limbs are directed laterally, toward the surgeon, so that the flank is oriented in a

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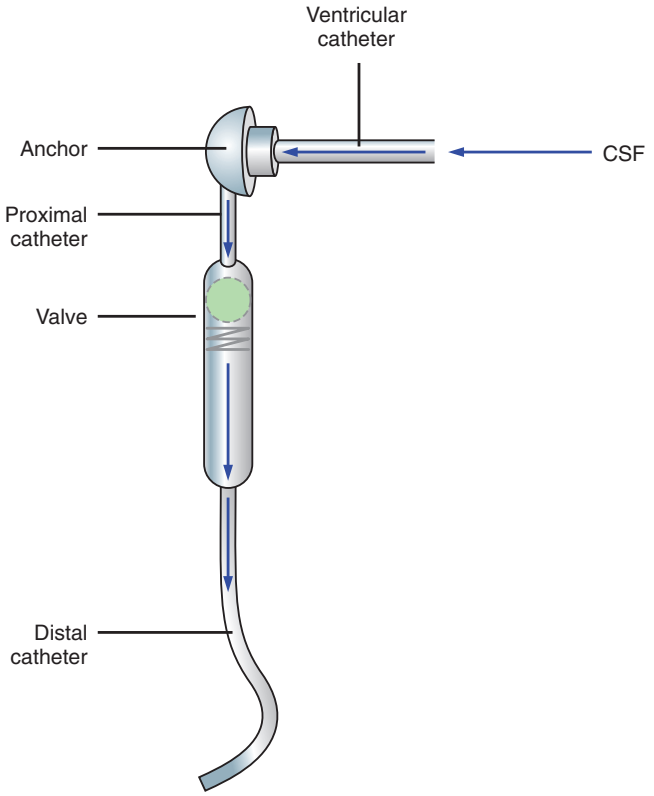


FIG. 39.10 Schematic of a typical ventriculoperitoneal shunt. *CSF,* Cerebrospinal fluid.

lateral plane. Lateral positioning of the caudal half of the patient facilitates the grid approach to the peritoneal cavity.

SURGICAL TECHNIQUE

Shave and prepare the head as described for rostrotentorial craniotomy (p. 1343). Also shave and prepare the entire lateral surface of the patient up to the level of the tuber coxae. For very small patients (e.g., Chihuahua, Yorkshire terrier), make two incisions: one curvilinear incision over the caudodorsal region of the parietal bone and one vertical incision caudal to the last rib (Fig. 39.11A). For larger patients (e.g., English bulldogs), make an additional incision approximately midway between these two incisions on the lateral thorax, if required. Use a high-speed drill to drill two holes in the parietal bone: one for the shunt and one for an anchoring suture. Incise the meninges over the area of shunt insertion with a No. 11 blade, and remove the small area of cerebrum underlying the meninges with a suction tip to access the lateral ventricle. Premeasure the amount of shunt to be inserted into the ventricle, and insert it into the dorsal aspect of the ventricle. After insertion of the rostral end of the shunt into the lateral ventricle, secure it with a Chinese finger-trap suture (see p. 923) via the anchor hole using 3-0 or 4-0 polypropylene (Prolene) suture (Fig. 39.11B). Perform a grid approach to the peritoneal cavity, and tunnel the distal end of the shunt from the cranial incision to the caudal incision using long Carmalt or Doyen forceps (Fig. 39.11C). After placement of the distal end of the shunt tubing into the peritoneal cavity, secure it to both the last rib and the abdominal musculature.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Shunts were described previously under Surgical Treatment. A high-speed drill is needed to drill holes into the parietal bone. To secure the shunt, 3-0 to 4-0 nonabsorbable suture (e.g., polypropylene [Prolene]) is needed. A long Carmalt or Doyen forceps is used to tunnel the shunt. Baby or small Gelpi retractors facilitate the grid approach to the peritoneal cavity. A Matthew-Senn retractor or a spay hook in the flank incision (once the peritoneal cavity is entered) used to elevate the body wall helps with shunt insertion into the peritoneal cavity

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative radiographs (Fig. 39.12) or a CT scan (Fig. 39.13) is performed to assess the accuracy of shunt placement. The patient is placed in a padded cage in an intensive care unit and is administered analgesics and antiseizure medications as needed.

PROGNOSIS

The prognosis for dogs and cats with congenital hydrocephalus is variable but is generally guarded to good with surgical management. In the author's experience, most severely clinically affected congenital hydrocephalic dogs treated medically do poorly. The prognosis for sustained clinical improvement in neurologic status after surgical shunting procedures varies in the literature from 50% to 90% for dogs. The success rate is approximately 75% to 80%. Potential postoperative surgical shunt complications in dogs and cats include shunt obstruction or dislodgment, mechanical damage to the shunt, and shunt infection. In one retrospective study of 36 hydrocephalic patients treated via ventriculoperitoneal shunt placement, 26 patients (72%) experienced improvement in clinical signs. In this study, most shunt-related complications and deaths occurred within the first 3 months of surgery, suggesting the need for particularly close monitoring of these patients during this postoperative time period.[7]

INTRACRANIAL ARACHNOID CYST

DEFINITION

IAC is a developmental brain disorder in which CSF is thought to accumulate within a split of the arachnoid membrane during embryogenesis. It is also termed an *intraarachnoid cyst* or *quadrigeminal cyst*.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The developing neural tube is surrounded by a loose layer of mesenchymal tissue called *perimedullary mesh;* this tissue eventually becomes the pia and arachnoid layers of the meninges. In normal development, pulsatile CSF flow from the choroid plexuses divides the perimedullary mesh into pia and arachnoid layers, creating the subarachnoid space. It is postulated that some aberration of CSF flow from the choroid plexuses during this stage of development forces a separation within the forming arachnoid layer, eventually leading to the creation of an IAC. The intraarachnoid location of IACs in people has been demonstrated via light and electron microscopy. The mechanisms by which an IAC continues to expand with fluid are unknown,

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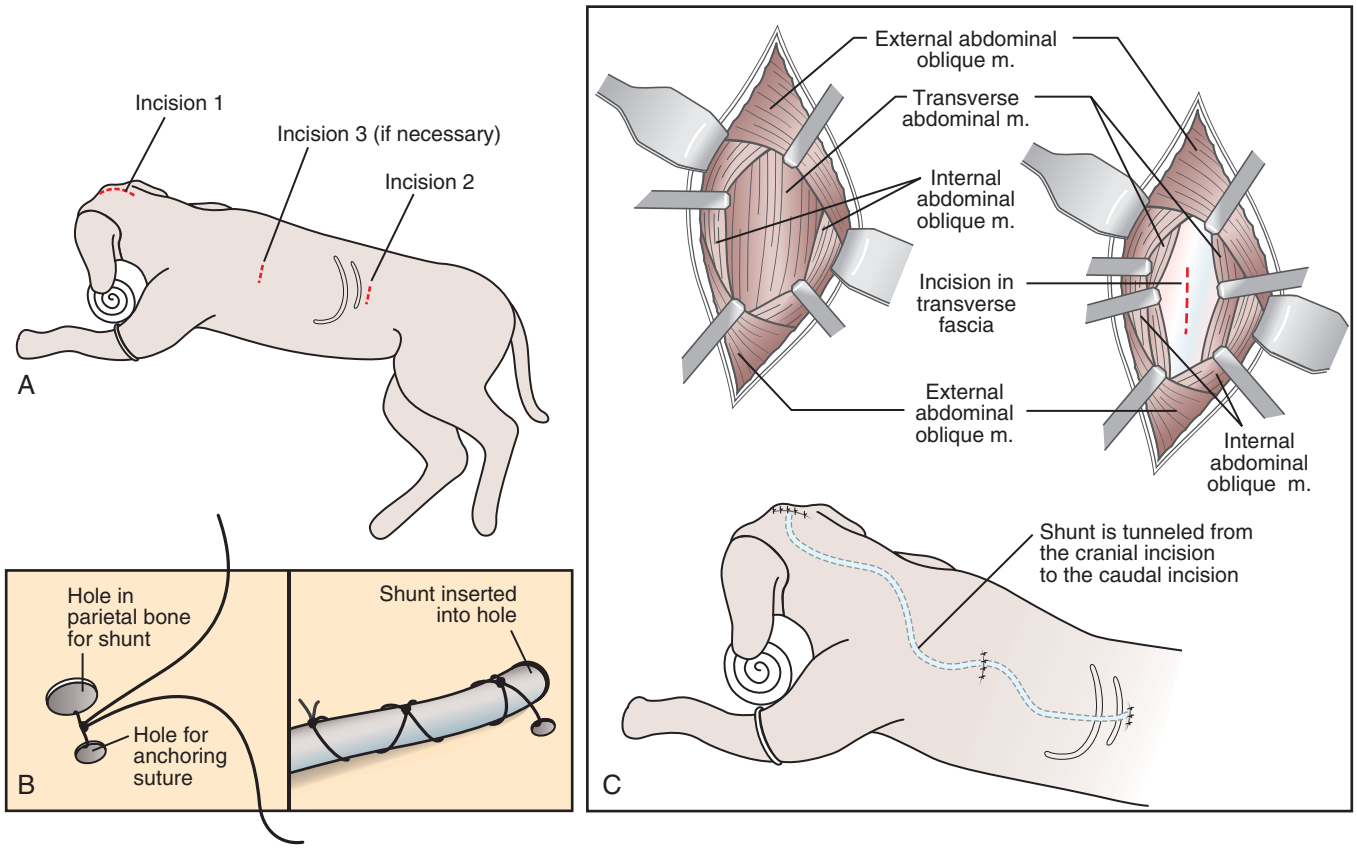


FIG. 39.11 (A) Surgical positioning and location of incisions for ventriculoperitoneal shunt (VPS) placement. (B), Insertion and anchoring of the rostral end of the VPS. (C) Grid approach to the peritoneal cavity following subcutaneous tunneling of the VPS.



FIG. 39.12 Lateral radiograph of a dog showing proper shunt placement.

but several theories have been proposed. Fluid may be secreted by arachnoid cells lining the cyst cavity. Evidence suggests that cells lining the IAC may have secretory capacity. Fluid movement into the cyst may also occur via an osmotic pressure gradient. Given that the fluid within the IAC is nearly identical to CSF, this theory is unlikely. In addition, cases in people in which small slits exist between the IAC and the subarachnoid space have been documented; these slits act as one-way valves, diverting CSF into the cyst during systole, which cannot return to the subarachnoid space during diastole.

Although IAC has been reported to occur in several locations in humans, all reported canine cases have been located in the caudal fossa. Because IAC is typically associated with the quadrigeminal cistern in dogs, these accumulations of fluid in this species are often called *quadrigeminal cysts.* Similar structures have been reported in cats. Also termed *intracranial intraarachnoid cyst,* IAC accounts for 1% of all intracranial masses in people and has been sporadically reported in dogs. IAC is often an incidental finding in humans; it has recently been suggested that this may also be the case for IAC in dogs.

DIAGNOSIS

Clinical Presentation

Signalment

A vast majority of reported IAC cases in dogs have been noted in small breeds, with a predominance of brachycephalic dogs. The breed most commonly reported is the shih tzu. A wide age

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FIG. 39.13 Computed tomography image of a dog's head showing the rostral end of the ventriculoperitoneal shunt in the lateral ventricle.

range has been reported at clinical presentation for dogs with IAC, with an approximate average age of 4 years (range, 2 months–10 years).

History

The most common clinical signs seen with IAC are forebrain (including seizure activity) and/or central vestibular (cerebellovestibular) dysfunction. Dogs may also present with a primary complaint of neck pain.

Physical Examination Findings

Dogs with IAC typically do not have obvious structural abnormalities of the skull or divergent strabismus, as are seen in patients with congenital hydrocephalus. Clinical signs of vestibular dysfunction (e.g., head tilt, strabismus, nystagmus) may be present. Most patients appear to be in pain on palpation of the head and neck regions.

Diagnostic Imaging

IAC is typically diagnosed via CT, or preferably MRI. IACs can be visualized using ultrasound imaging (via foramen magnum, temporal window, and/or persistent bregmatic fontanelle), especially in younger dogs. The characteristic appearance of IAC is a large, well-demarcated fluid-filled structure, isointense with CSF spaces and located between the caudal cerebrum and the rostral cerebellum (Fig. 39.14A–B). Because IAC may be an incidental finding, it is important to rule out concurrent inflammatory disease (i.e., CSF examination). It is often difficult or impossible to discern whether IAC in the presence of another brain disorder is purely an incidental finding. Because the presence of a large, fluid-filled structure within the cranial vault likely decreases intracranial compliance, some IACs may be contributory, rather than an incidental finding. The degree of brain compression by the IAC as measured on MRI is predictive of whether the patient will display clinical signs of dysfunction due to the IAC.



FIG. 39.14 (A) Midsagittal and (B) axial T2-weighted magnetic resonance images of a dog with an intracranial arachnoid cyst.

Because this disorder is believed to represent a developmental abnormality of the intracranial ventricular CSF system, it may occur concurrently with other fluid abnormalities (e.g., congenital hydrocephalus). The cyst may or may not communicate with the remainder of the ventricular system. When evidence of IAC and signs of another disease (e.g., GME) are noted in the same patient, optimal response may entail treating both conditions.

Laboratory Findings

As with congenital hydrocephalus (see p. 1348), laboratory findings are normal when IAC is the primary condition.

DIFFERENTIAL DIAGNOSIS

The differential diagnoses for IAC are essentially the same as for congenital hydrocephalus (see p. 1348). In IAC cases that exhibit obvious vestibular dysfunction, disorders that tend to cause vestibular dysfunction (e.g., GME) are higher on the differential diagnosis list. Additionally, for those IAC dogs that exhibit signs of cerebellar dysfunction, disorders such as cerebellar vermian hypoplasia (Dandy-Walker syndrome) and cerebellar abiotrophy need to be considered. If clinical signs of cerebellar dysfunction are of acute onset, cerebellar infarct should be considered a differential diagnosis.

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MEDICAL MANAGEMENT

Medical treatment for IAC is identical to that described for congenital hydrocephalus (e.g., glucocorticoids, diuretics, anticonvulsants if indicated; see p. 1349). Dogs with IAC tend to respond initially to medical therapy, but the response is often temporary.

SURGICAL TREATMENT (Video 39.3)

Preoperative Management

See p. 1338 for preoperative management of the brain surgery patient.

Anesthesia

See p. 1340 for anesthetic management of the brain surgery patient.

Surgical Anatomy

See Chapter 38 and p. 1342 for surgical anatomy of the brain and skull.

Positioning

Patient positioning is identical to that described for congenital hydrocephalus (see p. 1349).

SURGICAL TECHNIQUE

Surgical management of IAC in people is typically achieved via cyst fenestration or cystoperitoneal shunt placement. Both procedures have been reported in dogs with IAC. Most IACs in dogs are large enough that they can be accessed for fenestration through a caudally based rostrotentorial approach or a suboccipital approach. If extensive resection of the cyst wall is planned, this may be difficult to achieve with the limited exposure of a suboccipital approach. A combined rostrotentorial/suboccipital approach with sacrifice of the transverse sinus and insertion of a cystoperitoneal shunt may be preferred.

With the exception of the extent of the craniotomy defect and placement of the rostral end of the shunt, the procedure is identical to that described for congenital hydrocephalus. *For placement of the rostral end of the shunt in IAC cases, create a wide caudolateral craniotomy, incorporating the creation of a rostrotentorial and a suboccipital skull defect. Sacrifice the transverse sinus between these two defects and occlude it with bone wax.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

See congenital hydrocephalus (see p. 1350).

POSTOPERATIVE CARE AND ASSESSMENT

As with congenital hydrocephalus, postoperative imaging is performed to assess the accuracy of shunt placement (Fig. 39.15). The patient is placed in a padded cage and is administered analgesics and antiseizure medications (if necessary).

PROGNOSIS

The success rate for surgical management of IAC appears to be high in humans and in dogs, and whether fenestration or cystoperitoneal shunting is the preferred procedure remains controversial for both species.

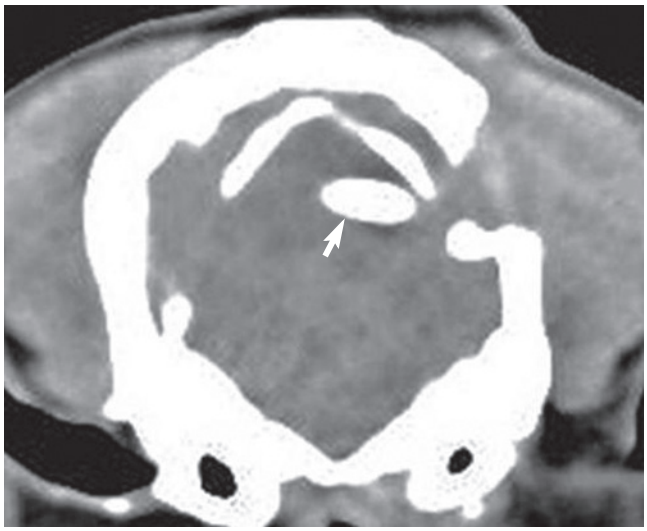


FIG. 39.15 Postoperative axial computed tomography image, demonstrating shunt position *(arrow)* and collapse of the intracranial arachnoid cyst.

CHIARI-LIKE MALFORMATION

DEFINITION

CLM, a disorder that is believed to be the canine analog of Chiari type I malformation of people, is characterized by malformation of the supraoccipital bone. Similar to the human disorder, the cranial cavity is too small to accommodate the contents of the caudal fossa (cerebellum and brain stem), resulting in overcrowding of the cerebellomedullary region of the brain (Fig. 39.16A–B).

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Increasing evidence suggests that CLM in dogs is a condition in which the entire skull is malformed, although the caudal aspect is most obviously abnormal on MRI, which often reveals a visible abnormality of the supraoccipital bone that causes an indentation of the caudal cerebellum. Additionally, an interruption of the dorsal subarachnoid space is typical at the level of the cervicomedullary junction. Herniation of the caudal aspect of the cerebellum through the foramen magnum is also often appreciable on sagittal magnetic resonance (MR) images in CLM cases (Fig. 39.17). Overall, CLM appears to represent a mismatch between the brain parenchyma residing within the cranial cavity and the volume available within this cavity to accommodate the parenchyma. In other words, there is too much brain parenchyma in too small a space; this is more apparent in the caudal fossa, but is an overall brain parenchyma/intracranial volume mismatch in affected dogs. Most dogs with CLM have cervical syringomyelia (SM) (fluid accumulation within the spinal cord parenchyma) evident on MRI (Fig. 39.18). The majority of dogs that have their entire spine imaged (i.e., not just the cervical region as is commonly done in some practices) have syrinx cavities in the thoracic and lumbar spinal cord regions, as well as in the cervical region. SM in this condition is believed to be secondary to a pressure differential between intracranial and spinal components,

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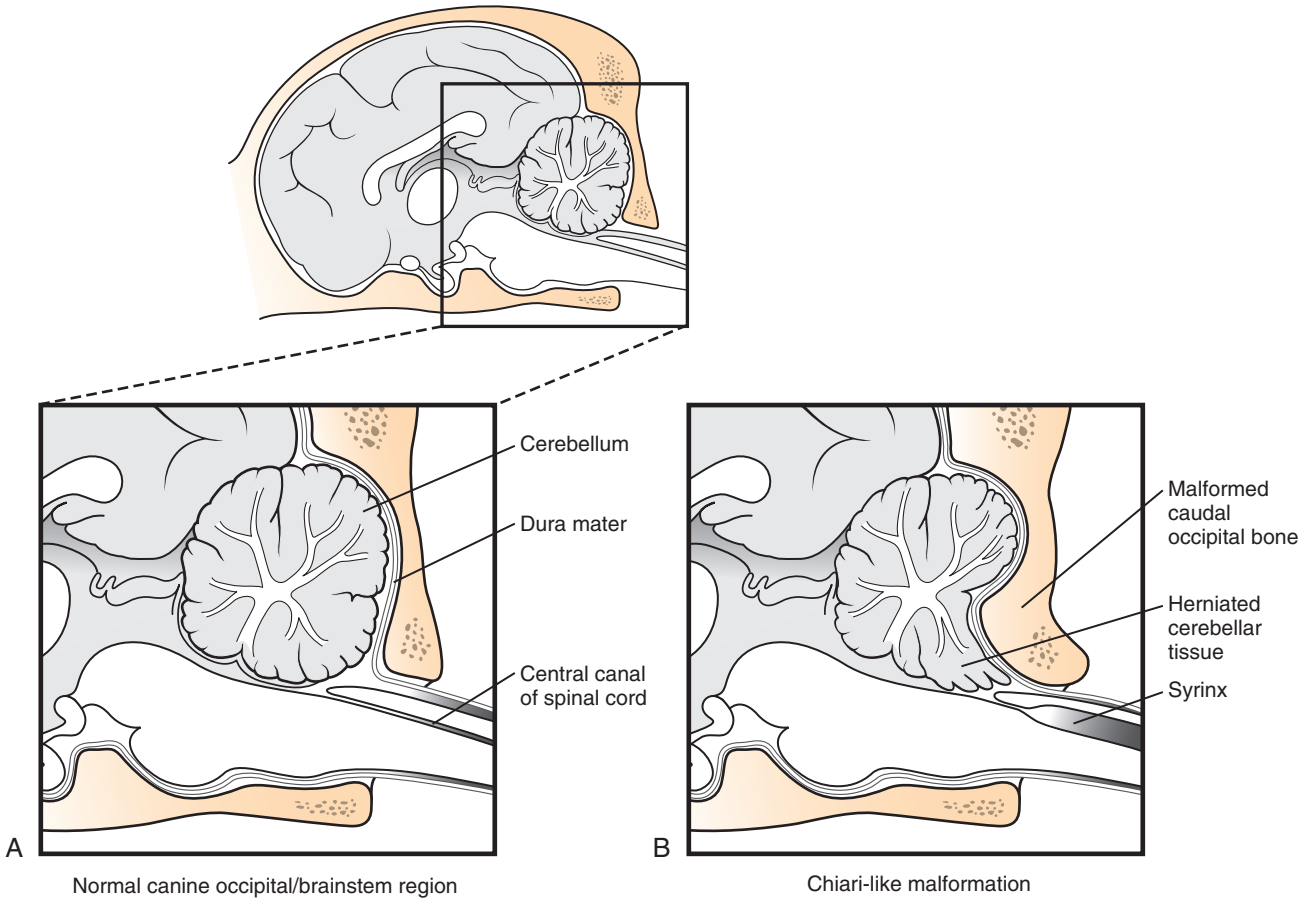


FIG. 39.16 (A) Schematic illustration of the normal shape of the caudal occipital region. (B) The typical shape of this region in a dog with Chiari-like malformation.

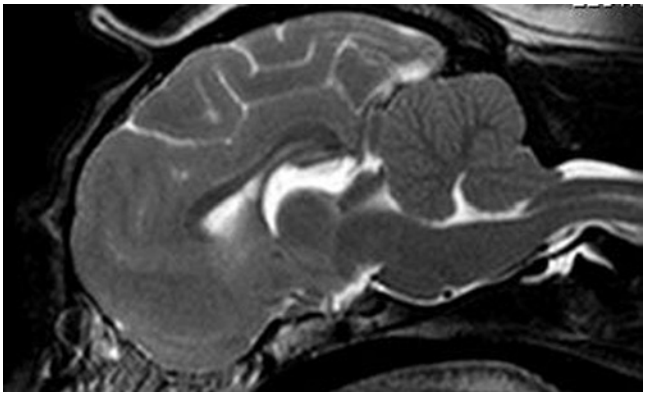


FIG. 39.17 Sagittal T2-weighted brain image of a dog with Chiari-like malformation, showing cerebellar herniation through the foramen magnum.

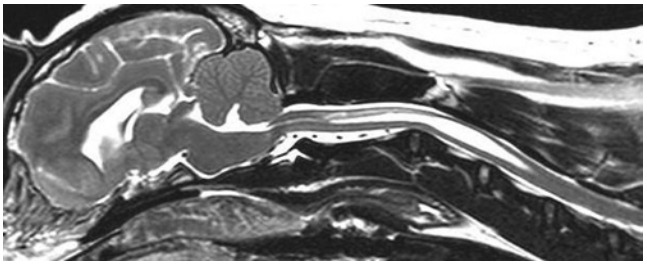


FIG. 39.18 Sagittal T2-weighted cervical spine image of a dog with Chiari-like malformation showing extensive syringomyelia in the cervical spinal cord.

because of abnormal flow dynamics at the cervicomedullary junction (see Chapter 40).

DIAGNOSIS

Clinical Presentation

Signalment

CLM is typically encountered in small-breed dogs; the Cavalier King Charles spaniel (CKCS) is the most commonly affected breed. Other breeds affected by this disorder include Brussels griffon, miniature poodle, Yorkshire terrier, Maltese, Chihuahua, bichon frise, Staffordshire terrier, pug, shih tzu, miniature dachshund, miniature pinscher, French bulldog, Pekingese, and Boston terrier. Anecdotal reports describe this disorder in several brachycephalic cats. The typical age range at presentation appears to have changed over time, with many dogs developing clinical signs within the first year of life. In general, although the age range at clinical presentation is broad, most dogs present by the time they are 4 years old. Dogs that present at younger than 2 years of age often have more severe clinical signs than older dogs. In recent years, we have seen an increasing number of younger patients (<1 year of age); whether this trend reflects

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increasing severity of the disorder with subsequent generations, increased awareness of the veterinary community and hence earlier diagnosis, or a combination of these two factors is unknown.

History

The most salient historical complaint for this disorder is pain, which is usually localized to the neck region. Incessant scratching activity, typically in the head and neck region, is a common historical complaint that is usually evident in the CKCS breed. Other historical complaints include balance loss, head tilt, tetraparesis, and scoliosis.

Physical Examination Findings

Clinical signs are variable, but the most consistent clinical feature is pain (hyperesthesia). Some of the clinical signs are attributable to constriction at the cervicomedullary junction, but many are due to SM. Clinical signs of this disease include neck pain, back pain, vestibular dysfunction, cervical myelopathy, incessant scratching activity, and scoliosis. In people with Chiari type I malformation, one of the most frequent clinical features is a severe suboccipital headache. Almost all dogs with CLM that the author has examined exhibit dramatic evidence of hyperesthesia when the caudal occipital region is lightly palpated with the thumb and index finger. Cervical myelopathy with associated neck pain and cerebellovestibular dysfunction (e.g., strabismus, decreased menace response with normal vision, head tilt, nystagmus) are most commonly encountered. In most cases, cerebellovestibular dysfunction is revealed during a neurologic examination and has not necessarily been observed by the pet owner. Many dogs with CLM have decreased to absent menace responses with normal vision, as well as varying degrees of positional ventrolateral strabismus. Scratching activity appears to be a very consistent clinical feature of CLM in the CKCS breed but is not as prevalent in other breeds with CLM. An unusual and distinctive feature of the scratching activity associated with CLM in dogs is that these patients typically do not make contact with the skin while scratching at the head and shoulder regions, so-called "phantom scratching." Scratching often occurs only on one side. Facial rubbing (pawing at the face and/or rubbing against objects) is encountered in some dogs and is considered to be a form of pain and/or paresthesia. Spinal hyperpathia (typically cervical), scratching activity, and scoliosis are all generally believed to be related to interference of the syrinx cavity with ascending sensory pathways in the spinal cord. Scratching activity and neck discomfort are often exacerbated by abrupt weather changes, stress or excitement, and physical contact with the neck/shoulder region (e.g., collar). Some dogs will experience pain during defecation. The presence of both pain and scoliosis is correlated with syrinx width in CKCS dogs with SM secondary to CLM. Some of the neck pain may be directly related to constriction at the cervicomedullary junction.

Diagnostic Imaging

The diagnosis of CLM is made by MRI, which is also the preferred imaging modality for diagnosing SM. The malformation is best visualized on a midsagittal view (preferably T2 weighted), which includes the caudal fossa and the cranial cervical spinal cord. Consistent findings on MRI associated with CLM include attenuation/obliteration of the dorsal subarachnoid space at the cervicomedullary junction and rostral displacement of the caudal cerebellum by the occipital bone (Fig. 39.19). A 2015 retrospective MRI study of brachycephalic dogs without clinical signs attributable to CLM/SM concluded that rostral cerebellar displacement is often (44%) present incidentally and is likely a normal anatomic variant associated with the brachycephalic skull. This MR feature also became more prominent with increased head extension.[8] Other common MRI findings in CLM include SM (usually C2 level caudally), herniation of the caudal cerebellum through the foramen magnum, and a kinked appearance of the caudal medulla. Phase-contrast MRI (cine-MRI) is often used to measure CSF flow in humans with Chiari type I malformation and has been evaluated for use in dogs with CLM. Occasionally, dogs with MRI findings consistent with CLM will have evidence of other congenital disorders, such as intracranial arachnoid (quadrigeminal) cyst, malformation/malarticulation of the C1 and/or C2 vertebrae, and hydrocephalus. Most small-breed dogs normally have large lateral ventricles as a breed characteristic (ventriculomegaly) and are not hydrocephalic.

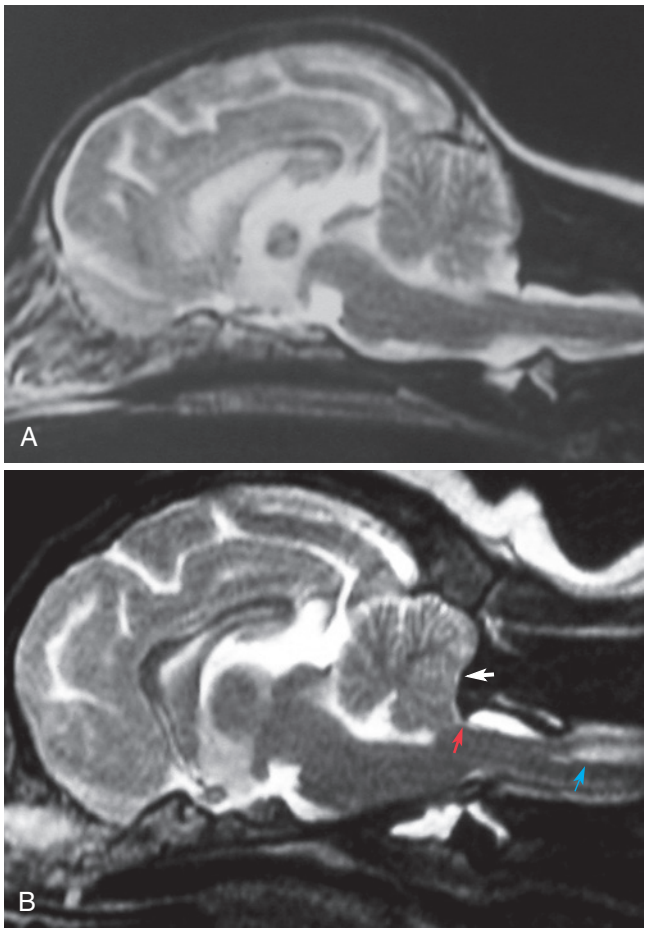


FIG. 39.19 Midsagittal T2-weighted brain magnetic resonance image of (A) a normal small-breed dog and (B) a dog with Chiari-like malformation (CLM). In the CLM dog, note rostral displacement of the caudal cerebellum by the supraoccipital bone *(white arrow)*, obliteration of the dorsal subarachnoid space at the cervicomedullary junction *(red arrow)*, and the syrinx cavity starting at the level of the C2 cervical vertebra *(blue arrow)*.

Laboratory Findings

In the absence of concurrent disease processes, CSF analysis is usually normal; occasionally, a mild mononuclear pleocytosis will be apparent.

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DIFFERENTIAL DIAGNOSIS

In addition to the questionable significance of some of the MR "abnormalities" associated with CLM (some may be breed-associated normal variants), clinical diagnosis of this disorder is made further complicated by the fact that a large subset of dogs with definitive MRI evidence of CLM and SM display no obvious clinical signs associated with their anatomic abnormalities. It is important to realize that, especially in the CKCS breed, other conditions may account for some, or all, of the clinical signs identified. More than 40% of CKCS dogs with MRI evidence of CLM/SM are asymptomatic for the disorder, according to their owners. The severity and rate of progression of CLM in dogs are variable, ranging from asymptomatic (i.e., finding evidence of CLM while imaging for some other reason, or as a breed screening study) to extreme pain and debilitation with rapid worsening over a short period. Additionally, some dogs with CLM have other, unrelated concurrent disorders (e.g., disc extrusion, inflammatory brain disease) that could explain observed clinical signs. In such situations, it may be difficult to discern whether CLM is the main problem or is contributory or an incidental finding. Finally, other disorders of the craniocervical junction (see Chapter 40) can occur concurrently with or can be mistaken for CLM in dogs.

MEDICAL MANAGEMENT

Medical therapy for dogs with CLM generally falls into three categories: analgesic drugs (implies relief of dysesthesia/paresthesia also), drugs that decrease CSF production, and glucocorticoid therapy. The most useful drug available for relief of scratching activity associated with SM has been gabapentin (see Table 39.1). It has been shown that neuropathic pain is accentuated over time owing to upregulation of α2δ-1 subunit voltage-gated calcium channels in dorsal root ganglion neurons and dorsal horn nociceptive neurons of the spinal cord. Gabapentin and the newer gabapentin analog, pregabalin, are believed to exert their antinociceptive effects by selectively binding to the α2δ-1 subunit and inhibiting calcium influx in these neurons. Side effects of gabapentin are minimal, usually restricted to mild sedation, pelvic limb ataxia, and weight gain.

Pregabalin (see Table 39.1) may be a more effective drug in relieving pain and scratching activity in CLM dogs. Because the half-life of elimination of pregabalin is nearly twice as long as that of gabapentin, twice a day dosing is possible. It is important to start at the low end of the dose range to avoid side effects of sedation and ataxia. Orally administered opiate drugs are sometimes helpful in alleviating neck and head pain in CLM dogs. Oral tramadol (2–4 mg/kg every 8–12 hours) may also be useful. A number of drugs aimed at decreasing CSF production have been used in CLM patients in an effort to diminish CSF pulse pressure. All information regarding efficacy of these drugs is anecdotal. They include omeprazole (a proton pump inhibitor), acetazolamide (a carbonic anhydrase inhibitor), and furosemide (a loop diuretic). More specific information regarding these drugs is covered in the congenital hydrocephalus discussion. Prednisolone is often used in medical management of CLM. Potential benefits include antiinflammatory effects, decreased CSF production, and decreased substance P (a nociceptive neurotransmitter) expression in spinal cord dorsal horn neurons. An initial anti-inflammatory dose of prednisolone (0.5 mg/kg orally every 12 hours) is often effective in controlling clinical signs. This dose should be tapered, if at all possible, to an every-other-day schedule within the first month of therapy. In most cases of CLM, medical therapy will diminish the severity of clinical signs, but resolution is unlikely.

SURGICAL TREATMENT (Videos 39.4 and 39.5)

Preoperative Management

See p. 1338 for preoperative management of the brain surgery patient.

Anesthesia

See p. 1340 for anesthetic management of the brain surgery patient. A guarded endotracheal tube should be used because of the extreme neck flexion often required for this procedure.

Surgical Anatomy

See Chapter 38 and p. 1342 for surgical anatomy of the brain and skull.

Positioning

Positioning is as described for suboccipital craniotomy (p. 1344). Because the head needs to be ventroflexed approximately 90 degrees, a guarded endotracheal tube should be used.

SURGICAL TECHNIQUE

In people with symptomatic Chiari type I malformation, surgical therapy is considered the treatment of choice, and FMD is the preferred surgical procedure. Adjunctive surgical procedures are occasionally performed in people who have had a suboptimal response to FMD; such procedures usually involve placement of a shunt to divert SM fluid from the spinal cord region to another location for absorption (e.g., pleural or peritoneal cavity, subarachnoid space). Although a high degree of success has been associated with surgical management of Chiari type I malformation in people, the reoperative rate varies from 8% to 30% for FMD; the most common problem necessitating reoperation is excessive scar tissue formation at the FMD site, causing compression at the cervicomedullary junction and effectively recreating the original disease state.

CLM is a surgical disorder. Based on more than 100 operated cases with long-term (i.e., more than 3 years) follow-up, the preferred surgical procedure for treatment of CLM in dogs is FMD with titanium mesh/PMMA cranioplasty. This procedure involves a suboccipital craniotomy and laminectomy of C1, with subsequent placement of a titanium mesh/PMMA plate on titanium screw anchor posts inserted around the circumference of the occipital bone defect (Fig. 39.20A–B). *Once the FMD is completed, incise the meninges (dura/arachnoid) on the midline and resect the meningeal tissue in the region of the FMD.* There is typically a fibrous band of tissue at the cervicomedullary junction that may be partially ossified. *Transect this band carefully with a probe (Gross ear hook and spoon) and a sharp No. 11 blade. Drill four to six guide holes in the occipital bone around the periphery of the skull defect using a 1.1-mm drill bit (Fig. 39.21A). Insert self-tapping 6-mm long (1.5-mm wide) screws into the guide holes for an approximate depth of 2 to 3 mm (Fig. 39.21B). Release the dog's head from the flexed position and reposition it at a normal resting angle. Fashion the skull plate using titanium mesh and PMMA and fix it to the back of the skull, using the titanium screw heads as anchor posts for the PMMA (Fig. 39.21C). Shape the plate somewhat like a guitar pick with the wide end of the pick toward the occiput. Apply only a thin layer of PMMA to both*

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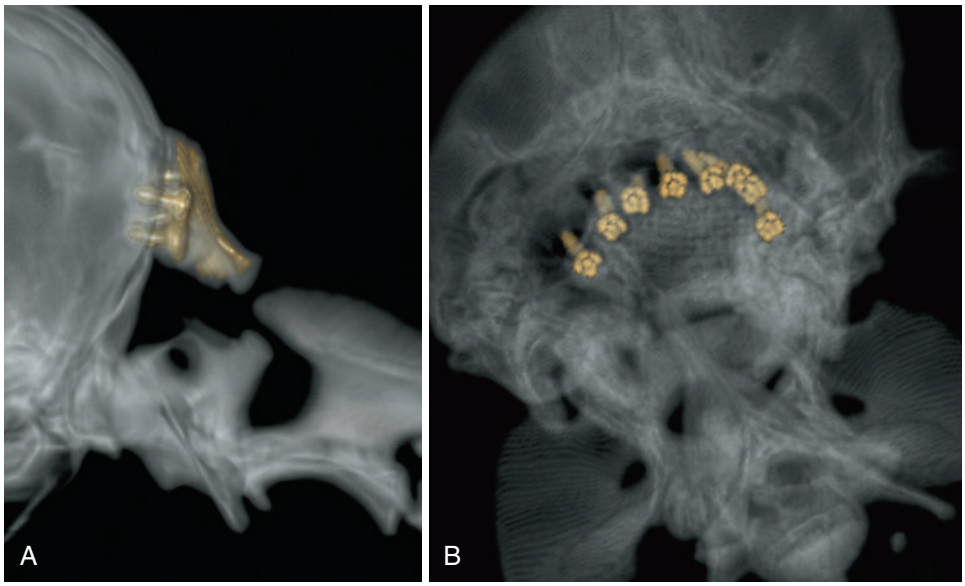


FIG. 39.20 (A) Lateral and (B) caudal three-dimensional reconstructed computed tomography postoperative views of a dog's skull following foramen magnum decompression with cranioplasty for Chiari-like malformation.

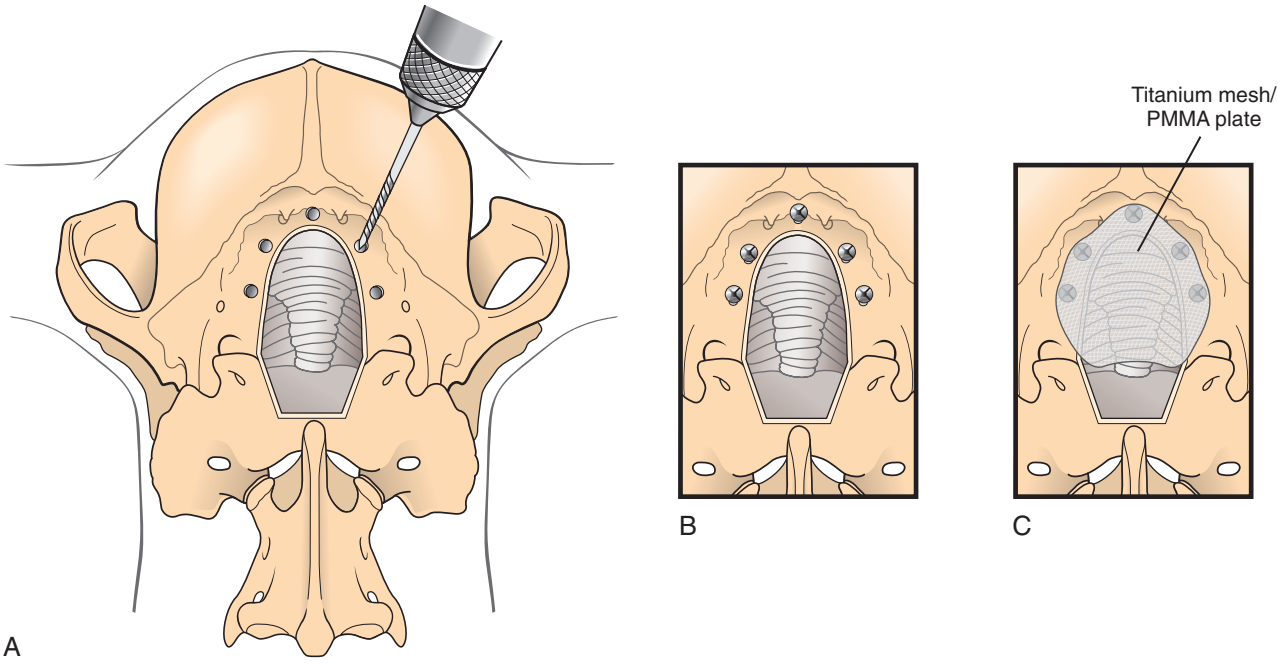


FIG. 39.21 (A) Typical pattern of guide hole placement for titanium screws. (B) Placement of self-tapping titanium screws in guide holes. (C) Placement of titanium mesh/polymethylmethacrylate plate to the foramen magnum decompression site. *PMMA,* Polymethylmethacrylate.

surfaces of the plate, with some PMMA extending beyond the edges of the titanium mesh to adhere to the titanium screw heads. Extend the caudal aspect of the plate slightly over the dorsal defect of C1; curve the tail aspect of the plate dorsally to avoid impinging on the medulla or the cranial cervical spinal cord. Close the skin routinely.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

A high-speed air drill, titanium mesh, titanium screws, and PMMA are required for this procedure. The drill guide holes can be made by using a minidriver or a specialized high-speed drill with a 1.1-mm drill bit attachment.

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POSTOPERATIVE CARE AND ASSESSMENT

The patient is placed in a padded cage and analgesics are administered (e.g., fentanyl continuous-rate infusion; see p. 144). Patients generally require some form of oral analgesic therapy for 1 to 2 weeks after surgery. Proper plate application is confirmed via radiographs or (preferably) CT before anesthetic recovery.

PROGNOSIS

The short-term surgical success (sustained improvement of neurologic status and/or pain/scratching relief) rate with FMD in dogs with CLM is approximately 80%, whether or not adjunctive cranioplasty is performed. There appears to be an inverse relationship between the length of time clinical signs are present before surgical intervention and the extent of postoperative improvement. Unfortunately, there appears to be a disease relapse rate ranging from 25% to 47% of cases treated with FMD alone; most of these relapses are suspected to be due to excessive postoperative scar tissue formation at the FMD site. After surgery, clinical signs of pain are routinely relieved, but scratching activity tends to persist. A cranioplasty procedure (based on a similar procedure used in human FMD surgery) was developed by the author and colleagues to discourage excessive postoperative scar tissue from recompressing the operative site. Our most recent analysis of more than 100 cases of FMD with cranioplasty determined the reoperative rate to be 7% (unpublished data). A 2015 report described long-term (1- to 3-year follow-up) success in 23 dogs with CLM/SM treated via FMD with duraplasty (using lyophilized swine submucosa) and autogenous fat grafting; the authors of this study reported some level of improvement in 94% of patients.[9]

Little information is available regarding the prognosis for CLM in dogs treated medically alone. Most dogs with CLM will respond favorably to medical therapy, although in many cases this response is temporary. In general, between one-third and one-half of dogs symptomatic for CLM/SM seem to worsen over 1 to 2 years from the time of diagnosis, based on prior small studies and the authors' experience. In one larger, long-term prospective study of 48 CKCS dogs that evaluated the response to nonsurgical (medical only) management over a mean time period of 39 months, 25% of patients either remained static or improved, whereas 75% of the dogs deteriorated clinically over time. Approximately 15% of the dogs were euthanized due to clinical signs of severe neuropathic pain.[10] Although the surgical success rate appears generally favorable for CLM in dogs, the recurrence rate due to excessive postoperative scar tissue formation has been unacceptably high with FMD alone. It appears that cranioplasty and duraplasty have improved the success rate of FMD, but no surgical procedure has yet been demonstrated to consistently eliminate clinical signs associated with Chiari malformation in humans or dogs.

BRAIN TUMORS

DEFINITIONS

Brain tumors include neoplastic diseases involving the brain, including those arising from the brain and meningeal coverings *(primary brain tumors),* and those arising from local or distant (i.e., metastatic) anatomic structures *(secondary brain tumors).*

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

A discussion of the mechanisms involved in cancer development is beyond the scope of this textbook. Brain tumors cause clinical signs of disease primarily by physically impinging on neighboring brain tissue and increasing ICP. Peritumoral edema adds to this phenomenon. Brain tumors may cause brain compression and increased ICP via obstructive hydrocephalus and hemorrhage, both of which can lead to rapid exacerbation of neurologic dysfunction. Primary brain tumors are commonly encountered in dogs and cats. In both species, meningioma is the most commonly reported tumor. Gliomas (e.g., astrocytoma, oligodendroglioma) are frequently reported in dogs and are occasionally reported in cats. Other primary brain tumors less commonly reported in dogs include choroid plexus tumor, primary CNS lymphosarcoma, primitive neuroectodermal tumor (which typically includes neuroblastoma), primary CNS histiocytic sarcoma (also termed *malignant histiocytosis*), and vascular hamartoma. Other primary brain tumors occasionally encountered in cats include, in addition to gliomas, ependymomas, olfactory neuroblastomas, and choroid plexus tumors. Case reports of medulloblastomas in both dogs and cats usually involve the cerebellum. Secondary brain tumors in dogs and cats include hemangiosarcoma, pituitary tumor, lymphosarcoma, metastatic carcinoma, and invasive nasal/frontal sinus tumor.

DIAGNOSIS

Clinical Presentation

Signalment

Dogs and cats diagnosed with brain tumors tend to be older. The median age for dogs with primary brain tumors is approximately 9 years, and for cats it is over 10 years of age. In general, large-breed dogs tend to develop brain tumors more commonly than small-breed dogs. Golden retrievers and boxer dogs appear to be predisposed to primary brain tumors. Golden retrievers are prone to developing meningiomas, whereas boxer dogs and other brachycephalic breeds (e.g., Boston terriers) are more likely to be diagnosed with glioma. More than half of brain tumors diagnosed in cats are meningiomas. In both dogs and cats, patients with meningiomas tend to be somewhat older at diagnosis than those with other brain tumors types. In dogs, astrocytomas are more likely than other primary brain tumors to occur in the diencephalon and cerebellum. Choroid plexus tumors may occur in the lateral, third, or fourth ventricle. The mean age of patients with secondary brain tumors is similar to that for patients with primary brain tumors (9–10 years). Mixed breed dogs are most commonly affected, followed by golden retrievers and Labrador retrievers.

History

Historical complaints vary according to species, tumor location, and tumor aggressiveness. Brain tumors are more commonly located in the rostral fossa; therefore abnormal behavior and seizures are the most common clinical complaints. Seizure activity is the most common clinical complaint associated with canine brain tumors, whereas behavior change is the most common clinical complaint associated with feline brain tumors. With slow-growing primary brain tumors (i.e., meningioma), the owner may retrospectively (i.e., after the diagnosis is attained) realize that the pet had been exhibiting signs of altered behavior

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for a protracted period (sometimes up to a year); initially, such vague clinical signs may be attributed by pet owners to "old age." Clinical signs associated with tumors in the caudal fossa usually include loss of balance and abnormal behavior (usually obtunded mental status).

Physical Examination Findings

Neuroanatomic localization of the brain tumors is typically reflected in the signs of neurologic dysfunction exhibited by the patient. However, it is not uncommon for dogs and cats with solitary brain neoplasms to display dysfunction of more than one region of the brain (i.e., combination of forebrain and brainstem dysfunction); this phenomenon is likely due to the size of some of these tumors and to the extent of peritumoral edema. Because approximately half of canine primary brain tumors occupy more than one anatomic region of the brain, it is possible to falsely conclude, based on neurologic examination, that a patient with a solitary brain mass has multifocal disease.

NOTE Nearly one-fourth of dogs with primary brain tumors have concurrent, unrelated neoplasia (e.g., pulmonary carcinoma, hemangiosarcoma), most of which involves the thoracic or abdominal cavity. Be certain to screen for concurrent unrelated neoplasia (via thoracic radiography and abdominal ultrasonography) before pursuing advanced diagnostics and definitive therapy for brain tumors.

In cats with a primary brain tumor, nonspecific presenting clinical signs (i.e., signs not obviously referable to neurologic dysfunction) occur in more than 20%. These clinical signs include lethargy, inappetence, and anorexia. Multiple intracranial meningiomas have been reported to occur in cats with some frequency (approximately 17%). Most dogs and cats with brain tumors display evidence of head and neck pain on palpation.

Diagnostic Imaging

MRI is the preferred imaging modality for the diagnosis of canine and feline brain tumors. In general, meningiomas appear as extraaxial, uniformly contrast-enhancing masses with broad-based attachment to the skull and well-delineated borders (Fig. 39.22), whereas gliomas tend to be intraaxial, nonuniformly contrast-enhancing masses with indistinct margins between mass and normal brain tissue (Fig. 39.23). Meningeal contrast enhancement evident on MRI of the brain has been described but is not specific for brain tumors. The "dural tail" sign is an MRI feature typically associated with meningiomas, in which a contrast-enhancing meningeal-associated "tail" is seen extending from the main tumor mass (Fig. 39.24).

Laboratory Findings

Because CSF analysis tends to yield fairly nonspecific information in brain tumor cases, obtaining CSF from dogs and cats when the imaging is highly supportive of a brain tumor may not be worthwhile.

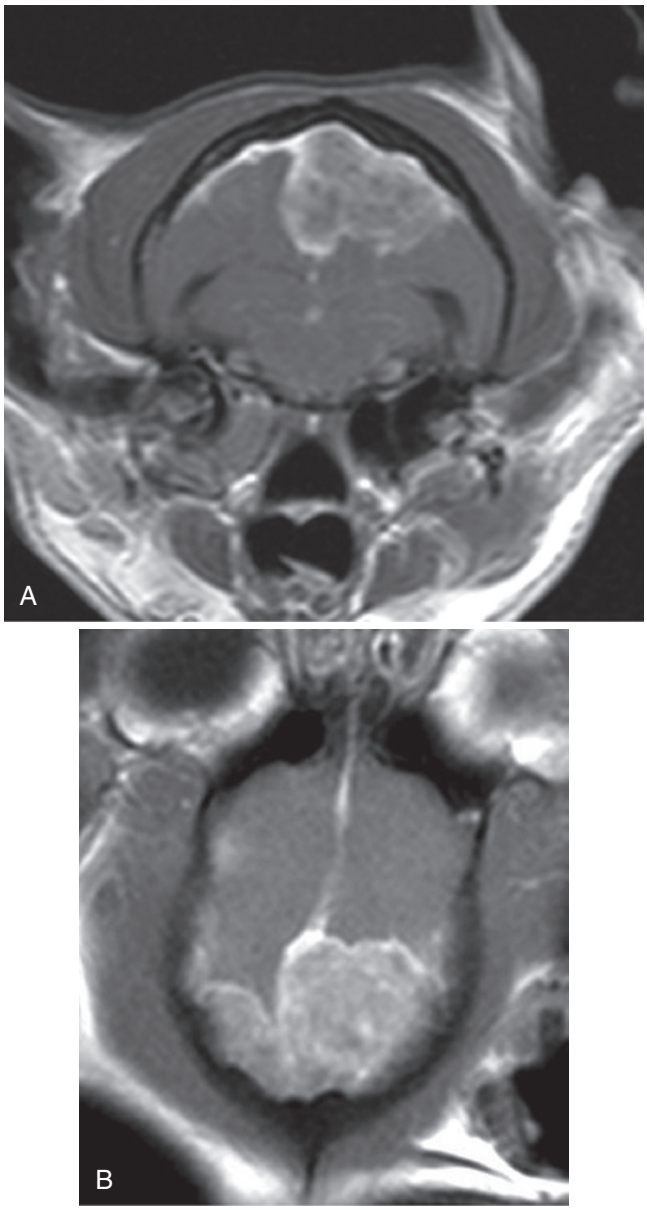


FIG. 39.22 (A) Axial and (B) dorsal (coronal) T1-weighted with contrast magnetic resonance brain images of a cat with a cerebral meningioma.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for brain tumors in dogs and cats depend primarily on clinical signs and neuroanatomic localization. For example, in an older dog with recent onset of seizures and no neurologic deficits, the main differential diagnosis other than brain tumors is late-onset idiopathic epilepsy. In an older dog with clinical signs of dementia and circling behavior, a major differential diagnosis would be the canine analog of Alzheimer's disease of people: canine cognitive dysfunction. Other differential diagnoses in suspected brain tumor patients include metabolic encephalopathy, inflammatory/infectious disease (e.g., GME), toxin exposure, and ischemic/vascular events (in cases with acute onset of neurologic dysfunction).

MEDICAL MANAGEMENT

Medical management for dogs and cats with brain tumors may be supportive or definitive. Supportive therapy refers to treatment that addresses the secondary effects of a tumor, not the tumor itself. Such therapy includes low doses of oral prednisolone (e.g.,

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FIG. 39.23 Axial T2-weighted magnetic resonance image of a dog with an intracranial astrocytoma.

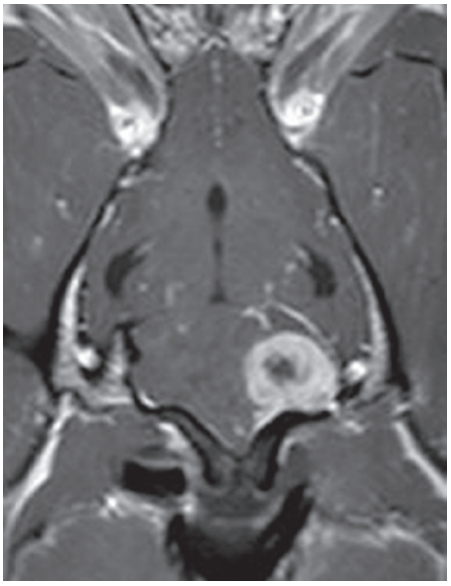


FIG. 39.24 Dorsal (coronal) T1-weighted with contrast magnetic resonance image of a cerebellar meningioma in a dog with a dural tail.

0.5 mg/kg every 12 hours) to decrease peritumoral edema and antiseizure medication, if necessary (see Table 39.1). Definitive medical therapy is directed at killing tumor cells and includes radiation therapy and chemotherapy. The traditional or conventional method of delivering definitive megavoltage radiation therapy of brain tumors is administration of fractionated daily doses of 2.5 to 4 Gy on a Monday through Friday (once a day) schedule for 4 weeks. Protocols vary, but most involve administration of a total cumulative dose in the range of 30 to 50 Gy. In recent years, stereotactic radiation therapy techniques (e.g., Gamma knife, Cyberknife) have been gaining in popularity; these methods of more anatomically precise (less chance of inadvertent radiation delivery to surrounding normal parenchyma) radiation delivery allow for fewer treatment sessions (one to five treatments) to deliver the same total dose as with traditional fractionated radiation therapy.

Oral chemotherapy is often used to treat dogs with brain tumors. Hydroxyurea is used for meningiomas and lomustine for gliomas (Box 39.1). Bone marrow suppression and delayed dose-related hepatotoxicity may occur with oral lomustine use. Although hepatoxocity has been reported in approximately 6% of cases, it may occur more frequently; this drug should be used cautiously. Hydroxyurea is an oral chemotherapeutic agent with efficacy against intracranial meningiomas in people (see Box 39.1). Proposed mechanisms of action of hydroxyurea include induction of tumor cell apoptosis and inhibition of ribonucleotide diphosphate reductase (with subsequent interference with DNA synthesis). Mild to moderate bone marrow suppression is a potential side effect of hydroxyurea use in people; to date, this side effect has not been an important issue in dogs.

BOX 39.1 Medical Therapy for Brain Tumors in Dogs

Prednisolone

0.5–1.0 mg/kg PO q12h; may be able to taper to every other day after 1 month if treated with chemotherapy and/or radiation therapy

Hydroxyurea

20 mg/kg PO q24h

Lomustine

60 mg/m^2 PO every 6–8 wk. Potential adverse effects include bone marrow suppression and hepatotoxicity.

Megavoltage Radiation

2.5–4.0 Gy administered to tumor or tumor bed (postoperatively) for 15–20 treatments (total of 30–50 Gy) vs. stereotactic radiation therapy (total dose delivered in 1–5 treatments)

Antiseizure Drugs

See Table 39.1.

SURGICAL TREATMENT

Preoperative Management

See p. 1338 for preoperative management of the brain surgery patient.

Anesthesia

See p. 1340 for anesthetic management of the brain surgery patient.

Surgical Anatomy

See Chapter 38 and p. 1342 for surgical anatomy of the skull and brain.

Positioning

See descriptions of specific surgical approaches (pp. 1342–1344) for patient positioning.

SURGICAL TECHNIQUE

Surgical approaches to the brain necessary to access brain tumors in various locations are discussed on pp. 1342 to 1344.

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Once the brain is exposed, sharply incise the meninges (dura/arachnoid) with a No. 11 blade, over the region of the mass using a combination of a probe (e.g., Gross ear hook and spoon) and a No. 11 blade. Create a meningeal flap using Bishop-Harmon forceps (no teeth) and tenotomy scissors to assist with tumor removal and to decrease bleeding tendencies (compared with incising the meninges directly over the mass). Incise below the mass, create a flap, and pull it dorsally. Using a combination of a probe and a lens loop, find a plane between the tumor and surrounding brain tissue, and attempt to free the tumor edges from the brain for the entire circumference of the mass. When applying traction to a tumor, always direct the traction away from the brain to minimize brain trauma and inadvertent increases in ICP. Use moistened, lint-free cottonoid sponges on the brain surface to keep it moist and decrease surface hemorrhage. In some firm tumors, place a stay suture into the mass and use it to help pull the mass outward while dissecting around its medial aspect. In some tumors (e.g., infiltrative tumors, tumors near the dorsal sagittal sinus), it may be beneficial to use a CUSA to assist in mass removal. *Once the tumor is removed, resect abnormally colored tissue in the tumor bed with suction or a CUSA. Control tumor bed hemorrhage with bipolar cautery, as well as placement of moistened Gelfoam and/or Surgicel into the defect. Do not close the incised or resected meninges unless the frontal sinus has been entered.* See Standard Surgical Approaches to the Brain (pp. 1342–1344) for a description of skull reconstruction. *Close subcutaneous tissues and skin routinely.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

In addition to a high-speed air drill, instruments that are essential for successful brain tumor removal include probes (e.g., Gross ear hook and spoon), lens loops, bipolar cautery, and Bishop-Harmon forceps. A CUSA may be helpful for removal of infiltrative masses and/or masses adjacent to large blood vessels. Intraoperative ultrasound is helpful in locating intraaxial neoplasms.

POSTOPERATIVE CARE AND ASSESSMENT

The patient is placed in a padded cage in an intensive care unit and is administered analgesics and antiseizure drugs as needed. They should be turned frequently. Postoperative vocalization is common in dogs after brain tumor removal, and dementia may be mistaken for pain, leading to overmedication in these patients. Oversedation and resultant prolonged recovery may predispose these dogs to atelectasis and subsequent pneumonia. Postoperative pneumonia is the most common and most severe postoperative complication of brain tumor removal in dogs and is better avoided than treated. PCV should be monitored frequently in cats after surgery.

PROGNOSIS

Most dogs and cats with brain tumors respond favorably to supportive therapy; however, such therapy is typically associated with very short survival times (approximately 1–4 months). In one study evaluating survival times in 51 dogs with primary brain tumors treated with supportive therapy alone, the median survival time after hospital discharge was 69 days; median survival time was significantly longer for dogs with supratentorial tumors (178 days), compared with those with infratentorial tumors (28 days).[11] For cats with surgical removal of meningiomas, the median survival time has previously been thought to be approximately 2 years; however, in a 2015 multicenter retrospective study of 121 cats with intracranial meningiomas that were surgically excised, the median survival time was 37 months.[12] The majority of deaths in the aforementioned study were due to causes unrelated to the meningioma. Regrowth of intracranial meningioma in cats usually occurs in the original tumor location. The success rate of reoperation of feline intracranial meningioma is similar to the reported success rate of first-time operation of these tumors; thus reoperation of a recurrent feline intracranial meningioma is a logical therapeutic option.

Survival rates reported for canine brain tumors with different modes of definitive therapy are quite variable. Part of this variability may be due to the inclusion of different tumor types and different variations of therapy within study populations. The tendency for these published rates to improve in general over time may reflect advancements in our ability to treat these tumors. In general, the prognosis for survival of dogs with intracranial neoplasia treated with surgery alone is poor. Canine meningiomas are generally more aggressive than feline meningiomas and tend to be more invasive. Canine meningiomas tend to recur within a year of surgery if adjunctive treatment is not pursued. Dogs with intracranial meningiomas treated with only hydroxyurea and prednisolone (i.e., no surgery or radiation therapy) have an average survival time of approximately 7 months, nearly twice the survival time of dogs treated with prednisolone alone. Survival times are longer for dogs with supratentorial versus infratentorial tumors. No information is available regarding survival times of dogs with intracranial gliomas treated by surgical removal. Dogs with gliomas treated with lomustine have survival times ranging from several months up to approximately 1 year. Megavoltage radiation therapy for meningiomas has generally been associated with average survival times between 1 and 3 years, although more recent studies (discussed later) suggest longer survival times. For gliomas, survival times reported for dogs treated with megavoltage radiation tend to average less than 1 year (approximately 10 months). Combined surgery and postoperative radiation therapy for canine meningiomas result in survival times of 1.5 to longer than 3 years. In one retrospective study of 31 dogs with intracranial meningioma treated via radiation (total dose of 45–54 Gy) alone (21 dogs, traditional fractionated protocol) or surgery and radiation (10 dogs), the overall median survival was 577 days (906 days when only dogs dying due to meningioma were considered); there was no significant difference in survival between the two groups.[13] In another retrospective study evaluating a stereotactic radiation delivery protocol (single median radiation dose of 15 Gy), 38 dogs with intracranial meningioma had a median survival time of 399 days (493 days when only dogs dying due to meningioma were considered).[14] Limited published data address the prognosis associated with the combination of surgery and oral chemotherapy for dogs with intracranial meningioma. These patients tend to survive for longer than a year, but not as long as those treated with postoperative radiation therapy or radiation therapy alone. In addition, small-breed dogs tend to have minimal postoperative complications (e.g., pneumonia) compared with large-breed dogs, and dogs with olfactory bulb meningiomas tend to have less postoperative morbidity and longer survival times than dogs with meningiomas located more caudally in the brain. With the exception of pituitary tumors and multilobular osteochondrosarcomas, the prognosis for secondary brain tumors in dogs and cats is generally considered to be poor.

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COMPLICATIONS

The major postoperative complication for intracranial meningioma in dogs is pneumonia; this complication occurs in roughly 20% of patients. Postoperative pneumonia in these patients has an approximately 50% mortality rate. The most common postoperative complication in cats with intracranial meningioma is anemia. The reason for the anemia is unknown, but it virtually always responds to blood transfusion. It is important to monitor cats' PCVs every 4 to 6 hours for the first 24 hours after surgery and to transfuse if indicated (usually if PCV drops below 20%).

TRAUMATIC BRAIN INJURY

DEFINITION

Traumatic brain injury is brain damage due to externally applied trauma; it is mediated by direct (e.g., force of impact) and indirect (e.g., hemorrhage, edema, secondary inflammatory processes) factors.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Severe traumatic brain injury in dogs and cats is an unfortunately frequent occurrence and is often associated with a guarded to poor prognosis, even with aggressive management. Much of the information regarding therapy for these patients is adapted from the human literature, although several veterinary-specific studies have been reported. In addition to the challenge of choosing and administering appropriate medical therapy for acute severe brain injury, a decision regarding whether or not surgical intervention is indicated for a particular patient is also essential. Although some differences in opinion have been expressed over what constitutes "appropriate" medical therapy for severe brain injury in veterinary and human patients, there is general agreement on most available treatment options. Less agreement has been noted among clinicians with respect to surgical intervention for severe brain injury, particularly in scenarios in which surgical therapy is used as a decompressive maneuver in the absence of intracranial hemorrhage.

Brain injury can be conceptually divided into primary and secondary injury. *Primary brain injury* occurs immediately following impact and initiates a number of biochemical processes, which result in *secondary brain injury*. Both primary and secondary brain injury contribute to increased ICP. Primary brain injury refers to the physical disruption of intracranial structures that occurs immediately at the time of the traumatic event. Such injury includes direct damage to brain parenchyma, such as contusions, lacerations, and diffuse axonal injury. Damage to blood vessels may result in intracranial hemorrhage and vasogenic edema. Skull fractures can contribute to continued trauma to the brain parenchyma and blood vessels, especially if they are unstable (Fig. 39.25). The extent of primary brain injury is a function of the force of impact. Acceleratory and deceleratory forces of both the impacting object(s) and the intracranial contents will affect overall tissue damage. Direct parenchymal damage associated with primary brain injury is generally beyond the control of the clinician. However, stabilization of skull fractures and evacuation of intracranial hemorrhage may decrease morbidity associated with primary injuries. In addition to continued hemorrhage and edema, damage caused by the primary brain injury activates a number of interrelated biochemical pathways that act in concert to perpetuate further brain tissue damage and subsequent increases in ICP. These processes constitute secondary brain injury.

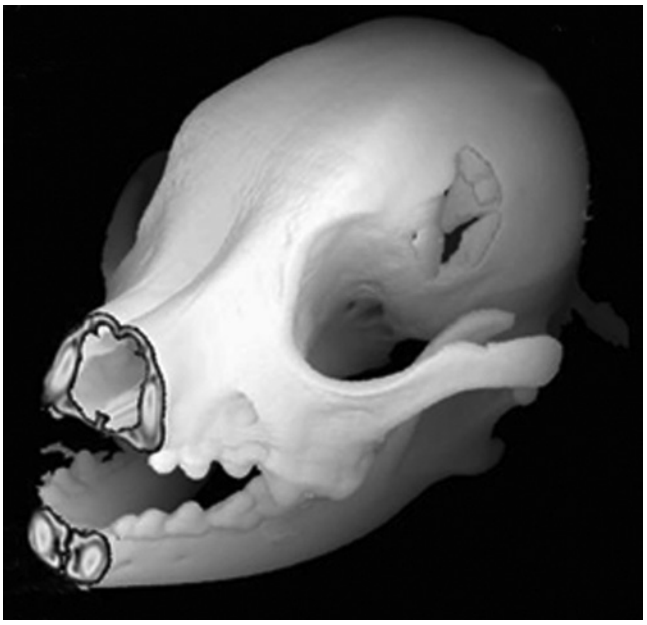


FIG. 39.25 Three-dimensional reconstructed computed tomography image of a dog with a depressed skull fracture. (Courtesy Dr. Charles Vite.)

Adenosine triphosphate depletion disrupts maintenance of cellular ionic homeostasis. Sudden, uncontrolled intracellular influx of sodium (Na^+) and calcium (Ca^{++}) occurs. Cellular swelling (cytotoxic edema) and depolarization result. Uncontrolled depolarization leads to the release of large amounts of glutamate, an excitatory neurotransmitter, into the extracellular environment. Glutamate causes further increases in intracellular Ca^{++} levels. Elevated Ca^{++} levels activate a number of tissue-damaging pathways, including the arachidonic acid cascade (phospholipase A_2 activation) and the xanthine oxidase (free radical producing) pathway. Iron (Fe^{++}) is a vital cofactor in the xanthine oxidase pathway, and free radical species generated via the Fenton reaction (e.g., hydroxyl and superoxide radicals) are preferentially damaging to cell membranes containing high levels of polyunsaturated fatty acids and cholesterol. Brain tissue is rich in both Fe^{++} and membranes with high levels of polyunsaturated fatty acids and cholesterol. Intraparenchymal hemorrhage also increases the amount of Fe^{++} available for perpetuation of oxidative damage. Free radical species are thus particularly damaging to neuronal membranes and probably play a major role in secondary brain injury. Their production is also induced by ischemia, arachidonic acid metabolites, catecholamine oxidation, and activated neutrophils. Other secondary autolytic processes induced after severe head trauma include the complement, kinin, and coagulation/fibrinolytic cascades. Elevated levels of nitric oxide and various cytokines (e.g., tumor necrosis factor, interleukins) also contribute to parenchymal injury in the damaged brain. Most of the mediators of tissue damage produced by these various reactions perpetuate their own continued production, as well as the production of other mediators. Maintenance of an ischemic environment perpetuates the previously mentioned processes and leads to the accumulation of lactic acid (via anaerobic glycolysis). Lactic acid

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accumulation leads to further damage to brain tissue. Hypotension and hypoxemia, extracranial conditions that are common in the traumatized patient, can worsen brain ischemia, thereby enhancing the events responsible for secondary brain injury. The end result of these secondary processes is increased ICP. In contrast to primary brain injury, the clinician has some control over secondary brain injury.

DIAGNOSIS

Clinical Presentation

Signalment

Traumatic brain injury affects both dogs and cats, of any age and both sexes.

History

The history typically consists of a witnessed traumatic event, such as automobile trauma (hit by car), attack by a larger animal, fall from a height, or a projectile injury (e.g., gunshot).

Physical Examination Findings

Depending on the extent of trauma involved, physical examination findings may range from no apparent extracranial abnormalities and minor aberration of consciousness (see p. 1323) to severe multiorgan injury and coma.

Diagnostic Imaging

Imaging of the patient's head is often indicated, especially in those animals that fail to respond to aggressive medical therapy, or that deteriorate after responding to such therapy. Skull radiographs are unlikely to reveal clinically useful information in cases of severe head trauma but on occasion may reveal evidence of depressed fractures of the calvaria (Fig. 39.26). CT is the preferred modality for imaging the head in cases of severe brain injury. CT is preferred over MRI in head trauma cases for several reasons. CT images are obtained much more quickly than MR images (an important advantage in the critical patient scenario), patients may be more closely monitored with standard monitoring systems during CT than during MRI because of the large magnetic field required for MRI, and acute hemorrhage and bone are better visualized with CT than with MRI (Fig. 39.27). MRI can, however, provide additional prognostic information for head trauma patients. In one retrospective case series of dogs experiencing head trauma, MRI images were obtained within 48 hours of injury; in this investigation, it was found that T2 and fluid attenuation inversion recovery (or FLAIR) sequences provided the most clinically useful information, and that injuries involving the caudal fossa or both the caudal and rostral fossae had poorer outcomes.[15] In a retrospective study evaluating MRI results (MR images obtained within 2 weeks after trauma) in 50 dogs that had experienced severe traumatic brain injury, the MRI grade (based on factors such as lesion location, presence of midline shift, herniation) was negatively correlated with prognosis.[16]



FIG. 39.26 Lateral skull radiograph of a dog's head following severe traumatic brain injury, showing displaced skull fractures.

Laboratory Findings

Emergency bloodwork (e.g., PCV/total solids, Azostix, blood gas) should be performed, specifically focusing on evidence of trauma-related abnormalities (e.g., anemia). Admission hyperglycemia has been shown to be a poor prognostic indicator in dogs with severe traumatic brain injury.

DIFFERENTIAL DIAGNOSIS

In cases of traumatic brain injury in which the occurrence of trauma is obvious (historical and physical evidence of external trauma), there are no reasonable differential diagnoses.

MEDICAL MANAGEMENT

Most cases of traumatic brain injury in dogs and cats present with varying degrees of cardiovascular shock. Emergency management of the shock state takes precedence (ABCs of shock therapy) over focusing on the brain injury. Specific therapies for suspected brain swelling are discussed on pp. 1338 to 1342.

SURGICAL TREATMENT (Video 39.6)

Preoperative Management

Once normovolemia and appropriate oxygenation/ventilation have been established, the brain injury patient should be more

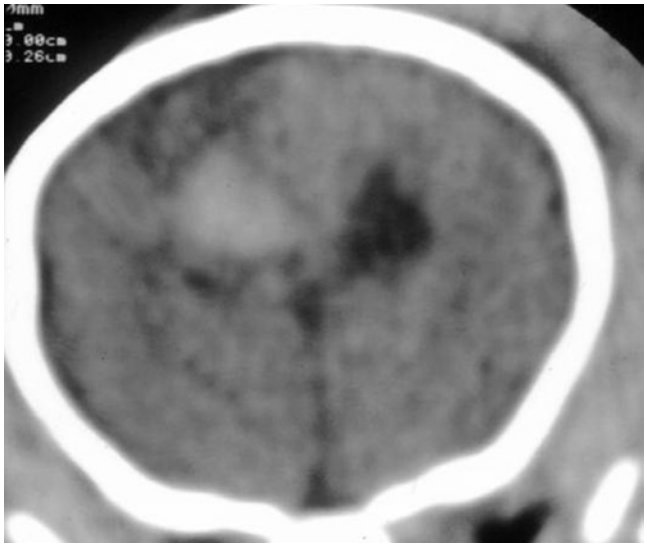


FIG. 39.27 Axial noncontrast computed tomography image of a dog that experienced traumatic brain injury, showing an area of intraparenchymal hemorrhage.

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carefully assessed for other injuries to the nervous system (e.g., vertebral fractures/luxations), as well as to other body systems (i.e., lungs, abdominal organs, musculoskeletal system). A complete neurologic examination should be performed at this time. Specific medical therapy for brain injury should begin coincident with the secondary assessment (see pp. 1338–1342). Additional bloodwork, as well as radiographs, may be warranted.

Anesthesia

See p. 1340 for anesthetic management of the brain surgery patient.

Surgical Anatomy

See Chapter 38 and p. 1342 for surgical anatomy of the skull and brain.

Positioning

See descriptions of specific surgical approaches (pp. 1342–1344) for patient positioning.

SURGICAL TECHNIQUE

Surgical approaches used to decompress the brain, remove depressed skull fractures, and evacuate intracranial hemorrhage are typically the rostrotentorial or suboccipital approaches, depending on the location of the lesion(s). These approaches are described on pp. 1342 to 1344. When surgical decompression is required for brain swelling in brain injury cases, even if large pieces of skull need to be removed, the craniotomy defect should not be reconstructed. The brain should be allowed to expand in such cases, rather than be confined in a closed compartment. If the patient does well, the skull can be reconstructed at a later date, if indicated.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Intraoperative ultrasound is a useful tool in brain injury cases in which an intraparenchymal hematoma needs to be located for surgical removal.

POSTOPERATIVE CARE AND ASSESSMENT

The patient is placed in a padded cage in an intensive care unit and is administered analgesics and antiseizure drugs as needed. Posttraumatic seizure activity is a concern in dogs experiencing traumatic brain injury, but does not appear to be a common phenomenon in cats.[17,18] Patients should be turned frequently.

PROGNOSIS

The prognosis for survival following severe traumatic brain injury remains guarded to poor overall. Nonetheless, many severely brain-injured dogs and cats make functional recoveries if provided enough time and appropriate postoperative nursing care.

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Surgery of the Cervical Spine

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Weakness of all four limbs due to a cervical myelopathy is **tetraparesis.** When voluntary motor ability is lost to all four limbs, the term used is **tetraplegia. Hemiparesis** and **hemiplegia** are similar terms that refer to weakness of or inability to move the thoracic and pelvic limbs on one side of the body, respectively. The two most common surgical approaches to the cervical spinal cord are the **ventral slot procedure** (creation of a bony defect in the ventral aspect of an intervertebral space in addition to removing a section of the intervening disc) and dorsal laminectomy. The ventral slot procedure provides a small window to the ventral aspect of the spinal cord, but lateral access is limited by both the size of the defect and the large vertebral venous sinuses (internal vertebral venous plexus) on either side of the defect. **Dorsal laminectomy** provides a wide decompression and access to the entire dorsal surface of the cord. Further removal of the articular processes (facets) laterally **(facetectomy)** and/or the pedicles allows for exposure of the nerve roots/spinal nerve and lateral surface of the spinal cord, respectively. Removal of parts of the articular processes to enlarge the intervertebral foramen is called **foraminotomy**. Removal of the dorsal lamina on one side is often performed, usually combined with facetectomy/foraminotomy; this is called **dorsolateral hemilaminectomy. Hemilaminectomy** is the unilateral removal of the lateral surface of the vertebral arch, including the articular processes. Although this can be done in the cervical region, the term is used more commonly when discussing surgery of the thoracolumbar spine (see Chapter 41). **Fenestration** is the creation of a defect (window) in the annulus fibrosus to the level of the nucleus pulposus.

GENERAL CONSIDERATIONS

The range of neurologic dysfunction possible with cervical myelopathies is broad, in terms of both severity and character. Many patients present with severe neck pain as the primary or sole clinical sign of dysfunction. Neck pain is typically more severe with cranial cervical spinal cord lesions than with caudal lesions. Loss of motor function occurs with compressive cervical myelopathies but is less common than with thoracolumbar myelopathies, probably due to the larger volume of the vertebral canal in the cervical region compared with the thoracolumbar region. Ambulatory patients with caudal cervical myelopathy tend to have obvious pelvic limb weakness and ataxia, with less severe, sometimes even subtle, thoracic limb dysfunction. In many such cases, the thoracic limbs move with short, stilted steps, whereas the pelvic limbs display obvious ataxia. This odd gait is often referred to as a *two-engine gait* because of the disparate nature of the thoracic and pelvic limb movements. Patients with cranial cervical myelopathies typically have more obvious thoracic limb dysfunction. A phenomenon called *central cord syndrome* is sometimes appreciated in dogs and cats with caudal cervical spinal cord lesions that are confined to the central region of the cord. In this syndrome, the thoracic limbs display severe lower motor neuron (LMN) weakness, but the pelvic limbs are minimally affected or neurologically normal. This occurs because the centrally located lesion interferes with LMNs innervating the thoracic limbs, but it spares the more peripherally located white matter upper motor tracts that innervate the pelvic limb LMNs. Central cord syndrome is most commonly associated with intraaxial lesions like syringomyelia and neoplasia (see Chapter 38 and pp. 1353 and 1396).

Descending tracts from the medullary respiratory centers traverse the cervical spinal cord to innervate the phrenic nerve LMNs (C5–C7) and the LMNs of intercostal muscles in the thoracic spinal cord. Damage to these tracts can lead to respiratory compromise. Caudal cervical spinal cord lesions that spare innervation to the phrenic nerve LMNs (e.g., lesions caudal to the C5–C7 segments) but disrupt normal activation of intercostal LMNs can lead to the phenomenon of *abdominal breathing.* In this scenario, the diaphragm bears the sole burden of moving air through the lungs because of the denervated chest wall, and this motion causes the abdomen to visibly move back and forth during respiration. Tonic input to the sympathetic LMNs in the thoracolumbar spinal cord that maintain normal blood pressure is provided by neurons in the medulla (rostroventrolateral medulla). Because these medullary neurons send their processes through the cervical spinal cord, cervical myelopathies may be associated with severe hypotension.

Two major surgical approaches to the cervical spinal cord have been identified: ventral and dorsal. A lateral approach to the cervical cord is rarely performed and is not discussed in this text. The ventral approach is technically less demanding than the dorsal approach, and considerably more bone is available for implants ventrally versus dorsally when vertebral stabilization is required. Exposure of the vertebral canal and the spinal cord is very limited with a ventral approach, and the likelihood of severe venous sinus hemorrhage is much greater than with a dorsal approach. The dorsal approach allows for wide decompression and access to dorsal and lateral aspects of the cervical spinal cord. This procedure is technically more difficult than the ventral approach, and patients with dorsal cervical laminectomies tend to have longer recuperative periods than those who undergo ventral slot procedures. The dorsal spinous processes in the cervical region are short (and are typically removed during

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ABSTRACT

Disorders of the cervical spine are common in small animal practice and can be challenging to treat. Type I disk ruptures are frequently encountered and can be successfully managed via both ventral (ventral slot) and dorsal (dorsal laminectomy, facetectomy) approaches. In addition to disk disease, other fairly frequently encountered cervical spinal cord problems include congenital abnormalities (e.g., atlantoaxial instability), spondylomyelopathy ("wobbler syndrome"), neoplasia and traumatic fracture/luxation.

Other surgical techniques for dealing with cervical spine abnormalities include implant placement for unstable segments (e.g., atlantoaxial instability, trauma).

KEYWORDS

Cervical spine
ventral slot
dorsal laminectomy
disk
atlantoaxial
spondylomyelopathy

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decompression), and the articular processes afford a small amount of bone stock for implant placement. In some instances, both a ventral and a dorsal approach are needed to accomplish the goals of surgery. Dorsally directed (interarcuate) branches of the internal vertebral venous plexuses (venous sinuses) at the articular process levels may bleed during the dorsal approach or during facetectomy/foraminotomy. However, this hemorrhage is easier to control because of the visualization afforded by the dorsal approach. Venous sinus hemorrhage that is severe and difficult to visually pinpoint is addressed by placing Gelfoam over the site of hemorrhage with slight pressure, using Bishop-Harmon forceps. Suction is used to evacuate blood around the site of hemorrhage. If the hemorrhage is less severe and can be localized to a small area, Surgicel can be used over the hemorrhage site. This is less obtrusive and will allow the surgeon to continue working in areas that are not bleeding. This method of hemorrhage control is particularly useful with approaches that are smaller (i.e., ventral slot approach).

Preoperative Management

Specifics of preoperative management depend largely on the disease process. In cases of suspected or known vertebral instability, the patient needs to be kept as still as possible and may need some external support (e.g., neck brace, secured to a board or other rigid surface). It is extremely important to emphasize the dangers of neck manipulation in such animals to everyone who may be handling the patient. Once the dog or cat is anesthetized, voluntary contraction of the cervical musculature will no longer be an available protective mechanism for that patient. If radiographs are obtained, overzealous neck flexion in an effort to demonstrate atlantoaxial (AA) instability may prove fatal. With increased availability of multislice computed tomography (CT) scanners and magnetic resonance imaging (MRI) units, safe and more accurate imaging methods are available for patients with cervical spine disorders.

A hallmark of many cervical myelopathies is neck pain, which is often severe. Oral prednisolone (0.5 mg/kg every 12 hours) is often effective in relieving pain in some cervical myelopathies. In chronic compressive myelopathies, this dose of prednisolone will also likely improve clinical signs through its effect on spinal cord edema. High doses of glucocorticoids should not be administered as an emergency or preemptive presurgical therapy for spinal cord injury. The "high-dose" methylprednisolone protocol has been ineffective for spinal cord injury and can have deleterious consequences (e.g., gastrointestinal ulceration/perforation). Polyethylene glycol (PEG) has been used for spinal trauma in dogs due to its ability (in experimental models) to fuse damaged cell membranes. However, a 2016 double-blinded, placebo-controlled study in dogs with acute severe spinal cord injury associated with thoracolumbar disc extrusions concluded that both PEG and methylprednisolone sodium succinate had no beneficial clinical effect.[1] Injectable opioid drugs (e.g., fentanyl) are usually effective analgesics in hospitalized patients (see Chapter 13). Gabapentin and pregabalin are oral calcium channel blocking drugs used as both antiseizure agents and antinociceptive agents (see Table 39.1 and pp. 149 and 1140). Pregabalin is the "next generation" of gabapentin. Although gabapentin is often an effective pain-relieving oral drug, pregabalin appears to be superior.

Anesthetic Considerations

Specific anesthetic concerns associated with cervical spinal cord surgery are hypotension, blood loss, cardiac arrhythmias, ventilatory compromise, and pain management. Because of the potential for venous sinus blood loss, blood should be available for transfusion (see Chapter 4). Severe hypotension, whether due to blood loss, lack of sympathetic tone, or both, can be treated with fluid support and/or positive inotropic drugs (see Table 4.5 and Box 19.6). Bradycardia may occur with vagal nerve stimulation during the ventral approach and can be treated with anticholinergic drugs (atropine or glycopyrrolate; see Chapter 12). Mechanical ventilation may be necessary both during and after surgery, depending on the severity of the myelopathy.

Taping the animal across the chest to maintain positioning at surgery can cause severe respiratory compromise if the tape is applied too tightly; it is best to avoid this practice and to rely on other methods (e.g., V trough, towels, vacuum apparatus) to maintain positioning for surgery. Excessive neck extension should be avoided during positioning because this may exacerbate compression in some situations (e.g., disc protrusions, caudal cervical spondylomyelopathy [CCSM]). The endotracheal tube should extend beyond the thoracic inlet during ventral approaches to the cervical spine so that the trachea is not damaged during retraction at surgery.

Anesthetic protocols vary but are similar to those used for fracture repair surgery (see Tables 32.1 and 32.2). Premedication with an opioid drug is often performed, as many of the cervical myelopathies are characterized by severe neck pain. If myelography is to be performed before surgery, the potential for postmyelographic seizures exists, especially in larger dogs. Antiseizure drugs are discussed in Chapter 39 (see Table 39.1).

ANTIBIOTICS

In dogs and cats undergoing surgery of the cervical spine, intravenous antibiotics (e.g., cefazolin, 22 mg/kg) should be administered perioperatively (30 minutes before the surgical incision is made and every 90–120 minutes during surgery).

SURGICAL ANATOMY

Seven cervical vertebrae are present, each of which has a body, laminae, pedicles, transverse processes, and articular processes. All but the first cervical vertebra (C1) have a dorsal spinous process. From C3 to C7, these processes slope in a cranial direction and increase in size from cranial to caudal. No intervertebral disc is found between the first and second cervical vertebrae. Distinctive features of the first (C1), second (C2), and sixth (C6) cervical vertebrae provide important landmarks for the surgeon; similarly, the large dorsal spinous process of the first thoracic vertebra (T1) is a distinct surgical landmark (Fig. 40.1). The first cervical vertebra (C1), the atlas, has a shortened body (the ventral arch); wide, shelf-like transverse processes (wings); and modified articular processes. The ventral tubercle of C1 feels like a distinctive "spike" when palpated during a ventral approach (Fig. 40.2), compared with the smooth "bump" of the ventral aspect of the intervertebral disc spaces. The dorsal spinous process of C2 (the axis) is a distinctive bladelike structure, the caudal aspect of which is very thick and partially overhangs the dorsal aspect of C3. The dorsal spinous process of C3 is extremely small and is sometimes nonapparent, especially in miniature and toy dog breeds. The dorsal lamina of C3 in these small breeds may be very short as well. It is important not to confuse C4 with C3 while performing a dorsal approach in the cranial cervical spine, especially in small dogs and cats. The articulation between C1

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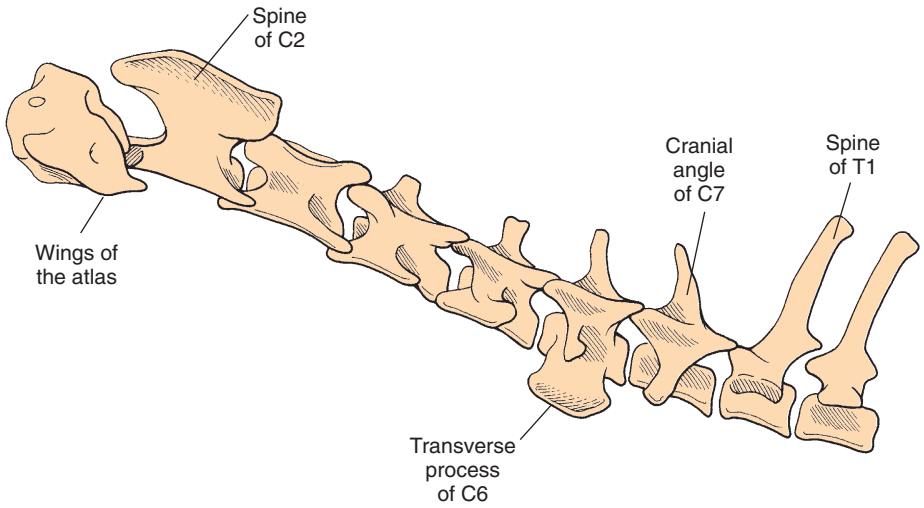


FIG. 40.1 Distinguishing anatomic landmarks of the cervical spine.

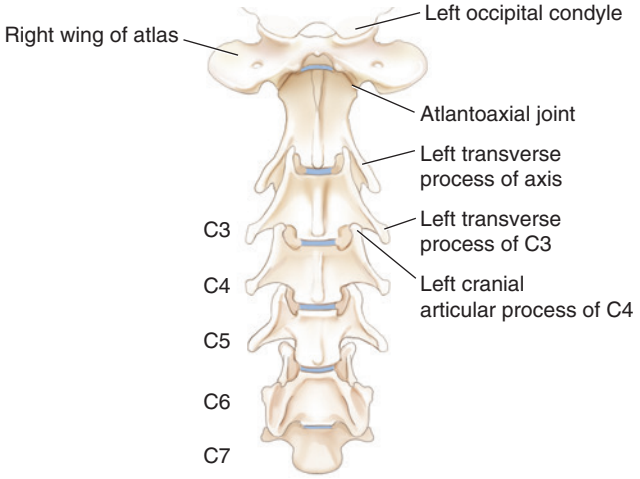


FIG. 40.2 Illustration of the ventral aspect of the cervical spine in the dog.

and C2 involves a cranial projection of the body of C2 (the dens) that rests on and is attached to (via several ligaments) the dorsal aspect of the C1 ventral arch (Fig. 40.3). The transverse processes of C6 are very prominent ventral structures that are easily palpated during the ventral approach. The dorsal aspect of the first rib as it articulates with T1 can also be palpated during the ventral approach; however, the surgeon must be cautious not to rupture the thin cupula pleura during palpation of this structure.

In addition to the venous sinuses on the floor of the vertebral canal, a vertebral artery is present on each side of the cervical spine. This large artery traverses the transverse foramina from C6 through C1 and is located ventral to the level of the articular processes from C2 through C6. The vertebral artery is usually not encountered during standard approaches to the cervical spine; disruption of this vessel during surgery can lead to rapid and extensive hemorrhage. Eight paired cervical spinal nerves are present. The paired first cervical spinal nerves exit from the lateral vertebral foramina of C1. The C2 through C7 spinal nerves exit through the intervertebral foramina cranial to the vertebra of the same number as the specific nerve (i.e., the C2 spinal nerve exits at the C–C2 foramen). The C8 spinal nerve exits the foramen formed by the C7 and T1 vertebrae. The nuchal ligament extends from the T1 dorsal spinous process to the caudal aspect of the C2 dorsal spinous process (Fig. 40.4). This structure is helpful during the dorsal approach both in verifying anatomic location and as a tool for remaining on midline (the nuchal ligament is a paired structure that can be split on the midline).

STANDARD SURGICAL APPROACHES TO THE CERVICAL SPINE

The main indication for performing a ventral approach to the cervical spine is to create a ventral slot for removal of ventrally located disc material. This approach is also used to stabilize cervical vertebral segments by placing implants in the vertebral bodies (e.g., AA instability, CCSM, traumatic fracture/luxation). When performing the ventral slot procedure, especially in small dog breeds, visualization of the limited surgical field can be challenging. The use of visualization aids, such as binocular magnification loupes or a high-definition video telescope system, can be of great benefit.[2] The dorsal approach is used to decompress the spinal cord and to remove dorsally or laterally situated compressive lesions (e.g., lateralized disc, hypertrophied yellow ligament, spinal tumors). The dorsal approach can be extended laterally by removing articular joint capsules (facets) and adjacent pedicles to improve access to the lateral vertebral canal.

Ventral Approach to the Cervical Spine

Shave the skin and aseptically prepare the area from approximately the midmandible level cranially to several centimeters past the manubrium caudally. The length of the surgical incision depends on the specific area to be operated. *Position the patient in dorsal recumbency with the head and neck in mild extension (Fig. 40.5). Secure the thoracic limbs by pulling them caudally and against the patient's trunk. Use a V trough or towels to position the neck area. Place a towel beneath the neck to facilitate extension. Secure the head with tape placed over the upper canine teeth.*

Assuming that the entire cervical region is to be exposed, make a ventral midline incision from the level of the larynx cranially to the level of the manubrium caudally (Fig. 40.6). Divide the

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sternocephalicus muscles to the level of the manubrium with Metzenbaum scissors or unipolar cautery if access to the caudal cervical spine is required. Divide the paired sternohyoideus muscles on midline (median raphe) with Metzenbaum scissors (Fig. 40.7). During this division, use bipolar cautery to address branches of the caudal thyroid veins that are in the dissection plane. Digitally separate the deep neck fascia, and visually identify then retract the trachea, esophagus, and left carotid sheath to the left and the right carotid sheath to the right. Once these structures are retracted, visualize the longus colli musculature (Fig. 40.8) and perforate the overlying fascial covering with Metzenbaum scissors; remove this fascia digitally using anatomic reference points discussed previously to identify the intervertebral space of interest. Bluntly separate the longus colli musculature on midline at the cranial and caudal aspects of the disc space (for spaces C2-C3 and caudally), using a straight Kelly or Mosquito forceps, while exposing the tendons of insertion of the longus colli muscles on the ventral tubercles of the vertebrae. Dissect these tendon insertions from the tubercles using Freer elevators (small dogs and cats), or cut them with Mayo scissors (large dogs). Continue the dissection of the longus colli musculature off the ventral aspect of the vertebral bodies to be exposed using Freer elevators or narrow Army-Navy osteotomes (large dogs). Once this dissection is complete, place Gelpi retractors in the cranial and caudal aspects of the area of interest, with the tips of the retractors beneath the longus colli musculature (Fig. 40.9). Control hemorrhage during this approach using bipolar cautery. When approaching the

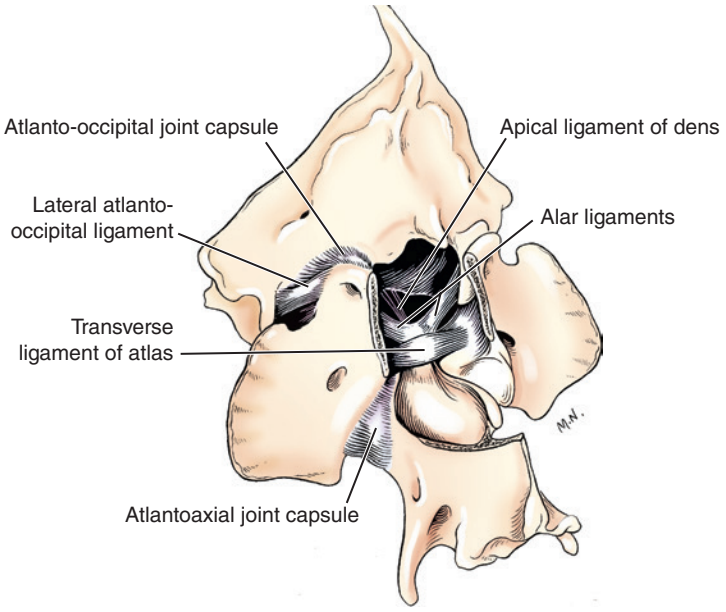


FIG. 40.3 Illustration of the ligamentous attachments between C1 and C2.

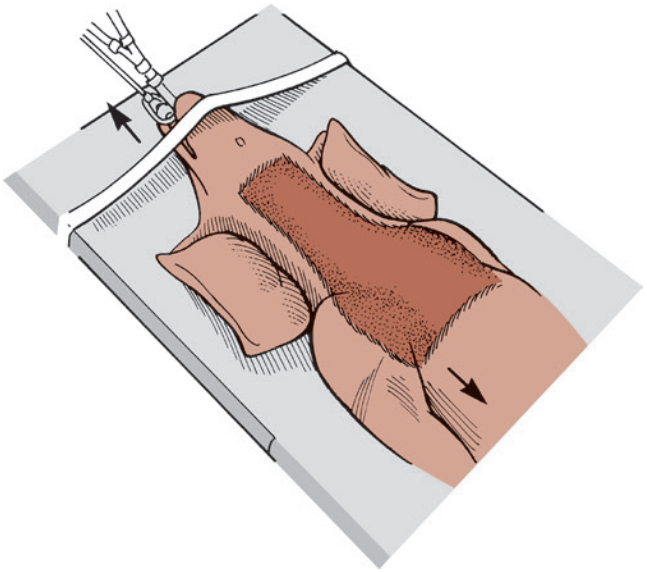


FIG. 40.5 Positioning for ventral approach to the cervical spine.

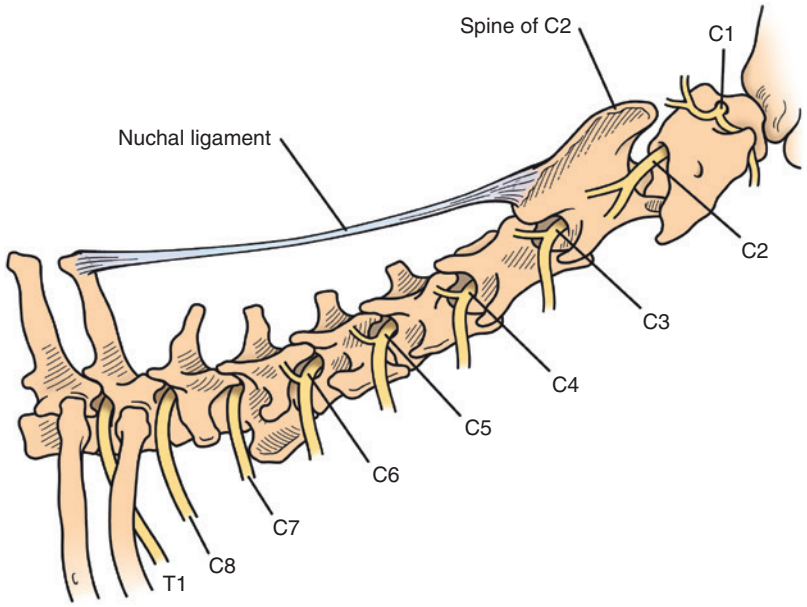


FIG. 40.4 Illustration of the cervicothoracic spine, showing spinal nerves and the nuchal ligament.

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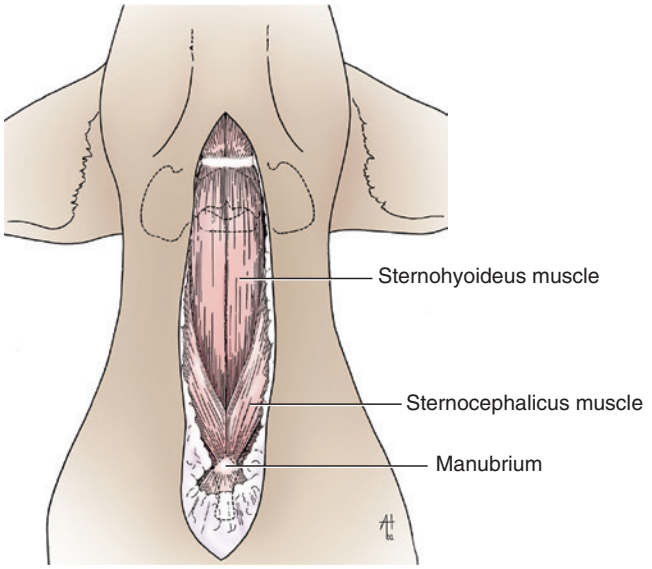


FIG. 40.6 A ventral midline skin incision is made from the larynx to the manubrium of the sternum, exposing the sternocephalicus and sternohyoideus muscles.

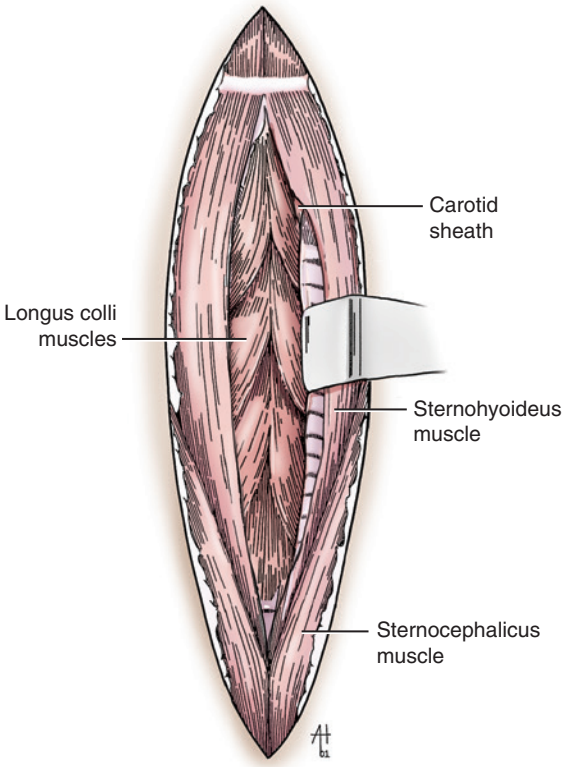


FIG. 40.8 Exposure of the longus colli musculature.

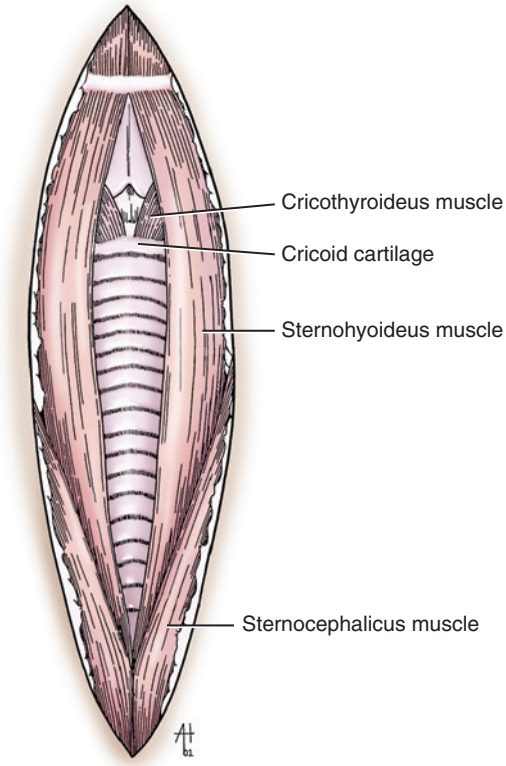


FIG. 40.7 Midline separation of the paired sternohyoideus muscles, exposing the trachea.

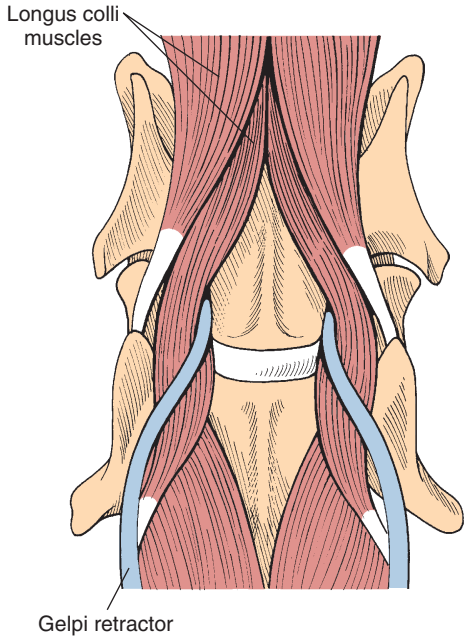


FIG. 40.9 Exposure of the vertebral bodies and the intervertebral disc.

C–C2 intervertebral space, transect the belly of the sternothyroideus muscle to facilitate exposure of this joint space. Using a No. 11 blade, incise the small muscle (rectus capitis ventralis) over the ventral surface of C1 and reflect it laterally with Freer elevators to expose the C1 ventral arch. To perform the ventral slot procedure (Videos 40.1 and 40.2), first fenestrate the disc of interest by resecting a rectangular section of the ventral annulus, and remove this piece of annulus with Lempert rongeurs, exposing the nucleus pulposus (Fig. 40.10A–B). Remove the ventral tubercle from the caudal aspect of the cervical vertebra forming the cranial aspect of the intended

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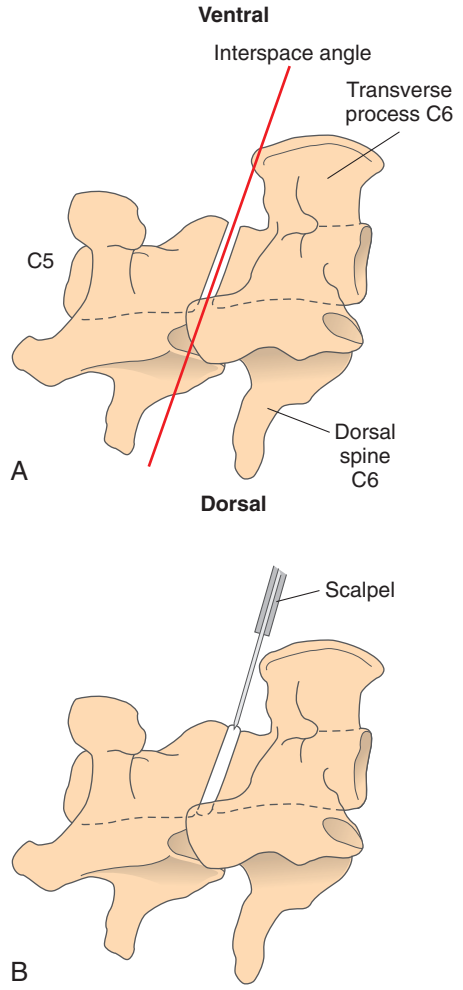


FIG. 40.10 (A) Note the slight cranial-to-caudal angle of the cervical vertebral interspaces. (B) To excise the ventral annulus, the scalpel blade should be inserted at this cranial-to-caudal angle.

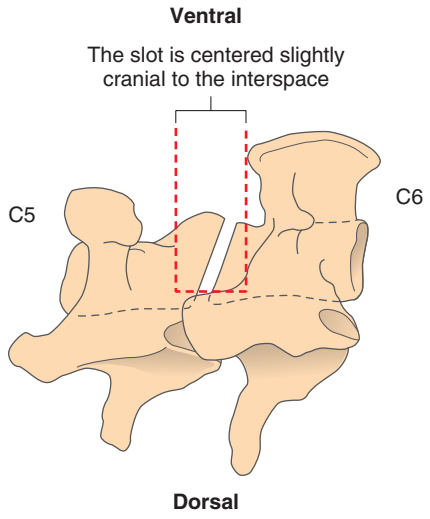


FIG. 40.11 Cranial and caudal extent of the ventral slot defect based on the caudal-to-cranial angle of the intervertebral space.

slot. Center the slot toward the vertebral body cranial to the disc because of the caudal-to-cranial direction of the intervertebral space from ventral to dorsal (Fig. 40.11). Do not exceed one-third the width or length of the vertebral bodies when creating the ventral slot. Using a high-speed air drill with a 4- to 5-mm burr, remove the outer cortical and inner cancellous bone layers, noting the spongy red nature of the cancellous layer during drilling. Stay on midline throughout creation of the slot to minimize the chance of rupturing the venous sinuses when entering the vertebral canal (Fig. 40.12A–B). If the cancellous bone bleeding is enough to interfere with visualization, use bone wax to stop the hemorrhage. When the bone again turns white, the inner cortical layer has been reached. *Use a smaller burr (e.g., 2–3 mm) to remove this final bone layer. Be cautious to avoid abruptly breaking through this layer.* The inner cortical bone takes on a flaky appearance, much like filo dough, just before the vertebral canal is penetrated. *Use a probe (e.g., Gross ear hook and spoon) to get under the dorsal annulus, and incise this last layer of the disc with a No. 11 blade. The adjacent periosteal layer is often incised concurrently if the inner cortical layer is thin enough. Once the inner cortical bone layer is thin enough, or if there is a window into the vertebral canal, use a 4-0 bone curette to enlarge the slot defect. In small-breed dogs, a distinct dorsal longitudinal ligament may not be apparent (may be removed with the periosteal layer); in larger dogs, this dull white fibrous structure may be distinct (Fig. 40.13). Carefully incise the dorsal longitudinal ligament with a No. 11 blade, if necessary, to gain entry into the vertebral canal (Fig. 40.14).* Fibrous (annular) material at the sides of the slot defect will have a frayed appearance, like irregularly cut rope. Calcified nucleus pulposus will appear white and granular, like miniaturized cottage cheese. The dura mater will be smooth and glistening and may be a bright white color or bluish-white (bruising from disc rupture). *Remove disc material within the vertebral canal using a probe and Bishop-Harmon forceps so that the dura is visualized. Use the probe to "sweep" underneath the edges of the slot to remove as much disc material as possible.* During disc removal and sweeping around the slot defect, it is common to lacerate a venous sinus. *Control venous sinus hemorrhage as previously described. After copious lavage of the surgical site, close the longus colli muscles with simple interrupted sutures. If a caudal exposure was performed, reappose the sternocephalicus muscles with simple interrupted sutures.* The sternothyroideus muscle does not need to be reattached if this muscle was severed during C1–C2 exposure. Subcutaneous and skin closure is routine.

Dorsal Approach to the Cervical Spine

The specific dorsal approach chosen differs depending on what part of the dorsal cervical spine is operated. For all variations of the dorsal approach, it is extremely important to stay on midline and to divide paired muscular and tendinous structures as much as possible; doing so will minimize hemorrhage and postoperative discomfort. Carefully identify dorsal cervical musculature layer by layer, using Gelpi retractors in the cranial and caudal limits of the surgical exposure as successive layers are reached.

For all of the dorsal approaches, place the patient in sternal recumbency with the neck gently flexed in a neutral position (Fig. 40.15). When the approach to the cranial cervical spinal cord is combined with suboccipital craniotomy (see p. 1344), flex the neck more acutely. The lateral limits of the shaved and aseptically prepared skin area extend to the middle third to half of the dorsal-ventral neck region on each side. The cranial limit of this

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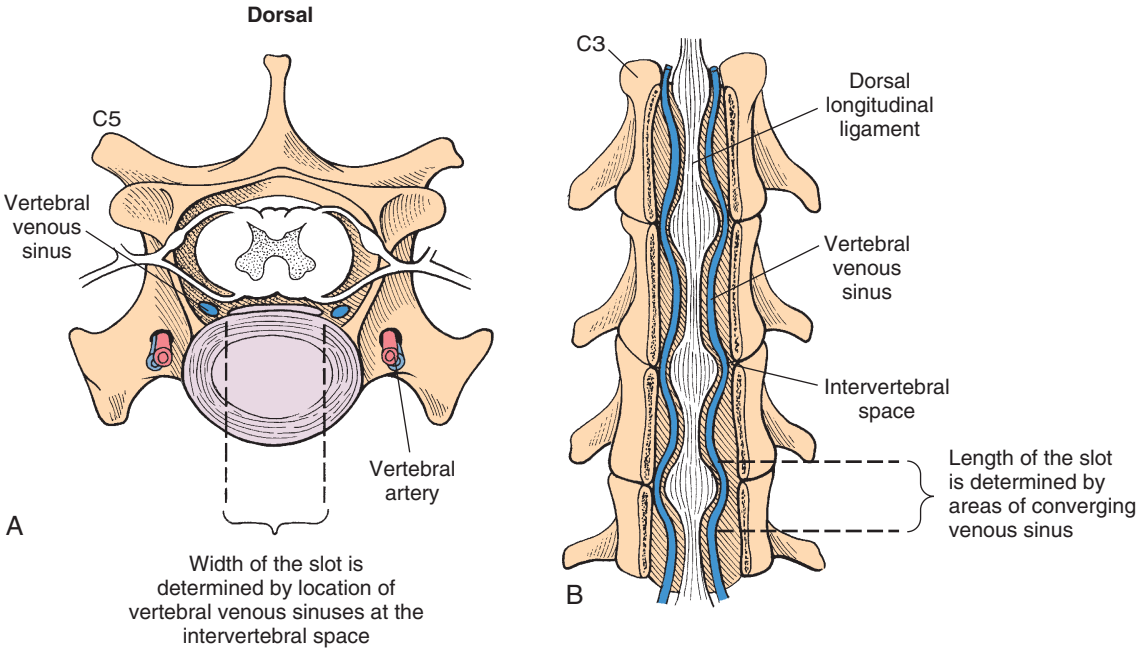


FIG. 40.12 (A) Cross section of the cervical spine showing vertebral venous sinus and vertebral artery relative to the intervertebral disc and spinal cord; the width of the slot is determined by this relationship. (B) Laminectomized cervical spine showing the shape of the vertebral venous sinus and the dorsal longitudinal ligament relative to the intervertebral spaces; the length of the slot is determined by this relationship.

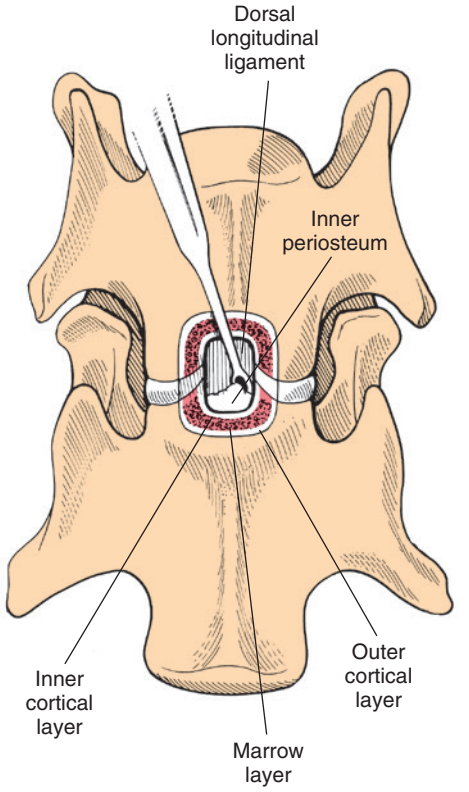


FIG. 40.13 The proper depth of the ventral slot defect is determined by identifying outer cortical, medullary, and inner cortical layers of bone; once the inner cortical layer is reached, use a 3-0 or 4-0 curette to expose the dorsal longitudinal ligament.

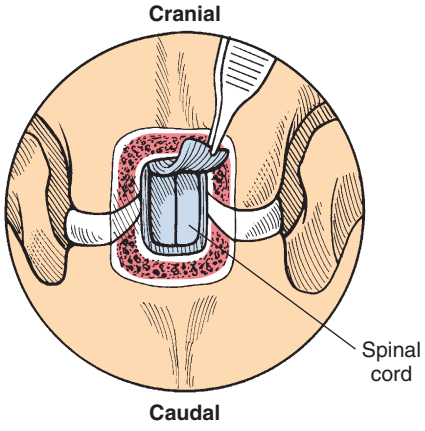


FIG. 40.14 Decompression is complete when the spinal cord is visualized.

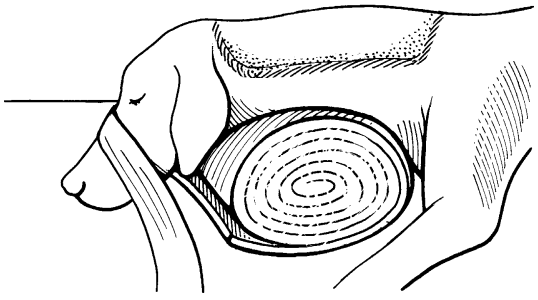


FIG. 40.15 To position an animal for cranial cervical laminectomy, gently flex and support the neck.

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area for cranial cervical and midcervical approaches is the external occipital protuberance. For midcervical and caudal cervical approaches, the caudal limit of the surgical site is approximately at the level of the dorsal spinous process of T2 or T3. The cranial limit of the surgical site for caudal cervical approaches is approximately at the level of C3.

For a cranial cervical approach, incise the skin on the dorsal midline from the external occipital protuberance to the midcervical region (C4 or C5 dorsal spinous process level). Locate the midline fibrous raphe of the superficial cervical musculature (Fig. 40.16). Incise this connective tissue with a No. 11 blade, and carefully continue the midline incision cranially and caudally with Metzenbaum or Mayo scissors, controlling hemorrhage with monopolar and/or bipolar cautery. Reflect and retract the superficial cervical musculature to expose the paired splenius muscles. Divide these muscle bellies, as well as those of the underlying biventer cervicis muscles, to expose the next layer of paired muscles: the rectus capitis dorsalis muscle cranially and the spinalis cervicis muscle caudally (Fig. 40.17). Identify the nuchal ligament extending caudally from the caudal aspect of C2 at this level. Using a combination of sharp and blunt dissection, elevate the rectus capitis dorsalis muscle off the C2 dorsal spine. Sharply transect the attachment of the nuchal ligament off the caudal aspect of C2, and divide the muscle bellies of the spinalis cervicis muscles over C3. Using a Freer elevator, bluntly elevate these muscles laterally, exposing the dorsal lamina of C3 (Fig. 40.18). The approach for access to the C1–C2 cervical region is discussed in Chapter 39 (suboccipital

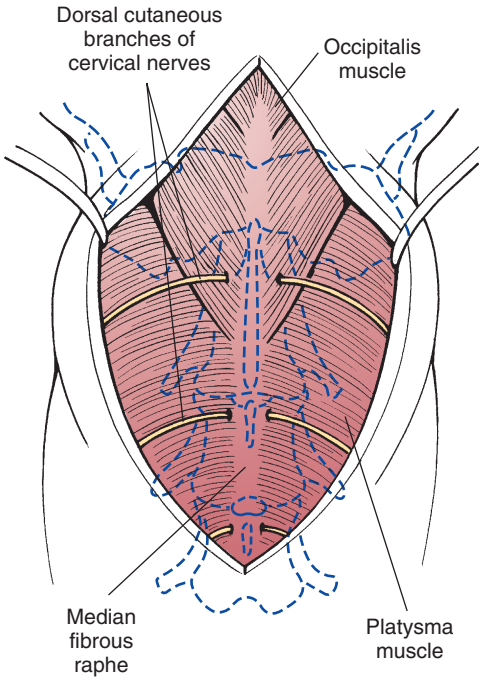


FIG. 40.16 During cervical laminectomy, dissect on the midline of the superficial cervical musculature to expose the median raphe and cutaneous nerves.

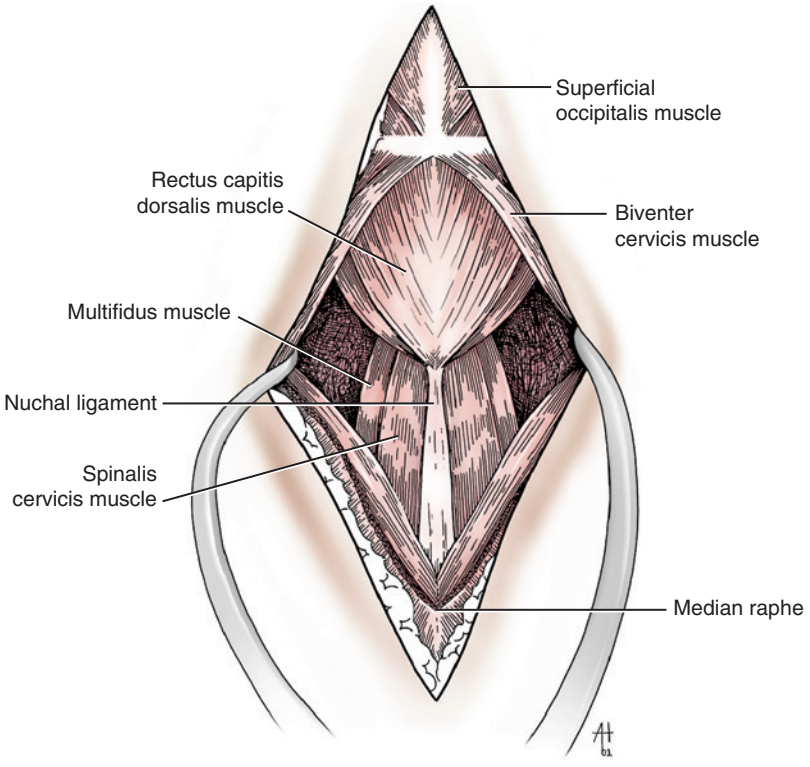


FIG. 40.17 Separation of the median raphe exposing superficial occipitalis muscle, biventer cervicis muscle, rectus capitis dorsalis muscle, spinalis cervicis muscle, multifidus muscle, and nuchal ligament.

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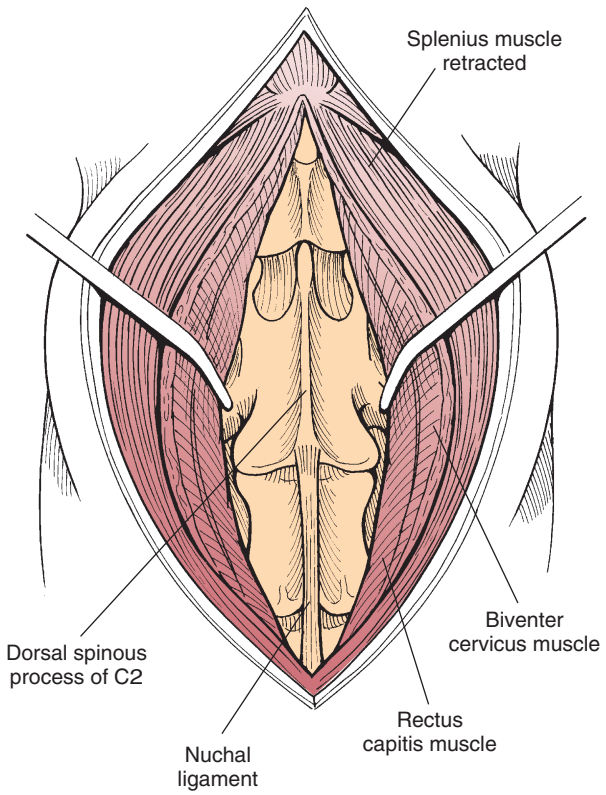


FIG. 40.18 During cranial cervical laminectomy, elevate muscle from the spinous processes and the laminae of C1–C2.

craniotomy) (see p. 1344). The initial dorsal approach to the midcervical and caudal cervical regions is similar to that for the cranial cervical approach, with incision of the median raphe of the superficial cervical muscles and retraction of these muscles laterally. *To access the dorsal laminae of C4 through C7, divide the nuchal ligament on midline and retract the paired components laterally with Gelpi retractors, exposing the underlying epaxial musculature. Using Mayo scissors, circumferentially cut the tendinous attachments of the spinalis cervicis and multifidus muscles to the dorsal spinous processes, then bluntly elevate these muscles off the dorsal lamina with Freer elevators (small dogs and cats) or Army-Navy osteotomes (large and giant dog breeds). Remove attachments of the multifidus muscles to the articular processes using a combination of periosteal elevators and bipolar cautery (Fig. 40.19A–B). Remove the dorsal spinous processes of the desired surgery site with rongeurs or bone cutters. Use a high-speed air drill to create the laminectomy defect.*

The defect should extend the length of the vertebrae of interest and should extend laterally to the level of the articular processes (Fig. 40.20). In medium- to giant-breed dogs, the bone will change in color and character as in the ventral slot procedure (i.e., outer cortical, inner cancellous, inner cortical). In cats and small-breed dogs, the dorsal lamina in the cervical region is often very thin, and an obvious inner cancellous bone layer may not be apparent.

HEALING OF THE SPINAL CORD

Three basic categories of change occur after acute injury to the spinal cord: (1) direct morphologic distortion of neuronal tissue,

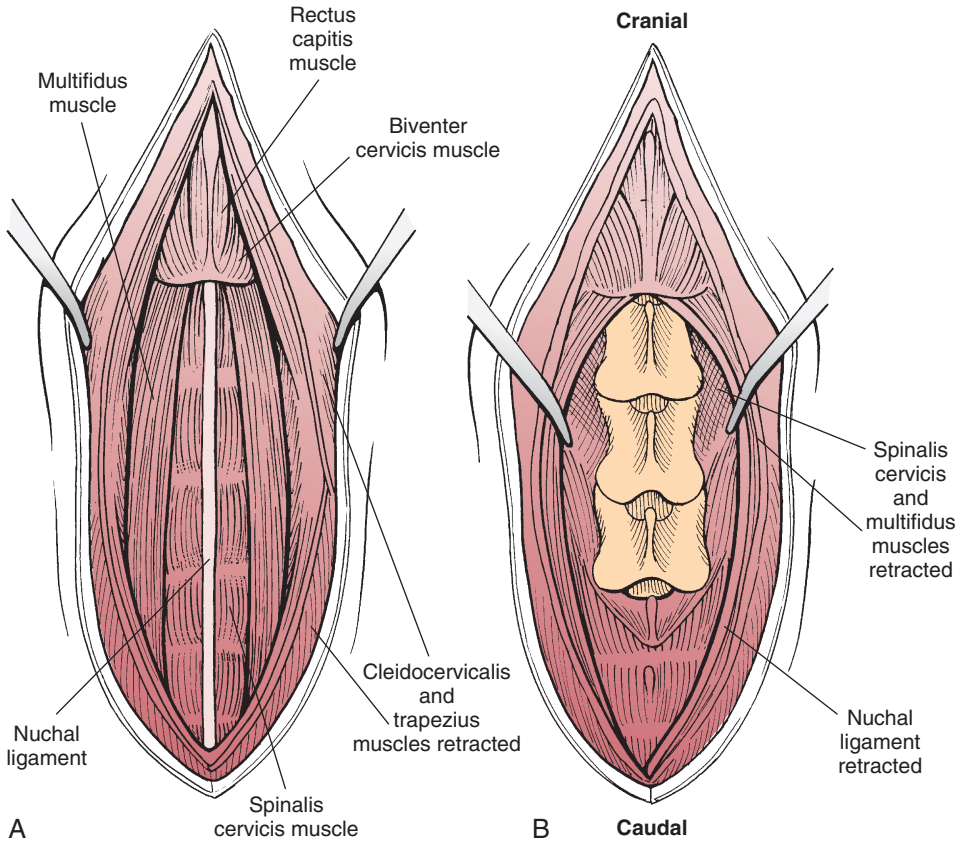


FIG. 40.19 (A) Identify the median fibrous raphe and the nuchal ligament during dorsal midcervical laminectomy and use them as landmarks. (B) Elevate the cervical musculature from the dorsal spinous processes and laminae of C3, C4, and C5.

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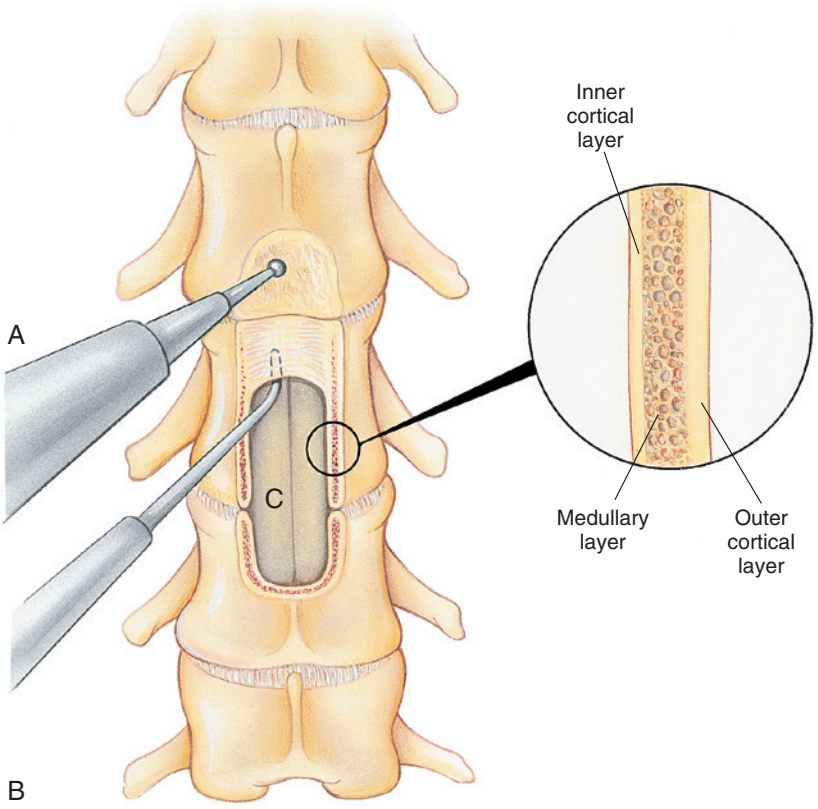


FIG. 40.20 (A) Use a pneumatic bone drill to expose outer cortical, medullary, and inner cortical layers of laminar bone. (B) Elevate the periosteum with a dental spatula to gain exposure to the vertebral canal. (C) Expose the spinal cord and nerve roots.

as well as severe axonal injury; (2) vascular changes; and (3) biochemical and metabolic changes. Controversy exists regarding the relative importance of these closely related categories in causing functional deficits. Direct morphologic damage to spinal cord tissue (e.g., laceration, compression, stretching) resulting in axonal interruption is believed to be irreversible and untreatable because central axons do not regenerate sufficiently to restore function.

In the case of reversible spinal cord injury, a characteristic series of events occurs during the repair process. Approximately 2 days after injury, a heterogeneous population of small cells (most likely of hematogenous origin), including polymorphonuclear, lymphocyte, macrophage, and plasma cells, invade the traumatized tissue. This is essentially an inflammatory response. Within 7 to 20 days, fibroblasts make their appearance and begin laying down scar tissue. A concurrent glial reaction consisting of astrocyte proliferation and expansion of processes occurs. Depending on the extent of spinal cord injury, moderate to extensive fibrosis, nerve fiber degeneration, and multifocal malacia may be seen. Axons that have been severed, compressed, or stretched start to regrow until they reach the edges of the scar mass. Regeneration of spinal cord axons sufficient enough to restore function has not been achieved; however, if enough axons remain intact, return to clinically acceptable motor function can be expected.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Neurosurgical instrumentation required for cervical spinal surgery is similar to that discussed for brain surgery (p. 1345). For dorsal laminectomies in the cervical region, deep right-angled Gelpi retractors are helpful in providing exposure. Angled burr guards are also needed to create a shelf under a ventral slot for anchoring polymethylmethacrylate (PMMA) in the PMMA plug technique for CCSM (see p. 1384).

POSTOPERATIVE CARE AND ASSESSMENT

Critical care observation for the first 24 hours postoperatively includes monitoring respiration, administering analgesics, and observing for gastric dilatation-volvulus (particularly in large- and giant-breed dogs) and seizure activity (particularly if the patient had preoperative myelography). Blood gas analysis is warranted in animals with ventilatory compromise. Postoperative analgesia is provided by low doses of opioids, thus reducing their respiratory depressant effects (see Table 13.2). Fluids should be administered at maintenance rates and the patient turned every 2 hours until sternal. Glucocorticoid administration is generally discontinued postoperatively. If neurologic status deteriorates after surgery, glucocorticoids may be given until the cause is determined or corrected. Ambulatory patients may be discharged 3 to 5 days after surgery and should be confined for 2 to 4 weeks. They should walk on a harness for 4 to 8 weeks. Nonambulatory patients are treated with frequent hydrotherapy, physiotherapy, an elevated padded cage rack, frequent turning, and bladder expression three to four times a day. They should be kept clean and dry to prevent decubital ulcers. Avoid indwelling urinary catheters because they are a frequent cause of urinary tract infection. A supporting cart helps nurse patients to an ambulatory status. Advantages of a

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cart include unhindered eating, drinking, micturition, and defecation. Additionally, animals with motor function are encouraged to ambulate, and an erect position facilitates physiotherapy. Nonambulatory patients may be discharged when owners are able to care for them. Neurologic examinations should be performed at 1, 2, 3, 6, 9, and 12 months postoperatively, or until improvement has ceased.

COMPLICATIONS

Complications associated with cervical spinal surgery are uncommon and include excessive hemorrhage from venous sinus laceration (may require blood transfusion), worsening of neurologic signs after surgery (usually transient, but permanent injury is a risk), postoperative infection, and seroma formation. Less common is vertebral instability and luxation from performing too wide a ventral slot. Recumbent patients are prone to urinary tract infection, particularly if they are receiving glucocorticoids.

SPECIAL AGE CONSIDERATIONS

Softer cortical bone of lamina, pedicles, and vertebral bodies of young animals requires less aggressive drilling and rongeuring than the denser, more compact bone of older dogs.

SPECIFIC DISEASES

CERVICAL DISC DISEASE

DEFINITIONS

Cervical disc disease refers to degeneration of the nucleus pulposus (**Hansen type I,** or **type I** degeneration) or annulus fibrosus (**Hansen type II,** or **type II** degeneration) components of cervical discs and the clinical consequences of such degeneration. Type I degeneration is also called *chondroid degeneration,* whereas type II degeneration is called *fibroid degeneration.* A third, less commonly encountered type of disc disease, hydrated nucleus pulposus extrusion (**HNPE**), has also been described (previously termed *discal cyst*); this is typically an acute-onset extrusion of hydrated nucleus pulposus material in the cervical region. HPNE likely represents a clinical variant of type I disc degeneration. There is evidence that there is some degree of nuclear degeneration in HNPE cases, suggesting that these are extrusions of an "almost healthy" nucleus pulposus.[3–9] **Radiculopathy** means pathology of the nerve roots and is often manifested as pain due to compression by disc material; when a radiculopathy leads to lameness or holding up a thoracic limb because of suspected irritation/pain, this is called a **root signature.**

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Degenerative disc disease is a common problem in dogs but a relatively infrequent clinical disorder in cats. Two commonly encountered types of disc degeneration (type I and type II) typically cause two distinct types of disc disease (Figs. 40.21 and 40.22). In *chondroid (type I) degeneration*, the normally gelatinous nucleus pulposus loses water-binding capacity, undergoes degradation of the glycosaminoglycan components, and often becomes calcified. The dorsal annulus often weakens, and the abnormal nucleus pulposus contents extrude through the weakened annulus into the vertebral canal. The severity of spinal cord damage caused by type I disc extrusion is believed to be related to the rate of extrusion (force of impact or concussion), the duration of compression, and the amount of disc material extruded. *Fibroid (type II) degeneration* involves progressive thickening of the dorsal annulus fibrosus, which protrudes dorsally into the vertebral canal over an extended period of time. Type I and type II degeneration can occur concurrently; these terms do not impose limitations on the pathologic behavior of abnormal discs. The phenomenon of "acute-on-chronic" disc rupture occasionally occurs, in which a dog with chronic signs of probable type II protrusion worsens rapidly owing to sudden extrusion of nucleus pulposus into the vertebral canal (type I extrusion). As previously mentioned, HNPE likely represents a variant of type I disc degeneration. The clinical scenario with HNPE is typically consistent with an acute compressive myelopathy. The compressive material in this type of extrusion is hydrated ("healthy") nucleus pulposus; it is thought that these extrusions arise from discs that are in an early stage of degeneration.[3–9]

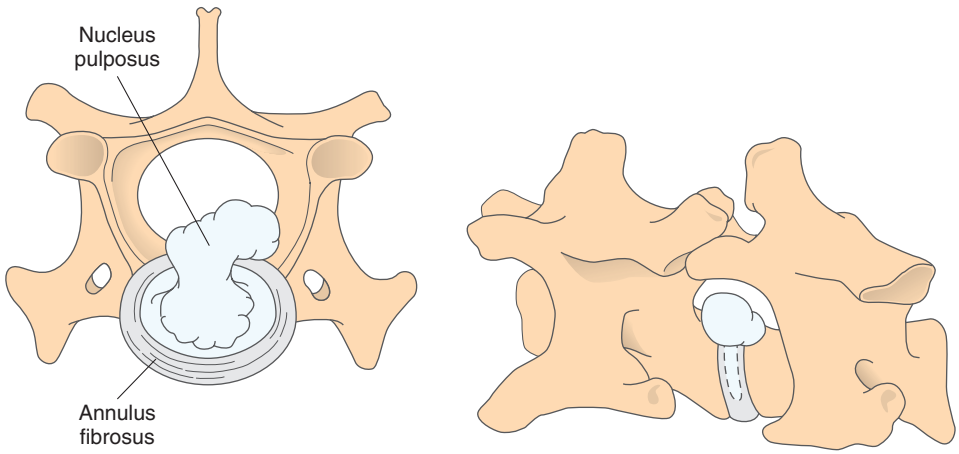


FIG. 40.21 Hansen type I disc degeneration is characterized by an acute massive extrusion of degenerate nuclear material in the vertebral canal.

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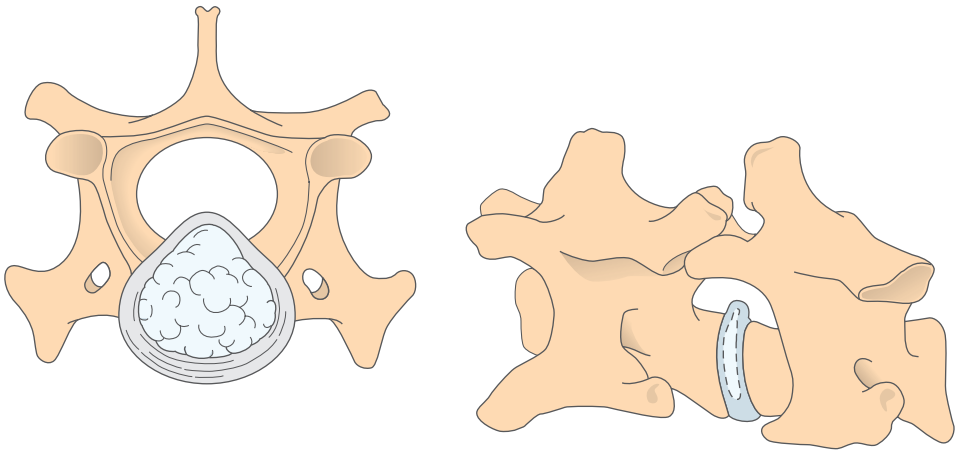


FIG. 40.22 Hansen type II disc degeneration is characterized by a slow chronic protrusion of degenerate dorsal annulus fibrosus in the vertebral canal.

DIAGNOSIS

Clinical Presentation

Signalment

Hansen type I extrusions typically occur in small-breed dogs, particularly the chondrodystrophic breeds (e.g., dachshund, beagle, bassett hound, shih tzu, Pekingese, Lhasa apso). The dachshund is by far the most commonly affected breed. Hansen type II disc protrusions typically occur in nonchondrodystrophic, larger breed dogs. Either type of disc disease can occur in any breed of dog, however, and both types have been reported in cats. Large dog breeds that seem to be most commonly encountered with type I disc extrusions include mixed breed dogs, German shepherds, Labrador retrievers, Doberman pinschers, and Rottweilers.

Hansen type I extrusions typically occur in dogs 3 years of age or older (peak ages in small dogs of 3–6 years) but may occur in dogs younger than this. A wide age range has been reported for type I disc extrusions in cats; they often occur in older patients. Hansen type II protrusions typically occur in dogs 5 years of age and older. A number of small and large breeds comprising a wide age range have been reported with HNPE of the cervical region; most of the reported cases were older dogs (median age, 9 years).[3–9]

History

Historical complaints related to type I and type II cervical disc disease include primarily neck pain and abnormal ambulation with all four limbs (tetraparesis, tetraplegia). Neck pain alone is the most common historical complaint, but it is often combined with historical evidence of a thoracic limb root signature. Some dogs have a history of unilateral neurologic dysfunction (i.e., hemiparesis). Hansen type I disc extrusion usually causes rapidly developing clinical signs (minutes to days), whereas Hansen type II disc protrusions typically cause chronically developing clinical signs (weeks to months, sometimes years). Owners sometimes mistake paroxysms of pain due to type I disc extrusions with seizure activity. In this scenario, the dog screams in apparent pain and falls over. HNPE cases are characteristically peracute to acute and tend to cause fairly severe neurologic dysfunction (nonambulatory tetraparesis or tetraplegia), often without obvious evidence of cervical hyperesthesia; this contrasts with typical calcified type I extrusions, which commonly lead to substantial cervical hyperesthesia that is less frequently associated with severe neurologic dysfunction.[3–9]

Physical Examination Findings

In small-breed dogs, type I cervical disc disease usually affects cranial cervical discs (C2–C3 most commonly) and typically causes severe neck pain, usually with inapparent or mild neurologic deficits. In one large retrospective study of 187 small-breed dogs with surgically managed type I cervical disc extrusions, several breed-related differences in extrusion sites were identified; dachshunds, beagles, shih tzus, and Pekingese were more likely to extrude discs at C2–C3, whereas caudal disc extrusions (C5–C6, C6–C7) were more likely in Yorkshire terriers and Chihuahuas. Yorkshire terriers and shih tzus were found to be older than other breeds at the time of surgery and also had a higher number of extruded discs.[10] In large nonchondrodystrophic dogs, the most common site for type I cervical disc extrusions is at the C6–C7 intervertebral disc space; these dogs also tend to present with acute onset of severe neck pain. In our experience, neck pain tends to be more severe with cranial versus caudal cervical disc extrusions. The patient often adopts a "nose-down" posture with an arched back (Fig. 40.23). When turning, these dogs tend to move the head and the neck as one unit rather than bending at the neck. Fasciculations of the neck musculature can often be appreciated, especially on palpation of the neck. Clinical signs of a root signature may be apparent. HNPE most commonly affects the C3–C4 and C4–C5 intervertebral discs, usually resulting in severe symmetric neurologic impairment (nonambulatory tetraparesis or tetraplegia) of acute onset (<24 hours), often without obvious cervical hyperesthesia. Type II cervical disc disease may result in clinically appreciable neck pain but rarely to the degree encountered in type I cervical disc disease. Type II cervical disc disease usually causes slowly progressive paresis. This can occur as an isolated process or as a component of CCSM. Impaired ambulatory function can range from unilateral thoracic limb lameness to tetraplegia with respiratory compromise.

Diagnostic Imaging

Historically, spinal imaging for pets with suspected disc disease consisted of plain radiographs followed by myelography (Fig. 40.24), both performed under general anesthesia. CT (or

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FIG. 40.23 Typical "nose-down" posture of a dog with severe cervical pain.

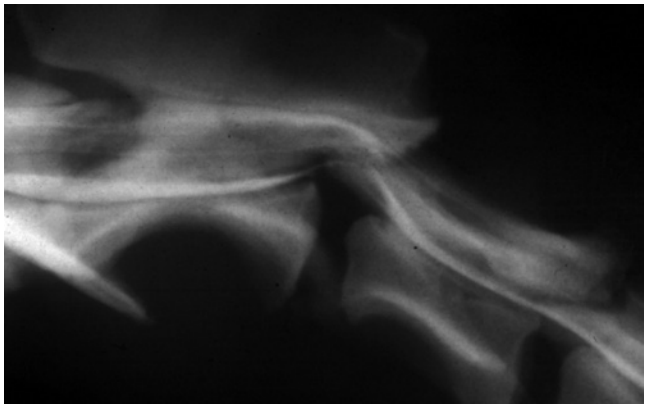


FIG. 40.24 Lateral myelographic image of a C2–C3 disc extrusion in a dog.

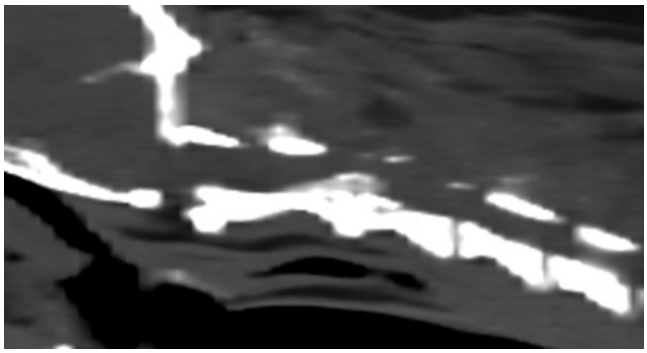


FIG. 40.25 Computed tomography sagittal reconstruction image of a dog with a cervical intervertebral disc extrusion.

CT/myelography combination) and MRI have been found to be useful imaging modalities in the diagnosis of intervertebral disc disease (Figs. 40.25 and 40.26). We consider MRI to be the current standard for spinal imaging of suspected disc extrusion/protrusion patients. In addition to providing superior anatomic detail in disc disease patients compared with myelography and CT, MRI is the best modality for diagnosing other spinal disorders that

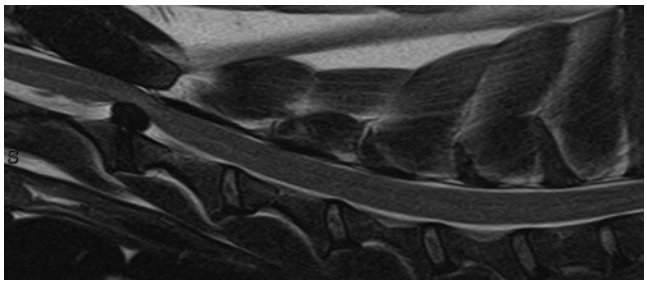


FIG. 40.26 Midsagittal T2-weighted magnetic resonance image of a dog with a cervical intervertebral disc extrusion.

may have similar clinical presentations as intervertebral disc disease and is associated with fewer side effects than myelography. Normally, the nucleus pulposus has high signal intensity on T2-weighted images (the annulus is hypointense). The nucleus pulposus of a degenerative disc is hypointense, and the distinction between the nucleus and the annulus fibrosus may be lost.

The typical imaging finding (myelography, CT, or MRI) in disc extrusion/protrusion is focal extradural spinal cord compression centered over a disc space. Sometimes, a disc extrusion will lacerate a venous sinus, leading to large extradural accumulations of hemorrhage (in addition to disc material), which can cause extensive cord compression or swelling. Occasionally, a type I disc extrusion will be lateral enough that myelographic results are normal. In such lateralized extrusions, hyperesthesia with or without unilateral limb lameness (ipsilateral to the extrusion) is a more likely clinical sign of dysfunction than overt proprioceptive or voluntary motor deficits. Another important consideration in such a clinical scenario is the presence of syringomyelia, which is not usually evident on myelography or CT imaging (requires MRI). In some cases of high-velocity concussive disc-related injury, severe motor deficits (due to a small amount of rapidly extruding disc material) may occur with minimal evidence of extradural compression on imaging studies; in such cases, evidence of spinal cord swelling is typically noted. This may be difficult to distinguish from a vascular event (see fibrocartilaginous embolic myelopathy discussion) if an imaging modality other than MRI is used. The appearance of HNPE lesions have been documented primarily on magnetic resonance (MR) images; the extruded material is hyperintense on T2-weighted images, hypointense on T1-weighted images, and may have contrast ring enhancement around the extruded material on T1-contrast (gadolinium) sequences. The extruded material is just dorsal to the dorsal annulus, between this structure and the dorsal longitudinal ligament. On axial T2-weighted images, HNPE has a characteristic "seagull" shape (Fig. 40.27). Occasionally, there may be parenchymal hyperintensity at the site of extrusion, thought to be reflective of spinal cord contusion. On CT images, HNPE material is hypodense and may exhibit peripheral ring enhancement with contrast administration.[3–9]

Laboratory Findings

Results of complete blood cell count (CBC) and serum biochemical profile analyses are typically normal. A stress leukogram may be observed on CBC results as a result of underlying disease, prior administration of glucocorticoids, or a combination of these factors. Hepatic enzyme elevations may be evident with dogs that have been treated with glucocorticoids. Urinary tract infection may be present, especially in dogs treated with

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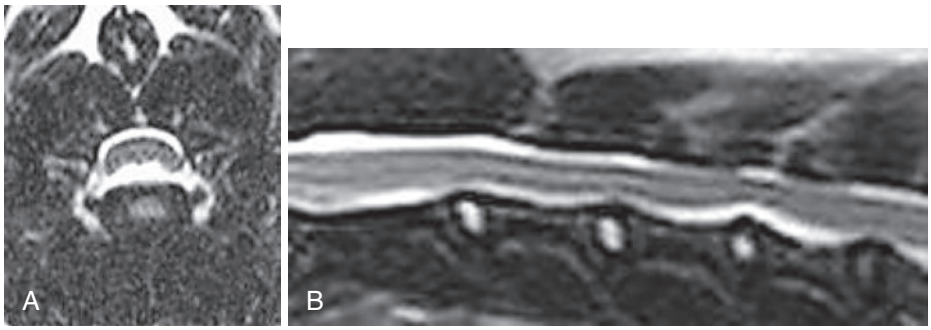


FIG. 40.27 (A) Axial and (B) midsagittal T2-weighted magnetic resonance images of a dog with a hydrated nucleus pulposus extrusion at C4–C5. Note the characteristic "seagull" appearance of the extruded material on the axial image (B).

glucocorticoids; however, pyuria and hematuria may or may not be present if the patient is still receiving steroids. Even if collected before myelography is performed, cerebrospinal fluid (CSF) is often not evaluated if clear evidence of a disc compression is present on imaging. Results of CSF analysis with type I disc extrusions are variable. Although CSF findings in type I disc extrusions have classically been characterized as normal to mildly inflammatory, moderate to marked pleocytosis with increased protein concentration is fairly common. In one retrospective study, 26 of 103 dogs (25%) with cervical disc extrusions had CSF pleocytosis ranging from 6 to 172 nucleated cells/μL.[11]

DIFFERENTIAL DIAGNOSIS

A tentative diagnosis of cervical disc disease is based on characteristic signalment, historical complaints, and clinical features. A definitive diagnosis requires spinal imaging, as well as surgical findings of extruded disc material. Other diseases that can cause similar or identical signs of cervical disc extrusion/protrusion include inflammatory/infectious conditions (e.g., corticosteroid-responsive meningitis, discospondylitis, granulomatous meningoencephalomyelitis), syringomyelia, traumatic fracture/luxation, neoplasia, and congenital abnormalities (e.g., Chiari-like malformation [CLM], AA instability, atlanto-occipital overlapping [AOO]). It is important for the surgeon to realize that, although it is uncommon, some patients with intracranial lesions will exhibit neck pain as the primary or sole clinical feature. If imaging of the cervical region in a patient with obvious neck pain is normal, imaging of the brain should be considered.

MEDICAL MANAGEMENT

Treatment of acute and chronic disc disease is a subject of considerable debate, but a number of guidelines have been established. Positives and negatives are associated with surgical and nonsurgical management of disc disease patients, and clients need to be informed of the benefits and risks associated with either approach.

Patients with suspected type I cervical disc extrusions are often first treated successfully nonsurgically if they exhibit mild to no neurologic deficits (i.e., mainly neck pain) and have not had repeated episodes of pain. Medical management traditionally consists of strict cage confinement for 3 to 4 weeks, with or without antiinflammatory medication. The cage or crate should be of such a size that the patient can change positions but cannot walk around or jump. Activity should be restricted to short walks to urinate/defecate, at which times the owner can assess the progress of the patient. Lateralized, or intraforaminal, disc extrusions are often associated with severe neck pain; however, some of these dogs may respond well to medical therapy as just described, as well as to perineural injections of glucocorticoid and bupivacaine. Medical therapy for such cases may be somewhat prolonged; in one report of 13 dogs with intraforaminal disc extrusions, 11 responded to medical management, with a median time period of 7.5 weeks to reach resolution of clinical signs without the need for analgesic drugs.[12,13] If the patient fails to improve or worsens at any time during the confinement period, surgical options should be considered.

Administering antiinflammatory drugs to a patient who is exhibiting signs of an extruded disc without concurrently confining that patient is contraindicated. Antiinflammatory drugs alleviate the patient's pain, and most dogs will subsequently become more active. Increased activity is thought to cause more pressure to be placed on the abnormal disc by adjacent vertebrae; subsequently, more disc material is extruded into the vertebral canal, and clinical signs acutely worsen. It is generally contraindicated to concurrently administer steroidal and nonsteroidal antiinflammatory drugs (NSAIDs) to disc disease patients because this combination substantially increases the chances of severe gastrointestinal complications. It is important to realize that subclinical gastroduodenal ulceration might be present in dogs with type I disc extrusions, even without administration of potentially ulcerogenic drugs (e.g., NSAIDs, dexamethasone); therefore the use of such drugs should be minimized (see also p. 428).

Traditional recommendations for medical management of dogs with type I disc extrusions have been called into question, especially those dealing with cage confinement and glucocorticoid administration. In one retrospective report evaluating medical management of dogs with presumptive type I cervical disc extrusions, no association was found between duration of cage confinement and success of medical therapy. Additionally, no beneficial effect of glucocorticoid administration on the success of medical management was found, whereas the use of NSAIDs was positively associated with successful outcome. These findings suggest that an extended cage confinement recommendation is often not adhered to by dog owners, and that it may be an unnecessary recommendation. The average confinement period was approximately 2 weeks in the aforementioned study, which may be a realistic recommendation.[14] In addition

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to antiinflammatory medications, oral opiates may be considered for dogs experiencing discomfort (e.g., tramadol, 2–4 mg/kg every 8–12 hours) and/or pregabalin (2 mg/kg PO every 12 hours).

Many dogs will respond favorably to medical management. In our clinical experience, 50% to 70% of ambulatory patients with presumptive type I cervical disc extrusions (cervical or thoracolumbar) will have an initial positive response to medical therapy, although many of these dogs tend to have recurrence of clinical signs. In the aforementioned retrospective study, sustained success was achieved with medical management in approximately half of patients, whereas initial success with subsequent disease relapse was experienced by approximately 30% of dogs. Medical failure was considered a failure from the onset of its institution in 18% of cases. The mean follow-up time from onset of disease until collection of data was approximately 3 years.

Based on a limited number of reported cases, medical therapy appears to be successful for managing dogs with HNPE; it has been suggested that the biochemical nature of the extruded material in these dogs allows for more rapid resorption compared with calcified disc material.[3–9]

Type II disc disease that is not associated with cervical spondylomyelopathy (CSM) is typically managed medically with restricted activity and antiinflammatory drugs. When surgical intervention is indicated for a disc disease patient, spinal imaging and CSF analysis are typically performed on the anesthetized patient immediately before surgery. Disc fenestration is considered to be an optional ancillary surgical procedure. Fenestration involves removing a segment of annulus fibrosus so that future extrusions will likely occur through that opening rather than into the vertebral canal. Fenestration is performed in the ventral aspect of the disc in the cervical region and in the lateral aspect of the disc in the thoracolumbar region. Disc fenestration is considered to be a prophylactic measure. The efficacy of fenestration remains to be definitively proven. At the present time, fenestration remains a potentially effective but unproven method of decreasing recurrence of disease for type I extrusions, and its use is a matter of clinician preference. Patients with type II disc disease are often controlled adequately for long periods of time with medical therapy.

SURGICAL TREATMENT

Indications for surgical intervention in patients with type I cervical disc extrusions include repeated episodes of pain, pain that is not responding to appropriate medical therapy, and moderate to severe neurologic deficits (tetraparesis, tetraplegia). These indications are guidelines; the decision regarding whether to operate or medically manage a dog with a suspected cervical disc extrusion with neck pain as the main or sole clinical abnormality should be predicated on the apparent severity of the individual patient's discomfort. Many patients with type I disc extrusions appear to be in extreme and unrelenting pain. Surgical removal of compressive disc material in such patients typically leads to immediate relief of neck pain. In addition, we have found that ventrally extruded type I disc material is most readily removed from a ventral slot early in the course of the dog's clinical signs. Adhesions between the extruded material and surrounding structures develop over time, so delayed ventral slot surgery (after 3–4 weeks) may not be as effective in relieving clinical signs. The extruded material is often very hard and immovable in such cases, and venous sinus hemorrhage can be extensive. In situations in which the dog's history of neck pain is protracted (several weeks or longer), it may be more logical to perform a dorsal decompression rather than chance a potentially ineffective ventral slot attempt. An acutely tetraplegic patient should be handled as a surgical emergency with deterioration in neurologic status, whether or not the patient is still ambulatory. The surgical procedure of choice for cervical disc extrusion is usually a ventral slot procedure. Occasionally, a dorsal laminectomy is indicated for cervical disc extrusions in which dorsal or lateral accumulation of disc material or extensive spinal cord swelling is observed. A high success rate has also been reported with surgical management of dogs with HNPE, based on a limited number of cases.[3–9] Surgery is sometimes required in cases of type II disc disease; the surgical procedures employed have generally been the same as those used for type I disc disease. Specific surgical options available for CSM are discussed elsewhere in this chapter.

Preoperative Management

Preoperative management of the patient undergoing cervical spinal surgery is discussed on p. 1366.

Anesthesia

See p. 1366 for anesthetic management of the cervical spinal patient. In addition, see Table 32.1 for anesthetic considerations in the stable orthopedic patient.

Surgical Anatomy

See p. 1366 for pertinent surgical anatomy of the cervical spine.

Positioning

Patient positioning for ventral and dorsal approaches to the cervical spine is discussed on pp. 1367 and 1370, respectively.

SURGICAL TECHNIQUE

The ventral slot procedure is described on pp. 1367 to 1370. The dorsal approach to the cervical spine is described on pp. 1370 to 1373. To remove intraforaminal disc material causing nerve root compression, a dorsolateral hemilaminectomy is performed, along with partial or complete removal of the articular processes (facets). *Once the dorsal lamina is removed to the level of the articular processes, incise the joint capsule of the articular processes with a No. 11 blade, and use a No. 11 blade and Bishop-Harmon forceps to remove this tissue. Once the anatomy of the articular processes is clearly visible, drill these processes off using a high-speed air drill. Protect the lateral surface of the spinal cord during removal of the articular processes by leaving a thin shelf of bone medially as you drill in a dorsal-to-ventral direction (Fig. 40.28), while rongeuring this bone off periodically during removal of the facets (articular processes). Drill off the caudal articular process to visualize the bulk of the cranial articular process ventral to it. Drill the remainder of the cranial articular process entirely, or leave a thin layer of the process to be rongeured off. Locate the spinal nerve roots and the spinal nerve once the cranial articular process is removed. Avoid the vertebral artery, which is ventral to the nerve roots and spinal nerve. Carefully sweep beneath the spinal cord and nerve roots with a probe to remove and free disc material or hemorrhage (Fig. 40.29).* Venous sinus bleeding may occur at this point, but these vascular structures are likely to be clearly visible. *Control venous sinus bleeding as previously described.*

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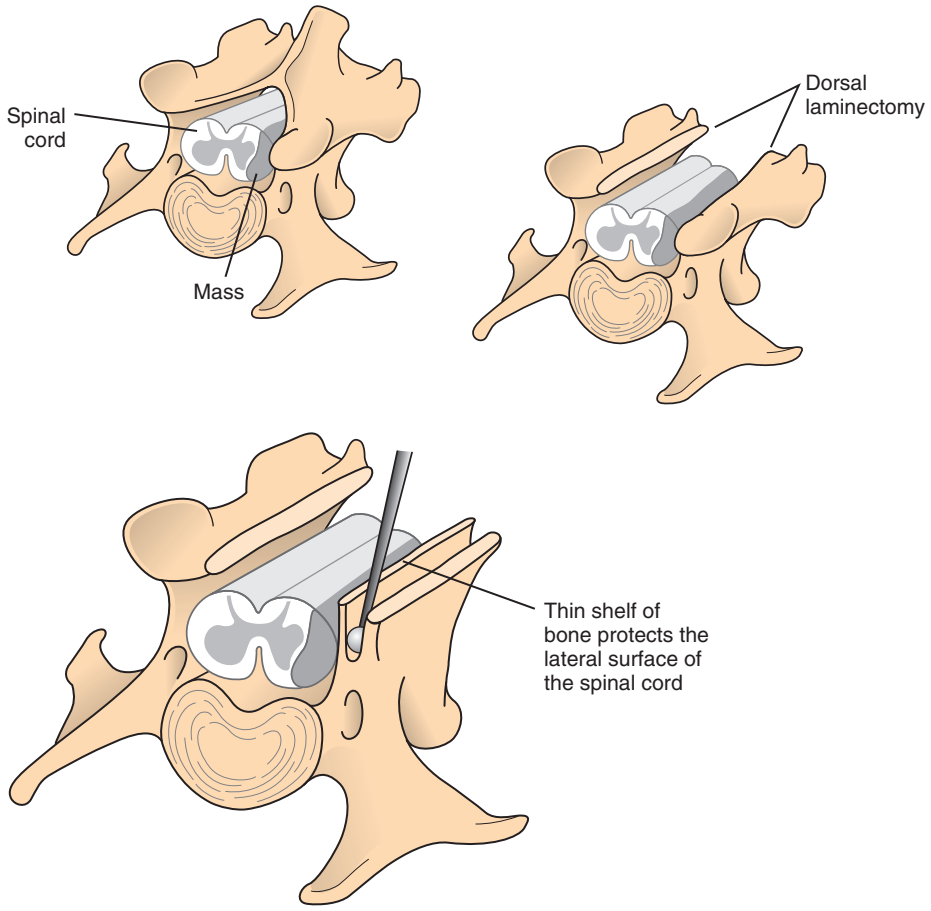


FIG. 40.28 Method of drilling off the articular processes in the cervical region while protecting the spinal cord.

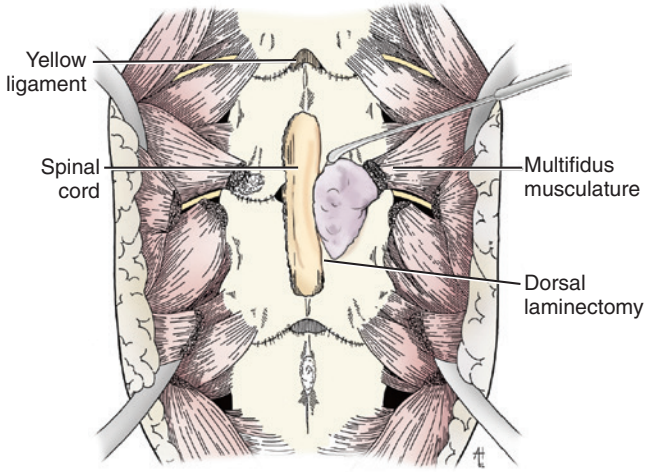


FIG. 40.29 Removal of lateralized type I disc material from a dorsal approach.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

To perform a ventral slot or a dorsal laminectomy, a high-speed air drill, Freer elevators, or Army-Navy osteotomes (large dogs), Lempert rongeurs, surgical Gelfoam, bone wax, and bipolar cautery are recommended.

COMPLICATIONS

Severe complications (e.g., loss of ventilatory ability) are rarely associated with surgery for cervical disc extrusions/protrusions. The most common complications are transient neurologic worsening after surgery and venous sinus hemorrhage (which may require transfusion). In one prospective report of 38 dogs with surgically addressed cervical type I disc extrusion, two dogs (5.2%) were euthanized in the early postoperative period due to severe aspiration pneumonia.[15] In a large (546 cases) retrospective study of dogs with cervical disc extrusions treated via ventral slot surgery, acute adverse events were documented in approximately 10% of the patients. These adverse events were considered to be major in 6.4% of cases (deterioration in neurologic status, persistent pain, intraoperative hemorrhage), and roughly half of this group required reoperation. Adverse events in this study were associated with NSAID administration, less experienced surgeons, C7–T1 extrusions, and intraoperative hypotension.[16] In one retrospective study evaluating dogs treated via dorsal cervical decompression, seven of 31 dogs with acute cervical disc extrusions (22%) experienced some level of postoperative neurologic deterioration.[17] As mentioned earlier, these dogs typically recover completely, although they tend to have a longer convalescent period than dogs treated via the ventral slot procedure. Seromas may form after the dorsal approach to the cervical spine. These typically resolve over 1 to 2 weeks with warm compressing.

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PROGNOSIS

The prognosis for functional recovery in type I cervical disc disease patients is generally good to excellent. In one study of surgically managed type I cervical disc extrusions in small- and large-breed dogs, the overall success rate was 99%.[18] Return to ambulation following surgery for nonambulatory tetraparetic or tetraplegic small- and large-breed dogs with type I cervical disc extrusions averages approximately 1 week (range, 4.5–7 days). In general, it appears from the available literature that recurrence of clinical signs of type I disc extrusion is substantially higher for medically treated versus surgically treated cases. In the study of small- and large-breed dogs mentioned earlier, recurrence of cervical spinal hyperesthesia after surgery was documented in 10% of patients (8% in the small dog group, 13% in the large dog group).[18] However, only 4% of dogs required a second surgery (2.1% in the small dog group, 8.7% in the large dog group).[18]

Cervical type I disc extrusions severe enough to cause compromised respiratory function occur uncommonly; such cases are often associated with a poor prognosis. However, with appropriate respiratory support (i.e., ventilator therapy) after surgery, most of these cases are likely to recover ambulatory function within 2 to 3 months. As the prognosis for HNPE cases appears to be favorable overall with medically and surgically managed cases, it is currently difficult to make generalized recommendations for treating these patients.

CERVICAL SPONDYLOMYELOPATHY (WOBBLER SYNDROME)

DEFINITIONS

CSM is a term used to describe two distinct clinical entities in dogs. One of these is often referred to as **disc-associated CSM (DA-CSM)**, and the other is termed **osseous-associated CSM (OA-CSM)**. DA-CSM refers to a combination of vertebral malformation and malarticulation typically affecting the caudal cervical vertebrae (C5-C6, C6-C7) and associated soft tissue structures (disc, articular process joint capsules, dorsal longitudinal ligament, ligamentum flavum), usually in middle-aged to older, large- and giant-breed dogs. This disorder is typified by protrusion of the disc (type II disc protrusion) at the affected intervertebral space(s). As the name implies, OA-CSM involves primarily bony proliferation associated with the articular processes and associated joint capsules, as well as the dorsal lamina and pedicles, usually without substantial disc protrusion. OA-CSM is also associated with progressive degenerative changes and proliferation of the articular process (facet) joints, including the synovial structures. OA-CSM can affect the spine from C2–C3 through T1–T2, although caudal cervical involvement is most commonly encountered.[19–23]

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The syndrome known as CSM encompasses both DA-CSM and OA-CSM and includes a number of different anatomic abnormalities (Table 40.1). In DA-CSM, chronic instability from the malformation/malarticulation between cervical vertebrae is thought to lead to hypertrophy of soft tissue supportive structures (including the intervertebral disc) over time, with subsequent impingement of the spinal cord. This disorder is typically encountered in middle-aged to older large-breed dogs (e.g., Doberman pinscher, Rottweiler) and is considered a type of type II disc protrusion accompanied by a variable level of intervertebral instability. The main location of spinal cord compression in such cases is usually ventral, associated with the protruded dorsal annulus. The C5-C6 and C6-C7 disc spaces are most commonly involved. Congenital bony stenosis of cervical vertebrae, or OA-CSM, most commonly occurs in young (usually between 1 and 3 years), primarily large- and giant-breed (most commonly) dogs (e.g., great Dane, mastiff, Bernese mountain dog, Doberman pinscher, bassett hound, boerboel [South African dog similar to a bullmastiff]). This latter disease process is frequently included in the category of CSM, or "wobbler syndrome," but it has several distinguishing characteristics from DA-CSM. In addition to affecting cranial cervical through caudal cervical sites, the vertebral canal in these dogs is often malformed and compromised. Hypertrophy of malformed articular processes is also a common feature, sometimes accompanied by cystic enlargement of the articular process joint capsule (extradural synovial cysts). In both

TABLE 40.1 Classification of Wobbler Syndrome

Classification	Age/Breed	Lesion Location	Cause of Compression	General Prognosis
Chronic degenerative disc disease	Adult, male Doberman pinschers	Compresses ventral aspect of spinal cord C5–C7	Disc degeneration and subsequent hypertrophy of ventral annulus fibrosus	Favorable
Congenital osseous malformation	Young great Danes and Doberman pinschers	Compresses spinal cord laterally or dorsoventrally C3–C7	Congenital malformation of vertebral bodies and articular facets	Favorable to guarded
Vertebral tipping	Adult, male Doberman pinschers	Compresses ventral aspect of spinal cord C5–C7	Dorsal malposition of the affected vertebral body in the vertebral canal	Favorable
Ligamentum flavum/ vertebral arch malformation	Young great Danes	Compresses dorsal aspect of spinal cord C4–C7	Hypertrophy and hyperplasia of the ligamentum flavum; vertebral arch malformation	Favorable to guarded
Hourglass compression	Young great Danes	Compresses spinal cord on all sides C2–C7	Hypertrophy of ligamentum flavum and annulus fibrosus; malformation or degenerative disc disease of articular facets	Favorable to guarded

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DA-CSM and OA-CSM, single or multiple site involvement can occur. [19–23]

DIAGNOSIS

Clinical Presentation

Signalment

As mentioned, there are two common scenarios for CSM in terms of typical signalment. Cases of caudal cervical malalignment/malarticulation, or DA-CSM, usually occur in middle-aged to older large-breed dogs, most commonly Doberman pinschers. In the OA-CSM scenario characterized by more obvious vertebral malformation, young adult giant breeds (e.g., great Dane, mastiff) are typically presented for progressive signs of cervical myelopathy. A clinical scenario similar to DA-CSM has also been described in small-breed dogs, most notably following ventral slot procedures. Instability in these dogs was likely due to excessive ventral slot size, with subsequent intervertebral instability.

History

The most common historical complaints relate to abnormal ambulation that progressively worsens over weeks to months. In many cases involving the caudal cervical region, the pelvic limbs are more notably affected than the thoracic limbs. Giant-breed dogs with OA-CSM frequently have multiple lesion sites, sometimes including cranial to midcervical involvement, and therefore occasionally display more obvious thoracic limb involvement.

Physical Examination Findings

Clinical signs of DA-CSM or OA-CSM affecting the caudal cervical region are typically consistent with a caudal cervical myelopathy, with the pelvic limbs usually more obviously affected than the thoracic limbs. If ambulatory, these patients typically exhibit a stiff, choppy, shuffling thoracic limb gait and an ataxic, wide-based, pelvic limb gait ("two-engine" gait). A thoracic limb posture characterized by elbow abduction and internal rotation of the digits (toe-in) may be appreciated in some dogs. Proprioceptive deficits are usually more appreciable in the pelvic limbs than in the thoracic limbs, and it may be difficult to distinguish these patients as having a C6-T2, rather than a T3-L3, myelopathy. Having these dogs walk with the head held in a mildly extended position may accentuate thoracic limb proprioceptive deficits. We have found that dogs with caudal CSM lesions that display subtle thoracic limb deficits are often resistant to walking with mild head extension, as if they realize they are not aware of the position of their thoracic limbs. These dogs may display neck pain, but it is often subtle. Low head carriage (flexed neck position) and resistance to lateral movement and extension of the neck may be appreciated. We have found that direct dorsal digital pressure applied to the ventral aspect of the vertebrae usually elicits discomfort in the affected cervical region(s) of dogs with CSM. Onset of clinical signs is typically slowly progressive over weeks to months, but is occasionally acute and associated with some minor traumatic event. As mentioned previously, OA-CSM patients may have cranial to midcervical lesions, and therefore may exhibit more obvious thoracic limb impairment than those patients with more caudally located lesions.

Diagnostic Imaging

The diagnostic imaging modality of choice for both DA-CSM and OA-SCM is MRI. Traction and flexed and extended sagittal MR views may provide clinically useful information in DA-CSM cases that can better identify lesions and help guide surgical planning.[24] Myelography has historically been used for both DA-CSM and OA-CSM; this imaging modality does not provide the detail afforded by MRI and often leads to transient neurologic worsening following the procedure. Myelography for DA-CSM has typically been performed using several stress (flexion, extension, linear traction) views (Fig. 40.30). These different views may be necessary (especially linear traction) to adequately define the specific abnormalities and to guide surgical decision making, if that route is chosen. Both CT and MRI have been demonstrated to be useful imaging modalities for CSM. As previously noted, MRI (Fig. 40.31), including linear traction views, for DA-CSM is preferred. In addition to providing a more detailed image of extradural compression, MRI provides valuable information regarding spinal cord parenchymal changes (e.g., edema). Hyperintense lesions of the spinal cord parenchyma on T2-weighted MRI have been demonstrated in more than half of DA-CSM Dobermans and seem to be correlated with more severe clinical presentations. Similarly, T2-hyperintense spinal cord parenchymal lesions were demonstrated in nine of 15 great Danes with OA-CSM in one study.[19] One or more of the following types of spinal cord compression at one or more disc spaces may be observed on spinal imaging for DA-CSM: ventral compression from a malaligned vertebral body; protruded dorsal annulus, or hypertrophied dorsal longitudinal ligament; dorsal compression from hypertrophied ligamentum flavum; and lateral compression from hypertrophied articular processes (facets) and associated joint capsule tissue.

Linear traction views are probably the most informative and the safest of the "stressed" views used to document whether a lesion is static or dynamic. Linear traction and flexion

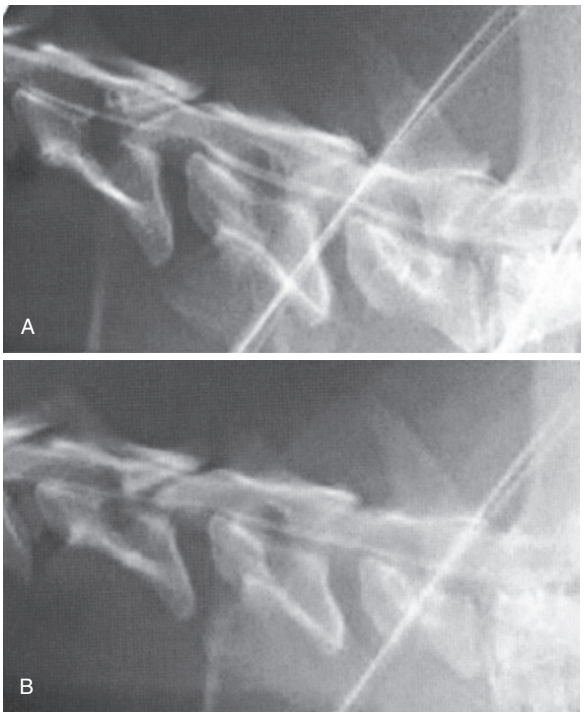


FIG. 40.30 Lateral myelographic images of a caudal cervical spondylomyelopathy lesion at the C5–C6 intervertebral disc space (A) before and (B) after application of linear traction.

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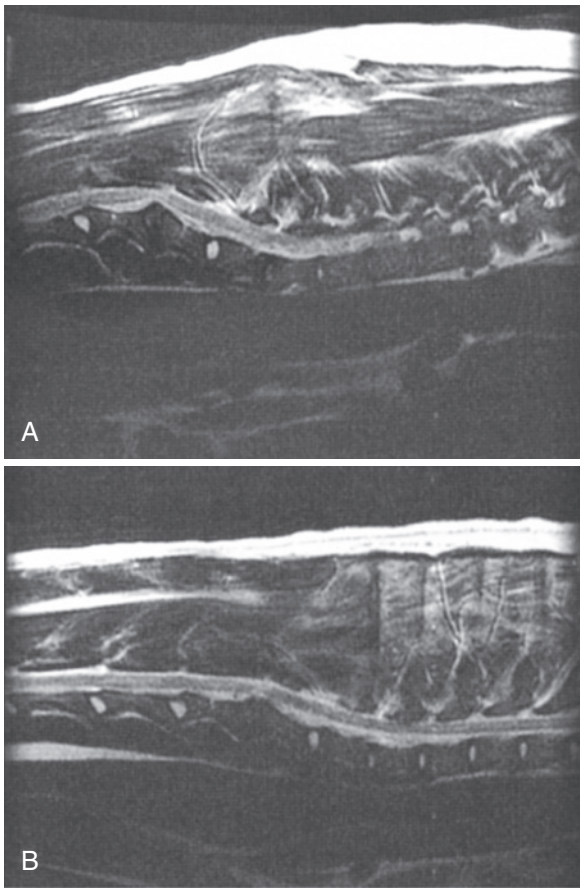


FIG. 40.31 Sagittal T2-weighted cervical spine magnetic resonance images from a dog with caudal cervical spondylomyelopathy (A) before and (B) after application of linear traction.

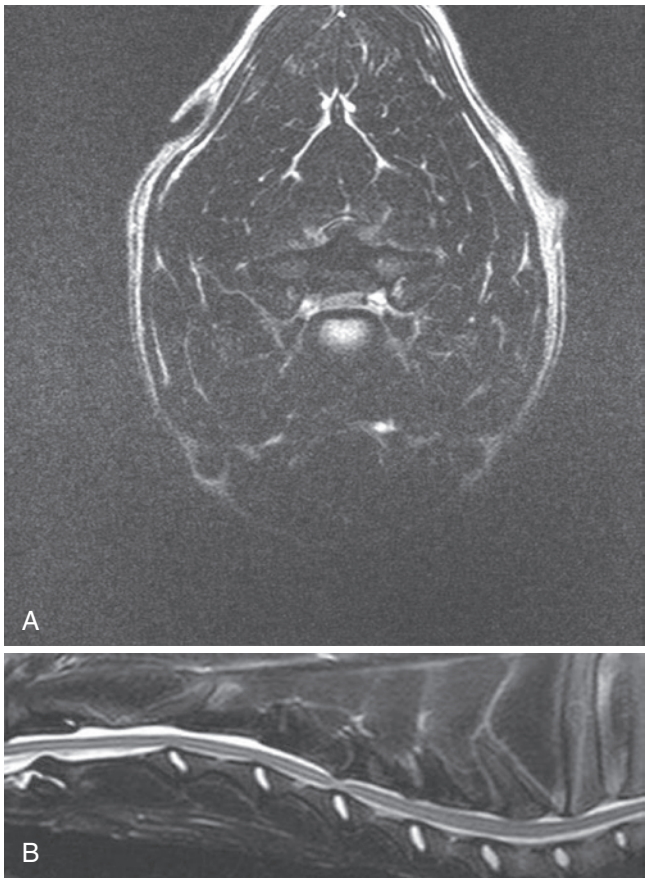


FIG. 40.32 (A) Axial and (B) midsagittal T2-weighted magnetic resonance images of a great Dane with a severely compressive osseous-associated cervical spondylomyelopathy lesion at C4–C5 and a smaller compression at C5–C6 (shown on midsagittal view).

(ventroflexed) views often relieve these compressive lesions, whereas extension views often exacerbate them. Extension views should be performed cautiously; the information gained from such views may not be worth the potential trauma they may induce. Change in the character of the lesion(s) with different views suggests that it is a dynamic, rather than a static (e.g., isolated type II disc protrusion), disorder. However, interpretation of these additional images is highly subjective, especially in cases in which lesions improve but do not dissipate on linear traction views. In many cases, assessment of whether a cervical lesion is static or dynamic is based on a number of clinical factors (e.g., history, degree of hyperesthesia), in addition to evidence of posttraction alleviation of spinal cord compression on imaging studies. MRI abnormalities typically observed in OA-CSM cases include bony abnormalities associated with the articular processes and associated joint capsule (e.g., joint capsule proliferation, extradural synovial cysts), as well as compressive bony abnormalities of the pedicles and lamina (Fig. 40.32). Spinal cord compression at these abnormal articulations is typically dorsolateral or lateral and is frequently bilateral. Stenosis of the intervertebral foramina at affected sites is also a common observation. As mentioned, T2-hyperintense parenchymal lesions are often visualized at the compression site(s). Most dogs with OA-CSM have more than one site of compression evident on MR images.[19–23]

In addition to the ambiguities associated with interpretation of traction views, one study comparing clinically normal Doberman pinschers with those with clinical signs of DA-CSM reported that four of 16 clinically normal dogs had MRI evidence of spinal cord compression. Twelve of these "normal" dogs had evidence of disc degeneration, and 11 had evidence of foraminal stenosis. The clinically affected dogs had significantly smaller vertebral canals and wider disc spaces than their clinically unaffected counterparts. These findings suggest that a relative vertebral canal stenosis combined with potentially more disc material protruding into the comparatively narrow vertebral canal may represent predisposing factors for Doberman pinschers with clinically apparent DA-CSM. In one study, transcranial motor evoked potentials elicited from the cranial tibial muscle in Doberman pinschers were found to be predictive of both MRI lesions and clinical disease due to DA-CSM.[25,26] Similar to DA-CSM, MRI abnormalities consistent with OA-CSM can be observed in clinically normal great Danes; however, there are distinct differences between abnormalities detected in clinically normal great Danes versus those with clinical features of OA-CSM. In one MRI study of great Danes with or without clinical signs of OA-CSM, one of 15 clinically normal dogs had obvious evidence of spinal cord compression compared with 15 of 15 dogs with clinical features consistent with OA-SCM. In this study, no clinically normal great Danes had evidence of spinal cord parenchymal T2-hyperintensity.[19] Additionally, it was demonstrated from a morphometric comparative study that clinically affected great Danes with OA-CSM demonstrate absolute vertebral

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canal stenosis and severe foraminal stenosis, compared with clinically normal great Danes.[21]

Laboratory Findings

Laboratory findings are typically normal in cases of CSM. As with cervical intervertebral disc disease (see p. 1377), some mild laboratory abnormalities may be associated with prior glucocorticoid administration.

DIFFERENTIAL DIAGNOSIS

Numerous disorders other than CSM can lead to clinical features of a progressive cervical myelopathy. The most likely differential diagnoses are type II disc protrusion and neoplasia (especially in older patients).

MEDICAL MANAGEMENT

Medical treatment, consisting of cage confinement (3–4 weeks), antiinflammatory medications (e.g., prednisolone), and potentially the use of a neck brace, can be attempted in these dogs. As with other disc-associated problems, the patient should be gradually returned to normal activity over 4 to 6 weeks if the initial cage confinement therapy is successful. Surgical therapy is often pursued because CSM is typically a progressive syndrome, and medical therapy has been traditionally regarded as ineffective or effective only transiently. However, one retrospective report compared medical therapy with surgical therapy in 104 dogs with DA-CSM and found that medical therapy was often effective for the disease. Specifically, it was found that the success rate for the surgically treated group was 81% and for the medically treated group 53.5%; no significant difference was noted between these groups. In addition, 26.5% of the medically treated group stayed the same (vs. 2% in the surgical group). No significant difference in mean survival time was noted between groups (46.5 months for medically treated, 48.2 months for surgically treated).[27] Although this study strongly supports the use of medical management as a viable treatment option for CCSM, it should be kept in mind that a variety of surgical procedures were represented in the surgical group, and 78% of them were ventral slots. Although performing a ventral slot alone has been advocated by some as an adequate surgical procedure for CCSM, the literature suggests a lower success rate and higher recurrence rate with this approach, in comparison with distraction-stabilization procedures.

The number of DA-CSM cases presenting for spinal imaging and surgical management has decreased dramatically over the last several years. Whether this decrease is due to improving success rates with medical management, a decrease in disease incidence in affected dog breeds, or some combination of these two factors is unknown. Conversely, the incidence of OA-CSM cases presented for spinal imaging and either medical or surgical management has sharply increased over the last several years. Medical management for OA-CSM is similar to that used for DA-CSM cases and has been demonstrated to provide long-term control of clinical signs, although neurologic deterioration is common.[23]

SURGICAL TREATMENT

Numerous surgical procedures have been advocated for dogs with DA-CSM. They fall into two major categories: ventral and dorsal procedures. Ventral procedures usually involve a ventral slot procedure at the affected space, combined with distraction-stabilization; this is often combined with cancellous bone grafting at that space. Dorsal laminectomy is generally considered to be the procedure of choice for OA-CSM and is also preferred in some cases of multiple site DA-CSM.

Preoperative Management

Preoperative management of the patient undergoing cervical spinal surgery is discussed on p. 1366.

Anesthesia

See p. 1366 for anesthetic management of the cervical spinal patient. In addition, see Table 32.1 for anesthetic considerations in the stable orthopedic patient.

Surgical Anatomy

See p. 1366 for pertinent surgical anatomy of the cervical spine.

Positioning

Patient positioning for ventral and dorsal approaches to the cervical spine is discussed on pp. 1367 and 1370, respectively.

SURGICAL TECHNIQUE

Ventral Procedures (Distraction-Stabilization)

Commonly employed distraction-stabilization procedures include pin or screw placement into vertebral bodies with a PMMA bridge, and PMMA "plug" insertion into the distracted ventral slot. We prefer a combination procedure utilizing the two techniques (Fig. 40.33).

After performing a ventral slot as previously described, undercut the cancellous bone with an angled burr guard and a 2-mm burr at the cranial and caudal aspects of the slot (Fig. 40.34). These divots will help hold the PMMA in place. *Put a small, thin fat graft over the spinal cord at the bottom of the slot. With an assistant providing traction via a rope around the upper canine teeth, place a small amount of PMMA into the slot, working it into the previously drilled divots with Freer elevators.* The PMMA should have a doughy consistency and should not be sticky when inserted into the slot.

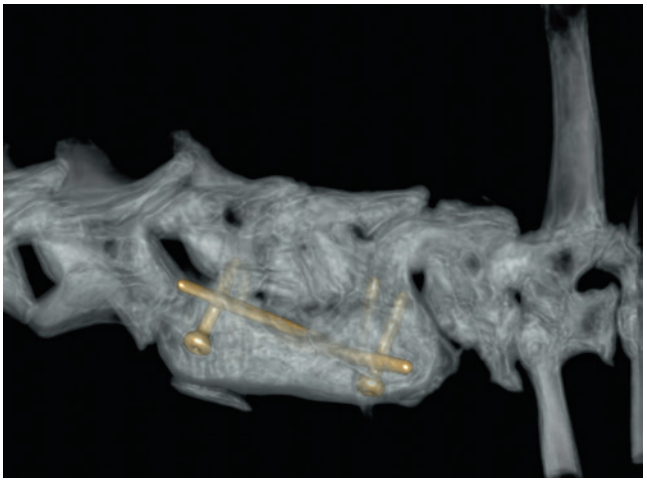


FIG. 40.33 Lateral view of a postoperative three-dimensional computed tomography reconstruction of a caudal cervical spondylomyelopathy lesion surgically addressed via a distraction-stabilization procedure.

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Lavage the surgical site copiously with cool saline as the PMMA plug hardens. Once the PMMA plug has hardened, have the assistant slowly release the applied traction. The PMMA plug will maintain distraction during the remainder of the procedure.

Using a 2-mm burr, drill two guide holes through the outer cortical bone of each ventral vertebral body, angling the hole at approximately 30 to 35 degrees from midline, starting the hole just lateral to midline. Self-tap 4-0, 22 mm (small-frame Doberman) to 24 mm (large-frame Doberman) partially threaded cancellous bone screws through these guide holes to approximately the level of the last thread (Fig. 40.35). Intraoperative radiographs are often helpful in determining proper screw depth. Once the screws have been inserted, place premeasured segments of intramedullary (IM) pins (e.g., 0.062-mm pins) across the screw heads to serve as extra support (Fig. 40.36). Finally, place a PMMA bridge over the screws and crossed IM pins, molding it with Freer elevators as it hardens. Close the incision routinely. Several modifications of this procedure can be performed, depending on surgeon preference. IM pins rather than screws can be used (Fig. 40.37). The potential advantage of IM pins is bicortical bone purchase with a stronger mechanical construct than cancellous screws. A potential disadvantage is inadvertent penetration of the vertebral canal or intervertebral foramen. Cortical screws or fully threaded screws can be used, and predrilling and tapping can be performed, depending on surgeon preference. Some surgeons prefer to stop the ventral slot for this procedure once the level of inner cortical bone is reached. We prefer to remove as much of the compressive fibrous material as possible; therefore a full slot is performed. The potential for insertion of PMMA into the vertebral canal exists with a complete ventral slot, but this is very unlikely if the PMMA is placed cautiously and at the right consistency (i.e., not liquid). Application of a spinal plate (polyvinylidene plate, cervical spine locking plate) to the ventral vertebral bodies with distraction and cancellous bone grafting has also been described (Figs. 40.38 and 40.39). With the cervical spine locking plate procedure, vertebral end plates are preserved and discectomy is performed rather than a ventral slot. Distraction during these ventral procedures can be provided by using a modified Gelpi retractor placed in predrilled holes in adjacent vertebrae.

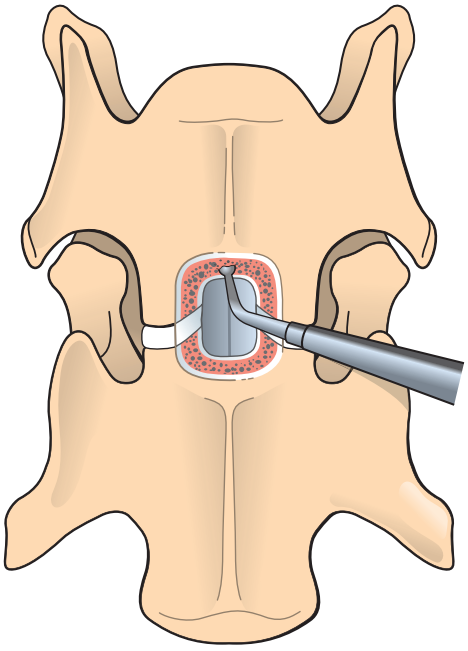


FIG. 40.34 Undercutting the ventral slot with an angled burr guard before placement of a polymethylmethacrylate plug.

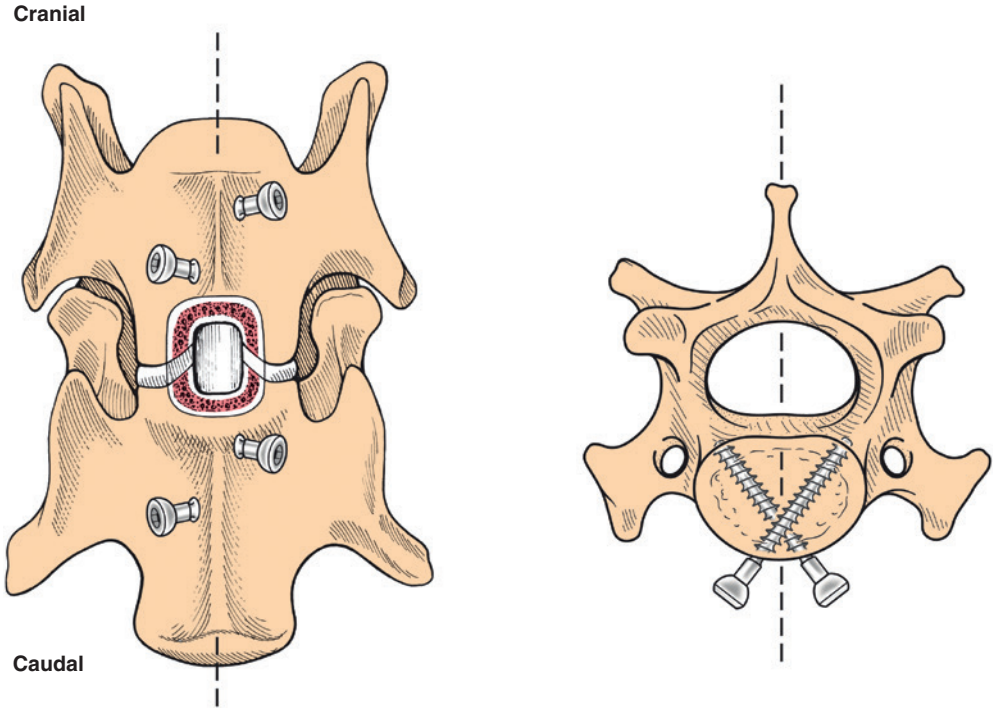


FIG. 40.35 Placement of cancellous screws in the vertebral bodies.

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NOTE Do not place the points of the Gelpi retractor in adjacent disc spaces because this may weaken the discs and predispose them to instability (Fig. 40.40).

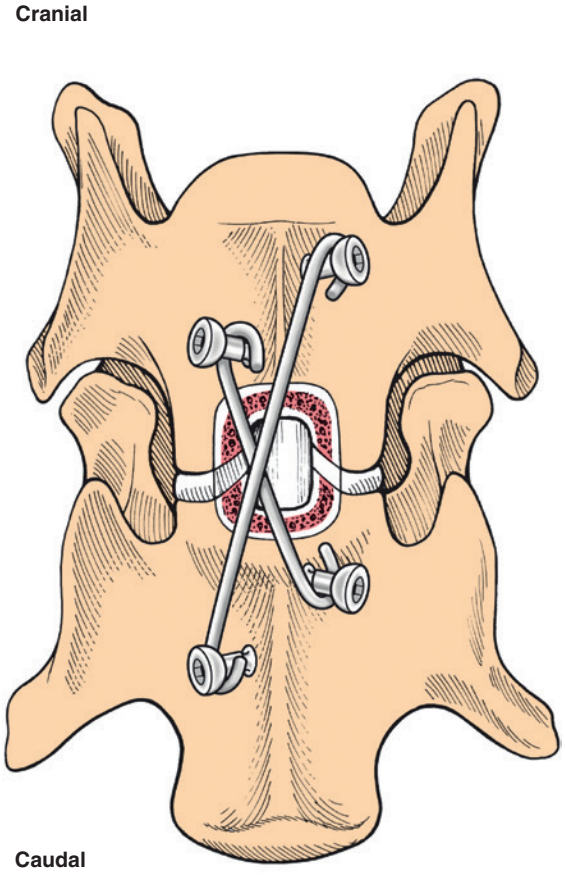


FIG. 40.36 Placement of segments of crossed intramedullary pins across the screw heads.

Dorsal Procedures (Laminectomy ± Stabilization)

Dorsal procedures involve a dorsal laminectomy for decompression, with or without articular facet screws or pins to help stabilize the joint space. The dorsal approach may be preferable when multiple disc spaces are affected or when the compression is primarily dorsal or lateral. The dorsal approach to the cervical spine is described on pp. 1370 to 1373.

To stabilize the articular processes with pins or screws, first open the joint capsule so that the articular surfaces are visualized. Using a No. 12 blade, scarify the articular surfaces of the joint. Using a mini-driver, insert the pin in the center of the caudal facet, aiming slightly laterally and ventrally and roughly perpendicular to the angle of the joint articulation. Drive the pin through the caudal and cranial facets (articular processes). Cut the pin to the desired length with a pin cutter. Stabilize the articular processes with a screw in a similar fashion, but predrill (mini-driver), tap, and measure the hole for the cortical screw before placing the screw with a screwdriver. The screw can be placed flush with the surface of the caudal facet or placed in lag fashion, or a variable amount of the proximal screw can be left extruding from the caudal facet (if it is to be used to tie in other hardware or PMMA).

Attempt to limit the pin or screw depth to just beyond the ventral aspect of the cranial facet to avoid underlying soft tissues (e.g., spinal nerve, vertebral artery). Premeasuring the distance (in millimeters) from the dorsal aspect of the caudal facet to the ventral aspect of the cranial facet on axial CT or MRI is helpful in choosing implant lengths. Facet pins or screws can be used alone (Fig. 40.41) or in combination with PMMA and Steinmann pins (Fig. 40.42).

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Basic instruments needed to perform ventral or dorsal procedures are covered on pp. 1374 and 1380. In addition to these instruments, a mini-driver, screws (± drill bits, taps, depth gauge), IM pins, specialized plates, and PMMA may be needed, depending on the specific procedures performed. Modified (blunt-ended) Gelpi retractors may also be needed for distraction.

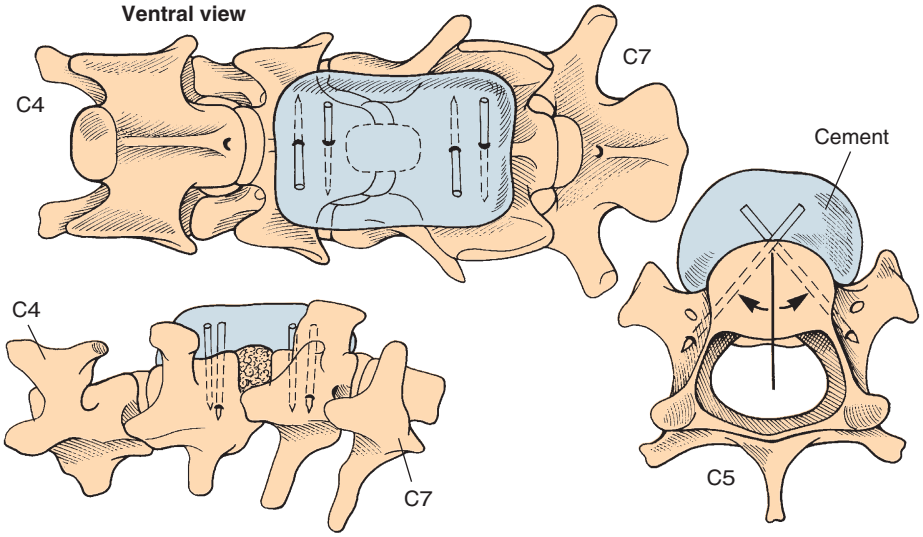


FIG. 40.37 Proper placement of Steinmann pins and polymethylmethacrylate to provide traction and stabilization to the affected intervertebral space.

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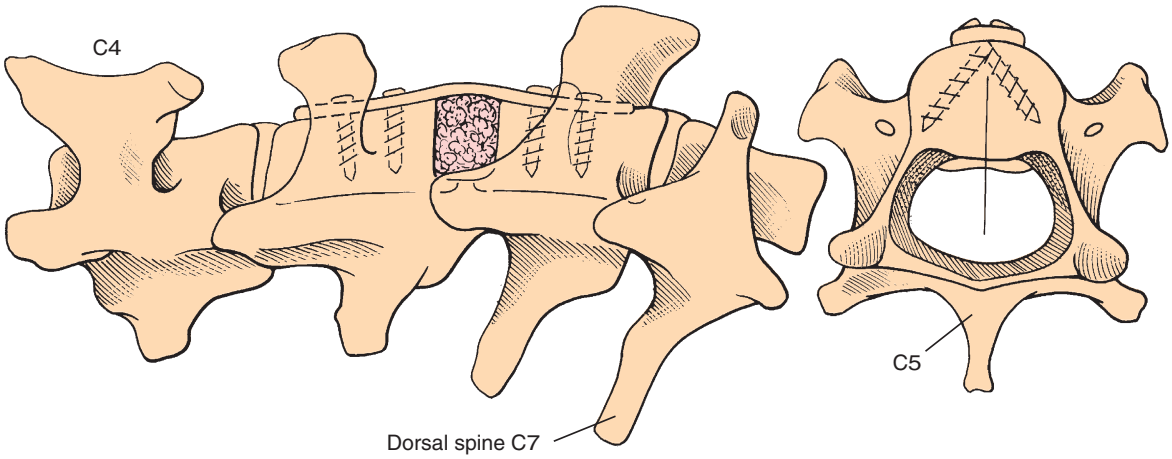


FIG. 40.38 Proper placement of full cortical allograft and polyvinylidene spinal plate to provide traction and stabilization to the affected intervertebral space.

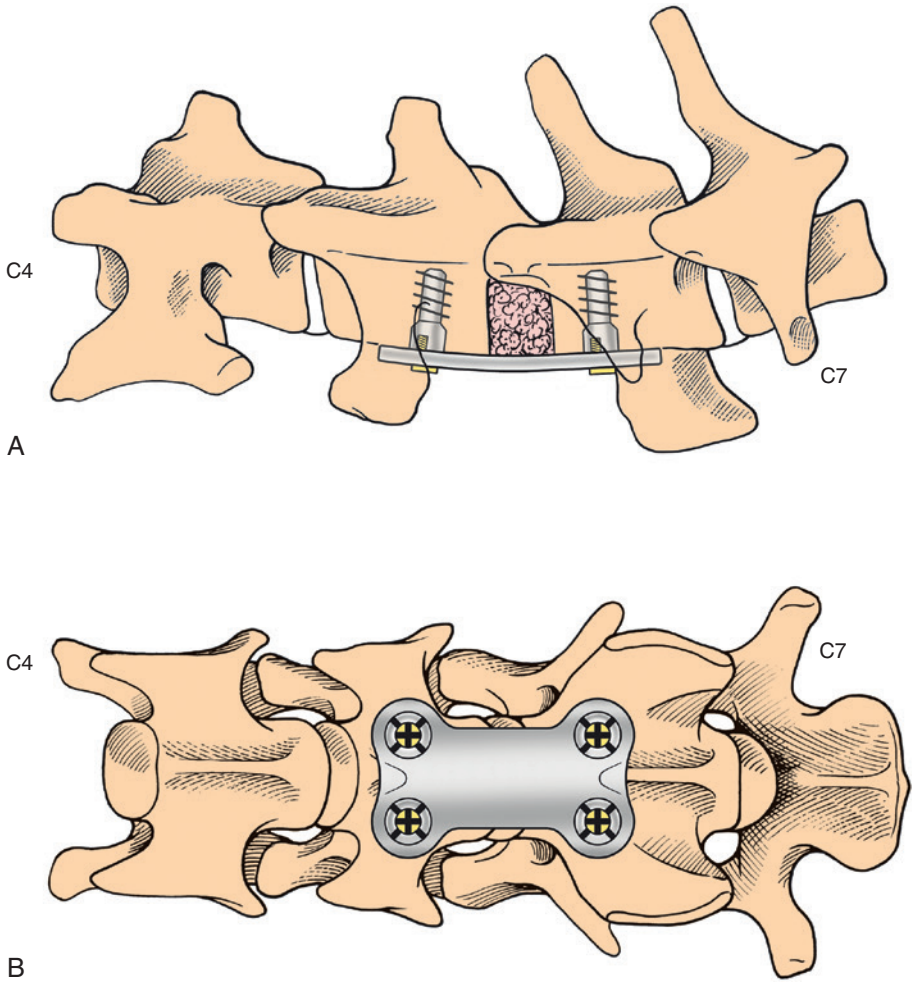


FIG. 40.39 Use of a spinal locking plate for caudal cervical spondylomyelopathy in the dog. Postoperative lateral (A) and ventrodorsal (B) views.

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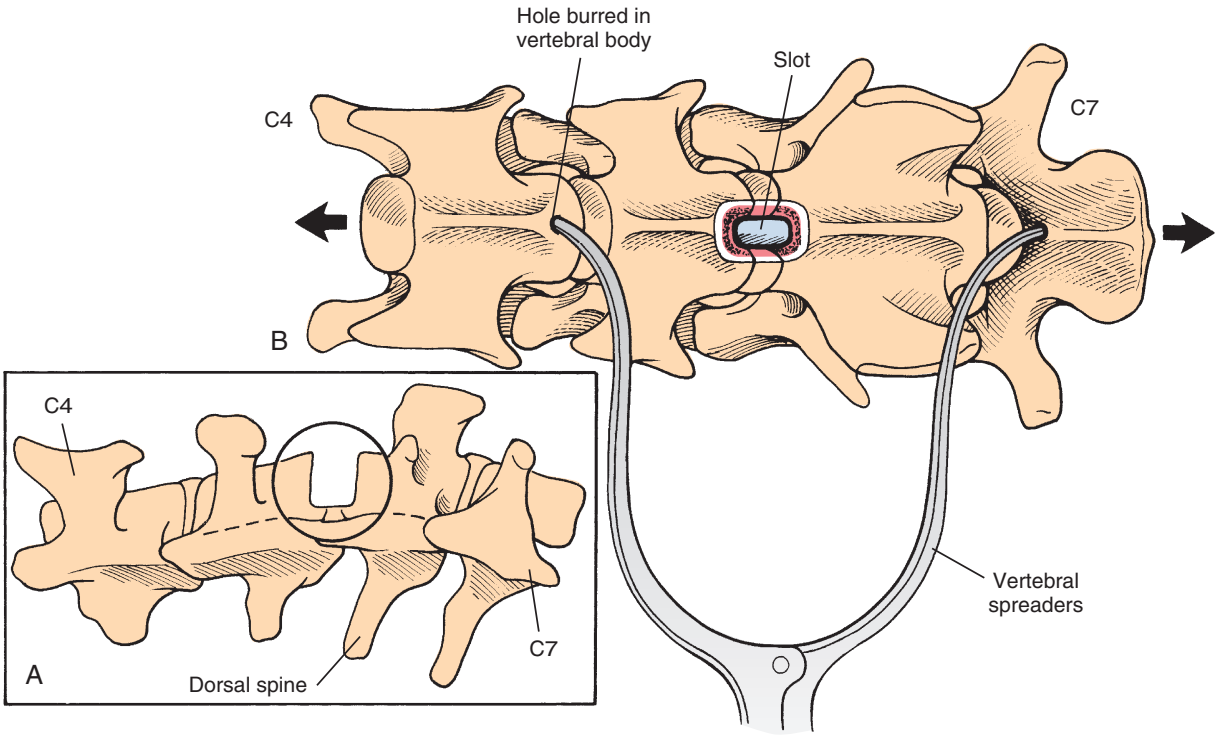


FIG. 40.40 (A) To decompress the spinal cord, perform a 75% transdiscal ventral slot at the affected intervertebral space. (B) Use vertebral spreaders to place the affected intervertebral space in 2 to 3 mm linear traction.

COMPLICATIONS

Complications associated with cervical spinal surgery are discussed on p. 1375. In addition to these complications, implant failure and infection are potential complications of distraction-stabilization procedures in DA-CSM patients. For dogs with OA-CSM treated via dorsal laminectomy, it is very common for dogs to experience transient neurologic worsening postoperatively, and some of these patients will be nonambulatory. Surgeons should be prepared to provide access to postoperative physical therapy/rehabilitation services for these dogs (see Chapter 11).

PROGNOSIS

The prognosis for dogs with both DA-CSM and OA-CSM treated surgically is generally good, but it is somewhat unpredictable compared with type I disc extrusions. Numerous reports have described various surgical procedures for DA-CSM, with success rates varying between approximately 70% and 90%. A distraction-stabilization technique using two string-of-pearl plates and a Fitz intervertebral traction screw was associated with significant neurologic improvement in 15 of 16 dogs by 6 weeks postoperatively; the majority of the dogs available for long-term follow-up had good to excellent outcomes.[28] Many dogs require a prolonged recuperative phase before regaining functional status, with attendant need for intensive nursing care. This is especially true for dogs undergoing dorsal laminectomy, which are more likely to worsen neurologically after surgery than those undergoing a ventral procedure. Although dogs undergoing distraction-stabilization procedures tend not to worsen neurologically after surgery to the extent exhibited by dogs undergoing dorsal laminectomy, prolonged recuperation is often required for these patients as well. Dogs that present nonambulatory and/or have more than one lesion on spinal imaging tend to have a less favorable prognosis than ambulatory dogs and those having only one demonstrable lesion. In general, dogs with CCSM that are ambulatory at presentation and have myelographic or CT/MRI evidence of a single affected intervertebral space have a favorable prognosis for functional recovery. The recurrence rate for DA-CSM has been reported to be up to 28% (most recent studies are <16%). The recurrence rate for the Fitz intervertebral traction screw/string-of-pearl plate procedure was 6.25%.[28] With ventral or dorsal procedures, surgical stabilization of one disc space may lead to abnormal biomechanical stresses on spaces cranial and caudal to those spaces. This is thought to lead to secondary lesions at these latter spaces over time and is referred to as the *domino effect*.

Information regarding prognosis for dogs with OA-CSM is somewhat limited compared with that available for dogs with DA-CSM. In one retrospective case series comparing medical and surgical (dorsal laminectomy) treatment of dogs with OA-CSM, it was found that long-term disease management was often achievable with either modality. Median survival for medically managed dogs in this study was approximately 3.6 years, compared with 5 years for surgically managed cases. Approximately 80% of surgically managed dogs improved neurologically and experienced good to excellent long-term outcomes. These dogs were also more likely to be euthanized for reasons unrelated

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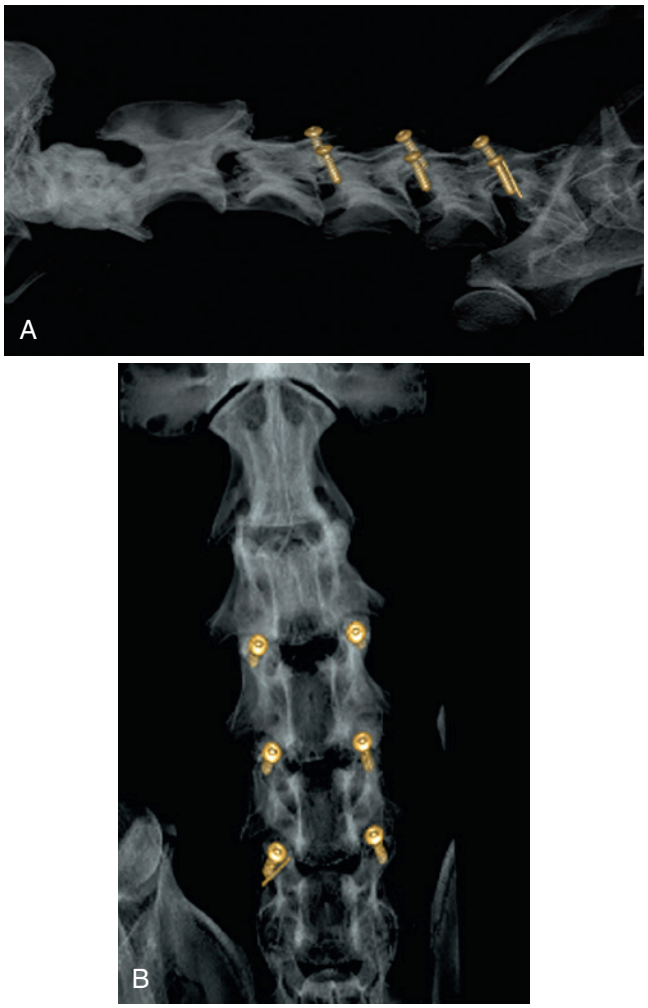


FIG. 40.41 (A) Lateral and (B) dorsal views of a three-dimensional computed tomography reconstruction, showing placement of articular facet screws in a caudal cervical spondylomyelopathy patient, following dorsal laminectomy.

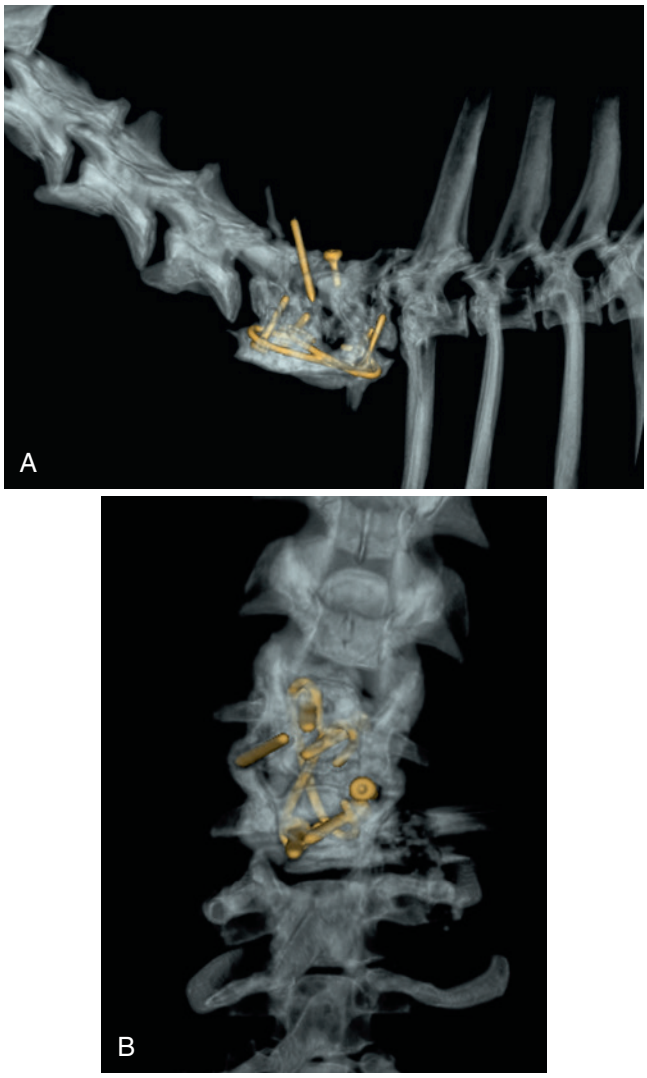


FIG. 40.42 (A) Lateral and (B) dorsal views of a three-dimensional computed tomography reconstruction, showing placement of articular facet pins, Steinmann pins, and polymethylmethacrylate following traumatic fracture/luxation.

to OA-CSM. The medically treated dogs generally worsened neurologically over time and were more likely to be euthanized due to OA-CSM.[23]

CRANIOCERVICAL JUNCTION ANOMALIES

DEFINITIONS

Craniocervical junction anomalies (CJAs) is an umbrella term used to describe a group of developmental disorders of the craniocervical junction region (supraoccipital bone, C1 and C2 vertebrae). These abnormalities include Chiari-like malformation (see p. 1353), AA instability, atlanto-occipital overlapping, and dorsal compression at C1–C2. Atlantoaxial instability refers to excessive motion at the C1–C2 joint, usually due to a hypoplastic or absent dens. **AOO** is a condition in which the atlas is displaced toward and into the foramen magnum. **Dorsal compression at C1–C2** is typically seen as soft tissue encroaching on the underlying spinal cord; this may be a reaction to instability at that joint. **Syringomyelia,** which is the accumulation of fluid within the parenchyma of the spinal cord, is usually secondary to one or more CJAs.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

AA instability is usually caused by hypoplasia or aplasia of the dens. Abnormal ligamentous support of the dens may also be involved. The instability may lead to dorsal subluxation or luxation of the axis, with resultant compression of the cranial cervical spinal cord. Associated malformations of the atlas or occipital bones may be observed in some patients. Some patients with AA instability have syringomyelia evident on MRI. An abnormality at the craniocervical junction in small- and toy-breed dogs that has recently been described is called *AOO.* In this malformation, the atlas (C1) is cranially displaced into the foramen magnum, and overlap of the occipital bone and the atlas occurs (Fig. 40.43). This displacement tends to compress the caudal aspect of the cerebellum and to elevate and compress the caudal medulla (medullary kinking). AOO is likely a form of basilar invagination. Basilar invagination is a human craniocervical junction disorder

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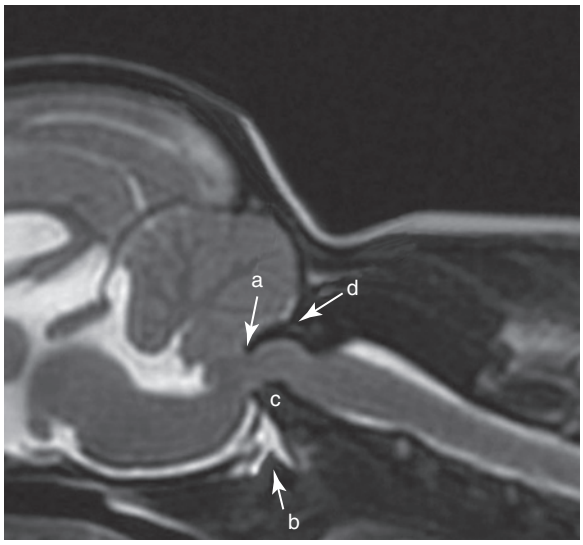


FIG. 40.43 Sagittal T2-weighted magnetic resonance image of a dog with atlanto-occipital overlapping and atlantoaxial subluxation. *a,* Cranial-most aspect of dorsal arch of C1; *b,* body of C1; *c,* dens; *d,* ventral extent of the supraoccipital bone.

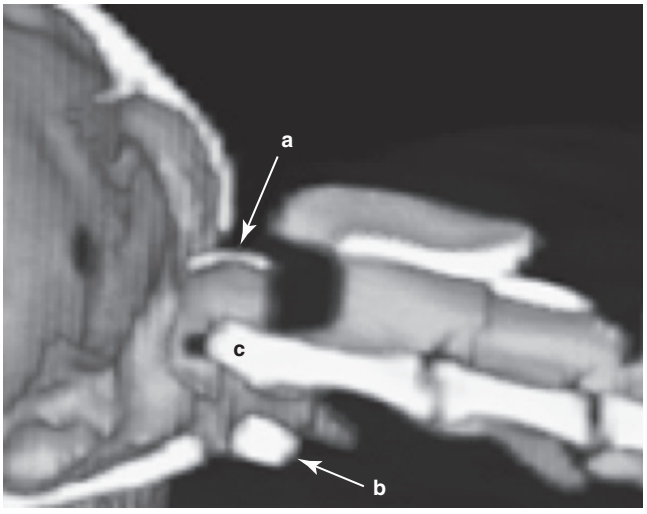


FIG. 40.44 Midsagittal reconstructed computed tomography image of the dog in Fig. 40.43, showing bony details of the abnormal anatomy. *a,* Dorsal arch of C1; *b,* body of C1; *c,* dens.

in which the atlas and the axis (C2) telescope toward the foramen magnum. We have seen this disorder as a sole entity and in combination with CLM or AA instability. Because bony detail is difficult to distinguish on MRI, it is likely that AOO has been underdiagnosed in dogs, and most cases have been incorrectly ascribed a diagnosis of CLM. The precise nature of this and other craniocervical malformations is typically apparent on CT imaging (Fig. 40.44). Similar to the AOO seen in small- and toy-breed dogs, we have seen a large number of dogs with dorsal compression at the level of C1–C2. This compression varies in severity, with some dogs having a mild divot in the dorsal subarachnoid space (Fig. 40.45) and others having severe cervical spinal cord compression (Fig. 40.46). At surgery, most of this

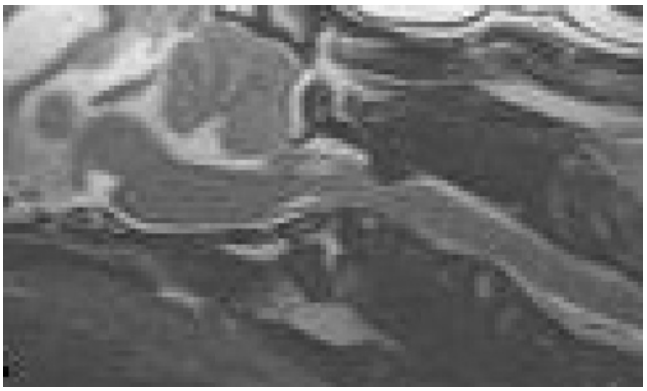


FIG. 40.45 Sagittal T2-weighted magnetic resonance image of a dog with a mild dorsal compressive lesion at C1–C2.

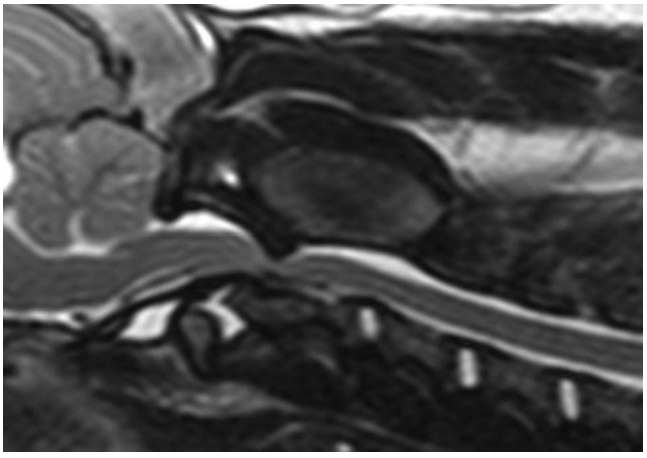


FIG. 40.46 Sagittal T2-weighted magnetic resonance image of a dog with a severely compressive dorsal lesion at C1–C2.

compressive mass appears to be soft tissue. This disorder is also likely a manifestation of instability at the C1–C2 junction, possibly a form of basilar invagination like the AOO problem. This can occur as a sole entity or in combination with CLM, AOO, or AA instability.

DIAGNOSIS

Clinical Presentation

Signalment

AA instability is usually seen in miniature and toy dogs, often younger than 2 years, but it has been reported in older dogs and in larger dog breeds. Commonly reported breeds with AA instability include Yorkshire terrier, Pomeranian, miniature and toy poodle, Chihuahua, and Pekingese. It occasionally occurs in cats. AOO and C1–C2 dorsal compression also appear to occur in predominantly miniature and toy dog breeds at young ages.

History

Historical complaints for dogs with AA instability, AOO, and C1–C2 dorsal compression typically include neck pain and varying degrees of ataxia of all four limbs. Nonambulatory tetraparesis and tetraplegia occur in severe cases.

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Physical Examination Findings

Dogs with these CJAs typically display evidence of cervical (neck) pain and often have varying degrees of tetraparesis. Some dogs may also have evidence of paraspinal pain locations other than the cervical region, suggesting the presence of syringomyelia in those areas.

Diagnostic Imaging

In most cases of AA instability, the abnormality is apparent on lateral radiographs of the neck (Fig. 40.47). Stressed views may be used to demonstrate AA instability, but they must be obtained with caution. Overzealous flexion of the neck to demonstrate instability of the C1–C2 joint space may have disastrous results. MRI is both safer for this disorder and preferable to radiographs in that concurrent craniocervical junction abnormalities and syringomyelia will also be identified if present (Fig. 40.48). MRI does not provide very good bony detail. In cases of CJA, we have found that MRI followed by CT imaging of the abnormal region at the craniocervical junction is often necessary to fully characterize the abnormal anatomy.

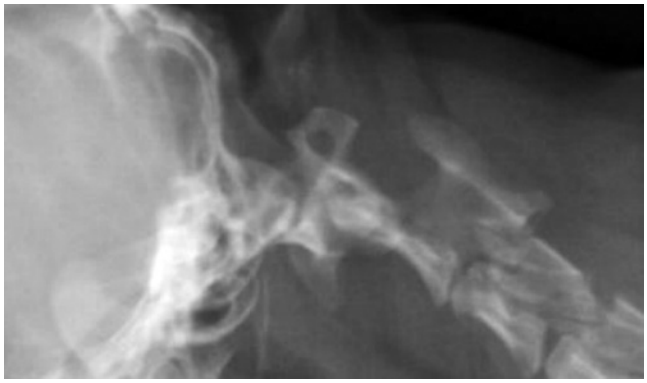


FIG. 40.47 Lateral radiograph of a dog's cervical spine, showing atlantoaxial joint instability.

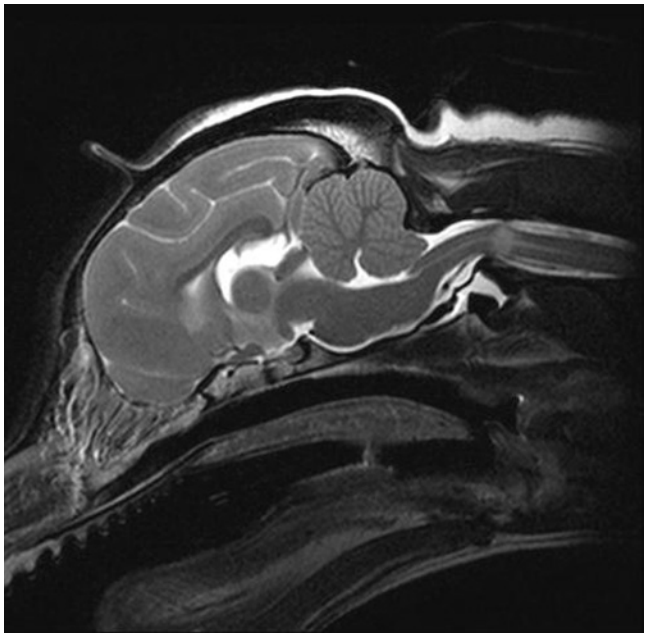


FIG. 40.48 Sagittal T2-weighted magnetic resonance image of a dog with atlantoaxial joint instability.

Laboratory Findings

As with other cervical myelopathies, results of CBC and biochemistry profile analysis are typically normal or indicative of prior administration of glucocorticoids. In very young patients, serum alkaline phosphatase levels may be elevated (bone isoenzyme).

DIFFERENTIAL DIAGNOSIS

The main differential diagnoses for dogs with CJAs are inflammatory/infectious disease (e.g., granulomatous meningoencephalomyelitis), traumatic luxation/fracture, and type I disc extrusion.

MEDICAL MANAGEMENT

AA instability is sometimes treated nonsurgically, often involving external splinting of the neck, with or without administration of antiinflammatory drugs (e.g., prednisolone). One retrospective study of AA dogs treated nonsurgically (all received external splinting) cited a good final outcome in 10 of 16 dogs (62.5%). Six of the dogs in this report (37.5%) died or were euthanized owing to neurologic deterioration or lack of improvement following splint removal.[29] In this study, the only factor significantly associated with outcome was length of clinical signs prior to therapy; dogs affected for less than 30 days were more likely to have positive outcomes than dogs affected for longer than 30 days. Reported cases of AOO or C1–C2 dorsal compression in the literature have been insufficient to allow general recommendations concerning therapy. Although most dogs with these malformations respond temporarily to medical management (similar to dogs with CLM), some dogs require surgical stabilization for relief of clinical signs.

SURGICAL TREATMENT

Surgical stabilization is the preferred treatment option for dogs that have clinical signs of neurologic dysfunction due to AA instability. Two main methods can be used to stabilize the AA joint, ventral and dorsal, with a number of variations available for each method. We do not recommend dorsal stabilization for AA instability as a primary mode of fixation; limited bone is available for implants, suture and wire fixation methods are prone to failure, and threading suture or wire beneath the atlas in a toy- or miniature-breed dog with a cranial cervical and/or caudal medullary constriction poses an unnecessary risk to the patient.

Preoperative Management

Preoperative management of the patient undergoing cervical spinal surgery is discussed on p. 1366.

Anesthesia

See p. 1366 for anesthetic management of the cervical spinal patient. In addition, see Table 32.1 for anesthetic considerations in the stable orthopedic patient.

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Surgical Anatomy

See p. 1366 for pertinent surgical anatomy of the cervical spine.

Positioning

Patient positioning for ventral and dorsal approaches to the cervical spine is discussed on pp. 1367 and 1370, respectively. In cases of suspected or known AA instability, it is extremely important to limit the patient's head and neck movement while under anesthesia and during patient positioning.

SURGICAL TECHNIQUE

The ventral approach to the C1–C2 space is described on p. 1344.

With a No. 11 or a No. 12 blade, incise the C1–C2 joint capsules bilaterally, exposing the articular surfaces. Incise the soft tissue attachment between C1 and C2, revealing the underlying dens (if present) and spinal cord. Sharply dissect redundant joint capsule tissue with a No. 11 blade and Bishop-Harmon forceps. Using a small towel clamp at the C2-C3 disc space or a speed-lock reduction forceps on the body of C2, lever the C1–C2 joint into reduction (Fig. 40.49). Typically, the cranial aspect of C2 is displaced in a cranioventral direction from the surgeon's perspective (ventral approach), craniodorsally from an anatomic perspective. *Apply mild but steady traction dorsally and caudally on C2 while putting slight downward pressure on the ventral arch of C1 to attain reduction. Distract the joint spaces a small amount and scarify the articular surfaces with a No. 12 blade. While maintaining reduction, place a pin or screw through the articular processes (facets) on one side.* In most toy and miniature dogs, the pin size will be 0.035 or 0.045 mm and the screw size will be 1.5 or 2.0 mm. *Insert the pin or screw in a medial-to-lateral direction, following the plane of the joint articulation, at an approximately 45-degree angle to the vertical plane (Fig. 40.50). Release the towel clamp or reduction forceps. Insert a pin or screw through the C1–C2 joint on the opposite side, using the same technique as the first side. Place additional pins or screws in the wings of C1 and the body of C2, taking care to direct these implants laterally, away from the spinal cord (Fig. 40.51).* Depending on surgeon preference, the screws/pins can be reinforced with small Kirschner wires (K-wires) and/or cerclage wire before PMMA is placed over the implants (Fig. 40.52).

Dorsal fixation methods for the AA joint typically involve suture or wire placed under the arch of C1 and attached to C2 (Fig. 40.53). We use dorsal stabilization in cases of AA instability as adjunctive fixation when a dorsal laminectomy of C1 or C2 is required, in addition to ventral stabilization. *In this scenario, place small (1.5–2.0 mm) screws through the dorsal lamina at the edge of the laminectomy defect. For safety reasons, use a specialized drill (e.g., Anspach, Midas Rex drill) with a 1.1-mm (for 1.5-mm screws) or a 1.5-mm (for 2.0-mm screws) drill-bit attachment for safety reasons.* These specialized drills can be held in one hand like a pencil and are much less unwieldy as a mini-driver. *Insert the screws (self-tap) 2 to 3 mm, and anchor them together with small K-wires and PMMA (Fig. 40.54).*

Both ventral and dorsal methods of stabilization of the atlanto-occipital region have been described in people for basilar invagination, and we have adapted these methods to several canine patients with favorable results (Fig. 40.55).

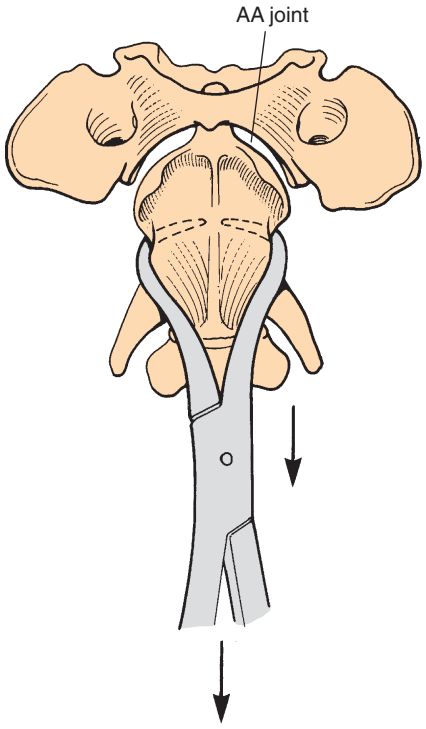


FIG. 40.49 Use orthopedic reduction forceps to grasp the body of C2. Use traction and countertraction to reduce the atlantoaxial subluxation. *AA,* Atlantoaxial.

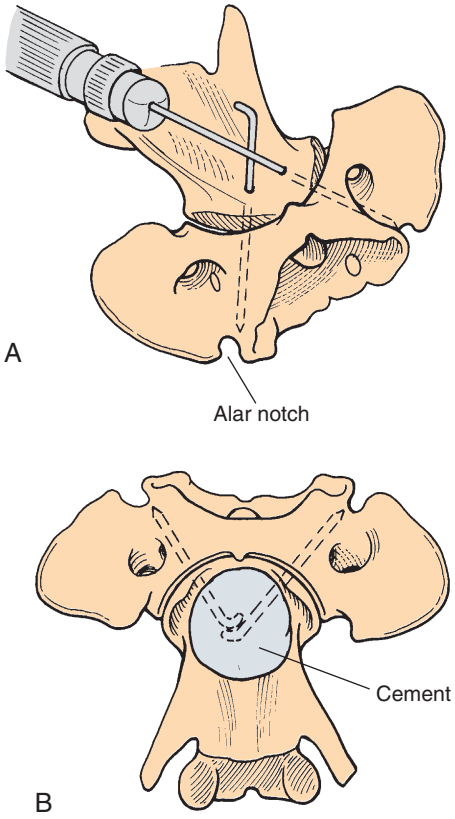


FIG. 40.50 (A) Place K-wires in cross-pin fashion; wires are directed toward the alar notch to ensure proper placement. (B) Notch or bend the exposed ends of the K-wires. Mold polymethylmethacrylate around both pin ends.

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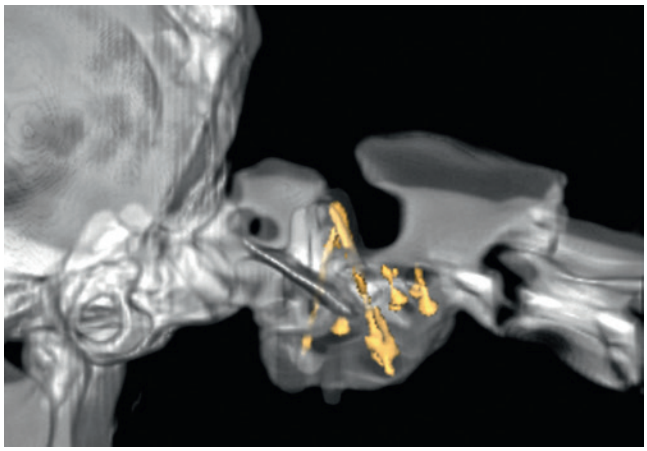


FIG. 40.51 Postoperative three-dimensional computed tomography reconstruction of a dog with atlantoaxial instability stabilized with multiple pins and screws and polymethylmethacrylate.

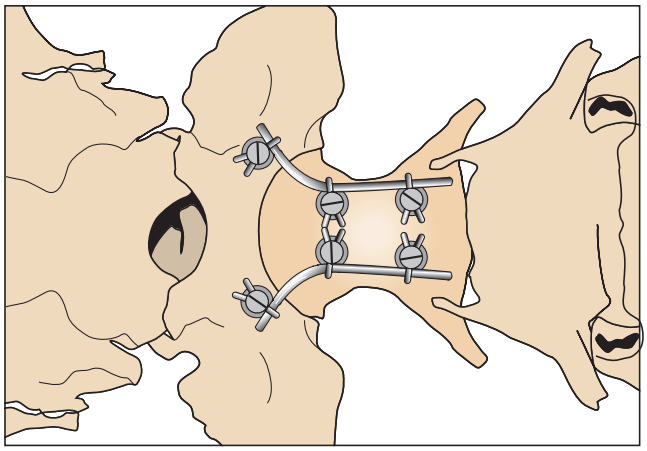


FIG. 40.52 Multiple cortical screws with Kirschner pins wired to the base of the screw heads can be used for atlantoaxial stabilization. Screws, wires, and pins are incorporated with polymethylmethacrylate.

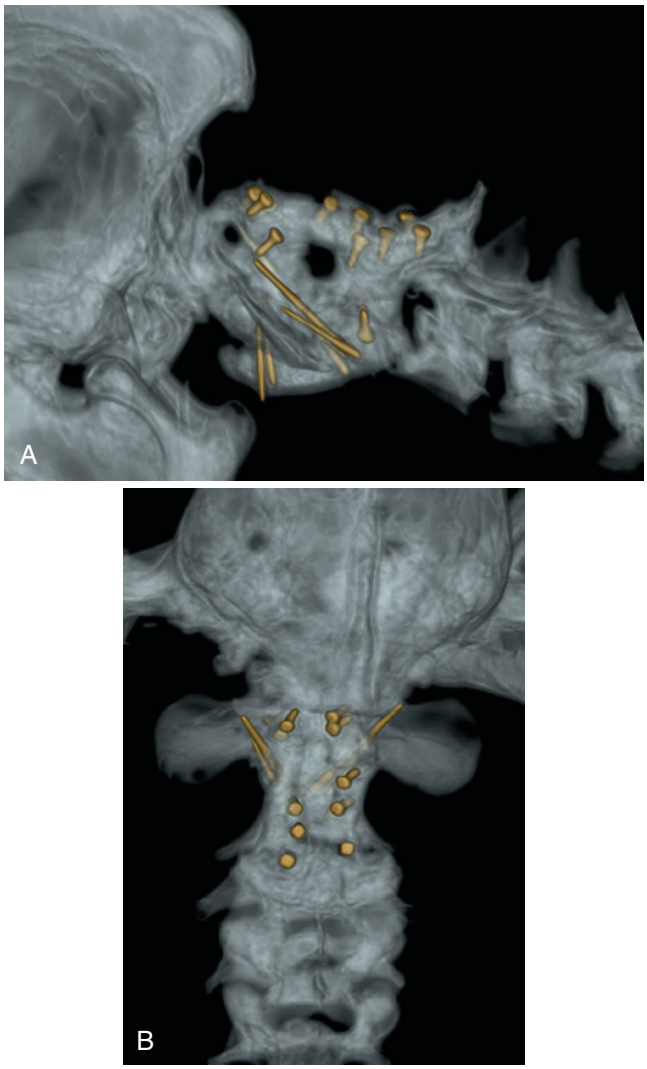


FIG. 40.54 (A) Lateral and (B) dorsal views of a three-dimensional computed tomography reconstruction from a dog with both ventral and dorsal fixation of atlantoaxial instability. A dorsal laminectomy at C1–C2 was also performed.

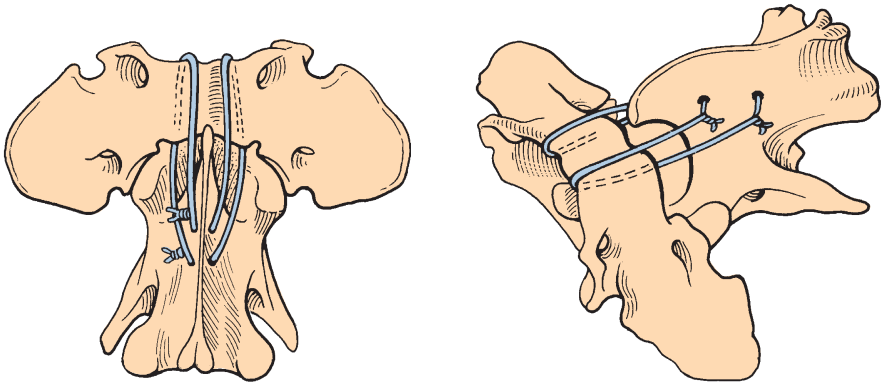


FIG. 40.53 For dorsal stabilization of atlantoaxial subluxation, pass wire or suture material under the dorsal arch of C1, then pass the wire or suture material through holes drilled in the dorsal spine of C2 and secure it.

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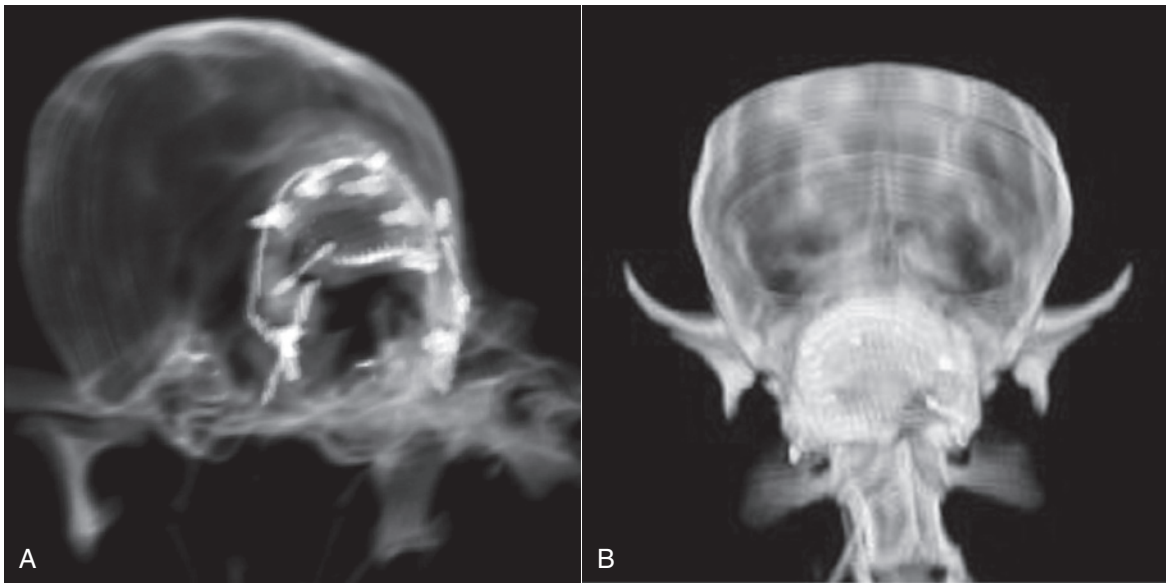


FIG. 40.55 (A) Caudolateral and (B) dorsal views of a three-dimensional computed tomography reconstruction from a dog with atlanto-occipital overlap decompressed and stabilized dorsally.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

In addition to standard instruments needed to perform a ventral or dorsal cervical approach (pp. 1345 and 1374), various surgical instruments may assist in performing surgery for CJAs. These include a specialized drill system (e.g., Anspach or Midas Rex), small (1.5–2.0 mm) screws (we typically use titanium screws in case reimaging with MRI is required in the future), small K-wires (titanium preferred), and PMMA.

COMPLICATIONS

Because of the proximity of the AA site to brainstem centers for cardiac and respiratory control, intraoperative deaths have been attributed to inadvertent damage to these medullary regions during AA stabilization. Postoperative complications involving upper respiratory function (e.g., coughing, gagging, laryngeal paralysis) occasionally occur with the ventral approach. Aspiration pneumonia is a potential postoperative complication that may be related to upper airway (i.e., laryngeal) or pharyngeal dysfunction. Anecdotally, we have found that relaxing the Gelpi retractors at frequent intervals (e.g., every 10 minutes) during surgery dramatically reduces the rate of postoperative upper airway complications. Overall, reported perioperative mortality rates for surgical management of dogs with AA instability vary from 0% to 30%, with most recent reports describing rates of approximately 5% to 10%. The complications to be expected with surgical management of AOO and C1–C2 dorsal compression are likely similar to those for AA instability; however, too few data are available regarding these surgical procedures for a complication rate to be calculated.

PROGNOSIS

Surgical success rates for AA instability vary in the literature from approximately 60% to over 90%; however, most recent reports regarding surgical treatment of this disorder describe a surgical success rate exceeding 80%. Similar to what has been reported for nonsurgical management of AA instability patients, length of clinical disease presence has been negatively associated with surgical success. Some investigators also suggest that the severity of neurologic dysfunction before surgery is inversely related to outcome; the prognosis for patients with AA instability is fair to good if mild to moderate neurologic deficits are present, and guarded if the deficits are severe (e.g., tetraplegia).

SPINAL ARACHNOID DIVERTICULUM

DEFINITION

Spinal arachnoid diverticula are typically solitary, dorsally or dorsolaterally located, focal accumulations of fluid that occur at the cranial cervical (most commonly over C2–C3 vertebral segments) or caudal thoracic regions of the spinal cord. In one large (122 dogs) retrospective study of spinal arachnoid diverticula, 65 were located in the cervical region and 60 in the thoracolumbar region.[30] Multiple or bilobed spinal arachnoid diverticula are frequently encountered, especially in the cervical region of Rottweilers. Ventrally located spinal arachnoid diverticula have also been reported.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The old name for this disorder (spinal arachnoid cyst) is a misnomer because the lesions are actually CSF-filled diverticula of the subarachnoid space rather than true cysts. These lesions have been described as meningeal cysts, leptomeningeal cysts, spinal arachnoid pseudocysts, and arachnoid cavitations. Proposed causes of these anomalous fluid accumulations are numerous, including congenital malformation, trauma, inflammation (arachnoiditis), and neoplasia. However, an underlying cause is often not found for spinal arachnoid diverticula. Accumulated fluid causes compression of adjacent spinal cord parenchyma, resulting in clinical signs of myelopathy.

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DIAGNOSIS

Clinical Presentation

Signalment

Spinal arachnoid cysts have been described in a considerable number of dogs and several cats. Rottweilers appear to be particularly predisposed to spinal arachnoid cysts in the cervical region. In this breed, dorsally located fluid accumulations in the cranial cervical regions are common. Multiple other dog breeds, including a number of small-breed dogs, have also been reported with this condition. In the large retrospective study mentioned earlier,[30] higher body weight was significantly associated with a cervical location of diverticula, there was a male predominance for the disorder, and pugs and French bulldogs were predisposed (in addition to Rottweilers). Pugs and French bulldogs were more likely to have a concurrent spinal disorder in the vicinity of the diverticulum, whereas only one of 17 Rottweilers had a concomitant spinal abnormality. Age at onset of clinical signs of neurologic dysfunction is quite variable, ranging from several months to 12 years of age. Most reported dogs developed signs of myelopathy in young adulthood. There is one report of seven young (3- to 4-month-old) pug dogs with C2-C3 diverticula and associated central canal dilation/syringomyelia of the cervical spinal cord.[31]

History

Dogs with cervical spinal arachnoid diverticula typically have historical complaints of slowly progressive ataxia and paresis of all four limbs. The reported young pug dogs initially showed signs of scuffing the dorsum of the front paws while ambulating, leading to skin abrasions; these dogs progressed to varying degrees of tetraparesis.[31]

Physical Examination Findings

Slowly progressive ataxia and tetraparesis are typical clinical features of this disorder. Dogs with dorsally or dorsolaterally located cranial cervical lesions (often Rottweilers) typically exhibit tetraparesis with hypermetria, primarily in the thoracic limbs (presumably via interference of the diverticulum with spinocerebellar tracts). Spinal hyperpathia does not appear to be a prominent clinical feature of this disorder but has been described.

Diagnostic Imaging

The diagnosis of spinal arachnoid diverticulum is based primarily on spinal imaging; myelographic diagnosis of this disorder is most commonly reported. Both CT and MRI (Fig. 40.56) have been successfully used to identify spinal arachnoid diverticula, and these modalities provide more detailed information regarding lesion lateralization and potential associated abnormalities (e.g., syringomyelia). The typical myelographic appearance is a bulbous, contrast-filled diverticulum continuous with the contrast column of the subarachnoid space, with a characteristic teardrop shape (Fig. 40.57).

Laboratory Findings

Results of CBC and biochemistry profile analysis are typically normal. Cerebrospinal analysis is usually within normal limits but may reveal a mild mononuclear pleocytosis with elevated protein concentration. Histopathology of resected "cyst" wall reveals meningeal tissue (dura-arachnoid).

DIFFERENTIAL DIAGNOSIS

The differential diagnoses for spinal arachnoid diverticula are diverse and include disc extrusion/protrusion, articular process (facet) hypertrophy, extradural synovial cyst, neoplasia, and infectious/inflammatory disease (e.g., discospondylitis). Ranking of these differential diagnoses in terms of likelihood is influenced heavily by patient age and breed.

MEDICAL MANAGEMENT

Medical management (e.g., glucocorticoid therapy) may be attempted but is unlikely to be successful in the long term.

SURGICAL TREATMENT

Surgical treatment involves resecting a portion of the meninges making up the "cyst" wall, thereby relieving the pressure exerted on underlying spinal cord parenchyma.

Preoperative Management

Preoperative management of the patient undergoing cervical spinal surgery is discussed on. p. 1366.

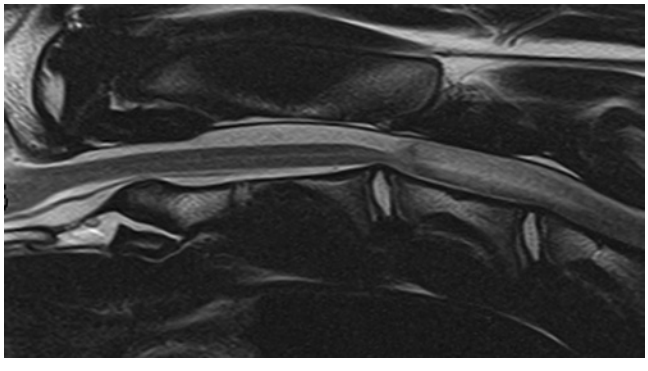


FIG. 40.56 Sagittal T2-weighted magnetic resonance images of a dog with a spinal arachnoid cyst.

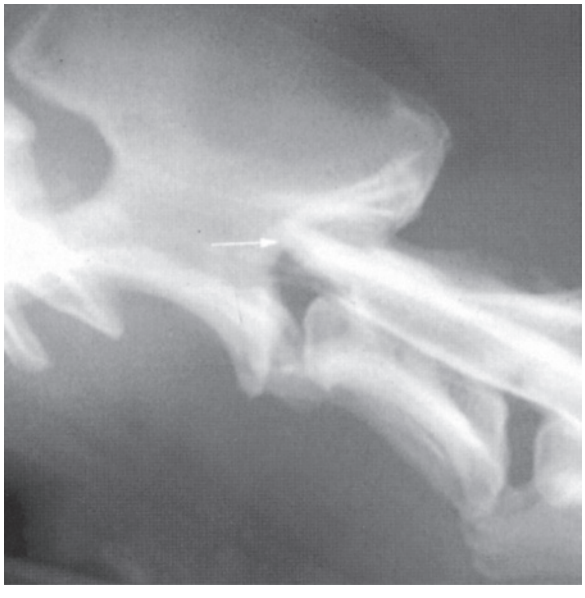


FIG. 40.57 Lateral myelographic image of a spinal arachnoid cyst *(arrow)* in the cervical region of a dog.

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Anesthesia

See p. 1366 for anesthetic management of the cervical spinal patient. In addition, see Table 32.1 for anesthetic considerations in the stable orthopedic patient.

Surgical Anatomy

See p. 1366 for pertinent surgical anatomy of the cervical spine.

Positioning

Patient positioning for the dorsal approach to the cervical spine is discussed on p. 1370.

SURGICAL TECHNIQUE

Cervical spinal arachnoid diverticula may be accessed via a dorsal laminectomy (p. 1373) or a dorsolateral hemilaminectomy (p. 1379). Decompression of the spinal arachnoid diverticulum is basically a durotomy over the abnormal region, usually combined with marsupialization of the "cyst" wall. *Bend a 25-gauge needle at approximately 120 degrees, with the bevel pointing upward. Grasp the hub of the needle with a Mosquito hemostat. Use the tip and bevel of the needle to "tent up" and perforate the dura-arachnoid at the level of the diverticulum (Fig. 40.58).* CSF should flow out of the perforation. *Place the blunt, angled tip of a nerve root retractor (e.g., Gross ear hook and spoon) into the perforation site. While gently pulling up on the dura-arachnoid, advance the retractor tip linearly along the diverticulum site, incising the meninges over the metal tip during advancement using the tip of a No. 11 blade (Fig. 40.59). Once the dura-arachnoid is incised over the diverticulum, sequentially grasp the edges of the cranial and caudal aspects of the incised meninges at either end of the meningeal defect, and incise (using a No. 11 blade or fine tenotomy scissors) the meninges perpendicular to the spinal cord, creating a dorsal and ventral flap that can be reflected dorsally and ventrally, respectively. Resect a portion of one of the meningeal flaps for biopsy. Perform marsupialization by attaching the edges of the meningeal defect to surrounding epaxial musculature using fine suture material (5-0 or 6-0 polydioxanone or polypropylene), Bishop-Harmon forceps, and Castroviejo needle holders, in an interrupted pattern.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

In addition to standard instrumentation used for dorsal cervical procedures, 5-0 or 6-0 polydioxanone or polypropylene, Castroviejo needle holders, and tenotomy scissors are useful for surgical treatment of spinal arachnoid diverticulum.

COMPLICATIONS

Complications associated with cervical spinal surgery are discussed on p. 1375.

PROGNOSIS

From the limited data available, surgical treatment of spinal arachnoid cysts in dogs and cats appears to have a good prognosis. Some evidence suggests that marsupialization of the diverticulum wall to surrounding tissues at surgery may help prevent recurrence.

SPINAL NEOPLASIA

DEFINITIONS

Spinal neoplasia refers to primary and secondary tumors of the vertebrae or spinal cord parenchyma. **Primary tumors** include those neoplasms that arise from spinal cord parenchyma (e.g., neurons, glial cells) or associated meningeal/ependymal tissue. **Secondary tumors** include tumors that arise from structures extrinsic to the spinal cord and its meningeal covering. Such tumors include primary or metastatic vertebral neoplasms, malignant nerve sheath tumors (MNSTs), and metastases to the extradural space or the cord parenchyma (intramedullary metastases).

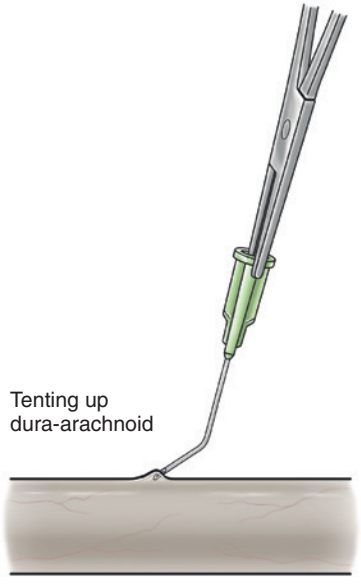


FIG. 40.58 Needle method for tenting-up the dura-arachnoid when performing a durotomy.

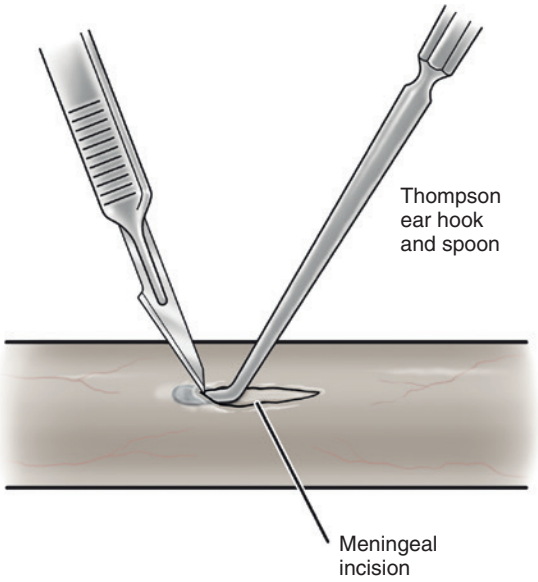


FIG. 40.59 Incision of the meninges over the tip of a probe, while the probe is advanced along the length of the diverticulum.

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GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Numerous tumors can affect the spinal cord of dogs and cats. As with brain tumors, tumors affecting the spinal cord can be conceptually divided into primary and secondary tumors. As with brain tumors, primary tumors are more common than metastatic tumors. It is often clinically useful to classify spinal cord neoplasms based on the relationship between the tumor and the meninges. Spinal cord tumors are typically classified as extradural, intradural/extramedullary, or intramedullary. Because some spinal tumors occupy more than one of these locations (e.g., may be both extradural and intradural/extramedullary), the additional location category of mixed compartment has been suggested. Spinal cord tumors exert their pathologic effects by causing compression or invasion of the spinal cord and by producing peritumoral edema, inflammation, and hemorrhage.

Extradural tumors represent the most frequently diagnosed category of spinal neoplasia. Extradural tumors include primary and secondary (metastatic or local invasion) vertebral and soft tissue tumors. Primary vertebral tumors such as osteosarcoma, chondrosarcoma, myeloma (plasma cell tumor), fibrosarcoma, and hemangiosarcoma are common extradural tumors encountered in dogs. Vertebral tumors reported in cats include osteosarcoma, fibrosarcoma, undifferentiated sarcoma, and plasma cell tumor. The most common primary vertebral body tumor in dogs and cats is osteosarcoma. Carcinomas account for the majority of secondary vertebral tumors in dogs. It may be difficult in some cases to ascertain whether a vertebral tumor is primary or metastatic. Other tumors may occur in the epidural space, without directly involving the vertebrae. Common among these are sarcomas, most frequently osteosarcomas and hemangiosarcomas. Lymphosarcoma can be primary or metastatic and is often located in the extradural space, particularly in cats. Lymphosarcoma is the most common spinal tumor of cats. Meningiomas and MNSTs are usually typically located intradurally but occasionally will exhibit an extradural pattern on myelography or other imaging modalities (CT/MRI). Metastatic carcinomas (e.g., mammary carcinoma, prostatic carcinoma) may localize to the extradural space. A number of fatty tumors have been reported to affect the spinal cord in dogs, including lipoma, myelolipoma, infiltrative lipoma, and liposarcoma. These all generally occur in an extradural location. Meningiomas and MNSTs are the two most common intradural/extramedullary neoplasms, with meningiomas predominating. Intramedullary tumors are infrequently encountered neoplasms that include primary spinal parenchymal tumors (e.g., astrocytoma, oligodendroglioma, ependymoma) and intramedullary metastases. The most common intramedullary metastases in dogs are thought to be hemangiosarcoma and lymphosarcoma. Mixed compartment tumors tend to occur in more than one of the three typical compartments listed previously. MNST is the most common type of neoplasm in this category, followed by lymphosarcoma and malignant fibrous histiocytoma.

DIAGNOSIS

Clinical Presentation

Signalment

In general, most patients with spinal neoplasia are older (e.g., >5 years), but some tumors (e.g., lymphosarcoma) are seen commonly in young animals. Spinal tumors appear to be much more common in larger dogs than in small-breed dogs. The median age of cats with spinal lymphosarcoma is 2 to 3 years.

History

Spinal tumors classically cause progressive signs of a myelopathy, but acute or subacute development of spinal cord dysfunction often occurs, especially with feline lymphosarcomas and intramedullary neoplasms. Rapid onset of clinical signs may be due to such factors as pathologic fracture of a cancerous vertebra, acute hemorrhage or necrosis of a tumor, or rapid growth of a neoplasm with subsequent damage to spinal cord parenchyma (more likely with intramedullary tumors). Spinal cord tumors are typically solitary and can occur anywhere along the length of the spine. Meningiomas and MNSTs arise most frequently in the cervical spinal cord, with MNSTs being especially prominent in the cervical intumescence area.

Physical Examination Findings

A prominent feature of extradural and intradural/extramedullary spinal neoplasia is spinal hyperesthesia, which often precedes the onset of proprioceptive and voluntary motor deficits. Spinal hyperesthesia is often not a prominent early clinical feature in patients with intramedullary spinal tumors, probably owing to lack of meningeal involvement. In MNSTs of the cervical intumescence, a history of unilateral thoracic limb lameness (on the side of the tumor) preceding the development of clinical signs of myelopathy is common.

Diagnostic Imaging

A tentative diagnosis of spinal neoplasia is typically based on signalment, history, clinical signs, and results of spinal imaging. In cases of vertebral neoplasia, bony lysis with loss of cortical outlines is often seen on imaging of affected vertebrae, with or without evidence of bony proliferation (Fig. 40.60). In most soft tissue spinal neoplasms, plain radiographs of the spine are normal. Myelography, CT, and MRI are usually helpful in diagnosis of spinal tumors and in therapeutic planning. An intramedullary myelographic pattern may be misleading in that an intradural/extramedullary mass with attendant cord swelling may cause an identical pattern on myelography or advanced imaging (e.g.,

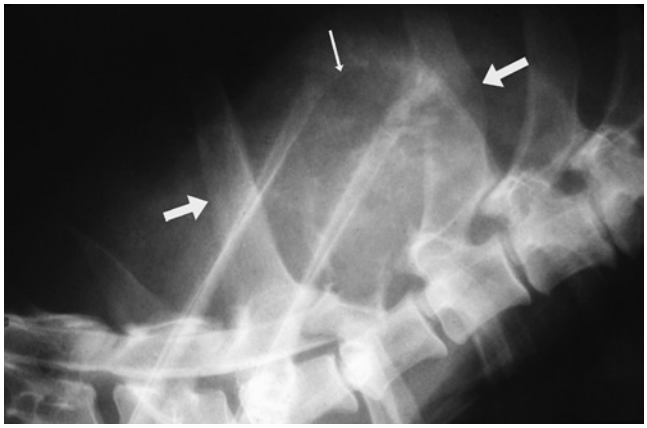


FIG. 40.60 Lateral myelogram of the cervical spine of a dog with a tumor of the T1 spinous process and dorsal lamina. Note the spinous processes of C7 and T2 *(large arrows)* and the absence of the spinous process of T1 *(small arrow)*. Profound spinal cord compression is illustrated by the inability of contrast to move distal to the level of T1.

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CT, MRI). Intradural/extramedullary spinal tumors will occasionally infiltrate the spinal cord parenchyma (mixed compartment mass), and this may contribute to the development of an intramedullary imaging pattern. It is important to realize that both meningiomas and MNSTs tend to be associated with an intradural extramedullary pattern. In addition, both tumors appear to have a predilection for the cervical spinal cord. In some cases of MNST, an enlarged intervertebral foramen evident on radiographs, CT, or MRI, or an enlarged nerve root identifiable with CT or MRI may help in distinguishing a tumor as an MNST rather than a meningioma. Absence of such distinguishing imaging results, however, does not rule out the possibility of MNST. Definitive diagnosis of spinal tumors in all cases requires histopathologic evaluation of affected tissue. This is not usually feasible without surgical intervention. However, fluoroscopic- or CT-guided needle biopsy may provide a diagnosis in some cases.

Laboratory Findings

Results of laboratory analyses are usually normal, but hyperglobulinemia and proteinuria may be evident in cases of myeloma. Most cats with spinal lymphosarcoma are feline leukemia virus positive, have leukemic bone marrow, and have multicentric neoplasia. With the possible exception of spinal lymphosarcoma, CSF evaluation rarely reveals neoplastic cells but may reveal increased protein levels, with or without elevated cell counts (more likely with tumors with meningeal involvement).

DIFFERENTIAL DIAGNOSIS

Other disorders that cause progressive cervical myelopathy include type II disc protrusion, syringomyelia, and CCSM. Because some cervical spinal cord tumors may have more rapid onset and progression of clinical signs, type I disc extrusions, CJAs, and infectious/inflammatory disorders need to be on the differential diagnosis list.

MEDICAL MANAGEMENT

As with brain tumors (see Chapter 39), therapy for dogs and cats with spinal tumors can be divided into supportive and definitive treatments. Supportive therapies are directed against secondary sequelae of the spinal tumor (e.g., cord edema, pain), whereas definitive therapies are aimed at elimination of neoplastic tissue. Supportive therapy consists of antiinflammatory doses of glucocorticoids (e.g., prednisolone, 0.5 mg/kg PO, every 12 hours), which can be increased or decreased as needed, with or without additional pain-relieving drugs (e.g., narcotics). Definitive medical therapy for spinal tumors, similar to brain tumor medical definitive therapy, consists primarily of megavoltage radiation therapy and oral chemotherapy. Chemotherapy is indicated for lymphosarcoma, myeloma, meningioma, and glioma.

SURGICAL TREATMENT

Preoperative Management

Preoperative management of the patient undergoing cervical spinal surgery is discussed on p. 1366.

Anesthesia

See p. 1366 for anesthetic management of the cervical spinal patient. In addition, see Table 32.1 for anesthetic considerations in the stable orthopedic patient.

Surgical Anatomy

See p. 1366 for pertinent surgical anatomy of the cervical spine.

Positioning

Patient positioning for the dorsal approach to the cervical spine is discussed on p. 1370. Most cervical spinal tumor removals will be performed from a dorsal approach. On occasion, partial or complete vertebral body removal with stabilization may be attempted for vertebral masses. This technique requires a ventral approach (see p. 1367).

SURGICAL TECHNIQUE

Ventral and dorsal approaches to the cervical spine are described on pp. 1367 and 1370, respectively. Tumors amenable to surgical removal are typically extradural or intradural/extramedullary in location, without invasion into vertebral bodies. Intradural tumors require a durotomy to be performed (see p. 1396). Some intramedullary tumors may have distinct borders and can be removed via myelotomy (incision through a thin layer of spinal cord parenchyma overlying the tumor).

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

In addition to standard instrumentation for spinal surgery, instruments such as lens loops, Bishop-Harmon forceps, and tenotomy scissors are often helpful in spinal tumor removal. A Cavitron ultrasonic aspirator may also be useful for removal of soft tissue spinal cord masses. In situations requiring extensive removal of surrounding bone (e.g., articular processes, vertebral body) with potential creation of instability, the surgeon should have access to necessary materials for stabilization procedures (e.g., pins, screws, locking plates, PMMA).

COMPLICATIONS

Complications associated with cervical spinal surgery are discussed on p. 1375. In addition to these complications, implant failure and infection are potential complications of stabilization procedures.

PROGNOSIS

The prognosis for dogs and cats with spinal neoplasia treated with supportive therapy alone is poor. Although data are lacking, these patients will likely be euthanized because of progressive spinal cord dysfunction within several weeks to several months of diagnosis, depending on the tumor type. For most canine and feline spinal tumors, meaningful prognostic information based on large numbers of cases in which definitive therapy was pursued is lacking. With the exception of lymphosarcoma, absence of prognostic information pertaining to feline spinal neoplasia is notable. In one report, prognostic data concerning nonlymphoid spinal neoplasia in 11 cats were described. Several cats experienced prolonged remissions following surgical intervention, including one cat with osteosarcoma, one with chondrosarcoma, one with an MNST, and one with meningioma, with survival times of approximately 57 months, 12 months, 73 months, and 47 months, respectively. The median survival time for four additional cats with meningioma was approximately 6 months after surgery.[32] In another report of 26 cats with nonlymphoid spinal tumors

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treated surgically, the median survival time for benign tumors (e.g., meningioma) was 518 days, and for malignant tumors (e.g., osteosarcoma) the median survival time was 110.5 days.[33] Although based on a small number of cases, nonlymphoid spinal neoplasia may often be associated with a favorable prognosis in cats treated surgically. Evidence suggests that osteosarcomas may be less aggressive in cats than in dogs. The prognosis for most cases of vertebral neoplasia in dogs (e.g., osteosarcoma, chondrosarcoma) is considered poor. Surgical decompression may provide some temporary relief of clinical signs. However, vertebral neoplasms often cause such extensive bony destruction by the time of diagnosis that any further destabilization due to surgical intervention (e.g., laminectomy) may hasten the development of a pathologic fracture/luxation. Radiation therapy and chemotherapy are usually unsuccessful in the treatment of these tumors. In one report of dogs with vertebral tumors treated via various methods (e.g., surgery, radiation therapy, chemotherapy), the median survival time was 135 days. Sustained remissions (>1 year) are likely in patients with plasma cell tumors (myeloma) of the vertebrae that are treated with chemotherapy.[34] Although chemotherapy or radiation therapy is the recommended definitive therapy for spinal lymphoma, most feline and canine patients will be euthanized within 3 months because of progressive or recurrent disease, despite therapy. Limited available information on spinal meningiomas suggests that remission times exceeding 6 months are likely with surgical therapy alone; adjunctive radiation therapy is likely to extend remission times for this tumor to 12 months or longer.

The prognosis for surgical removal/debulking of canine spinal MNSTs appears to be poor. Median postoperative survival is approximately 5 to 6 months, with a disease-free interval of only approximately 1 month. The efficacy/inefficacy of postoperative radiation therapy for spinal MNSTs has not yet been established for dogs or cats. Intramedullary spinal neoplasia is uncommon. The prognosis for dogs and cats with these tumors is considered poor, although definitive therapy has not been thoroughly described. Several reports have described surgical removal of lipomatous spinal cord masses in dogs. Although the overall success rate of surgery appears to be favorable, even with infiltrative lipomas and liposarcomas, postoperative radiation therapy may be advisable with these latter two tumor types to achieve good long-term results.

SPINAL TRAUMA

DEFINITIONS

Similar to brain injury, spinal trauma is conceptually divided into primary injury and secondary injury. **Primary injury** refers to that which occurs immediately after impact, whereas **secondary injury** describes the ensuing biochemical processes that potentiate further spinal cord damage. Disruption of supportive structures of the spinal column can occur as a true **fracture** if the vertebrae are damaged, as **luxation** (displacement from normal spinal alignment) without fracture if the damage occurs to soft tissues alone, or as a combination of fracture and luxation. **Concussive** spinal cord injury describes injury to the spinal parenchyma sustained from blunt force trauma that does not necessarily require fracture/luxation of supporting bony and ligamentous structures.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Fracture/luxation of the cervical spine in dogs and cats is most commonly the result of vehicular trauma. Other causes include projectile injuries (e.g., gunshot), bite wounds (e.g., "big dog–little dog" altercations), and running head-first into a stationary object. Pathologic fracture/luxation may occur with minor trauma in some cases owing to weakened bone (e.g., vertebral neoplasia) or abnormal anatomy (e.g., AA instability). Fracture luxation of the cervical spine occurs less commonly than in the thoracolumbar spine, and the C2 vertebra and the C1–C2 articulation represent the most common areas for fracture/luxation. This is thought to be due to the concentration of stresses at the level of the C2 vertebra caused by cranial and caudal anatomic attachments (Fig. 40.61). Following the initial traumatic event, further impact injury to the spinal cord can occur if unstable segments continue to move and repetitively traumatize the spinal cord parenchyma in the damaged area of the spine.

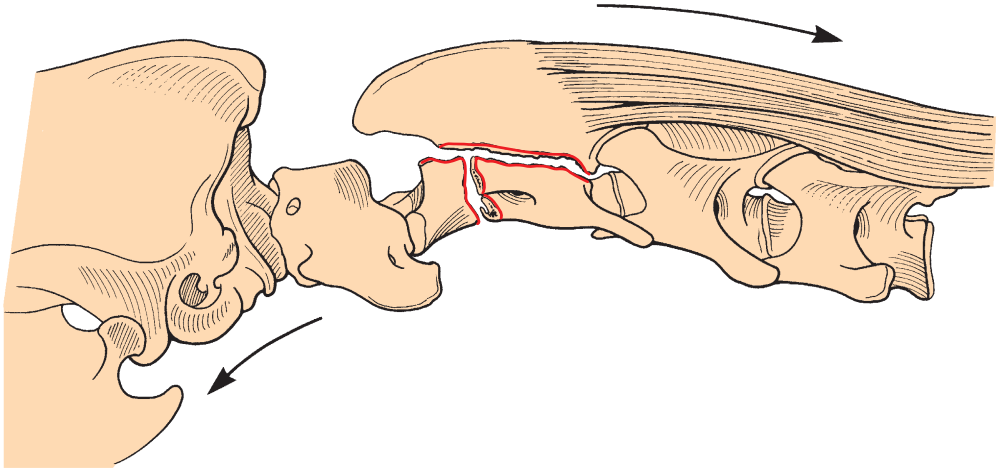


FIG. 40.61 A static-kinetic relationship is produced between the cervicocranium and the lower cervical spine; the axis is the point of stress concentration resulting in fracture/luxation.

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DIAGNOSIS

Clinical Presentation

Signalment

Although most spinal cord injuries occur in younger animals (<5 years old), no age, breed, or sex predilection for spinal trauma has been noted.

History

The history of trauma is often definitive, in that the incident (e.g., hit by car) was actually witnessed. In some cases, spinal trauma is inferred when an animal is found with neurologic deficits consistent with cervical myelopathy and other external evidence of trauma (e.g., abrasions, bite wounds). Most patients will present to the hospital soon after trauma with a brief history of neck pain and variable levels of proprioceptive and motor deficits in all four limbs.

Physical Examination Findings

As with brain injury patients, dogs and cats that have experienced traumatic spinal injury often present to the hospital in a state of shock. The basic tenets of shock management need to be followed before an in-depth assessment of neurologic status is performed. However, when the clinician suspects cervical spinal trauma, it is imperative to keep the patient as immobile as possible until obvious instability is ruled out or corrected. It is important to perform as thorough a neurologic examination as possible (see Chapter 38), but certain aspects of the examination (e.g., hopping, wheelbarrowing) should be left out until more is known about the stability of the cervical spinal column. Gentle palpation of the cervical spine may help localize the region of injury.

Diagnostic Imaging

Several methods of imaging the spinal column of the traumatized patient are known; radiographs are the most commonly performed on an emergency basis. It is important to obtain imaging of the entire spinal column because multiple fractures or luxations may be present. Radiographs may be taken in the awake patient, but subtle lesions may be difficult to evaluate in some animals if they are moving excessively. Sedation or anesthesia allows more detailed imaging but removes the patient's ability to protect the fracture/luxation site by contracting surrounding musculature. If possible, we prefer to obtain lateral radiographs of the entire spine in the conscious patient (after addressing any systemic concerns associated with trauma) to screen for overt fractures/luxations that are present. Following this, further radiographic views or CT images of the spine may be obtained. We prefer CT of the entire spine in these patients for several reasons. With current CT machines, scans are extremely rapid and the entire spine of a large dog can be completed under sedation within minutes. Patient repositioning is not required, and images can be reconstructed in multiple planes and three-dimensionally (Fig. 40.62). CT is more reliable than radiographs in identifying both the number and the extent of fractures/luxations. In addition, measurements for implants can be easily and accurately made from CT images. In the anesthetized patient, myelography can be performed as an adjunct to radiographs or in conjunction with CT (CT/myelogram). Use of contrast with radiography or CT in cases of spinal trauma is a matter of clinician preference. In our experience, contrast in such cases rarely provides much useful clinical information. MRI can also be used to assess cervical spinal cord trauma; it is our opinion that, although MRI may provide superior soft tissue detail in cervical spinal cord trauma, CT is generally preferable to MRI for assessing spinal injury in such cases. Classification of a cervical fracture/luxation as stable or unstable can be obvious in some cases but is more subjective in others. The three-compartment model (Fig. 40.63) is often used to assess the likelihood of instability, with disruption of two or three of the compartments constituting evidence of instability. This model is a potentially useful guideline but should not be viewed as an absolute determinant. Other factors, such as patient demeanor (i.e., a very active and hyperexcitable patient may not do well with conservative fracture management), location of fracture lines in reference to tension and compression forces, and spinal cord impingement by bone fragments or disc material, need to be considered when decisions concerning medical versus surgical management are made.

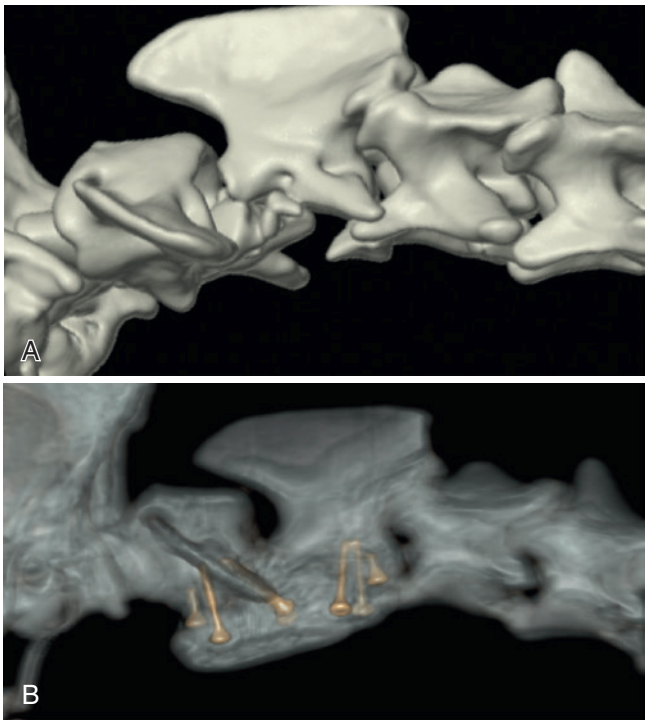


FIG. 40.62 Three-dimensional reconstructed computed tomography image of a dog with a C2 vertebral body fracture (A) before and (B) after surgical reduction and fixation with cortical screws and polymethylmethacrylate.

Laboratory Findings

Results of laboratory analyses (e.g., CBC, biochemical profile) are typically unremarkable in patients with cervical spinal trauma, or are reflective of the experience of a traumatic event (e.g., stress leukogram).

DIFFERENTIAL DIAGNOSIS

In cases of cervical spinal trauma in which the traumatic event was witnessed or clear evidence of associated trauma is found, no other differential diagnoses need to be considered. In situations that involve relatively minor trauma, it is important to consider the possibility of a pathologic fracture. Animals that are found by their owners with sudden onset of cervical myelopathy with no known traumatic event may have experienced ischemic insult to the spine (see p. 1448). In these cases, the absence of overt spinal hyperesthesia is a common historical and clinical feature.

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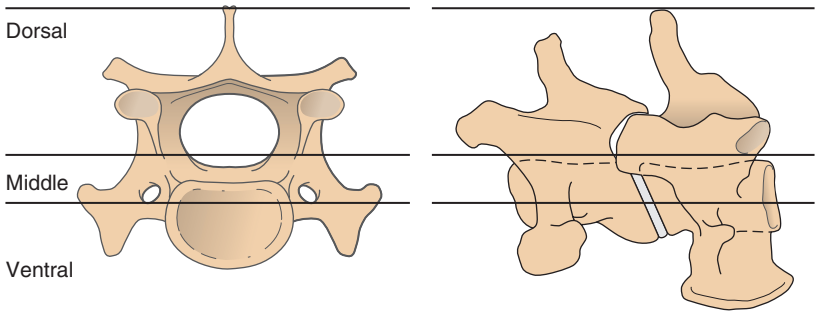


FIG. 40.63 Three-compartment model for evaluating the stability of a vertebral fracture/luxation.

TABLE 40.2 Anatomic Variations of the Atlas and Axis Important to Surgical Repair of Atlantoaxial Instability

Vertebrae	Anatomy	Surgical Significance
C–atlas	Thin dorsal arch (laminae); no dorsal spinous process No vertebral body, just thin ventral fovea Ventrolateral diarthrodial joints Prominent ventral tubercle Prominent wings	Poor implant-holding power dorsally Poor implant-holding power ventrally Good purchase for implants ventrally; used to evaluate anatomic reduction Landmark for surgical localization Moderate implant-holding power dorsally and ventrally
C2-axis	Prominent dorsal spinous process Thin central vertebral body Prominent cranial articular processes Prominent caudal vertebral body Dens	Moderate implant-holding power dorsally, patient age and size dependent; landmark for surgical localization Poor implant-holding power ventrally Good purchase for implants ventrally; used to evaluate anatomic reduction Good ventral purchase for implants May fracture or displace into vertebral canal
C–C2	Bilateral ventrolateral diarthrodial joints Dorsal atlantoaxial ligament	Good purchase for implants ventrally; used to evaluate anatomic reduction Moderate purchase of implants used to replace the ligament

MEDICAL MANAGEMENT

Providing treatment for shock and immobilizing the spine are the initial goals of medical therapy. In addition, analgesics for pain should be administered as needed. In patients that are deemed viable candidates for nonsurgical management of cervical spinal trauma, strict cage confinement with or without a neck brace is typically indicated. Although many factors go into the decision to medically manage a cervical spine trauma case, this mode of management is usually chosen for ambulatory patients with minimal displacement of vertebrae or fracture segments. It is also chosen for patients with no evidence of fracture/luxation or spinal cord compression, even if nonambulatory (e.g., suspected concussive cord injury). Neurologic deterioration during medical therapy is an indication to pursue surgical stabilization, with or without decompression.

Specific medical therapeutic interventions to combat secondary spinal injury processes are lacking. Glucocorticoid therapy has not been shown to improve outcome in clinical spinal cord injury, and excessive use of glucocorticoids in such cases can have major detrimental effects (e.g., gastrointestinal ulceration/perforation). Evidence has shown that intravenous PEG therapy is not beneficial for victims of spinal cord injury (see p. 1366).[1]

SURGICAL TREATMENT

Surgical treatment is provided with two objectives: stabilization and decompression. The most pressing goal is to provide stabilization, and decompression should generally not be performed in spinal fracture/luxation scenarios without some level of stabilization being provided. Decompression is performed dorsally (for spinal cord parenchyma) or dorsolaterally (nerve root impingement), and stabilization is most commonly performed ventrally. Dorsal stabilization can be performed in the cervical region, but the bone available for implants is sparse. Cranial cervical vertebral bodies have substantially less bone available for ventral implants than caudal vertebrae. Anatomic considerations and their impact on implant selection are summarized in Table 40.2. In our experience, presurgical CT measurements are invaluable in selection of implants for individual patients.

Preoperative Management

Preoperative management of the patient undergoing cervical spinal surgery is discussed on p. 1366. For trauma patients, blood should be available for transfusion. Hemorrhage from lacerated venous sinuses can occur at the time of trauma or during fracture/luxation reduction.

Anesthesia

See p. 1366 for anesthetic management of the cervical spinal patient. In addition, see Table 32.2 for anesthetic considerations for the acute trauma patient.

Surgical Anatomy

See p. 1366 for pertinent surgical anatomy of the cervical spine.

Positioning

Patient positioning for the ventral approach to the cervical spine is provided on p. 1367. Positioning for the dorsal approach to the cervical spine is described on p. 1370.

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SURGICAL TECHNIQUE

The surgical technique or techniques used will depend on several factors, including the location of the injury, the type and shape of the injury, whether or not decompression is needed, and the experience/preference of the surgeon. Dorsal and ventral approaches to the cervical spine are described on pp. 1370 and 1367, respectively. Types of implants are described in other sections of this chapter (CJA, CCSM) and include pins/screws and PMMA (Fig. 40.64), spinal plates (Fig. 40.65), and articular process (facet) screws (Fig. 40.66). In some cases, ventral stabilization is required in addition to dorsal or dorsolateral decompression. In these situations, ventral stabilization is performed, followed by dorsal or dorsolateral decompression. Dorsal stabilization can be added as well but should be considered as adjunctive rather than primary stabilization. From the ventral approach, fracture/reduction stabilization can often be facilitated by the use of ASIF small fragment reduction forceps.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

In addition to standard instrumentation required for cervical spine surgery, cancellous and cortical bone screws, spinal plates, pins (positive-profile thread), PMMA, and ASIF small fragment reduction forceps should be available for cervical spinal stabilization.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative evaluation and management of cervical spinal trauma patients are very similar to those for dogs and cats with other spinal disorders. Particular attention should be paid to managing pain and limiting excessive movement. Although stabilization procedures should leave the fracture/luxation "stable at the table," all fixations are subjected to repetitive mechanical forces that may threaten implant integrity in the early stages of healing.

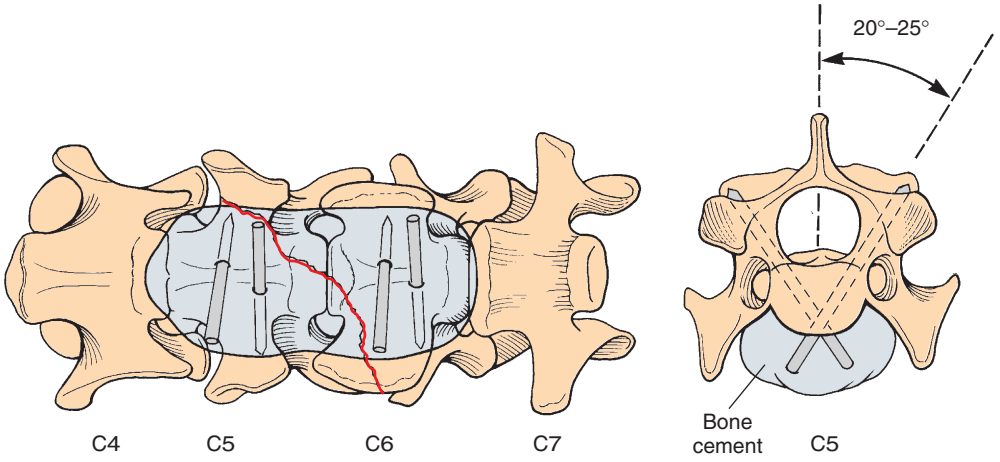


FIG. 40.64 Ventral stabilization of C3–C7 fracture/luxation using Steinmann pins and polymethylmethacrylate in the ventral bodies. Note that the pins are angled away from the vertebral canal.

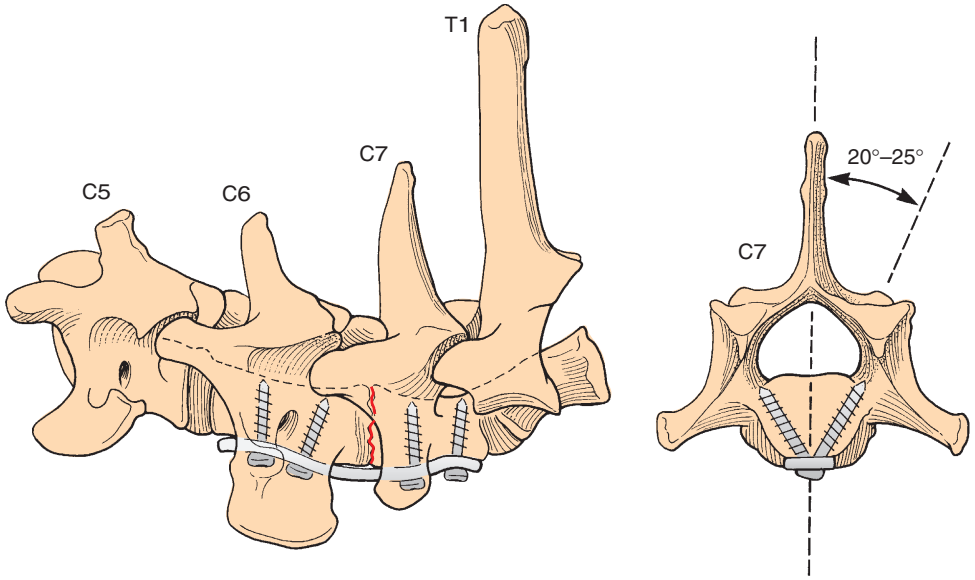


FIG. 40.65 Ventral stabilization of C3–C7 fracture/luxation using plastic spinal plates on the ventral vertebral bodies. Note that the screws are angled away from the vertebral canal.

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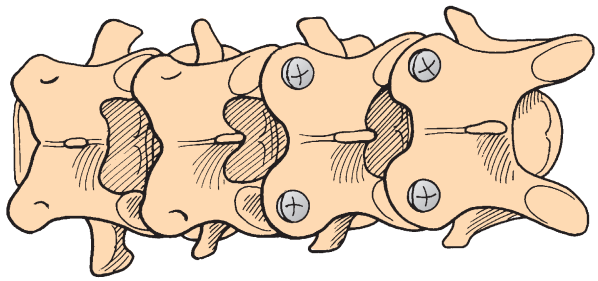


FIG. 40.66 Dorsal stabilization of C6–C7 fracture/luxation using articular facet screws.

PROGNOSIS

In general, the prognosis for most medically and surgically treated cervical spinal trauma patients is favorable. For surgically managed patients, prognosis is highly dependent on appropriate preoperative and postoperative care, in addition to the specific mode of surgical treatment applied.

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41

Surgery of the Thoracolumbar Spine



GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

Weakness of both pelvic limbs due to a thoracolumbar myelopathy is **paraparesis.** The term used to describe loss of voluntary motor ability to both pelvic limbs is **paraplegia.** The most commonly performed surgical approaches to the thoracolumbar spine are **hemilaminectomy** and **dorsal laminectomy.** Hemilaminectomy is unilateral removal of the lamina, articular processes (facets), and part of the pedicle. Dorsal laminectomy is removal of the dorsal lamina bilaterally, including removal of the dorsal spinous process. Portions or all of the articular processes can be removed during a dorsal laminectomy, depending on the desired exposure and risks of causing instability. Hemilaminectomy and dorsal laminectomy can be combined to create a **dorsolateral hemilaminectomy.** A scaled down version of hemilaminectomy that involves removal of portions of the pedicle cranial and caudal to the intervertebral foramen with preservation of the articular processes is called **pediculectomy;** this procedure has limited clinical application and will not be discussed in detail. **Partial lateral corpectomy** is removal of part of the vertebral body ventral to the vertebral canal; the main indication for this procedure is removal of chronic type II disc protrusions.

GENERAL CONSIDERATIONS

As with cervical myelopathies, a broad range of clinical severity is associated with thoracolumbar myelopathies in dogs and cats. In contrast to cervical myelopathies, inability to walk on the pelvic limbs (nonambulatory paraparesis, paraplegia) is the most common clinical scenario in thoracolumbar myelopathies, rather than spinal hyperpathia with minimal to no neurologic deficits. It is much more common for dogs and cats to experience upper motor neuron (UMN) bladder dysfunction with thoracolumbar myelopathies than with cervical myelopathies. In general, most patients that retain or regain (postsurgically) voluntary motor ability to the pelvic limbs will have voluntary control of micturition. Bladder management is a vital component of patient care in treatment for thoracolumbar myelopathies.

Preoperative Management

Preoperative management of the patient with thoracolumbar myelopathy is similar to that described for the patient with cervical myelopathy (see Chapter 40). If instability is known or suspected (e.g., spinal trauma), the patient should be immobilized on a rigid surface, and excessive motion to the thoracolumbar spine should be prevented. A thorough neurologic examination is essential, as is a complete history of the patient's neurologic status before hospital admission. In paraplegic patients, it is imperative to accurately assess the ability of the dog or cat to perceive noxious stimuli applied to the toes of the pelvic limbs. Absence of deep pain perception (DPP) (see p. 1336) is a negative prognostic indicator, especially in trauma cases. The length of time that DPP has been absent is suspected to be inversely related to prognosis, emphasizing the importance of an accurate history. Assessment of neurologic dysfunction as UMN or lower motor neuron in nature is important for localization purposes but does not affect prognosis. Similarly, the presence of Schiff-Sherrington posture (see p. 1330) is helpful in lesion localization but is not a prognostic indicator.

Anesthetic Considerations

Anesthetic management of the patient with thoracolumbar myelopathy is similar to that described for the patient with cervical myelopathy (see p. 1366). In addition, see Tables 32.1 and 32.2 for anesthetic considerations in orthopedic patients.

Antibiotics

Intravenous antibiotics (e.g., cefazolin, 22 mg/kg) should be administered perioperatively (30 minutes before initiation of surgery and every 90–120 minutes thereafter during surgery) and discontinued after surgery or within 24 hours.

Surgical Anatomy

There are 13 thoracic vertebrae and seven lumbar vertebrae. All of these vertebrae have similar components (vertebral body, lamina, pedicle, articular processes, transverse processes, dorsal spinous process), but these components differ substantially in size and shape along the vertebral column (Figs. 41.1 and 41.2). Of particular importance to the surgeon are the differences between the first 10 and the last three thoracic vertebrae and their articulations with each other. The articular processes from T1 through T10 are oriented dorsoventrally like the cervical articular processes (from C2–C3 caudally). Unlike the cervical articular processes, however, these thoracic articular processes do not present an obvious distinct anatomic boundary between what is articular process and what is lamina. From the T10–T11 articulation caudally, the joint spaces of the articular processes are located in a sagittal plane, with the cranial articular process joint surface facing medially and the caudal articular process joint surface facing laterally. The tubercles of the ribs articulate with the transverse processes of the thoracic vertebrae of the same number. The dorsal spinous processes of the thoracic vertebrae decrease in height from cranial to caudal and change from a caudal to a cranial orientation typically after the 11th

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ABSTRACT

The thoracolumbar region is the most commonly operated area of the spine in small animal practice, principally for type I disk extrusions. Detailed knowledge of the surgical approaches to the thoracolumbar spinal cord, particularly hemilaminectomy, is requisite for neurosurgeons. Although less commonly encountered in clinical practice, surgery is sometimes required in the cranial thoracic region (e.g., for congenital kyphotic vertebral malformations); surgical intervention in this area of the vertebral column is technically demanding. Other topics that neurosurgeons should be familiar with are thoracolumbar neoplasia and fracture/luxations, including implant placement.

KEYWORDS

Thoracolumbar spine
hemilaminectomy
dorsal laminectomy
disk
kyphotic malformation

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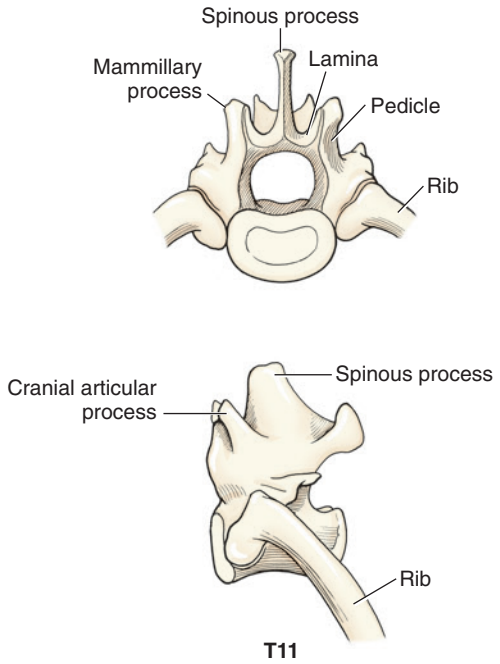


FIG. 41.1 T11 vertebra showing vertebral body. (From Evans HE, de Lahunta A. *Miller's Anatomy of the Dog.* 4th ed. St. Louis: Elsevier; 2012.)

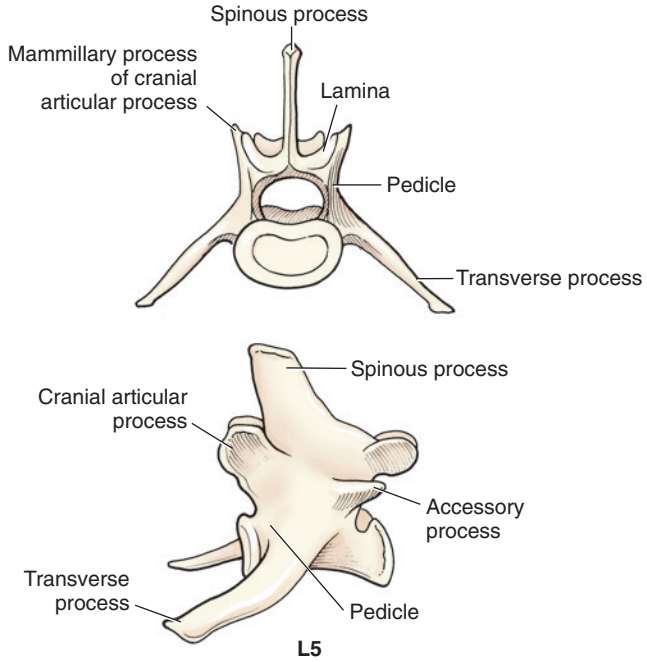


FIG. 41.2 L5 vertebra showing vertebral body. (From Evans HE, de Lahunta A. *Miller's Anatomy of the Dog.* 4th ed. St. Louis: Saunders; 2012.)

(anticlinal) thoracic vertebra, the dorsal spinous process of which is oriented dorsally. From midthoracic to approximately the fifth or sixth lumbar vertebra, there is a distinct accessory process lateral to and at the ventral base of the cranial articular process. From the T11–T12 space caudally, the intervertebral foramen is ventral to the accessory process; from T10–T11 cranially, the

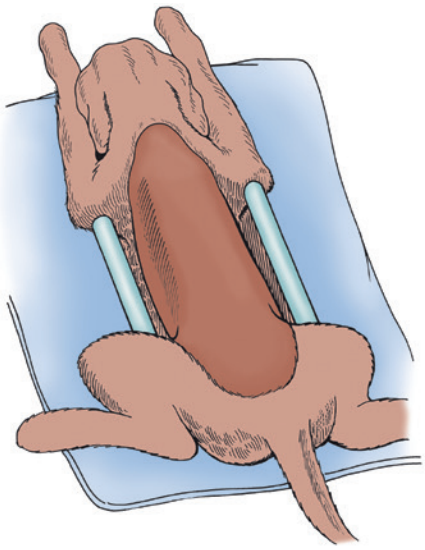


FIG. 41.3 Positioning a dog for a dorsal approach to the cranial and midthoracic spine.

intervertebral foramen is located between the accessory process and articular processes. At the T11–T12 articulation, there is typically a space between the accessory and articular processes; caudal to this articulation, the accessory process is flush with the articular processes. The lumbar vertebrae have large distinctive transverse processes that are oriented cranially and slightly ventrolaterally. The "elbow" of these processes (the cranial aspect of the base of the process near the junction with the pedicle) represents an important surgical landmark for implant placement. The dorsal spinous processes of the lumbar vertebrae are directed slightly cranially; they are largest in the midlumbar region. The dorsal spinous process of the seventh lumbar vertebra is palpably shorter than that of the sixth lumbar vertebra.

Large, paired venous sinuses (internal vertebral venous plexus) run longitudinally on the ventrolateral aspect of the vertebral canal. The thick dorsal longitudinal ligament is located on the ventral aspect of the vertebral canal. Nerve roots, branches of the venous sinuses, and radicular arteries pass laterally through the intervertebral foramina.

STANDARD SURGICAL APPROACHES TO THE THORACOLUMBAR SPINE

DORSAL APPROACH TO THE CRANIAL AND MIDTHORACIC SPINE

The most common indications for the dorsal approach to this region of the thoracic spine include removal of tumors and stabilization/decompression of congenital vertebral anomalies (e.g., "hemivertebrae"). In the latter scenario, it is essential to understand that stabilization of the abnormal region is mandatory. *Position the patient in sternal recumbency with the thoracic limbs pulled cranially (Fig. 41.3) and close to the body. Alternatively, cross the thoracic limbs.* The goal is to abduct the dorsal scapulae so that they do not interfere with the approach.

Make a dorsal midline incision from the caudal cervical to the caudal thoracic region (depending on the specific region of interest and the specific procedure to be performed). Incise the subcutaneous

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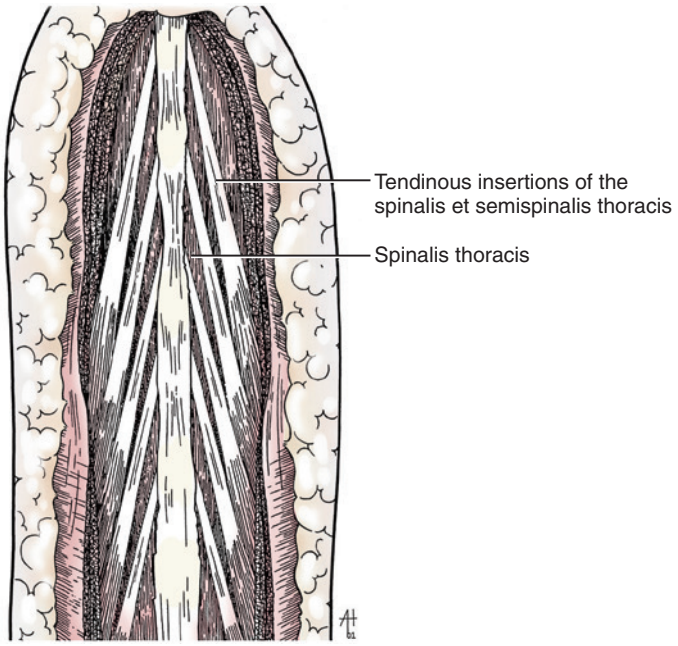


FIG. 41.4 Approach to the cranial and midthoracic spine after retraction of trapezius and rhomboideus muscles. Exposed are the tendons of the spinalis et semispinalis thoracis, and the spinalis thoracis.

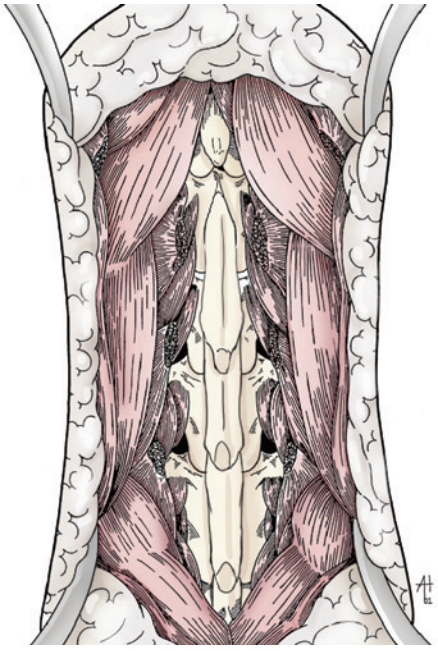


FIG. 41.5 Approach to the cranial and midthoracic spine after elevation of the multifidus musculature and when performing a hemilaminectomy with undercutting of the dorsal spinous process.

tissue to expose the median raphe. Incise the raphe on either side of the transverse processes, controlling hemorrhage with monopolar or bipolar cautery. Retract the trapezius and underlying rhomboideus muscles laterally to expose the serratus dorsalis musculature. Incise the fascia of this musculature along either side of the median raphe close to the dorsal spinous processes, and reflect these muscles laterally to expose the spinalis et semispinalis thoracis musculature and associated tendinous insertions on the dorsal spinous processes (Fig. 41.4). Sharply incise the attachments of these muscles to the dorsolateral aspects of the dorsal spinous processes bilaterally, and then elevate these and deeper epaxial muscles off the lateral aspects of the dorsal spinous processes using Freer elevators (small dogs and cats) or Army-Navy osteotomes. Insert Gelpi retractors at the caudal and cranial aspects of the incision at this time, and remove epaxial musculature from the articular processes to the level of the rib tubercle (if indicated) using a combination of sharp and blunt dissection and bipolar cautery. Several options are available for performing decompression at this point. In our opinion, the dorsal spinous processes should be preserved and a full dorsal laminectomy avoided if at all possible. In this region of the spine, the articular facets probably contribute more to spinal stability than in other regions of the spine, and these facets are difficult to distinguish from laminae at their medial aspects. *Perform a hemilaminectomy using a high-speed air drill, taking care to avoid the intervertebral foramen while drilling. Drill through outer cortical and inner cancellous bone as described for dorsal laminectomy of the cervical region (see p. 1370), then carefully drill the inner cortical layer until it is thin and flaky (like phyllo dough).* In small-breed dogs and cats, the bone in this region of the spine may not have an obvious cancellous layer. *Once the inner cortical layer is thin enough, remove it with a probe (e.g., Gross ear hook and spoon) or fine-tipped Lempert rongeurs. If necessary, undercut the lamina beneath the dorsal spinous process to midline or slightly past midline for increased exposure (Fig. 41.5). If a complete dorsal laminectomy is performed, stabilize the spine. Close the incision routinely.*

DORSAL APPROACH TO THE THORACOLUMBAR SPINE

This approach is the most frequently used in veterinary spinal surgery. It provides access to dorsal, lateral, and ventrolateral aspects of the vertebrae and spinal cord. Dorsal laminectomy, hemilaminectomy, and combinations of these two procedures can be performed via this approach. Indications for this approach include removal of compressive masses (e.g., disc material, hemorrhage, neoplasia, fracture fragments) and application of implants. *Position the patient in sternal recumbency with the thoracic and pelvic limbs in a flexed position (Fig. 41.6). Create a dorsal midline incision approximately three vertebral lengths cranial and caudal to the region of interest. Incise through subcutaneous fat and fascia to reveal the thoracolumbar fascia. Incise the thoracolumbar fascia (unilaterally for a hemilaminectomy, bilaterally for a dorsal laminectomy) just lateral to a dorsal spinous process at the caudal aspect of the incision, and continue the fascial incision cranially using Mayo scissors. Angle the scissor blades away from you to avoid a flap of fascia that will hang into the approach. Use Freer elevators or Army-Navy osteotomes to subperiosteally elevate the multifidus musculature off the lateral aspects of the dorsal spinous processes (Fig. 41.7). Remove any remaining muscular attachments with Metzenbaum scissors. Working caudally to cranially, place a Freer elevator or a small osteotome beneath the multifidus muscle caudal to the articular processes (facets), and pull the muscle dorsally and cranially over the facets, elevating the*

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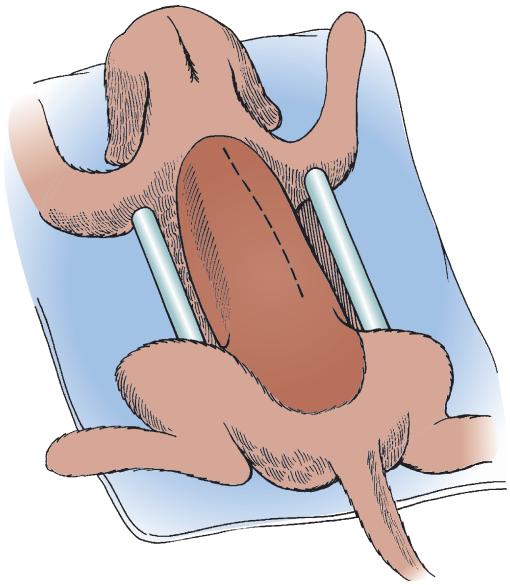


FIG. 41.6 Positioning the patient for a dorsal approach to the thoracolumbar spine.

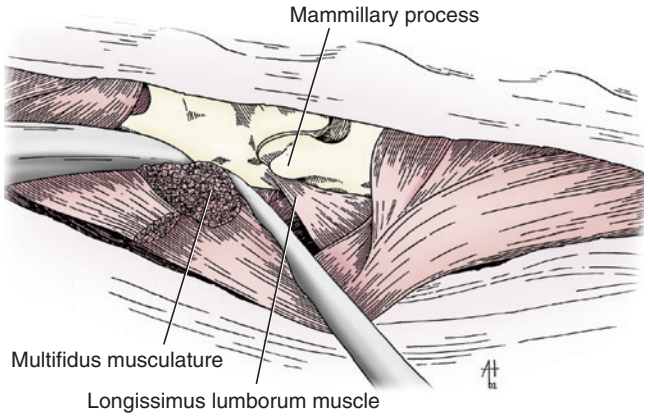


FIG. 41.8 Use of periosteal elevators to remove the attachments of the multifidus musculature to the mammillary process on the cranial articular process (facet). The tendinous attachment of the longissimus lumborum muscle to the accessory process is identified.

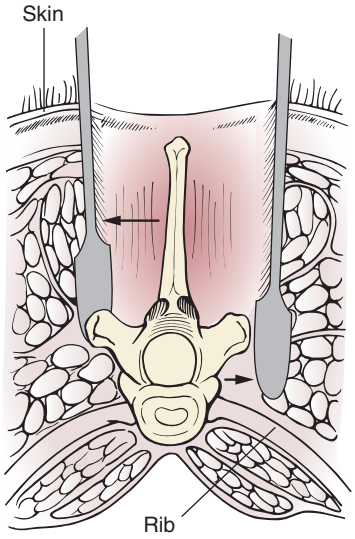


FIG. 41.7 Elevation of the multifidus musculature off the dorsal spinous processes.

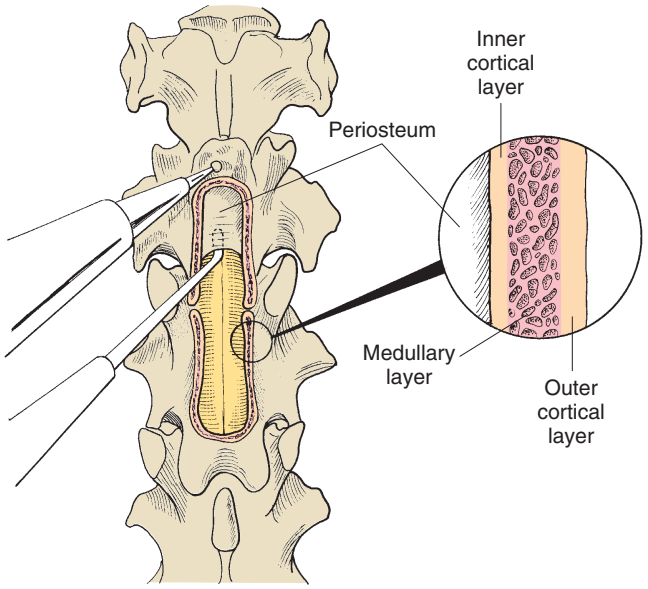


FIG. 41.9 For dorsal laminectomy, carefully drill through outer cortical and cancellous medullary bone layers to reach inner cortical bone. Use a paintbrush stroke to drill this last layer until periosteum is reached. Use a small probe and/or the tips of Lempert rongeurs to remove this final layer.

muscle attachments off the facets with another elevator or osteotome. At the cranial aspect of the facets, locate the white fibrous attachment of the multifidus musculature to the mammillary process (on the cranial articular facet), and remove this attachment with Mayo scissors and/or bipolar cautery (Fig. 41.8). Repeat this process for the remainder of the facets to be uncovered. Place Gelpi retractors in the cranial and caudal aspects of the incision. When performing a hemilaminectomy, place one end of the Gelpi retractor in the interspinous space, the other beneath the multifidus musculature (Videos 41.1 to 41.9). When performing a dorsal laminectomy place both ends of the Gelpi retractors beneath the multifidus musculature. If the dissection already described has been performed bilaterally, exposure is sufficient for performing a dorsal laminectomy. *To perform a dorsal laminectomy, remove the dorsal spinous processes over the intended laminectomy site using bone cutters or double-action rongeurs. Use a high-speed air drill to remove the outer cortical, inner cancellous, and inner cortical bone layers as previously described (Fig. 41.9). To perform a hemilaminectomy, locate the tendons of insertion of the longissimus lumborum musculature, and transect these tendons from their insertions on the accessory process with sharp Metzenbaum scissors or bipolar cautery (Fig. 41.10).* Spinal nerves and blood vessels are located beneath these tendons and should be avoided. *Remove portions of the articular processes using a Lempert or Ruskin rongeur*

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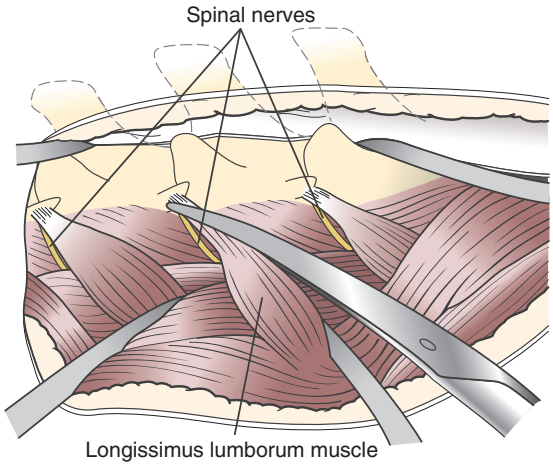


FIG. 41.10 Resection of the tendons of the longissimus lumborum muscles from the accessory processes to improve exposure for hemilaminectomy. The spinal nerve is located beneath the tendon of the longissimus muscle.

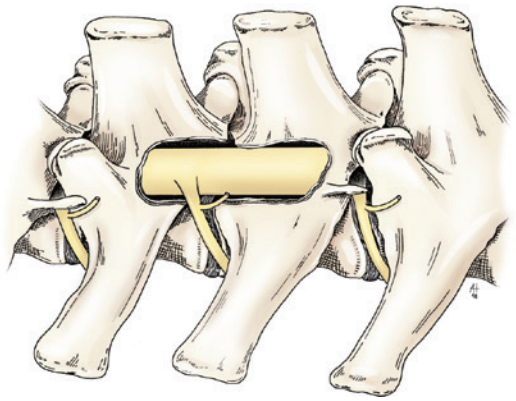


FIG. 41.11 Diagrammatic representation showing the location and extent of a typical left-sided hemilaminectomy.

to make this region more level with the pedicle. Use a high-speed air drill to remove the pedicle, the articular processes, and part of the lamina (undercut the dorsal spinous process).

The bone transition of outer cortical–inner cancellous–inner cortical is similar to that described for dorsal laminectomy. Because of the curvilinear shape of the area to be removed, the dorsal and ventral aspects of the hemilaminectomy are thicker than the middle section. Concentrating on the middle section will likely result in the creation of a narrow hemilaminectomy defect. We prefer to create a wide, rectangular defect (Fig. 41.11) to provide adequate decompression and to allow for ready removal of compressive tissue. We also prefer to leave the accessory process intact until the hemilaminectomy is completed (decreased chance of hemorrhage); this process is easily "snipped" off using the tips of Lempert rongeurs, if indicated. The venous sinuses are likely to be visualized with this approach. *If venous sinus hemorrhage occurs, control the hemorrhage with Surgicel or Gelfoam as described for cervical spinal surgery (see p. 1366).*

A miniaturized version of the hemilaminectomy, called a *pediculectomy* (Fig. 41.12), can also be performed via this approach. *Close the incision routinely.*

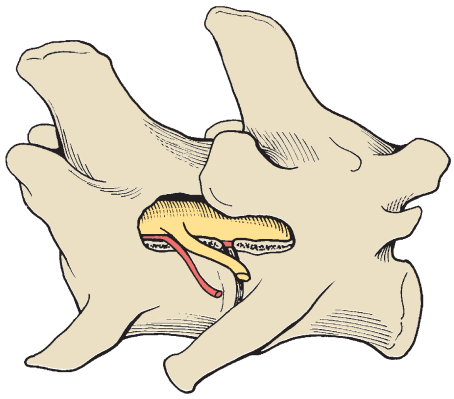


FIG. 41.12 Remove pedicular bone cranial and caudal to the intervertebral foramen during pediculectomy. Cranial and caudal articular processes (facets) are preserved.

LATERAL AND DORSOLATERAL APPROACHES TO THE THORACOLUMBAR SPINE

There are limited applications for these approaches to the spine, which include access to intervertebral discs for fenestration, exposure for pediculectomy, and exposure for lateral corpectomy. These approaches can be performed with the patient in sternal, oblique sternal, or lateral recumbency, depending on surgeon preference. *Create a skin incision lateral to the dorsal midline at approximately the levels of the rib heads and transverse processes (depending on the specific region to be exposed) for the lateral approach and at 1.0 to 2.0 cm lateral to the midline for the dorsolateral approach (Fig. 41.13). Incise through subcutaneous fat and fascia to reach the thoracolumbar fascia. Incise the thoracolumbar fascia to expose an underlying second layer of fat and the thoracolumbar musculature. For the lateral approach, identify and bluntly dissect between the fascicles of the iliocostalis lumborum muscles to expose the rib head or the transverse process (Fig. 41.14). Clear the musculature off the pedicle and disc space using a Freer elevator, and retract the iliocostalis lumborum fascicles with Gelpi retractors (Fig. 41.15). For the dorsolateral approach, bluntly separate between the longissimus lumborum and multifidus lumborum at the septum between these muscle masses (Fig. 41.16) in a craniomedial direction until the tendons of the longissimus muscles are revealed, inserting on the accessory processes. Elevate the musculature off of the intended region of exposure as described for the lateral approach, using Freer elevators. Close the incision routinely.*

HEALING OF THE SPINAL CORD

For healing of the spinal cord, see p. 1373.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

The neurosurgical instrumentation required for surgery of the thoracolumbar spine is essentially the same as that used for brain (p. 1345) and cervical spine surgery (p. 1374). For some thoracolumbar fracture/luxation scenarios, both basic plating equipment (see pp. 1016–1023) and specialized plates (e.g., string-of-pearl plates, Lubra plates, Auburn plates) may be required, depending on surgeon preference.

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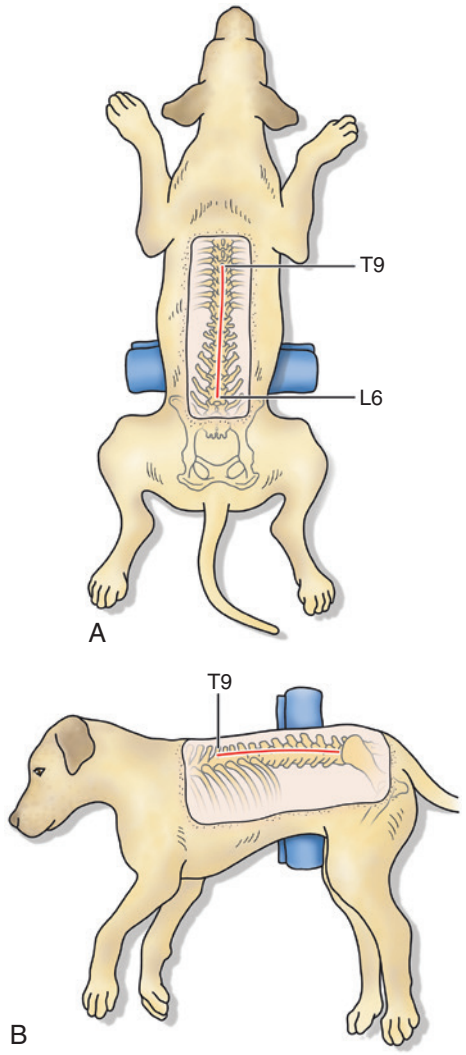


FIG. 41.13 Approximate level of skin incision for (A) dorsolateral and (B) lateral approaches to the thoracolumbar spine.

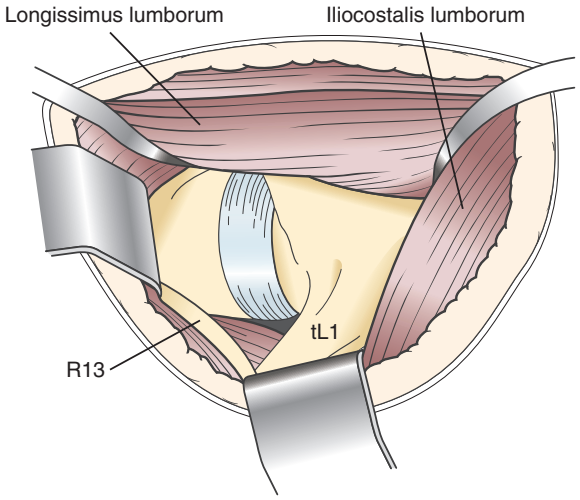


FIG. 41.15 Dorsal retraction of the longissimus lumborum muscle belly and caudal retraction of the iliocostalis lumborum muscles, exposing the T13-L1 intervertebral disc. *R,* Rib; *t,* transverse process.

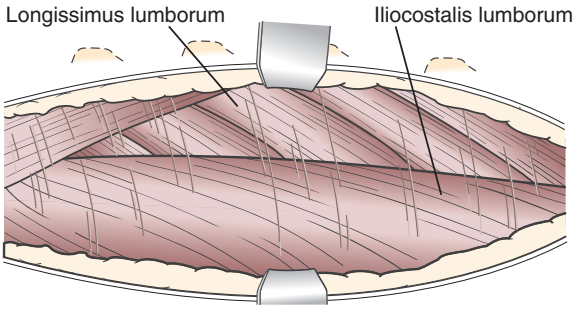


FIG. 41.16 Dorsolateral approach to the thoracolumbar spine. Incision of deep thoracolumbar fascia exposes the muscle separation between multifidus lumborum and longissimus lumborum musculature.

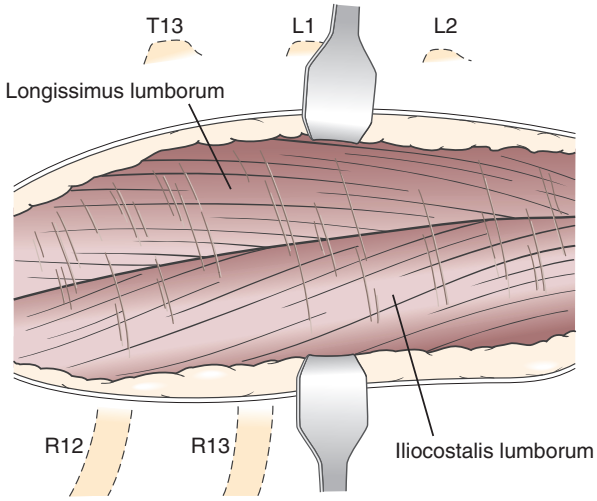


FIG. 41.14 Lateral approach to the thoracolumbar spine. *R,* Rib.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative care of the thoracolumbar surgical patient is similar to what is described for surgery of the cervical spine. Patients with thoracolumbar spinal cord disease are more likely to be nonambulatory than dogs with cervical lesions and more likely to have UMN urinary incontinence. Therefore physical therapy and attentive bladder management is commonly required for these patients. It is prudent to keep patients hospitalized until they are able to urinate on their own, as many owners have difficulty with bladder expression. The majority of dogs and cats with thoracolumbar spinal cord injuries who have voluntary pelvic limb motor function are able to urinate voluntarily. In patients with no DPP to the pelvic limbs, functional recovery is often prolonged or unlikely. In these scenarios, teaching pet owners about bladder management and principles of physical therapy is often necessary. A number of carts are available for patients who are expected to be nonambulatory paraparetic or paraplegic for extended periods of time.

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COMPLICATIONS

Complications associated with thoracolumbar spine surgery are similar to those described for cervical spine surgery (see p. 1375). When working in the cranial to midthoracic spine, especially if near the rib heads, pneumothorax is a possible complication. Inadvertent damage to thoracic or lumbar nerve roots during surgery may lead to a transient visible bulge of a section of abdominal musculature on the operated side. Although uncommonly reported, very wide dorsal laminectomies may predispose to the development of a constrictive laminectomy "membrane" (scar tissue).

SPECIAL AGE CONSIDERATIONS

See p. 1375.

SPECIFIC DISEASES

THORACOLUMBAR DISC DISEASE

DEFINITIONS

As discussed with cervical disc disease (see p. 1375), thoracolumbar disc disease refers to degeneration of the nucleus pulposus (**Hansen type I,** or **type I** degeneration) or the annulus fibrosus (**Hansen type II,** or **type II** degeneration) and the clinical consequences of this degeneration. Type I degeneration is also called *chondroid degeneration,* and type II degeneration is called *fibroid degeneration.* Hydrated nucleus pulposus extrusion has been documented in the thoracolumbar region in dogs[1] but is more commonly reported in the cervical spine. Hydrated nucleus pulposus extrusion is discussed in more detail in Chapter 40.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The pathophysiology of disc disease is discussed in Chapter 40 under cervical disc disease (p. 1375). Type I disc extrusions in dogs and cats (much less common) are usually ventral or ventrolateral (Fig. 41.17). Type I disc disease in the thoracolumbar region is more frequently encountered than cervical disc disease. Disc problems cranial to the T10–T11 disc space are uncommon, probably because of the stabilizing influence of the intercapital ligament (Fig. 41.18). This ligament passes over the dorsal annulus from rib head to rib head in all but the first pair and the last two pairs of ribs. However, cranial thoracic disc extrusions (T1–T9 region) do occur, most commonly in large-breed dogs, and most frequently affecting the German shepherd breed.[2,3] Type I disc extrusions usually occur between vertebral levels T11 and L3. The T12–T13 and T13–L1 discs are the most common sites for type I disc extrusions to occur in small-breed dogs. English cocker spaniels are more likely to experience midlumbar and caudal lumbar disc extrusions, in comparison with dachshunds.[4] There are a number of distinguishing clinical features of French bulldogs that experience thoracolumbar type I disc extrusions that differ from dachshunds. French bulldogs tend to experience lumbar disc extrusions more commonly than dachshunds, especially if there is a concurrent kyphotic/kyphoscoliotic thoracic vertebral malformation in the thoracic vertebral region. In one retrospective study comparing type I intervertebral disc extrusions between 47 French bulldogs and 671 dachshunds, 89% of the French bulldogs had an unrelated (to the disc extrusion) and clinically insignificant thoracic vertebral anomaly (vs. one dachshund). In this study, several significant clinical features were identified in French bulldogs versus dachshunds: younger age at clinical presentation (median of 3 years vs. 5 years), a higher male predominance, and more likelihood of developing progressive hemorrhagic myelomalacia in patients without DPP in the pelvic limbs.[5] In larger dogs, the L1–L2 and L2–L3 disc spaces are the most common sites for type I extrusions. The L4–L5 intervertebral disc space appears to be the most common site for thoracolumbar disc extrusion in cats.

DIAGNOSIS

Clinical Presentation

Signalment

The typical signalment for patients with thoracolumbar disc disease is essentially the same as that described in Chapter 40 (see p. 1376). Although most dogs that present with type I disc extrusions are small breeds, large-breed dogs are also occasionally afflicted by type I disc extrusions. Large dog breeds that seem to be most commonly encountered with type I disc extrusions include mixed breed, German shepherds, Labrador retrievers, Doberman pinschers, and Rottweilers.

History

Historical complaints associated with thoracolumbar disc extrusion are usually related to pain or varying degrees of pelvic limb weakness. The most common neurosurgical emergency encountered in small animal practice is acute onset of nonambulatory paraparesis or paraplegia due to the sudden extrusion of a thoracolumbar disc.

Physical Examination Findings

Although patients with signs of back pain with minimal to no neurologic deficits are occasionally encountered, type I thoracolumbar disc extrusions more typically result in acute paraparesis or paraplegia. This may be due to limited epidural space in the thoracolumbar vertebral canal compared with the cervical region. These patients often exhibit back pain in the general area of the disc extrusion. We occasionally encounter patients with type I disc extrusions in the caudal lumbar region that exhibit a pelvic limb root signature on the side of the extrusion; this phenomenon appears to be more common in spaniel breeds. Type II thoracolumbar disc protrusions typically cause progressive signs of paraparesis, often with some degree of back pain.

Diagnostic Imaging

Diagnostic imaging for suspected disc extrusion/protrusion is discussed in Chapter 40 under cervical disc disease (see p. 1376). In general, detailed spinal radiographs alone are often of limited value in a typical case of a type I disc extrusion.[6] Spinal radiographs may not reveal the site of active disc extrusion (i.e., there may be collapsed disc spaces of no clinical consequence in addition to the one causing clinical signs of disease), do not provide information regarding spinal cord swelling or hemorrhage, and often require sedation or general anesthesia for proper positioning and technique. Survey spinal radiographs are useful in ruling out vertebral neoplasia or discospondylitis but otherwise will not contribute to case management in the typical type I disc extrusion scenario (e.g., young adult dachshund with acute onset of myelopathy). In our opinion, anesthetizing a patient with a

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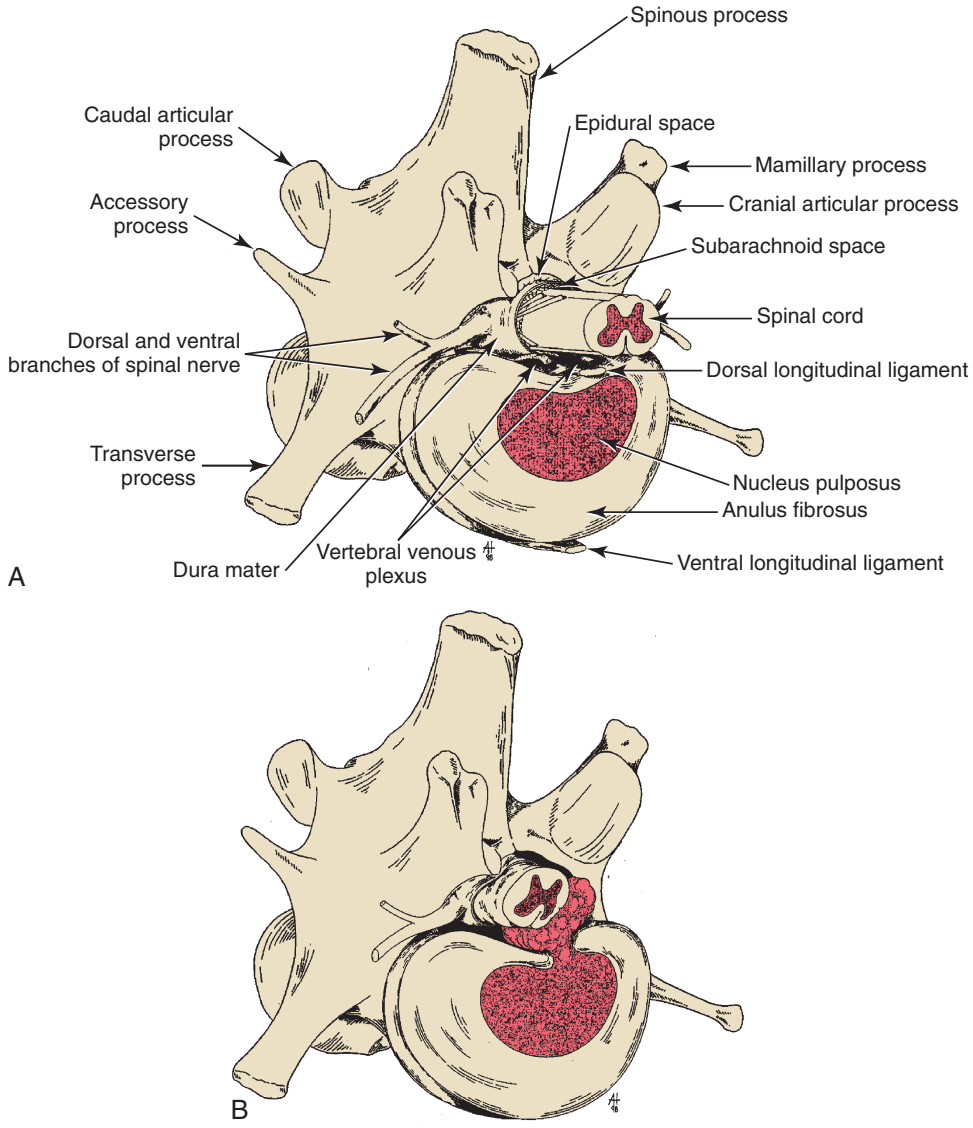


FIG. 41.17 Anatomic structures associated with a (A) normal and (B) extruded thoracolumbar disc.

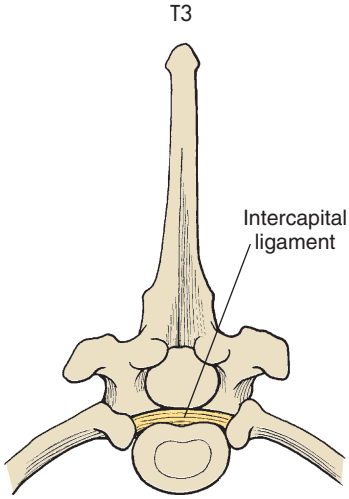


FIG. 41.18 Attachment of the intercapital ligament to each rib head. The ligament spans the dorsal annulus fibrosus.

suspected type I disc extrusion for imaging should not be undertaken unless surgical intervention is planned to follow the imaging, should a compressive lesion be identified. Myelography (Fig. 41.19) is still performed for diagnosing acute type I disc extrusions in dogs, but magnetic resonance imaging (MRI) is performed more frequently for this purpose (Fig. 41.20), and we consider it the imaging mode of choice. Computed tomography (CT) alone or with contrast (CT/myelogram) is another viable imaging modality for diagnosing type I disc extrusions (Fig. 41.21). In one prospective study of dogs with surgically confirmed intervertebral disc extrusions, MRI was found to be more sensitive (98.5%) than CT (88.6%) in correctly identifying the site of disc herniation and also more accurate than CT in discerning disc extrusions from disc protrusions.[7] Certain specific MRI characteristics have been shown to be useful in differentiating disc extrusions (type I) from disc protrusions (type II); protrusions tend to be on midline (extrusions are often lateralized), are typically confined to the disc space (extrusions are more dispersed), and are associated with partial disc degeneration

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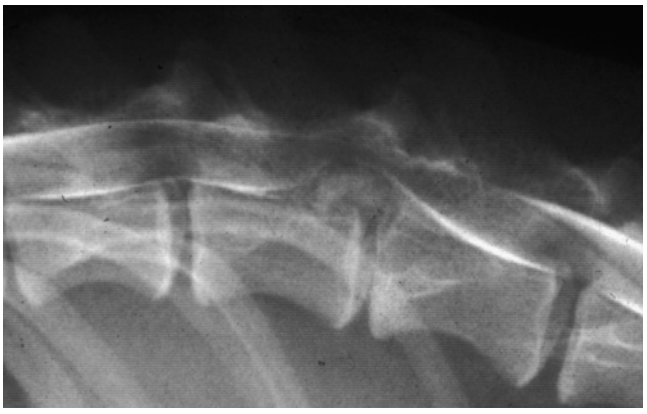


FIG. 41.19 Lateral myelogram showing a type I disc extrusion at T13–L1.

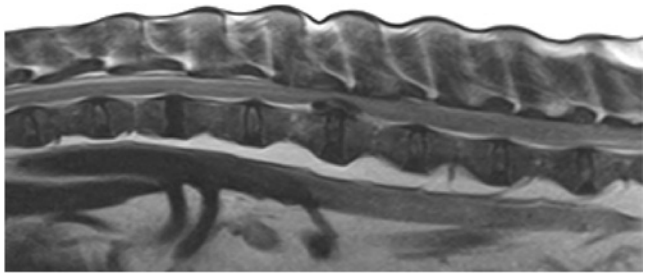


FIG. 41.20 Midsagittal T2-weighted magnetic resonance image showing an L2–L3 disc rupture.

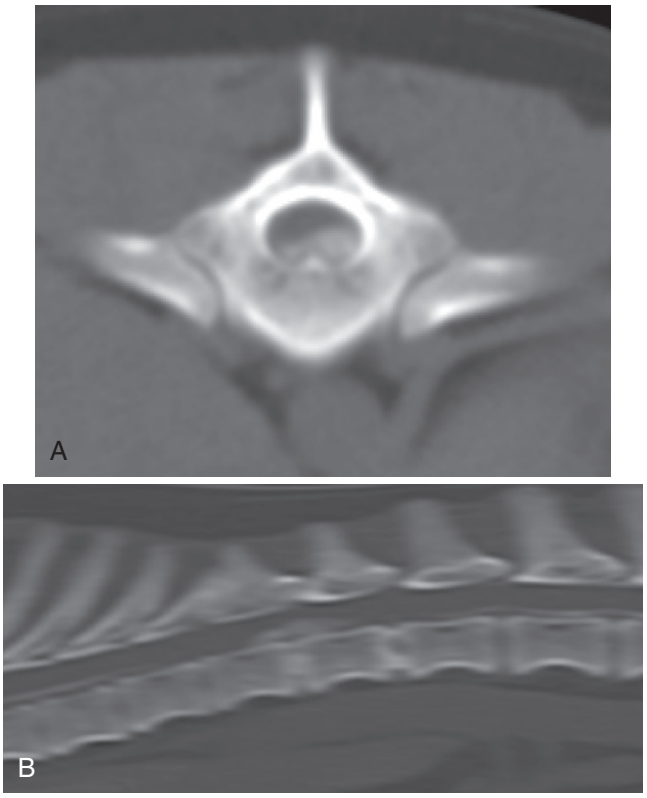


FIG. 41.21 (A) Axial and (B) sagittal reconstructed computed tomography images of a dog with a type I disc extrusion at T13–L1.

(extrusions are usually associated with a completely degenerated disc).[8,9] Several reports have described intramedullary disc extrusions. In this scenario, nuclear material extrudes with enough force to penetrate the meninges and enter the spinal cord. A characteristic MRI feature in such a case is a linear tract extending from the affected disc space into the spinal cord.

Laboratory Findings

Results of complete blood count and biochemistry profile analysis are typically normal. A stress leukogram may be observed on complete blood count results due to the underlying disease, prior administration of glucocorticoids, or a combination of these factors. Hepatic enzyme elevations (especially alkaline phosphatase) may be evident with dogs that have been treated with glucocorticoids. Urinary tract infection (UTI) may occur, especially in dogs treated previously with steroids. If the patient is still receiving glucocorticoids, it might not have pyuria or hematuria despite severe infection. Even if collected before a myelogram, cerebrospinal fluid (CSF) is often is evaluated if clear evidence of a disc compression is seen on imaging. Results of CSF analysis with type I disc extrusions are variable. Although CSF findings in type I disc extrusions have classically been characterized as normal to mildly inflammatory, moderate to marked pleocytosis with increased protein concentration is a common finding.

DIFFERENTIAL DIAGNOSIS

Multiple differential diagnoses are known for patients with suspected disc extrusions/protrusions, and their likelihood depends primarily on the signalment and history. Degenerative myelopathy is a potential differential for progressive type II disc protrusion in older dogs with progressive signs of T3–L3 myelopathy, especially in breeds at risk for this disorder (e.g., German shepherds, Pembroke Welsh corgis). Similarly, spinal neoplasia is a viable differential diagnosis in this situation. In some brachycephalic breeds, especially in young dogs, the presence of kyphotic/kyphoscoliotic vertebral malformations (e.g., hemivertebrae) is a possible cause for thoracolumbar myelopathy. As mentioned previously, such malformations are often incidental findings and may divert the clinician's attention away from the active lesion site (e.g., a disc extrusion elsewhere in the thoracolumbar spine). When such malformations are the cause of myelopathy, the anatomic abnormality is typically striking (an acute kyphotic angle [Cobb angle]) and there is evidence (MRI, myelography) of spinal cord compression; kyphotic/kyphotic vertebral abnormalities are discussed in more detail later in this chapter. Isolated thoracolumbar syrinx formation is not very common but can also cause a T3–L3 myelopathy; such cases are usually associated with local aberrations of laminar CSF flow Inflammatory disorders (e.g., discospondylitis, spinal granulomatous meningoencephalomyelitis, epidural empyema) may mimic signs of intervertebral disc extrusion. Ischemic/vascular events (e.g., fibrocartilaginous embolic myelopathy) can also mimic acute type I disc extrusion, especially in the early stages of that disorder. Most patients with vascular events to the spinal cord exhibit minimal to no evidence of spinal hyperpathia, in contrast to that exhibited by patients with type I disc extrusion.

MEDICAL MANAGEMENT

Medical management for patients with type I or type II disc disease is discussed in detail in Chapter 40 (under cervical disc disease,

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pp. 1378–1379) and is identical to that for thoracolumbar disc disease. As in type I cervical disc extrusion patients, the validity of prolonged (e.g., 3–4 weeks) confinement has been questioned. In one large retrospective study,[10] the duration of crate confinement did not affect outcome, and the use of glucocorticoids had a negative effect on outcome. In this study, 54.7% of dogs were considered treatment successes, 30.9% initially responded to medical therapy then relapsed, and 14.4% were considered treatment failures. The mean and median duration of crate confinement in successfully and unsuccessfully managed dogs in this study was approximately 2 weeks. In another retrospective study concerning medical treatment of ambulatory dogs with suspected thoracolumbar disc extrusions (back pain with mild neurologic deficits), all 78 dogs had an initial positive response to medical treatment, but 50% of them (39 dogs) subsequently experienced recurrence of clinical signs (median follow-up time, 25 months).[11] Other reports of medically treated dogs with presumptive thoracolumbar disc extrusions presenting primarily for back pain cite recurrence rates of approximately 30% to 40% as well. Enforced rest is thought to minimize further disc extrusion into the vertebral canal, while allowing tears in the annulus fibrosus to heal (preventing further disc extrusion). The inflammatory reaction caused by the extruded disc material is thought to subside during this enforced resting period. If confinement therapy is successful, the patient should be allowed to return gradually to a normal level of activity over a period of 4 to 6 weeks. Owners need to be informed that the patient may acutely worsen during confinement and, especially with thoracolumbar disc disease, may become a surgical emergency.

SURGICAL TREATMENT

Indications for surgical intervention for patients with type I disc extrusions include recurrent or unrelenting back pain, worsening neurologic status despite the retention of ambulatory status, and nonambulatory paraparesis or paraplegia. In these scenarios, the time course of events is commonly acute and rapidly progressive. Indications for surgical intervention are similar for patients with type II disc protrusions, although such cases are usually much more chronic in nature.

Preoperative Management

Preoperative management of the patient undergoing thoracolumbar spinal surgery is discussed on p. 1404.

Anesthesia

See p. 1366 for anesthetic management of animals undergoing surgery of the spine. In addition, see Table 32.1 for anesthetic considerations in the stable orthopedic patient.

Surgical Anatomy

See p. 1404 for pertinent surgical anatomy of the thoracolumbar spine.

Positioning

Patient positioning for approaching the thoracolumbar spine is discussed on p. 1406.

SURGICAL TECHNIQUE

Hemilaminectomy is the most commonly performed surgical procedure for decompressing the spinal cord and removing extruded disc material and hemorrhage for type I disc extrusion patients. Dorsal laminectomy and combined hemilaminectomy/dorsal laminectomy procedures are also occasionally performed in these patients. Hemilaminectomy and dorsal laminectomy procedures are described on pp. 1406 to 1408. Removal of type I disc material and durotomy are described in Chapter 40 under cervical disc disease (see pp. 1375–1381). Fenestration is typically performed by retracting the spinal nerve and associated vessels cranially with a Freer elevator and incising the lateral annulus with a No. 11 blade (Fig. 41.22). Multiple methods and indications have been proposed for thoracolumbar disc fenestration. Most recent data support fenestration of degenerated discs within the confines of the surgical approach for a spinal decompression and disc removal as an *adjunctive preventative* procedure (i.e., to prevent further disc

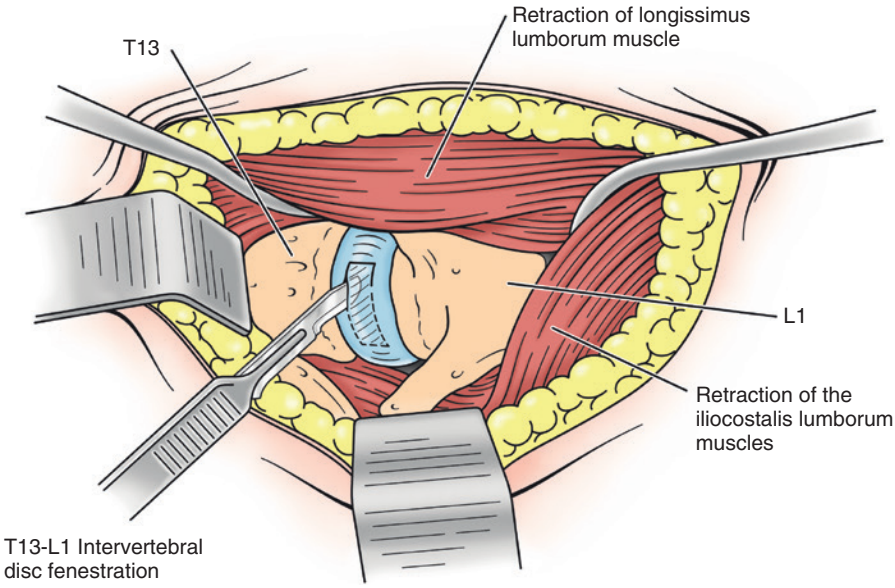


FIG. 41.22 Fenestration of a thoracolumbar disc with a No. 11 scalpel blade.

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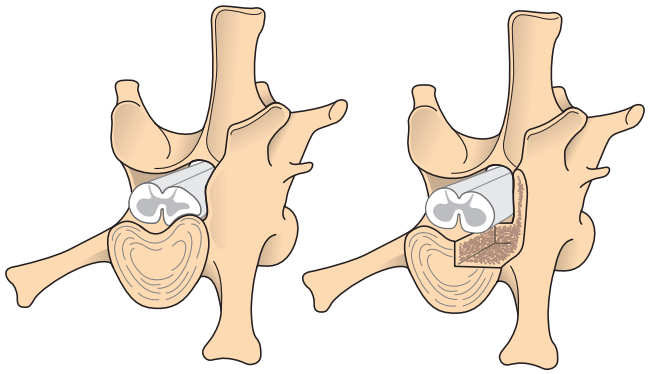


FIG. 41.23 Schematic illustration of the lateral corpectomy procedure (caudal vertebra shown).

extrusions). In one retrospective study of 662 dogs with thoracolumbar disc extrusions treated with hemilaminectomy with or without fenestration of adjacent disc spaces, a recurrent episode of disc extrusion was 26 times more likely to occur in fenestrated versus nonfenestrated discs.[12] Fenestration is never recommended as a sole procedure. Partial lateral corpectomy is a more recently described procedure, primarily for addressing chronic type II disc protrusions in dogs, in which a lateral slot is created ventral to the spinal cord and the protruded disc (Fig. 41.23). *Position the dog in lateral or ventral recumbency, and perform a dorsal, lateral, or dorsolateral approach to the spine (clinician preference), as previously described (pp. 1405–1408). Expose the lateral aspect of the annulus fibrosus and adjacent vertebral bodies, and retract the spinal nerve and associated blood vessels cranially with a probe (e.g., Gross ear hook and spoon). Fenestrate the disc of interest with a No. 11 scalpel blade. Use a high-speed air drill to create the lateral slot in the vertebral bodies and intervening disc.* The dimensions of the slot are somewhat dependent on presurgical imaging findings but are approximately as follows: one-fourth the caudal vertebral length for the caudal margin; one-fourth the cranial vertebral length for the cranial margin; one-half the vertebral body height for the ventral margin; and one-half to two-thirds the vertebral body width for the depth of the slot defect. *Drill through outer cortical and inner cancellous bone along the longitudinal axis of the spine, as well as through the intervening disc. Control hemorrhage from the cancellous bone with bone wax. Carefully drill to the level of the inner cortical bone dorsally until it is thin enough to flake off with a probe or remove with Lempert rongeurs. Remove this final layer of periosteum and remaining annulus material to expose the dorsal longitudinal ligament. Control venous sinus hemorrhage (if necessary) with Gelfoam and/or Surgicel. Close the incision routinely.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

To perform a hemilaminectomy or a dorsal laminectomy, a high-speed air drill, Freer elevators and/or Army-Navy osteotomes (large dogs), Lempert rongeurs, Surgical, Gelfoam, bone wax, sterile cotton applicators, Gelpi retractors, Bishop-Harmon forceps, a Thompson ear hook and spoon (probe), and bipolar cautery are recommended.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative care and assessment for the patient undergoing surgery for thoracolumbar disc disease is similar to what has been described for other thoracolumbar disorders (see p. 1409). Intravenous analgesic drugs are administered as needed for the initial 24 to 48 hours after surgery, and oral analgesic drugs are continued, if needed. Patients are generally hospitalized for a minimum of 72 hours. In general, patients can be sent home before regaining ambulatory status, but we prefer to delay hospital discharge (if possible) until the patient is urinary continent (if continence was lost). Patients are generally rechecked 2 weeks after hospital discharge and every 1 to 2 months thereafter until fully recovered.

COMPLICATIONS

Potential complications of decompressive spinal surgery for disc extrusion/protrusion include neurologic worsening, hemorrhage, postoperative infection, and seroma formation. Postoperative UTIs are common in dogs with thoracolumbar disc extrusions. In many cases, UTIs are subclinical and may occur before surgery and up to several months following surgery. These findings emphasize the need to routinely monitor such patients for UTI and to treat them accordingly. Commonly incriminated organisms include *Escherichia coli, Enterococcus* species, and *Staphylococcus pseudintermedius*. Female dogs are more likely to develop UTI than males, and dogs not administered perioperative antibiotics are more likely to develop a UTI. Other factors that are significantly associated with an increased chance of UTI included nonambulatory status (especially with advancing age), inability to urinate voluntarily, and length of hypothermia while under anesthesia. In a 2016 retrospective study of 38 dogs that underwent hemilaminectomy for thoracolumbar disc extrusion, there was a 20.5% prevalence of UTIs over a 6-week evaluation period; in this study, no difference in likelihood of UTI development was found when dogs treated perioperatively with intravenous cefazolin were compared with those treated with subcutaneous cefovecin (Convenia).[13] The majority of UTIs experienced by postoperative thoracolumbar disc disease patients are not characterized by multiple drug-resistant organisms.

PROGNOSIS

Functional recovery for surgically treated patients with type I thoracolumbar disc extrusions and intact pain perception (nociception) to the pelvic limbs is expected in roughly 80% to 95% of cases (reports range from 72%–100%). In one large retrospective study involving 620 dogs with thoracolumbar disc extrusions and intact pelvic limb pain perception, 606 (97.7%) were ambulatory after surgery.[14] The majority of patients who are nonambulatory after surgery (with intact pelvic limb pain perception) will reach ambulatory status within 2 weeks postsurgery. No difference in outcome has been reported between dogs with UMN and lower motor neuron pelvic limb dysfunction with thoracolumbar disc extrusions. Neither the degree of spinal cord compression nor whether disc extrusions are focal or dispersed on MRI is associated with prognosis in surgically treated dogs with type I thoracolumbar disc extrusions. The prognosis appears to be similarly favorable for cats with intervertebral disc extrusions treated surgically.

Loss of clinically detectable DPP (see p. 1336) in the pelvic limbs occurs with some frequency in type I thoracolumbar disc disease and is associated with a guarded to poor prognosis. In this subset of patients, the length of time elapsed from loss of DPP to surgical decompression has been reported to be inversely associated with prognosis. Unfortunately, an accurate estimate of when DPP was

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lost in a particular patient is often not attainable in clinical practice, and the time of onset of paraplegia is often substituted. Most reports suggest a functional recovery rate of approximately 50% (between 25% and 78%) for paraplegic dogs that have lost DPP to the pelvic limbs. In the large retrospective study mentioned earlier, 110 of 211 dogs (52%) with paraplegia and loss of pelvic limb DPP became ambulatory postsurgery.[14] There is a tendency (based on some of the older published literature) for better functional results to be expected when dogs with absent DPP are operated on within 12 hours of losing DPP. Conversely, there is a long-held belief that paraplegic dogs with loss of DPP for more than 48 hours are not viable surgical candidates; based on more recent clinical studies, this contention appears to be inaccurate. In one prospective study of 78 dogs with paraplegia and lack of DPP due to thoracolumbar disc extrusions, there was no significant association between the timing of surgery in relation to the onset of paraplegia and eventual outcome; in other words, the long-held dogma of a 48 hour "window" of expected successful surgical intervention for dogs with no DPP was disproven. In this study, 58% of dogs regained ambulatory status postsurgery.[15] It is our opinion that timely surgical intervention is warranted in such cases, as surgery may alleviate ongoing spinal cord compression; however, assigning a 48-hour cut-off point for such intervention is not justifiable. Similarly, as concussive parenchymal spinal cord (at the moment of impact) injury is not likely to be affected by surgical intervention, and the relative contribution of such injury (vs. compression) is typically unknown, the concept of a 12-hour "window of opportunity" is probably inaccurate. The development of focal or diffuse myelomalacia (liquefaction of the spinal cord parenchyma) is a concern with dogs that have lost pain perception and can usually be ruled in or out only at surgery (via a durotomy). Unfortunately, even with surgical confirmation of myelomalacia in dogs with no DPP, some level of subjectivity remains. Dogs with focal myelomalacia may still recover function, although the likelihood of this is unknown and performance of a durotomy may not have an effect on outcome. We routinely perform durotomies in dogs with no DPP to inspect the spinal cord for prognostic purposes. Dogs with no DPP before surgery that eventually recover ambulatory function tend to do so over longer time periods than dogs that had DPP before surgery. Although loss of DPP to the pelvic limbs is a negative prognostic indicator, the surgeon should be cautioned against being too "heavy-handed" in these situations.

The prognosis for functional recovery of dogs with chronic thoracolumbar disc protrusions treated via partial lateral corpectomy appears to be favorable in the majority of cases. In one retrospective study of 107 dogs with chronic intervertebral disc disease, 91.4% of dogs were ambulatory at final follow-up.[16] This procedure has also been demonstrated to be a viable option for acute disc extrusions.[17] Partial lateral corpectomy can also be performed in more than one site, for dogs with multiple disc lesions. In one retrospective study of 17 dogs, the use of multiple lateral corpectomies was found to be effective and without clinical evidence of vertebral instability.[18]

HEMIVERTEBRA (KYPHOTIC/KYPHOSCOLIOTIC VERTEBRAL MALFORMATIONS)

DEFINITIONS

Hemivertebra is a term widely (and often inappropriately) used to describe a number of vertebral body abnormalities that lead to kyphosis and scoliosis of the vertebral column. Another commonly used term for these malformations is "butterfly vertebra," although this term refers specifically to vertebrae with a butterfly shape when viewed dorsoventrally. These vertebral anomalies are usually characterized by lack of complete formation of the vertebral body. A more comprehensive radiographic classification system for thoracic vertebral anomalies in dogs has recently been proposed and is based on schemes similar to those used in humans (Fig. 41.24).[19] According to this scheme, kyphosis is more likely to develop with ventral hypoplasia (ventral wedge shape), ventral aplasia, and ventrolateral aplasia of the vertebral body. Although these malformations can be incidental findings in many cases, the resultant wedge-shaped vertebra or vertebrae sometimes cause an unstable segment with spinal cord compression.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

In cases of kyphotic/kyphoscoliotic vertebral malformations, part of the vertebra fails to form properly, usually the vertebral body. The abnormal vertebra usually has a wedge or cuneiform shape. Malformations of the vertebral bodies in the thoracic spinal region of dogs are generally believed to be due to failure of vertebral ossification centers to form, fuse properly, or both during embryonic or fetal development. The etiology of this abnormal development is not fully understood, but factors such as genetic defects, teratogenic insults and abnormal intersegmental blood supply to the developing vertebral column have been implicated. In humans with vertebral body malformations, genetic mutations have been identified in genes responsible for regulating normal fetal vertebral segmentation. In both humans and dogs, the vertebral malformations that tend to cause neurologic deficits are primarily those that lead to kyphosis (dorsal curvature) of the vertebral column. The midthoracic region of the vertebral common is most commonly affected, usually between T6 and T9. The malformed vertebra or vertebrae lead to varying degrees of abnormal angulation of the spine, most commonly kyphosis and scoliosis. In addition to compression of the spinal cord at the level of the malformation, instability may play a role in the development of myelopathy in clinically affected dogs.[20]

DIAGNOSIS

Clinical Presentation

Signalment

Thoracic vertebral body anomalies leading to kyphosis most commonly occur in small-breed dogs, particularly the brachycephalic screw-tailed breeds (English bulldog, French bulldog, pug, Boston terrier). The abnormal shape of the tail in these breeds is thought to be due to similar malformations in the coccygeal vertebral bodies. These thoracic vertebral anomalies are occasionally encountered in other small-breed (e.g., Yorkshire terrier, West Highland white terrier, dachshund, Pekingese, Chihuahua, Maltese) and large-breed (e.g., Doberman pinscher, German shorthaired pointer) dogs. This is thought to be a heritable trait in the screw-tailed breeds, and the disorder in German shorthaired pointer dogs is believed to be autosomal recessive. There is a wide age range at the time of clinical presentation, with approximately 60% being less than 1 year old and 40% greater than 1 year old. There does not appear to be any obvious sex predilection for this disorder.

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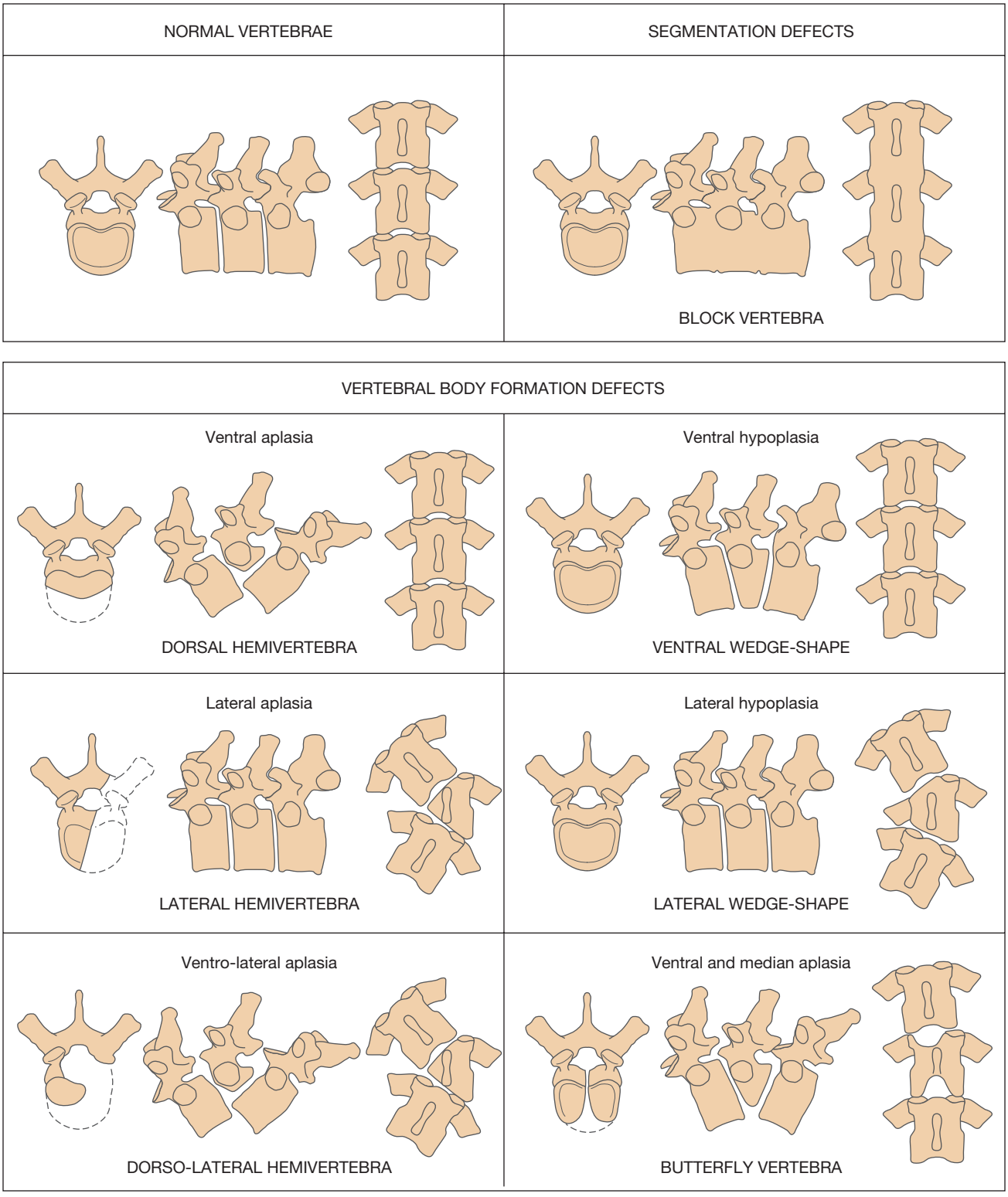


FIG. 41.24 Proposed radiographic classification system for thoracic vertebral anomalies in dogs based on schemes similar to those used in humans. (Modified from Gutierrez-Quintana R, Guevar J, Stalin C, et al. A proposed radiographic classification scheme for thoracic vertebral malformations in brachycephalic "screw-tailed" dog breeds. *Vet Radiol Ultrasound.* 2014;55[6]:585–591.)

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History

The typical history for a dog with a kyphotic vertebral body malformation is a progressive T3–L3 myelopathy. The rate of progression and the severity of dysfunction are variable with this disorder. Some dogs may become nonambulatory over a period of days, and others may progress over weeks to months. We have operated on several affected dogs who had suffered an acute type I disc extrusion in the vicinity of the abnormal vertebral segments. In one small retrospective study (14 dogs), it was found that dogs with kyphoscoliotic thoracic vertebral malformations tended to have degenerative discs adjacent to the malformed vertebral segment.[21]

Physical Examination Findings

Affected dogs typically exhibit clinical signs consistent with a T3–L3 myelopathy. Neurologic severity can vary from back pain with mild ataxia to paralysis and loss of DPP to the pelvic limbs.

Diagnostic Imaging

Imaging options for the diagnosis of kyphoscoliotic vertebral malformations include radiography, myelography, CT, and MRI. Survey radiographs are typically obtained initially, identifying the kyphotic segment. In dogs considered surgical candidates, we prefer to perform MRI first, followed by CT imaging of the area of vertebral malformation. MRI gives superior soft tissue detail (Fig. 41.25), as well as axial images. CT alone or combined with myelographic contrast agent provides excellent bony detail, especially when three-dimensional reconstruction images are created. We have found preoperative CT images to be most useful for surgical planning in these cases. In the vast majority of dogs, the malformed vertebra or vertebrae responsible for the deviation of the vertebral column will be located in the mid to caudal thoracic vertebral region, usually between T6 and T9. In one report,[19] the most commonly affected vertebra was T7, followed by T8 and T12. Scoliosis is typically not associated with neurologic dysfunction. The degree of kyphosis has been associated with the presence of neurologic dysfunction in both dogs and humans. The degree of angulation of the vertebral segment associated with the anomalous vertebra or vertebrae can be quantified by measuring the Cobb angle (Fig. 41.26). The Cobb angle is derived from the intersection of two lines: one at the cranial aspect of the kyphotic vertebral segment and the other at the caudal aspect of the segment. In one retrospective study, a significant difference in kyphotic Cobb angles was appreciated between dogs with and without neurologic dysfunction; the majority of dogs with neurologic dysfunction had a Cobb angle associated with the kyphotic vertebral segment greater than 35 degrees.[22]

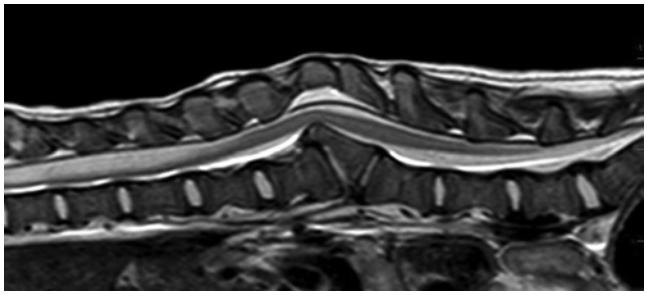


FIG. 41.25 Sagittal T2-weighted magnetic resonance spinal image of a dog with a hemivertebra and spinal cord compression.

Laboratory Findings

Laboratory findings are typically normal. Some mild laboratory abnormalities may be associated with prior glucocorticoid administration.

DIFFERENTIAL DIAGNOSIS

In young patients, differential diagnoses include intervertebral disc extrusion, inflammatory/infectious disease, and trauma. It is extremely important to differentiate dogs with acute type I disc extrusions and incidental thoracic vertebral anomalies from dogs whose vertebral anomalies are the cause of the myelopathy. Neoplasia is also a differential diagnosis but is more likely with older patients.

MEDICAL MANAGEMENT

Medical management of dogs with hemivertebra is similar to that described for patients with intervertebral disc disease (p. 1378), but more prolonged confinement or limited activity is recommended because of suspected instability.

SURGICAL TREATMENT

Preoperative Management

Preoperative management of the patient undergoing thoracolumbar spinal surgery is discussed on p. 1404.

Anesthesia

See p. 1366 for anesthetic management of animals undergoing surgery of the spine. In addition, see Table 32.1 for anesthetic considerations in the stable orthopedic patient.

Surgical Anatomy

See p. 1404 for pertinent surgical anatomy of the thoracolumbar spine.

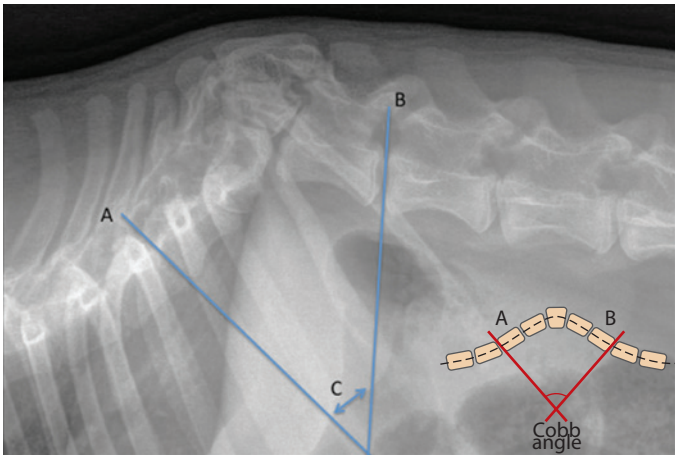


FIG. 41.26 Lateral thoracic radiograph from a dog with a vertebral malformation. The intersection of two lines (*A*) cranial and (*B*) caudal to the malformation show the Cobb angle of 47 degrees (*C*). (From Dewey CW, Davies E, Bouma JL. Kyphosis and kyphoscoliosis associated with congenital malformations of the thoracic vertebral bodies in dogs. *Vet Clin North Am Small Anim Pract.* 2016;46[2]:295–306.)

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Positioning

Patient positioning for approaching the thoracolumbar spine is discussed on pp. 1405 to 1408.

SURGICAL TECHNIQUE

The two most commonly employed methods to stabilize kyphoscoliotic vertebral segments are modified segmental spinal fixation (spinal stapling) and vertebral body pins with polymethylmethacrylate (PMMA). These patients generally have unstable vertebral segments, and decompression without stabilization is contraindicated.

For modified segmental spinal fixation or pin/PMMA fixation, a dorsal approach to the cranial to midthoracic spine is performed with bilateral exposure of the dorsal spinous processes, laminae, and rib tubercles (see p. 1405). At least two, and preferably three, thoracic segments cranial and caudal to the region of suspected instability are included in the fixation. *For modified segmental spinal fixation, drill holes through the bases of the dorsal spinous processes to be included in the fixation, using a 0.045-inch or 0.062-inch (1.143 or 1.575 mm) Kirschner wire (K-wire) (the holes must accommodate the passing of 18- to 20-gauge orthopedic wire). Stay above the dorsal lamina when drilling the holes to prevent entrance into the vertebral canal. Pass lengths of orthopedic wire through the holes and lay them flat. Allow enough length of orthopedic wire on either side of the dorsal spinous processes to comfortably twist them around the "staples" to be placed alongside these processes. Choose two intramedullary (IM) pins of appropriate thickness for the patient (this needs to be "eyeballed" for each patient) to make the "staples." Using a handheld plate bender or a wire twister, bend the ends of the IM pins so that they can be placed around the cranial and caudal dorsal spinous processes at the limits of the fixation.* We prefer to place two staples facing opposite directions (Fig. 41.27), with one staple located inside the other (e.g., one staple spanning two vertebrae cranial and caudal to the site of instability, the other spanning three vertebrae). *Place these IM pins on top of the previously placed orthopedic wires, to lie against the dorsal lamina. Tighten the orthopedic wires around the IM pins using a wire twister. In the cranial and midthoracic region, orthopedic wires looped around the rib heads can be incorporated into the fixation. In the caudal thoracic and lumbar region, orthopedic wire passed through the articular processes (predrilled holes) can also be used. To perform pin/PMMA fixation, select the appropriately sized positive profile threaded pins to place in the vertebral bodies.* Because of limited room for error in pin placement in this region and the abnormal anatomy present in these patients, presurgical measurement for implant placement based on axial MRI or CT images is recommended. *In the cranial and midthoracic spinal region, insert pins into the transverse process, slightly less than halfway from the rib articulation (tubercle of the rib articulating with the transverse process of the vertebra) medially to the upward slope of the lamina as it meets the dorsal spinous process laterally. This is also at the level of or just medial to the mammillary process (a dorsally projecting knoblike eminence on the transverse process). Angle the pins fairly acutely, approximately 20 to 30 degrees from vertical (Fig. 41.28). The proper angle of insertion into the vertebral body is very close to the joint angle between the rib tubercle and the transverse process. Placing a small K-wire into this joint space may help in achieving the correct trajectory for implant placement (Fig. 41.29). Insert the pins to engage the far cortex of the vertebral body, but the tips of the pins should not extend more than a few millimeters past the bone. Insert these pins bilaterally. Place PMMA over the pins bilaterally, with or without an IM pin within the PMMA for reinforcement (Fig. 41.30).*

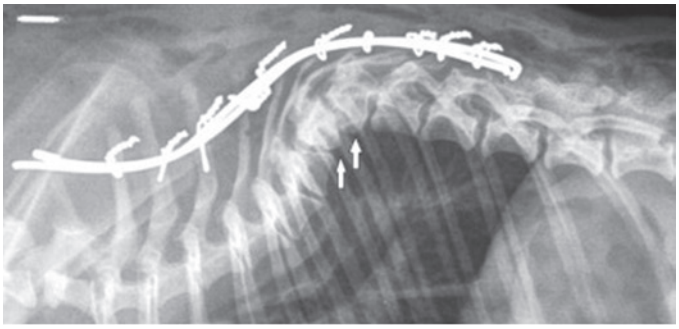


FIG. 41.27 Lateral radiograph showing postoperative segmental spinal implants to stabilize the vertebral column in a young dog with a thoracic vertebral malformation. Image shows the use of wire to stabilize the segments. (From Charalambous M, Jeffery ND, Smith PM, et al. Surgical treatment of dorsal hemivertebrae associated with kyphosis by spinal segmentation stabilization, with or without decompression. *Vet J.* 2014;202[2]: 267–273.)

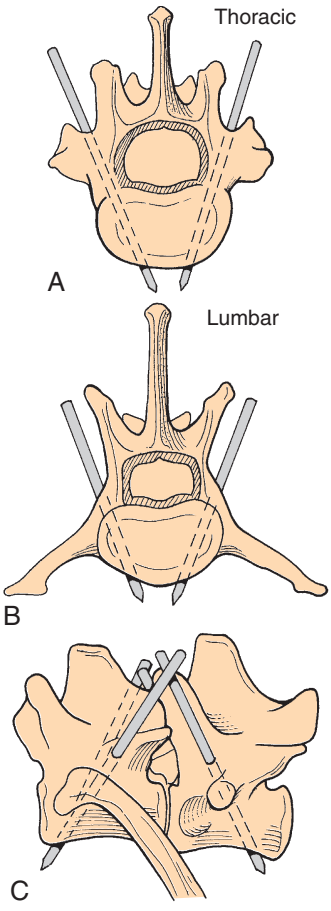


FIG. 41.28 Approximate angles of insertion of pins for (A) thoracic and (B) lumbar vertebrae. (C) Lateral view at thoracolumbar junction.

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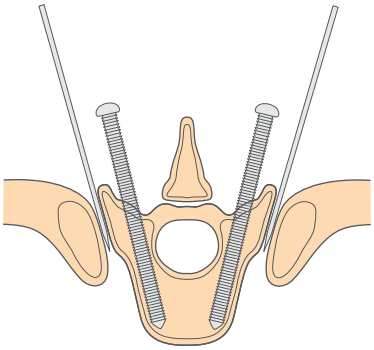


FIG. 41.29 Illustration depicting the placement of a vertebral body pin in a cranial-midthoracic vertebra. A small K-wire has been placed in the joint between the rib tubercle and transverse process to estimate the proper angle of insertion.

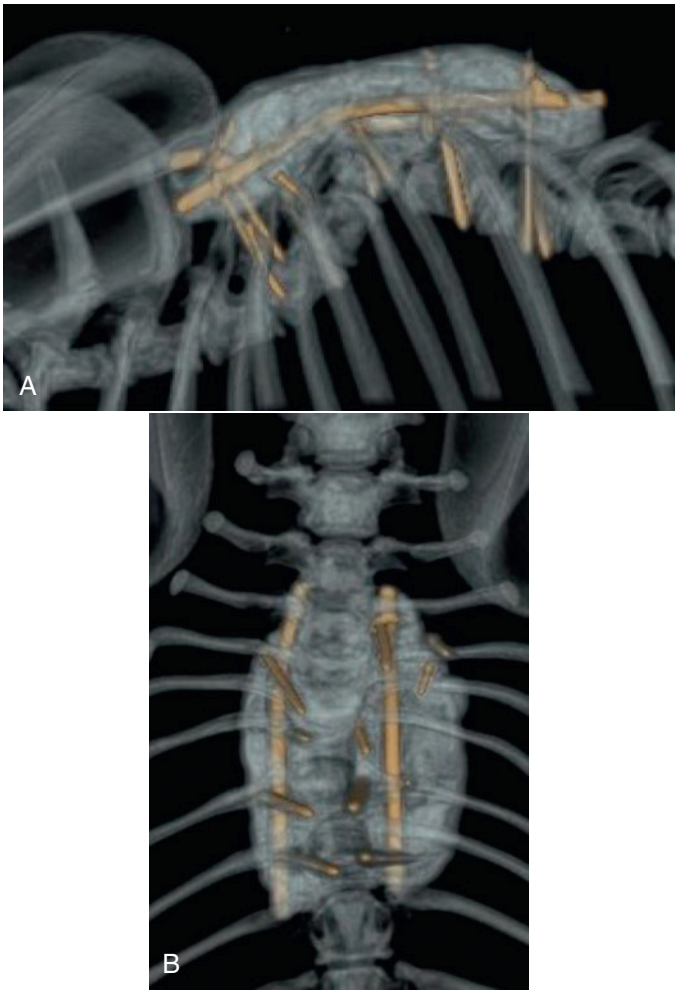


FIG. 41.30 (A) Lateral and (B) ventral views of three-dimensional reconstructed computed tomography images showing postoperative vertebral body pin and polymethylmethacrylate stabilization following dorsal laminectomy to decompress the spinal cord in a young dog with a thoracic vertebral malformation.

The PMMA should be doughy and should not stick to the surgeon's fingers when it is ready to be placed. *Close the incision routinely.*

We prefer a surgical technique that is a hybrid of the two techniques described earlier (Fig. 41.31). A hemilaminectomy is performed over the kyphotic segment. Vertebral body pins are placed in the side of the hemilaminectomy, and a spinal staple is placed on the opposite side. In this method, the trajectory of the spinal cord can be visualized while placing vertebral body pins. Regardless of the surgical method of vertebral stabilization chosen, either postoperative radiographs or CT imaging is recommended to assess the adequacy of implant placement.

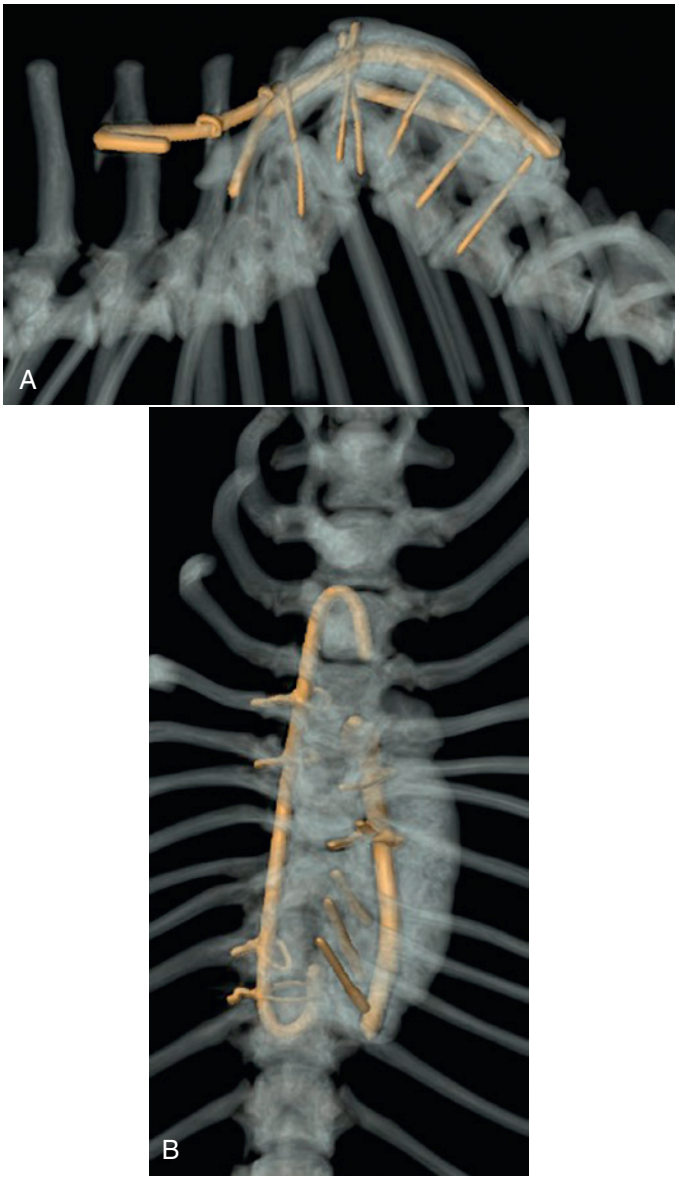


FIG. 41.31 (A) Lateral and (B) ventral views of three-dimensional reconstructed computed tomography images showing postoperative hybrid stabilization using two of the most common techniques following hemilaminectomy to decompress the spinal cord in a young dog with a thoracic vertebral malformation.

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SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Basic instruments needed to perform thoracolumbar spinal surgery are covered on pp. 1374 and 1414. In addition to these instruments, a mini-driver, IM pins, cerclage wire, and PMMA will be required.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative care and assessment is similar to that described for thoracolumbar disc disease (see p. 1409). Longer periods of analgesic drugs are often required owing to implant placement, and longer periods of confinement/limited activity are imposed to prevent premature loosening or breaking of implants.

COMPLICATIONS

Complications associated with thoracolumbar spinal surgery are discussed on p. 1410. Potential complications of pin/PMMA fixation include inadvertent penetration of the vertebral canal and iatrogenic spinal cord damage. In addition to these complications, implant failure and infection are potential complications of this surgery. Pneumothorax may occur during surgery, especially if wires are placed around the rib heads.

PROGNOSIS

Limited reports have described the surgical management of dogs with kyphoscoliotic vertebral anomalies experiencing clinical signs of myelopathy. However, stabilization of the abnormal vertebral segment, with or without decompression, has been successful in most reported cases.[20]

SPINAL NEOPLASIA

DEFINITIONS

Spinal neoplasia refers to primary and secondary tumors of the vertebrae and/or spinal cord parenchyma. **Primary tumors** include those neoplasms that arise from spinal cord parenchyma (e.g., neurons, glial cells) or associated meningeal/ependymal tissue. **Secondary tumors** include tumors that arise from structures extrinsic to the spinal cord and its meningeal covering. Such tumors include primary or metastatic vertebral neoplasms, malignant nerve sheath tumors, and metastases to the extradural space or the cord parenchyma (intramedullary metastases).

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Spinal neoplasia is discussed in detail in Chapter 40 (see pp. 1396–1399).

DIAGNOSIS

Clinical Presentation

Signalment

General signalment aspects for spinal neoplasia are discussed in Chapter 40 (p. 1397). Spinal nephroblastoma is a thoracolumbar neoplasm (occurs between 10th thoracic and third lumbar vertebral levels) of dogs that is typically diagnosed between 6 months and 3 years of age. German shepherds and retrievers appear to be predisposed to developing nephroblastoma.

History

As with cervical spinal neoplasms, thoracolumbar spinal cord tumors most often have slowly progressive signs of myelopathy (e.g., back pain, paraparesis). However, some neoplasms may have more rapid onset and progression of neurologic dysfunction (e.g., lymphoma in cats).

Physical Examination Findings

Focal back pain and varying degrees of pelvic limb ataxia and weakness are characteristic physical examination findings in dogs with thoracolumbar spinal neoplasia.

Diagnostic Imaging

Diagnostic imaging for suspected thoracolumbar neoplasia is identical to that described for cervical spinal neoplasia (see p. 1397). Nephroblastoma is typically in an intradural extramedullary location (Fig. 41.32).

Laboratory Findings

As with cervical spinal neoplasia, specific laboratory abnormalities are unlikely with thoracolumbar spinal neoplasia (see p. 1398).

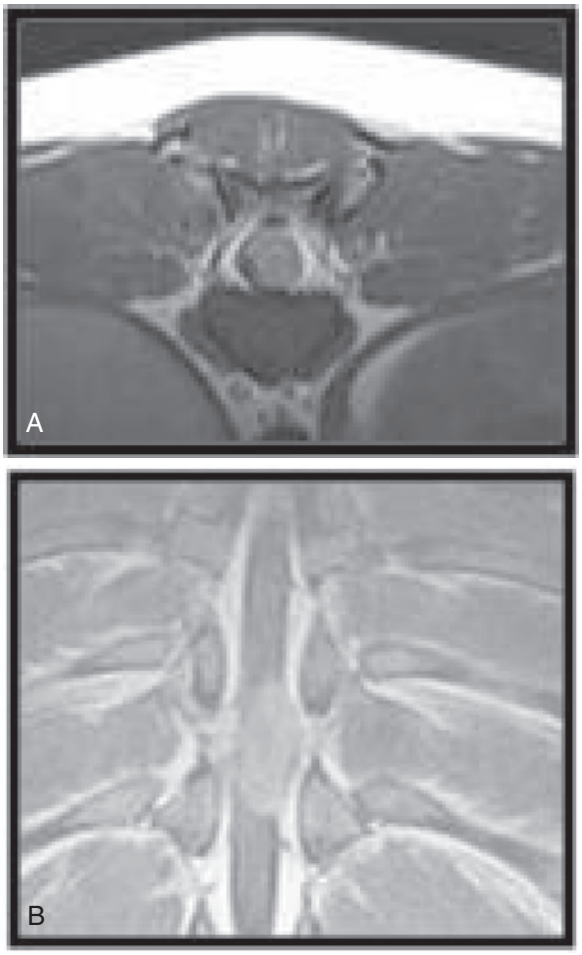


FIG. 41.32 (A) Axial and (B) dorsal T1-weighted with contrast images of a spinal nephroblastoma in a dog.

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DIFFERENTIAL DIAGNOSIS

The main differential diagnoses for thoracolumbar neoplasia are disc protrusion/extrusion and inflammatory/infectious disease (e.g., discospondylitis; see p. 1456). Tentative diagnosis of specific tumor type is based on specific tumor location on imaging studies in combination with signalment information (e.g., extradural mass in a young cat is most likely lymphoma).

MEDICAL MANAGEMENT

Medical management of spinal neoplasia is discussed in Chapter 40 (see p. 1398).

SURGICAL TREATMENT

Preoperative Management

Preoperative management of the patient undergoing thoracolumbar spinal surgery is discussed on p. 1404.

Anesthesia

See p. 1366 for anesthetic management of animals undergoing surgery of the spine. In addition, see Table 32.1 for anesthetic considerations in the stable orthopedic patient.

Surgical Anatomy

See p. 1404 for pertinent surgical anatomy of the thoracolumbar spine.

Positioning

Patient positioning for approaching the thoracolumbar spine is discussed on pp. 1405 to 1408.

SURGICAL TECHNIQUE

Hemilaminectomy, dorsal laminectomy, or a combination of the two procedures is used to access spinal cord and nerve root tumors. These approaches are described on pp. 1405 to 1408. Spinal stabilization may be necessary if vertebral masses are debulked or removed. Goals of surgery are to decompress the spinal cord and remove neoplastic tissue for both reduction of cancer load and diagnosis.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Basic instruments needed to perform thoracolumbar spinal surgery are covered on pp. 1374 and 1414. Additional instrumentation often required for effective tumor removal is discussed under cervical spinal neoplasia (see p. 1398).

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative care and assessment is similar to that described for thoracolumbar disc disease (see p. 1409).

COMPLICATIONS

Similar to other thoracolumbar surgeries, potential complications include neurologic worsening postsurgery, intraoperative hemorrhage, and postoperative seroma formation. Other specific complications include inability to remove tumor tissue and implant-associated complications if implants are used (e.g., vertebral body tumor debulking with stabilization).

PROGNOSIS

Prognosis for different types of spinal neoplasia is discussed in Chapter 40 under cervical spinal neoplasia (see p. 1398). The prognosis for dogs with nephroblastoma undergoing surgical removal of the neoplasm has historically been thought of as favorable; however, the long-term prognosis after surgical resection may not be as positive as was previously believed. The median survival for dogs treated with surgery was reported as 70.5 days based on five dogs in one study, and 380 days in another study based on six dogs.[23,24] Whereas there is considerable variability in the survival times of dogs in these two studies, the assumption that most dogs with this neoplasm are effectively cured with surgical removal is clearly not valid and postoperative radiation therapy should be pursued in these dogs to help improve survival times.

SPINAL TRAUMA

DEFINITIONS

Spinal trauma is defined in Chapter 40 (see p. 1399).

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The pathophysiology of spinal trauma is discussed in Chapter 40. The thoracolumbar spine is the most common site for fracture/luxation in dogs and cats, and the most common cause is automobile trauma. Fracture/luxation of the caudal thoracic and caudal lumbar spinal regions is most frequently encountered, presumably because these regions are more mobile compared with vertebral segments cranial and caudal to them. It is very common for dogs and cats with thoracolumbar trauma to have associated injuries to other parts of the body (e.g., pneumothorax, pulmonary contusions, other orthopedic injuries), and a second region of spinal fracture/luxation is present in approximately 20% of these patients.

DIAGNOSIS

Clinical Presentation

Signalment

As with cervical spinal trauma (see p. 1400), no breed or sex predilection has been noted for thoracolumbar spinal trauma, but this type of injury tends to occur more commonly in younger animals (less than 5 years old).

History

As with cervical spinal trauma (see p. 1400), there is often a known history of automobile trauma in patients with thoracolumbar spinal injury. In some cases, the patient may have gone missing for some period of time before being discovered with evidence of back pain and paraparesis/paraplegia. Dogs and cats that have experienced external trauma may present for reasons other than thoracolumbar spinal trauma (e.g., pelvic fractures, appendicular fractures, brain injury). Neurologic deficits in these patients referable to a T3–L3 myelopathy may be subtle or overshadowed by the presenting problem. It is important to maintain a high index of suspicion for thoracolumbar spinal injury in such cases.

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Physical Examination Findings

In addition to following the basic principles of shock management, the two most important aspects to consider when managing the dog or cat with suspected thoracolumbar spinal injury are (1) evaluate the patient for concurrent injuries (e.g., pneumothorax, pulmonary contusions, other orthopedic injuries) and (2) limit movement of the patient in case instability is present. If instability is suspected or confirmed, the patient should be immobilized (i.e., secured to a rigid structure), especially if that patient is to be transported elsewhere for care following emergency management of shock. If instability is suspected or confirmed, the neurologic examination is modified to prevent excessive motion of unstable vertebral segments. The spine is carefully palpated for areas of abnormal contour and hyperesthesia. When the toes of the pelvic limb are checked for the presence or absence of DPP, it is important that the spine does not move when the dog or cat withdraws the limb; this movement may cause motion of the fracture/luxation site and subsequent pain, which may be falsely interpreted as pain sensation coming from the toes being stimulated by the examiner.

Diagnostic Imaging

Diagnostic imaging for spinal fracture/luxation is discussed in Chapter 40 for cervical spinal injury (p. 1400). The amount of vertebral displacement apparent on imaging is not necessarily indicative of the extent of parenchymal damage. After fracture/luxation occurs, segments can move back into some degree of realignment, even if displacement at the time of injury was complete, causing spinal cord transection. We prefer complete spinal radiographs initially and CT images preoperatively and postoperatively if surgery is to be performed.

Laboratory Findings

As with dogs and cats with cervical spinal injuries (p. 1400), patients with isolated thoracolumbar spinal injuries often have unremarkable laboratory findings (e.g., stress leukogram, elevated hepatic enzyme levels). Injury to other organ systems may result in other laboratory abnormalities (e.g., abnormal arterial blood gas values with pulmonary injury, elevated blood urea nitrogen and creatinine with damage to the urinary tract).

DIFFERENTIAL DIAGNOSIS

As with cervical spinal injury (see p. 1400), there are no reasonable differential diagnoses if the traumatic event was witnessed. Concurrent traumatic disc extrusion is not common but should be considered. Thoracolumbar fracture/luxation following minor trauma should prompt a search for an underlying pathology (e.g., tumor, infection, hyperparathyroidism). As mentioned, other associated traumatic injuries need to be identified and addressed in dogs and cats with known thoracolumbar spine trauma.

MEDICAL MANAGEMENT

General medical management for spinal trauma is discussed in Chapter 40 (see p. 1401). Medical management for dogs and cats with thoracolumbar fracture/luxation is typically chosen for cases with minimal evidence of displacement and suspected low level of instability (e.g., compression fractures). In some cases, this therapeutic route is chosen primarily for financial reasons. Medical management for thoracolumbar fracture/luxation generally includes strict confinement, a back brace, and drugs for pain management. Several options for constructing a back brace are available. The brace should be well padded (e.g., cast padding) and should incorporate a stiff material (e.g., fiberglass, metal rods). The surgeon should assume that the brace affords a small amount of additional stabilization to the patient; strict confinement is still paramount even with a well-constructed brace. Some patients may need to be sedated during the confinement period, depending on their demeanor. Multiple options are available for parenteral and oral analgesic therapy in dogs and cats (see Chapter 13).

Thoracolumbar spinal trauma patients treated via medical therapy alone may have protracted periods of recumbency and inactivity. It is extremely important to provide adequate padding (to avoid pressure sores), appropriate bladder care, frequent turning (to avoid pressure sores, atelectasis, and subsequent pneumonia), range-of-motion physiotherapy (to prevent muscle contracture), and proper nutrition and hydration. Use of a paraparetic cart is contraindicated in these patients; the cart may create a lever arm at the site of instability.

SURGICAL TREATMENT

Preoperative Management

Preoperative management of the patient undergoing thoracolumbar spinal surgery is discussed on p. 1404.

Anesthesia

See p. 1366 for anesthetic management of animals undergoing surgery of the spine. In addition, see Table 32.2 for anesthetic considerations in the orthopedic trauma patient.

Surgical Anatomy

See p. 1404 for pertinent surgical anatomy of the thoracolumbar spine.

Positioning

Patient positioning for approaching the thoracolumbar spine is discussed on pp. 1405 to 1408.

SURGICAL TECHNIQUE

Depending on the specific site, approach the spine bilaterally as described on pp. 1405 to 1408 (dorsal approach to the cranial and midthoracic spine, dorsal approach to the thoracolumbar spine). If a hemilaminectomy, a dorsal laminectomy, or a combined procedure is indicated, perform the desired procedure as described on pp. 1406 to 1408. During exposure, avoid excessive motion of the unstable segment, using bipolar cautery and sharp dissection if necessary. Place penetrating towel clamps or bone-holding forceps (e.g., speed-lock reduction forceps) on the dorsal spinous processes cranial and caudal to the fracture/luxation, and use these to gently reduce the fracture/luxation. Visualize the adjacent articular process articulations (i.e., in the nondisplaced regions) to help judge the appropriate level of reduction (in the caudal thoracic and lumbar spine, the cranial process or facet generally sits more dorsally than the caudal articular process). From medial to lateral, place a small K-wire (0.035 or 0.045 inch [0.889 or 1.143 mm]) through the articular processes at the luxation site if possible (caudal process engaged first, then cranial process) once reduction is achieved (Fig. 41.33); this should maintain reduction while implants are

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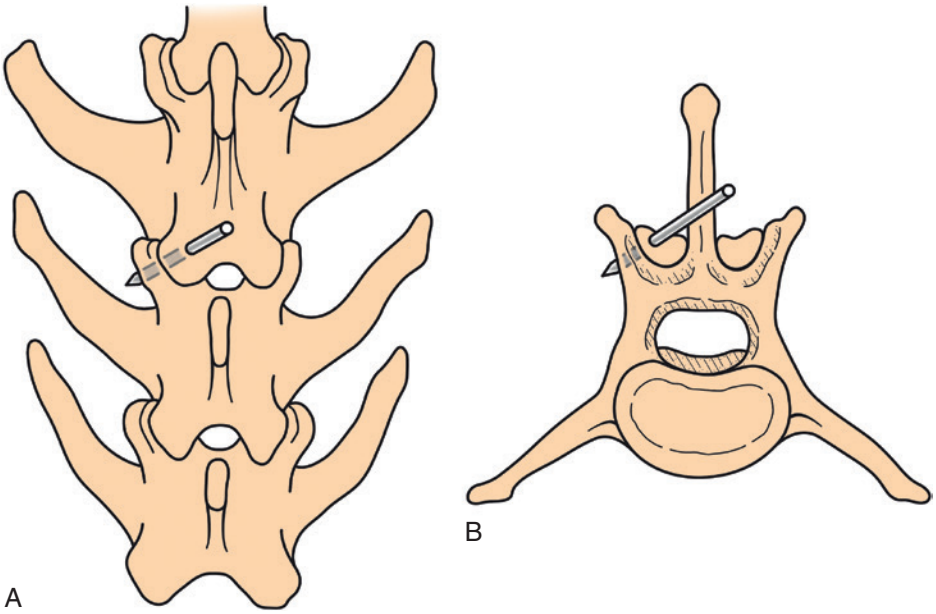


FIG. 41.33 Technique for temporary stabilization using a K-wire across the articular processes. (A) Dorsal view. (B) Axial view.

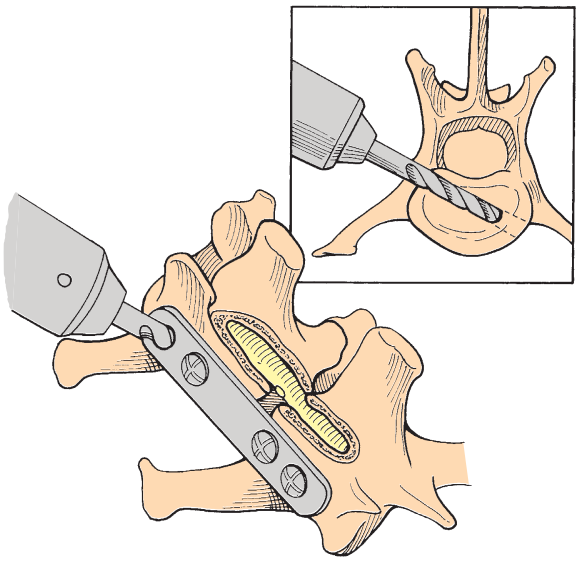


FIG. 41.34 Angle for screw placement in the lumbar vertebrae *(inset).*

being placed. Pin placement in the thoracic vertebrae and the technique for modified segmental spinal fixation are described under hemivertebra treatment (see p. 1418). Pin placement in the lumbar vertebrae is similar, but a less acute angle is taken; typically, 45 or more degrees from vertical (an angle of approximately 50–70 degrees) is used to predrill for plate screws (Fig. 41.34). The target area for vertebral implants in the lumbar region is between the base of the accessory process dorsally and the "elbow" of the transverse process ventrally. Screws (usually cortical) can be used in lieu of pins, depending on surgeon preference. When screws are used, they can be self-tapped after a guide hole is drilled, but we prefer to tap the holes first. Pin/PMMA or screw/PMMA fixation for thoracolumbar vertebral fracture/luxation is generally performed bilaterally (Fig. 41.35). Vertebral body plates can be placed in caudal thoracic vertebrae and lumbar vertebrae. In the thoracic region, the rib heads may need to be drilled down or disarticulated to accommodate the plate (Fig. 41.36). *When using plates in the lumbar region, drill a ventral plate "groove" parallel to the long axis of the spine at the base of the transverse process (Fig. 41.37) to help conform the plate to the vertebra and to prevent slipping during plate application. Engage a minimum of four cortices with screws on either side of a fracture/luxation when plates are used.*

Rhizotomy of the nerve roots is not always required when vertebral body plates are applied but may be necessary in some cases. Because of the risk of damaging nerve root contributions to the femoral nerve caudal to the L4 vertebra, some surgeons do not apply plates caudal to this vertebra. Plating beyond the L4 vertebral level also becomes technically difficult owing to interference by the ilial wings. Plating of the dorsal spinous processes using plastic or metal plates is a less commonly pursued option for thoracolumbar spine fracture/luxation (Fig. 41.38); incorporation of three vertebral segments cranial and caudal to the site of instability is recommended with this mode of stabilization. Closure is routine for all these methods of stabilization. Resection of epaxial musculature to effect closure usually is not required.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

In addition to standard instrumentation required for thoracolumbar spinal surgery (see p. 1414), positive-profile threaded pins, cortical and cancellous screws, bone plates and plating equipment, PMMA, and ASIF small fragment reduction forceps should be available. Depending on surgeon preference, specific dorsal spinous process plating systems (e.g., plastic spinal plates, steel Auburn plates) may also be desired.

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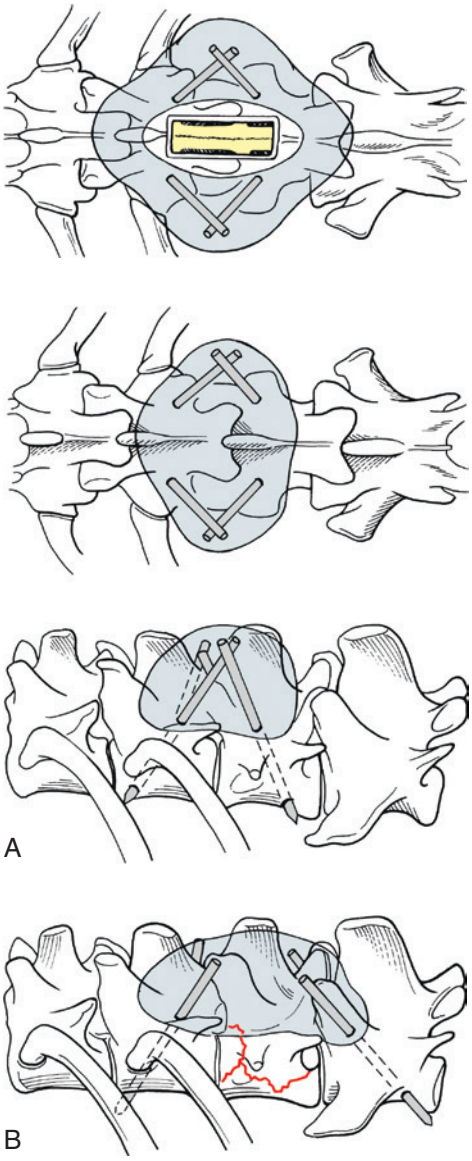


FIG. 41.35 Bilateral fixation with pins and polymethylmethacrylate (A) with and (B) without concurrent performance of a laminectomy.

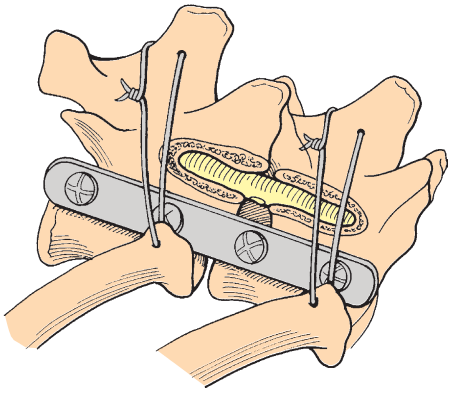


FIG. 41.36 Disarticulation of the rib heads to facilitate vertebral body placement in the caudal thoracic spine. The ribs have been wired to the dorsal spinous processes.

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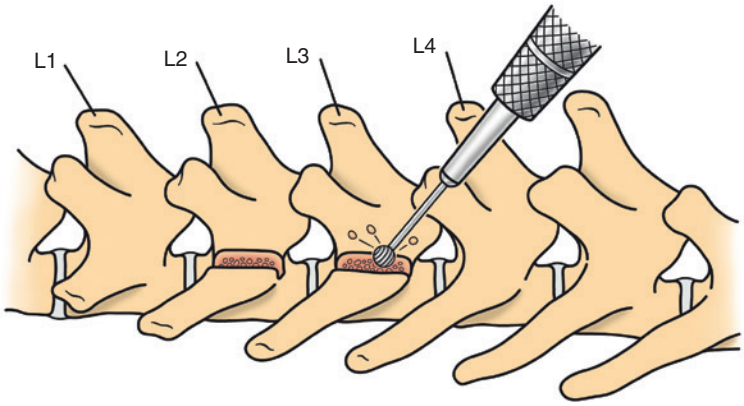


FIG. 41.37 Ventral plate groove created in the base of the transverse process of a lumbar vertebra.

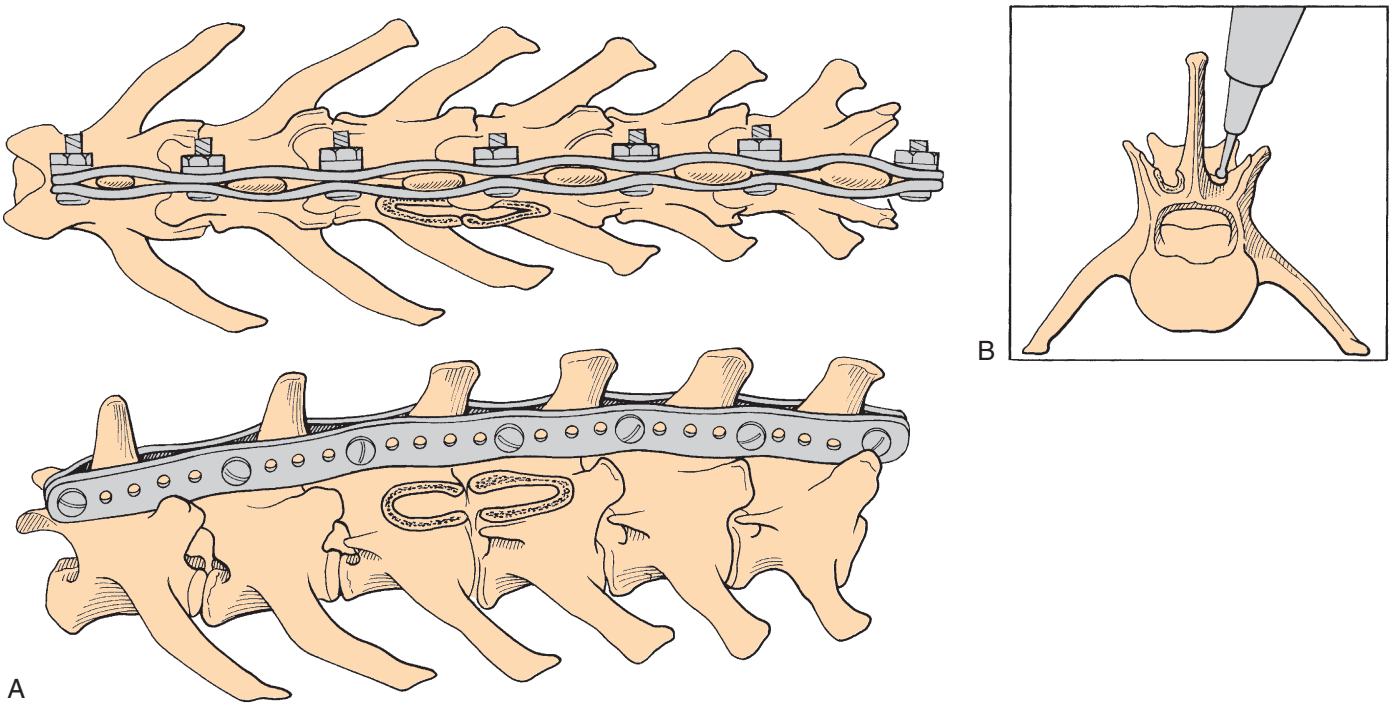


FIG. 41.38 (A) Place plastic dorsal spinous process plates on each side of the dorsal spinous processes. Place nuts and bolts through the plates and between spinous processes. (B) To facilitate uniform plate contact of all dorsal spinous processes spanned by the plate, remove any points of laminar or articular facet contact by the plates with a high-speed pneumatic drill.

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative care and assessment of the thoracolumbar spinal trauma patient is similar to that described for the cervical spinal trauma patient (see p. 1402).

COMPLICATIONS

Complications of surgery for thoracolumbar spinal cord injury include neurologic worsening after surgery, improper implant placement (e.g., iatrogenic damage to the spinal cord, poor bone purchase), implant failure, implant migration, iatrogenic pneumothorax (with cranial and midthoracic vertebral fixation), postoperative infection, and postoperative seroma formation.

PROGNOSIS

In dogs and cats with preservation of DPP to the pelvic limbs, the prognosis for medical or surgical management is generally favorable. Unlike the situation with thoracolumbar intervertebral disc extrusion, loss of deep pain sensation with external trauma (e.g., automobile trauma) is associated with a very poor prognosis; estimates for functional recovery in such patients are generally less than 10%.

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Surgery of the Cauda Equina

GENERAL PRINCIPLES AND TECHNIQUES

DEFINITIONS

The **cauda equina** is the leash of nerve roots derived from the terminal spinal cord segments from L7 caudally (Cd1–Cd5) that travel through the vertebral canal in the lumbosacral area. The **lumbosacral area**, or **lumbosacral junction**, is the bone (e.g., L7 vertebra, sacrum) and connective tissue (e.g., L7–S1 articular facet joint capsules, interarcuate ligament, disc) enclosing the cauda equina. **Dorsal laminectomy** is removal of the lamina; this usually entails partial or complete removal of the dorsal spinous process of L7 and cranial parts of the sacral spine. **Facetectomy** is partial or full removal of the L7–S1 articular processes (facets), and **foraminotomy** is the removal of specific regions of these processes to enlarge the L7–S1 intervertebral foramen. Both facetectomy and foraminotomy are used in this region of the spine to relieve pressure on the L7 nerve root if necessary. **Dysesthesia** and **paresthesia** are similar terms that describe abnormal sensations caused by irritation of nerve roots and/or nerves. Descriptions of such abnormal sensations in people include pricking, burning, and tingling. It is believed that dogs with cauda equina lesions have similar sensations because affected patients often stare or bite at areas around the rump and pelvic limbs.

GENERAL CONSIDERATIONS

The most common clinical sign associated with lesions of the cauda equina is pain (hyperesthesia). Hyperesthesia may arise from compression and/or inflammation of meninges and nerve roots of the cauda equina (particularly the L7 nerve roots), bony components of the lumbosacral area, L7–S1 disc, or L7–S1 articular process (facet) joint capsules. Hyperesthesia may be manifested in a number of ways. Some patients exhibit obvious discomfort when rising or sitting down. Others may be reluctant to jump or climb stairs. A unilateral or bilateral pelvic limb lameness, which may be exacerbated by increased activity, may also indicate hyperesthesia in the area of the lumbosacral region and cauda equina. With some conditions, the patient may appear to be in constant pain (e.g., discospondylitis, vertebral tumor), whereas in others, careful palpation of the lumbosacral area is required to elicit a painful response. Dogs and cats with cauda equina lesions often appear painful on extension of the coxofemoral joints. This may be because the extension is pulling on nerve roots that are already irritated by a disease process at the lumbosacral junction. Alternatively, these patients may have concurrent hip dysplasia that is not of clinical significance, or of lesser clinical significance than the cauda equina disorder. Because clinical signs of cauda equina disease can mimic those of hip dysplasia, it is important not to confuse the two disorders, especially in dogs that are predisposed to both disorders and may have radiologic evidence of hip dysplasia (e.g., German shepherds). There is evidence that the presence of lumbosacral transitional vertebrae is a predisposing factor for developing both degenerative lumbosacral stenosis (DLSS) and hip dysplasia.[1,2]

Proprioceptive deficits are also often evident in patients with cauda equina lesions. Interference with afferent proprioceptive fibers at the level of the cauda equina can produce various degrees of proprioceptive deficits to the pelvic limbs. This can be as mild as delayed proprioceptive positioning reactions or as severe as pelvic limb ataxia with dragging of the dorsal aspect of the toes (knuckling). Dogs with cauda equina lesions are not typically ataxic in the manner that ataxia is usually described; these dogs do not display truncal ataxia when walking; rather, they misstep occasionally and stumble. Proprioceptive deficits may or may not be symmetric. Voluntary motor deficits to the muscles innervated by the sciatic nerve and caudal (coccygeal-tail innervation) nerves may be clinically detectable, as the cord segments and nerve roots of the cauda equina give rise to these nerves. Pelvic limb weakness may be apparent with damage to sciatic nerve contributions, and decreased to absent tail tone and movement may occur with damage to caudal segments and/or nerve roots. During the stance phase of the pelvic limb gait, patients with cauda equina lesions will sometimes either internally or externally rotate the hind paw. This phenomenon may be due to lack of proprioception, but may also be due to interference with innervation to muscles responsible for hip rotation.

In some cases, the patient may exhibit an abnormally low tail carriage, which the owner often notices. Abnormal reflex activity such as decreased to absent withdrawal and gastrocnemius reflexes may be seen. In our experience, most dogs with progressive cauda equina compression (e.g., DLSS) have poor pelvic withdrawal reflexes only at the level of the hock. The patellar reflex is typically normal or may appear hyperreflexive. Caudal thigh muscles normally inhibit the action of the quadriceps muscle group when the patellar reflex is elicited. Removal of this tonic antagonistic influence by disrupting the nerve supply to the caudal thigh muscles may result in an apparently hyperactive patellar reflex (i.e., patellar pseudohyperreflexia). A decreased to absent perineal reflex may result from cauda equina lesions. Urinary and fecal abnormalities, with varying degrees of urinary and fecal incontinence, may occur with damage to sacral segments and/or roots. Bladder dysfunction is classically lower motor neuron (LMN) in

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ABSTRACT

The lumbosacral region is most commonly afflicted by a disorder referred to as degenerative lumbosacral stenosis, which mainly affects middle-aged to older dogs. Standard surgical procedures for the lumbosacral spine include laminectomy, foraminotomy, facetectomy, and methods for surgical stabilization. A minimally invasive procedure for degenerative lumbosacral stenosis is described.

KEYWORDS

Cauda equina
lumbosacral
degenerative
stenosis
nerve roots
facetectomy
foraminotomy
dorsal laminectomy

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nature. Nociceptive (pain perception) deficits in areas of the pelvic limbs, perineum, and tail may occur with severe lesions of the cauda equina; these are usually traumatic in nature.

Preoperative Management

Specifics of preoperative management depend largely on the disease process. In cases of DLSS, most patients will benefit from analgesic drugs prior to scheduling for surgery. There are multiple options for oral analgesic drug therapy for these patients, including nonsteroidal antiinflammatory drugs (NSAIDs), tramadol, and either gabapentin or pregabalin (see Table 34.3). Pregabalin appears to be effective in relieving pain associated with nerve root compression in dogs with DLSS. In our opinion, there is no advantage of glucocorticoids over NSAIDs in patients with cauda equina lesions, and there are potential disadvantages of glucocorticoid use.

Patients with radiographic evidence of discospondylitis (see p. 1457) should be treated with appropriate antibiotic regimens (e.g., oral cefadroxil, 22 mg/kg every 8 hours or clindamycin, 11 mg/kg every 8 hours). The L7–S1 disc space is the most common site for discospondylitis, and some dogs will have this infectious process in addition to DLSS. It is prudent to control the infection before considering surgical decompression; in some cases, treatment of the infection resolves clinical signs of disease and surgery is not needed. In traumatic fractures/luxations of the lumbosacral area, treatment is similar to that described for trauma to other regions of the spine (see pp. 1399 and 1421). These patients need to be stabilized from a cardiovascular standpoint and evaluated for injury to other organ systems. Additionally, these patients need to be kept as immobile as possible to avoid further damage to the nerve roots of the cauda equina.

Anesthetic Considerations

Anesthetic management of the patient with a cauda equina lesion is similar to that described for patients with other spinal surgical disorders (see p. 1366).

Antibiotics

Intravenous (IV) antibiotics (e.g., cefazolin, 22 mg/kg IV) should be administered perioperatively (30 minutes before the initiation of surgery and every 90 to 120 minutes thereafter during surgery) and either discontinued after surgery or within 24 hours. In cases of suspected bacterial discospondylitis, oral antibiotics are administered long-term (months).

Surgical Anatomy

The lumbosacral junction has many features in common with other regions of the lumbar spine (see p. 1404), but there are several unique features (Fig. 42.1). The dorsal spinous process of L7 is considerably shorter than that of L6, and the intervertebral foramen of the lumbosacral junction is dorsoventrally flattened in comparison with cranial lumbar vertebrae. The lateral aspects of this foramen have been referred to as the *lateral recesses* and contain the L7 nerve root as it runs caudally toward the L7–S1 intervertebral foramen. The sacrum is composed of three fused vertebrae, and the fused dorsal spinous processes form a median sacral crest. The dorsal lamina of the sacrum is considerably thinner than the L7 dorsal lamina, and in small dogs and cats it may not have a visually distinguishable inner medullary cancellous bone layer. The prominent ilial wings of the pelvis project dorsally over the dorsal aspect of the sacrum and cranially to the level of the L6–L7 disc space.

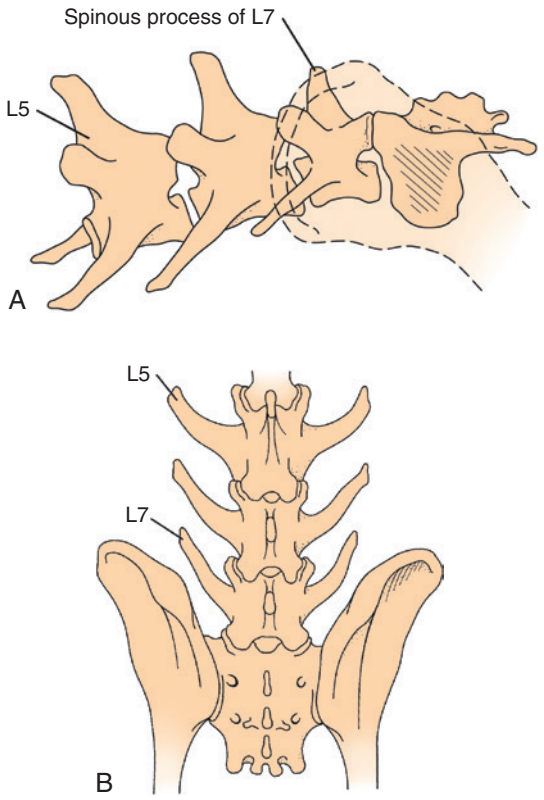


FIG. 42.1 Anatomy of the lumbosacral junction. (A) Lateral view. (B) Dorsal view.

Clinically relevant soft tissue structures in the lumbosacral junction include the termination of the spinal cord and the cauda equina nerve roots, as well as supportive joint and ligamentous tissues and blood vessels. In medium- to large-breed dogs, the spinal cord ends *(conus medullaris)* at the L6 or L7 vertebral level (Fig. 42.2). In small dogs and cats, the spinal cord terminates more caudally, around the S1 vertebral level. The *filum terminale* is a meningeal extension beyond the conus medullaris that attaches to the caudal (coccygeal) vertebrae. As in the rest of the vertebral canal, paired venous sinuses (internal vertebral venous plexuses) are located on the floor of the canal. *Radicular vessels* (artery and vein) travel through the L7–S1 intervertebral foramina along with the L7 nerve root. Small blood vessels also exit the dorsal sacral foramina; these are usually cauterized at surgery or occluded with bone wax. Supportive connective tissues at the lumbosacral junction include the intervertebral disc and dorsal longitudinal ligaments ventrally, the articular process (facet) joint capsules laterally, and the ligamentum flavum (interarcuate ligament) and interspinous ligaments dorsally.

SURGICAL TECHNIQUE

Dorsal laminectomy is used to access the cauda equina, with or without removal of articular process tissue (foraminotomy or facetectomy). Crucial to the performance of a dorsal laminectomy in this region of the spine is proper positioning. The patient should be positioned in a manner that opens the L7–S1 space. The pelvic limbs are flexed and positioned along the patient's abdomen (Fig. 42.3).

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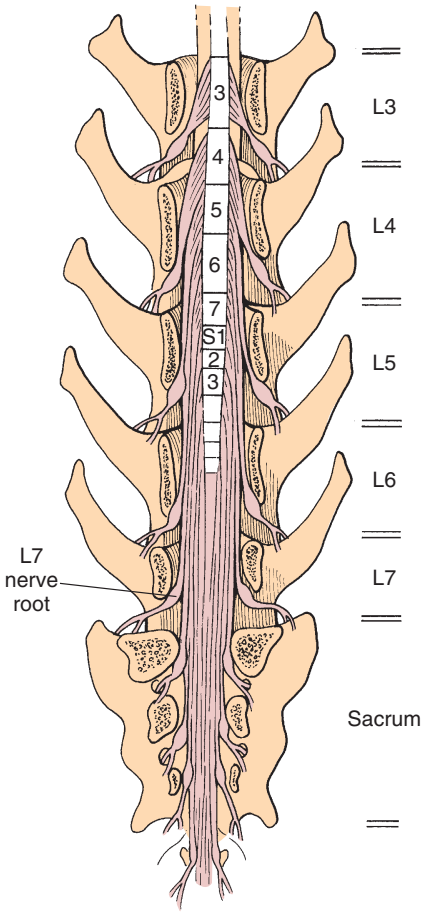


FIG. 42.2 Anatomic relationship of vertebral bodies to spinal cord termination and cauda equina in a medium- to large-breed dog.

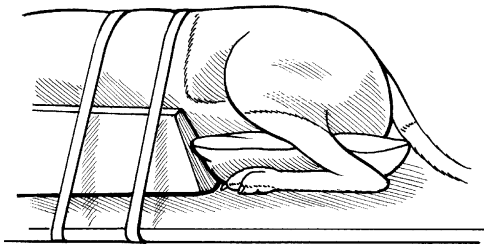


FIG. 42.3 Positioning patients with the pelvic limbs tucked under the abdomen helps accentuate the L7–S1 interarcuate space.

Dorsal Laminectomy

Position the patient in sternal recumbency with the pelvic limbs pulled cranially, as shown in Fig. 42.3. Maintain this position with sandbags, towels, a deflated air cushion, or a V trough. Using the iliac crests and L6 dorsal spinous process as landmarks, create a midline incision from the dorsal spinous process of L5 to the end of the sacral crest (base of the tail). Sharply incise through subcutaneous tissue and fat to reach the thick lumbodorsal fascia. Incise this fascial layer on midline and around the dorsal spinous processes of interest with a No. 11 blade, Mayo scissors, or a combination of the two. Elevate the multifidus lumborum (cranially) and sacrocaudalis dorsalis medialis (caudally) muscles from the dorsal

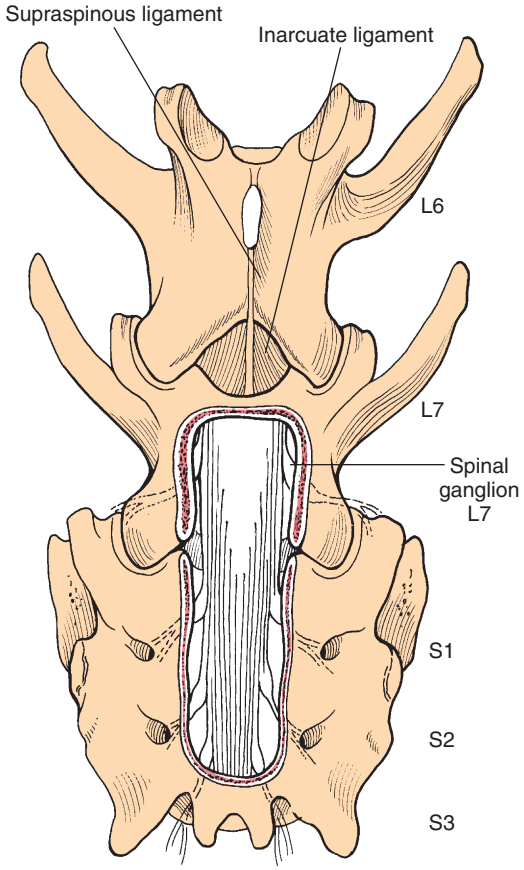


FIG. 42.4 L7–S1, S2 dorsal laminectomy provides exposure of the filum terminale and cauda equina.

spinous processes and median sacral crest using either Freer periosteal elevators (small dogs and cats) or Army-Navy osteotomes (large dogs). Continue this periosteal elevation to expose the dorsal lamina of L7 and the sacrum. Exposure of the L7–S1 junction is facilitated by also exposing L6–L7. Fully expose the articular processes of L7–S1 laterally to the level of the medial aspect of the ilium on each side. Place Gelpi retractors cranially and caudally. Sharply excise the interarcuate ligament between L7 and S1. Identify the interarcuate space of L7–S1 and carefully excise ligamentum flavum tissue with a No. 11 blade and a nontoothed Bishop-Harmon forceps so that the bone edges of caudal L7 and cranial S1 are visualized. Remove the dorsal spinous process of L7 and the cranial half of the median sacral crest with a bone cutter and/or rongeur. Use a high-speed air drill to create the dorsal laminectomy defect, drilling through outer cortical and inner cancellous bone to reach the inner cortical layer. The lateral aspects of the L7 dorsal lamina are thickest, and the dorsal lamina of the sacrum is considerably thinner than that of L7.

Once the inner cortical bone is soft enough, remove it with fine-tipped Lempert rongeurs and/or flake it off with the spatula end of a Gross ear hook and spoon. Remove any remaining periosteum and/or interarcuate ligament (ligamentum flavum) and overlying epidural fat to expose the cauda equina (Fig. 42.4). In some chronic cases of DLSS, the ligamentum flavum and/or endosteum may be adhered to the nerve roots; in this case, carefully dissect the connective tissue off the nerve roots with a No. 11 blade. Using a probe (e.g., Gross ear hook and spoon), feel under the edges

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of the laminectomy defect, especially laterally and in the region of each intervertebral foramen. If compression of the L7 nerve root is still apparent after dorsal laminectomy and removal of compressive annulus tissue, perform a facetectomy or foraminotomy.

Facetectomy and Foraminotomy

Perform a facetectomy by sharply incising and removing the articular process joint capsule tissue of L7–S1, then drilling off the facets from dorsal to ventral using a high-speed air drill. Remove the most ventral aspect of the facets with a fine-tipped Lempert rongeur to avoid ensnaring the L7 nerve root with the burr. To perform a foraminotomy, visually bisect the dorsoventral length of the L7–S1 articular facets. Using a small (1- to 2-mm) burr, drill a longitudinal trough through the facet tissue at this bisection and then snip off the ventral layer of bone (dorsal limit of the intervertebral foramen) with a fine-tipped Lempert rongeur. An angled burr guard is recommended for this procedure. Attempting a foraminotomy by drilling within the intervertebral foramen is not recommended for fear of damaging the L7 nerve root. *Close the site routinely. To help prevent seroma formation, use an interrupted subcuticular pattern for closure.*

HEALING OF THE CAUDA EQUINA

The nerve roots of the cauda equina are more resistant to injury compared with spinal cord parenchyma and respond to injury similar to the response of peripheral nerves. Nerve injury is typically classified according to severity. From least severe to most severe, nerve injury is classified as class I (neurapraxia), class II (axonotmesis), and class III (neurotmesis) (Table 42.1). Class I injury or neuropraxia refers to a transient lack of nerve function, with little or no structural damage to the axons or their supportive connective tissue structures. This temporary dysfunction may be due to ischemia (no structural damage) and/or mild paranodal demyelination. The degree of motor and proprioceptive dysfunction is variable, but nociceptive function is preserved for the most part (large-diameter axons are preferentially affected). Spontaneous recovery is expected within days to a month, depending on the degree of demyelination. Neurogenic muscle atrophy is unlikely, as the axons are structurally intact. With class II injury (axonotmesis), some or all of the axons of the nerve are disrupted structurally, but the connective tissue support (e.g., Schwann cell basal lamina, endoneurium) remains intact. These axons can regrow along the connective tissue scaffold. Substantial motor, proprioceptive, and nociceptive dysfunction is expected, the extent of which depends on the number of axons damaged. Neurogenic muscle atrophy is likely with this class of injury. Class III injury or neurotmesis denotes complete severance of the axons of the nerve, as well as the connective tissue support. These axons will not regrow (no guiding scaffold) without surgical intervention. Complete motor, proprioceptive, and nociceptive (i.e., no pain perception) dysfunction occurs with this class of injury. Neurogenic muscle atrophy is to be expected. A severe class II injury may be clinically indistinguishable from a class III injury. In addition to the extent of nerve injury, the length of nerve required to regenerate affects prognosis. Axons grow at a rate of 1 to 4 mm per day. In people with nerve injuries, muscle motor end-plate degeneration tends to occur if the damaged nerve does not reestablish contact with the target muscle within 18 months. Therefore proximal nerve injuries have more guarded prognoses than distal injuries, even if axonal regeneration occurs.

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Suture material and special instruments for cauda equina surgery are the same as those used for thoracolumbar spinal surgery (see pp. 1374 and 1414).

POSTOPERATIVE CARE AND ASSESSMENT

Postoperative care and assessment are similar to that described for thoracolumbar spinal surgery patients. Short leash walks are allowed after surgery for dogs with DLSS, but longer walks, running, or going up and down stairs may lead to seroma formation. Patients with fracture/luxation at the lumbosacral area should have minimal exercise for the first 2 to 3 weeks after surgery (short walks to urinate and defecate) and gradually return to normal activity over a 2- to 3-month period. Appropriate bladder care and prevention of fecal soiling of the perineal region are necessary in cauda equina patients that have lost, or partially lost, urinary or fecal continence, respectively.

COMPLICATIONS

Severe complications are not commonly encountered in dogs undergoing surgery for cauda equina disorders. As in any spinal

TABLE 42.1 Causes and Characteristics of Nerve Injuries

Nerve Injury Classification	Causes	Characteristics
Class I (neurapraxia)	Ischemia (no structural damage) and/or mild paranodal demyelination	Transient lack of nerve function Little to no structural damage to axons Variable motor and proprioceptive dysfunction Nociceptive function is generally preserved Spontaneous recovery within days to a month
Class II (axonotmesis)	Mainly seen in crush injury	Structural disruption of axons Connective tissue support remains intact Axons may regrow along connective tissue scaffold Substantial motor, proprioceptive, and nociceptive dysfunction Neurogenic muscle atrophy
Class III (neurotmesis)	Occurs with severe contusion, stretch, or laceration	Complete severance of axons of the nerve and connective tissue Axons will not regrow without surgical intervention Complete motor, proprioceptive, and nociceptive dysfunction Neurogenic muscle atrophy

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cord surgery, neurologic worsening that results from manipulation of neural tissue at surgery is always a risk, albeit a small one. The most common complication following surgery of the cauda equina is development of a seroma at the surgery site. Current recommendations are to apply a warm compress to the seroma region several times per day. This usually resolves the problem. Intermittent or continuous drainage is also an option but may predispose the patient to developing a wound infection. In patients with LMN bladder dysfunction, recurrent urinary tract infections are a probable complication after surgery, and meticulous attention to bladder emptying must be paid to minimize this problem. Complications possible with trauma patients with implants include implant failure, inappropriate implant placement (e.g., into the vertebral canal), and implant-associated infection.

SPECIAL AGE CONSIDERATIONS

There are few clinically important age considerations associated with surgery at the cauda equina. In immature patients with traumatic fracture/luxation at L7–S1, the surgeon should be aware that the implant-holding capacity of immature bone is inferior to that of an adult. Older dogs are more likely to develop discospondylitis than younger dogs, so one should be aware that some older patients with suspected DLSS may have discospondylitis as a contributing or primary condition.

SPECIFIC DISEASES

DEGENERATIVE LUMBOSACRAL STENOSIS

DEFINITIONS

DLSS is a compressive disorder of the cauda equina caused by protrusion of supportive tissues (disc annulus, articular process joint capsule, interarcuate ligament) into the vertebral canal. Compromise of one or both L7–S1 intervertebral foramina, with subsequent compression of the L7 nerve root(s) is also a major component of this disorder. **Transitional vertebrae** are malformed vertebrae that possess shape characteristics of two different vertebral types. The L7 vertebra in dogs with DLSS often has a segmented appearance like the sacrum; this is called *sacralization* of the lumbar vertebra. The sacrum in these dogs sometimes has characteristics of a lumbar vertebra (e.g., transverse process unilaterally or bilaterally); this is called *lumbarization* of the sacrum.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

The pathogenesis of DLSS involves type II degeneration and subsequent protrusion of the L7–S1 intervertebral disc into the vertebral canal (Fig. 42.5). There are often other degenerative changes at the lumbosacral junction, indicative of chronic instability. Ventral spondylosis is often appreciated at the lumbosacral junction in these patients, but by itself, this radiographic finding has little clinical significance. Some of the degenerative changes, other than disc protrusion, that may lead to compression of the cauda equina include collapse of the intervertebral disc space at L7–S1 and subluxation of the L7–S1 articular facets (articular processes); the craniodorsal aspect of the sacrum may become displaced ventral to the caudodorsal aspect of the L7 vertebra; hypertrophy and folding inward (ventrally) of the

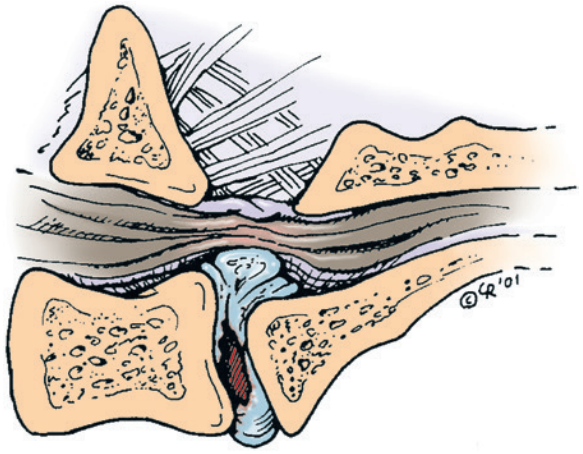


FIG. 42.5 Schematic illustration depicting a protruding disc at the L7–S1 intervertebral disc space.

interarcuate ligament (ligamentum flavum) located between the dorsal laminae of L7 and S1 may occur; and hypertrophy of soft tissue structures (e.g., joint capsule) as well as osteophyte formation associated with the L7–S1 articular processes (facets) may be observed. It has been shown that extension of the lumbosacral junction (lordosis) results in a significant decrease in the volume of the L7–S1 intervertebral foramina, compared with neutral and flexed positions; flexion of the lumbosacral junction has the opposite effect, increasing the volume of the L7–S1 foramina. In one volumetric computed tomography (CT) study, the dynamic narrowing of the L7–S1 intervertebral foramina in extension was more pronounced in German shepherd dogs with clinical features of DLSS compared with German shepherd dogs without clinical evidence of DLSS.[3]

Reasons for the chronic degenerative processes involved in DLSS are unknown. In German shepherds (as well as other large-breed dogs), the presence of transitional vertebrae at the lumbosacral junction has been associated with development of DLSS. Dogs with transitional lumbosacral vertebrae are approximately eight times more likely to develop DLSS than dogs without such anomalous vertebrae. Dogs with transitional lumbosacral vertebrae also develop clinical signs of DLSS 1 to 2 years earlier than dogs with lumbosacral stenosis that do not have these abnormal vertebrae. In addition to having a high incidence of transitional vertebrae at the lumbosacral junction, German shepherds have been shown to have different orientation of vertebral articular processes (facets) in the caudal lumbar and lumbosacral regions of the spinal column and a greater degree of facet joint tropism in this region, compared with other large-breed dogs. Facet joint tropism means an asymmetry in the angle of orientation between left and right articular processes (facets) at a given level of articulation. These anatomic differences, as well as the propensity for German shepherds to have transitional lumbosacral vertebrae, may partly explain the breed prevalence for DLSS.

DIAGNOSIS

Clinical Presentation

Signalment

This is a common disease syndrome that typically affects adult (usually middle-aged to older), large-breed dogs. According to

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some reports, there is a male predilection for this disease. The German shepherd appears to be particularly predisposed to DLSS.

History

The majority of dogs with DLSS have chronic (weeks to months) histories of pain and unilateral or bilateral pelvic limb lameness, with or without pelvic limb weakness. Pain is often localized to the back, but in many cases the owners are not sure if the pain is associated with the back or the pelvic limbs. Other historical findings include difficulty rising, inability or unwillingness to climb stairs or exercise, scuffing of the toenails of the pelvic limbs, muscle atrophy in the caudal thigh region, urinary and/or fecal incontinence, and/or abnormal (usually low) tail carriage with decreased tail movement and biting/chewing at the tail base and/or paws of the pelvic limb.

Physical Examination Findings

Clinical signs are variable, but lumbosacral pain is an early and consistent finding. Pain may be manifested in a number of ways, such as reluctance to rise or sit, and unilateral or bilateral pelvic limb lameness. Pain and lameness may be acute or chronic and may be persistent or episodic. These clinical signs may be misinterpreted as being due to orthopedic disease, most notably hip dysplasia. Many of these dogs have radiographic evidence of hip dysplasia, making this a particularly easy trap to fall into when the only obvious clinical sign of an abnormality is pain and/or pelvic limb lameness. If untreated, clinical signs of dysfunction may progress to proprioceptive loss in the pelvic limbs, voluntary motor weakness (in the distribution area of the sciatic nerve), and urinary/fecal incontinence, often in that order. Deficient pelvic limb withdrawal reflexes are most apparent at the hock level in cases of DLSS; flexion at the hip and stifle regions often appears normal. Although typically indicative of progressively severe cauda equina compression, urinary and/or fecal incontinence occasionally comprises the initial primary clinical complaint in cases of DLSS. Careful palpation of the lumbosacral area, including lordosing the caudal spine while pressing on the lumbosacral area, will usually elicit a pain response in affected patients. Most dogs afflicted with this disease also have obvious deficits on proprioceptive positioning tests of the pelvic limbs.

Diagnostic Imaging

The definitive diagnosis of DLSS is made by imaging of the lumbosacral area and demonstrating compression of the cauda equina. Magnetic resonance imaging (MRI) (Fig. 42.6) and CT (Fig. 42.7) are the imaging procedures of choice for DLSS, with all other imaging procedures rapidly becoming historical in nature. For MRI evaluation of the L7–S1 intervertebral foramina, oblique parasagittal imaging planes have been recommended over standard parasagittal planes to more accurately evaluate the dimensions of the foramina.[4] Although degenerative changes at the lumbosacral junction (e.g., spondylosis, malalignment of the sacrum with the L7 vertebra) may often be appreciated on plain radiographs of the region, additional imaging is necessary to demonstrate cauda equina compression. A number of procedures have been advocated in the past, including myelography, epidurography, vertebral sinus venography, and discography. Because the terminal thecal sac (subarachnoid space) of the spinal cord in large-breed dogs often ends cranial to the lumbosacral junction, myelography may not consistently provide useful information. *Vertebral sinus* venography is considered to be both a technically difficult and unreliable imaging procedure. *Discography* is an accurate imaging method for DLSS but provides information primarily limited to the intervertebral disc. *Epidurography* is also considered to be a relatively accurate contrast procedure to identify cauda equina compression. Combination discography/epidurography has also been demonstrated to be a useful diagnostic imaging method for DLSS. There is evidence that dogs without clinical signs of disease at the cauda equina may have imaging findings consistent with a diagnosis of DLSS and that the degree of cauda equina compression evident on imaging does not necessarily correlate with disease presence or severity. It is apparent that evidence on imaging studies consistent with a diagnosis of DLSS may be an incidental finding in some dogs. It is also apparent that the extent of cauda equina compression appreciated on such imaging studies is not necessarily indicative of disease severity in patients that exhibit clinical signs of DLSS. Middle-aged to older, large-breed dogs often demonstrate hyperesthesia on palpation of the lumbosacral junction during a neurologic examination that has nothing to do with the presenting complaint or ultimate diagnosis. In other words, some dogs with DLSS may have subtle or inapparent clinical signs that can only be elicited on palpation. It is imperative to realize that focusing on the lumbosacral junction in some patients may lead to overlooking another spinal disorder that is primarily responsible

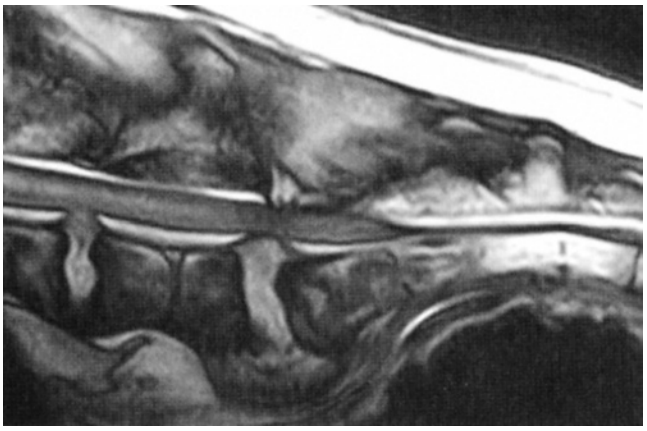


FIG. 42.6 Sagittal magnetic resonance image (T2 weighted) of a dog with degenerative lumbosacral stenosis. There is protrusion at both the L6–L7 and L7–S1 intervertebral disc spaces.

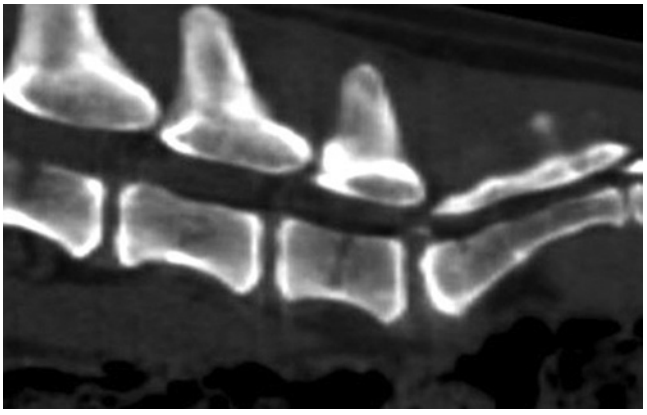


FIG. 42.7 Midsagittal computed tomography reconstructed image of a dog with degenerative lumbosacral stenosis.

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for the clinical deficits. In patients with subtle deficits (e.g., pelvic limb proprioceptive deficits and spinal hyperesthesia), it may be worthwhile to have survey image of the thoracolumbar region (e.g., T2-sagittal magnetic resonance survey) to ensure that the lumbosacral lesion is not an incidental finding. Concurrent electromyography performed on the epaxial, tail, and pelvic limb musculature may improve the accuracy of diagnosis with the various imaging modalities. Tibial nerve somatosensory evoked potentials have also been shown to be abnormal in dogs with clinical evidence of DLSS.[5]

Laboratory Findings

Laboratory findings in dogs with DLSS are generally nonspecific. A stress leukogram may be evident in patients who have received glucocorticoid therapy.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for DLSS include infectious/inflammatory disease (e.g., discospondylitis, vertebral osteomyelitis), neoplasia (vertebral or soft tissue), and trauma (fracture/luxation). Hip dysplasia can cause many signs similar to DLSS, but this can be ruled out as a sole explanation for the patient's clinical signs with a neurologic examination.

MEDICAL MANAGEMENT

Medical therapy for DLSS consists of enforced rest and antiinflammatory/analgesic medication, and is often recommended for dogs with signs of hyperesthesia only. Nonsurgical therapy is often either transiently effective or ineffective for this disease. However, in one retrospective study with long-term follow-up (mean and median of approximately 3 years), 55% of medically managed dogs with DLSS had successful outcomes.[6]

SURGICAL TREATMENT

In patients with neurologic deficits, or patients for whom pain is refractory to nonsurgical management, surgery is the preferred mode of therapy. Surgery usually consists of a dorsal laminectomy over the L7–S1 interspace, sometimes combined with removal of hypertrophied soft tissue (e.g., annular disc material). It should be noted that there is no evidence that resection of disc material (annulectomy and partial discectomy) improves clinical outcomes over dorsal laminectomy alone, but that there is evidence from cadaver studies that dorsal laminectomy combined with discectomy results in significant destabilization of the L7–S1 articulation, including dynamic collapse of the intervertebral foramina.[7,8] Enlargement of the L7–S1 intervertebral foramen (foraminotomy) or removal of the articular facets (facetectomy) may also be required if compression of the L7 nerve root is appreciated. Many surgeons do not advocate routine surgical stabilization of the lumbosacral joint, but this may be advisable in some cases (e.g., bilateral facetectomy). There are numerous published techniques for stabilization of the lumbosacral region, some of which are discussed under Trauma. Recently, a minimally invasive transilial vertebral blocking (MTV) procedure has been described for treatment of dogs with DLSS. In this technique, the patient is secured with the lumbosacral joint in a flexed position, and a premeasured transilial bolt is fluoroscopically (C-arm) placed via a detachable target pin (using the L7 dorsal spinous process as a buttress), fixing the L7–S1 space in distraction (Fig. 42.8). In a prospective evaluation of 59 dogs with DLSS treated with the MTV procedure, significant increases in L7–S1 end-plate distance and the mean areas of the L7–S1 foramina were documented via CT measurements.[9]

Preoperative Management

Preoperative management for cauda equina surgery is discussed on p. 1428.

Anesthesia

Anesthesia for patients undergoing spinal surgery is discussed on p. 1366.

Surgical Anatomy

The pertinent surgical anatomy of the lumbosacral area and cauda equina is discussed on pp. 1404 and 1428.

Positioning

Positioning for dorsal laminectomy at L7–S1 is discussed on p. 1429.

SURGICAL TECHNIQUE

The surgical techniques for dorsal laminectomy at L7–S1, facetectomy, and foraminotomy are described on pp. 1428 to 1430. Removal of protruding annulus fibrosus may be indicated in some instances. *To remove protruding dorsal annulus fibrosus of the L7–S1 disc, gently retract the nerve roots medial to the L7 nerve root on one side to expose the disc on that side. Use 25-gauge needles to act as retractors by placing the needles into the disc, holding the needle hub with a straight Mosquito hemostat during insertion (Fig. 42.9). Cut a wedge of annulus out of the disc using a sharp No. 11 blade, and remove the wedge with either straight Mosquito hemostats or the tips of a Lempert rongeur. Remove the needles and repeat the procedure on the opposite side. Control venous sinus hemorrhage, if necessary, as described on p. 1366.*

SUTURE MATERIALS AND SPECIAL INSTRUMENTS

Suture material and special instruments for dorsal laminectomy or foraminotomy/facetectomy at the lumbosacral junction are the same as those used for thoracolumbar spinal surgery (see pp. 1374 and 1414).

COMPLICATIONS

Complications of surgery at the lumbosacral junction are discussed on p. 1430.

PROGNOSIS

The prognosis for functional recovery from this disorder is generally good to excellent with surgical intervention and fair for medical treatment alone (see Medical Management). Successful outcomes after surgery have been reported in the majority of cases. It appears that although improvement in continence may occur in dogs with presurgical fecal and/or urinary incontinence (over weeks to months following surgery), resolution of incontinence is not likely to occur in most cases. Incontinence fails to resolve after decompressive surgery in half to two-thirds of cases. Improvement or resolution of incontinence has been shown to be much less likely in dogs that have been incontinent for

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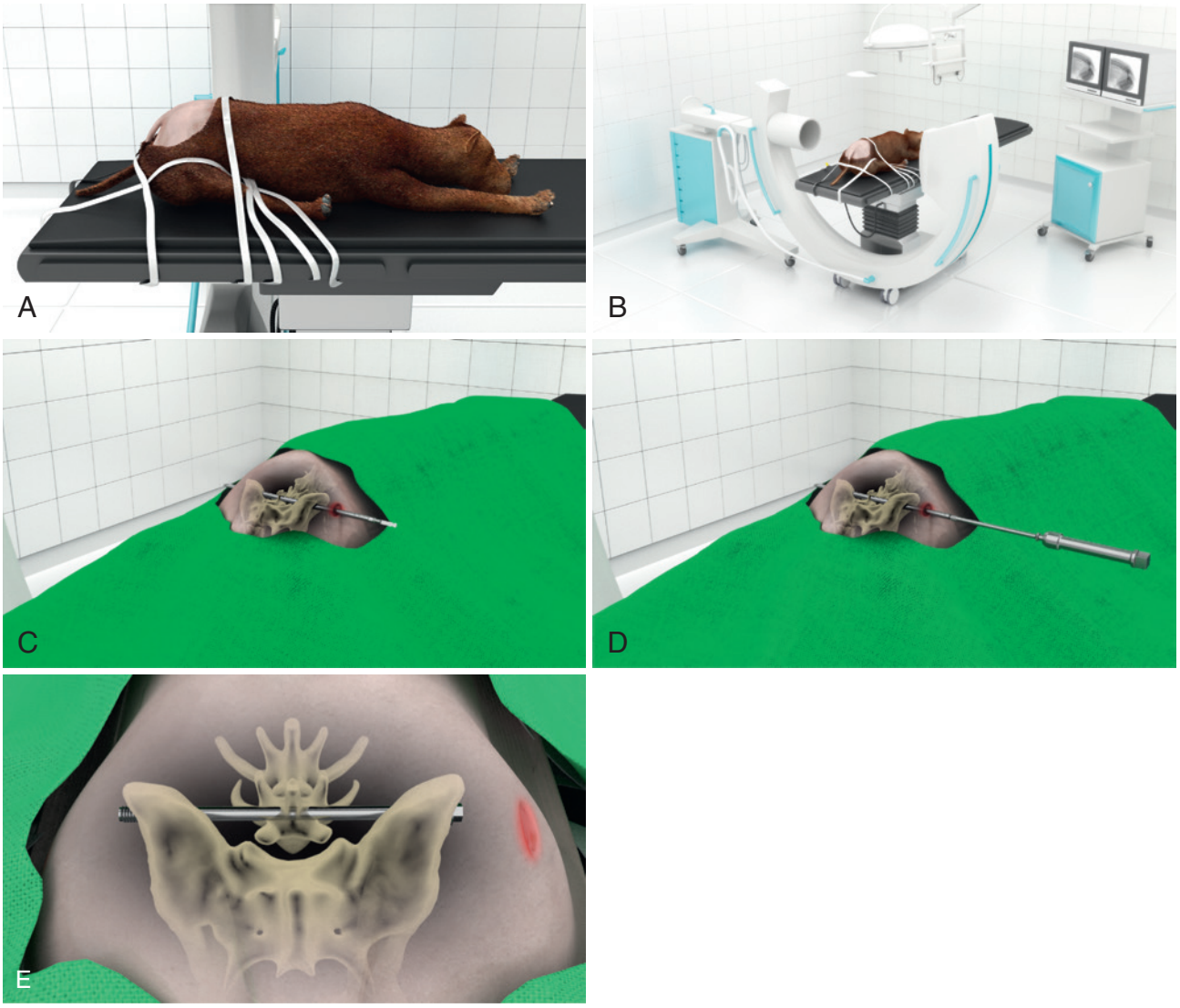


FIG. 42.8 Schematic steps of the minimally invasive transilial vertebral blocking procedure. (A) Positioning the patient. (B) Positioning the C-arm (fluoroscope). (C) Drilling the target pin (through a skin incision). (D) Target pin in place, connected to implant. (E) Implant in position, target pin disconnected. (From Müller F, Schenk HC, Forterre F. Short-term and long-term effects of a minimally invasive transilial blocking procedure on the lumbosacral morphometry in dogs measured by computed tomography. *Vet Surg.* 2017;46[3]:354–366.)

more than 1 month, compared with those that have shorter histories of incontinence. Recurrence rates for DLSS vary between 3% and 18%. Recurrence is probably more likely in very active dogs (i.e., working dogs). For the 39 dogs in the prospective MTV study available for long-term follow-up (1 year), all had improved by at least one neurologic grade (using a three grade scoring system: mild, moderate, severe dysfunction); in addition, 78% of all owners indicated via questionnaire that they would recommend the procedure.[9]

NEOPLASIA

DEFINITIONS

Neoplasms involving the lumbosacral vertebra and nerve roots are similar to those of the cervical and thoracolumbar regions (see pp. 1396 and 1429). As in other areas of the spine, tumors of the cauda equina can be primary (e.g., malignant nerve sheath tumor) or secondary (e.g., L7 or S1 vertebral body tumor).

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Tumors of the lumbosacral vertebrae and nerve roots are rare. Tumor type, location (e.g., spine, surrounding soft tissue, or nerve root), and classification (e.g., extradural, intradural-extramedullary, or intramedullary) are similar to neoplasms of the cervical, thoracic, and lumbar spine (see pp. 1396 and 1429). Regardless of tumor type or location, once the encroaching mass becomes large enough to cause cauda equina compression and ischemia, any of the neurologic signs associated with DLSS or fracture/luxation may occur.

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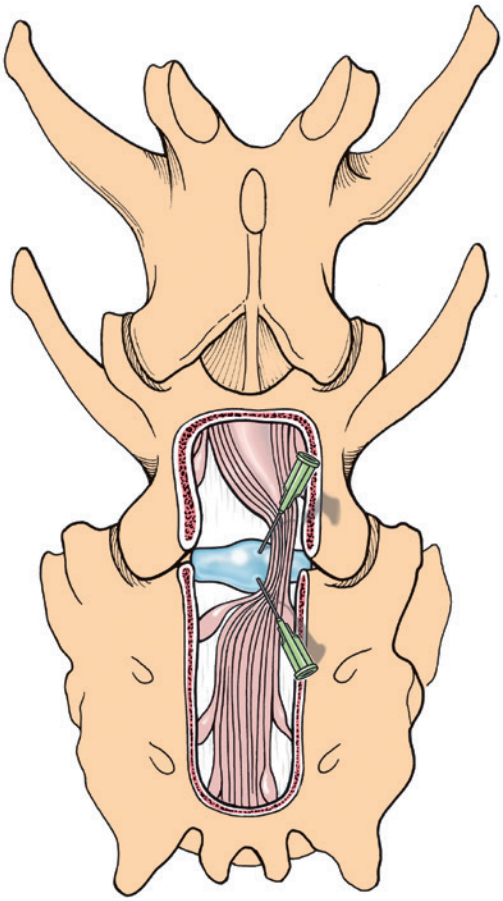


FIG. 42.9 Retract nerve roots laterally with care to facilitate excision of hypertrophied dorsal annulus fibrosus. Use needles placed into the disc to maintain nerve root retraction.

DIAGNOSIS

Clinical Presentation

Signalment

There is no sex or breed predilection for patients with tumors of the lumbosacral area or cauda equina. Generally, patients with spinal tumors are older than 5 years of age. An exception is solitary or multiple cartilaginous exostoses, which usually occur in patients less than 1 year of age. Another exception is lymphosarcoma, which often affects young animals.

History

History depends on the specific location of the neoplasm as well as both the degree and time course of cauda equina compression. Patients may present with variable degrees of back pain, with or without LMN ambulatory paraparesis. Patients with nerve root tumors of the cauda equina usually have a chronic history of pelvic limb lameness (i.e., monoparesis from root signature). As the tumor enlarges and compresses more of the cauda equina, paraparesis becomes evident. Patients with vertebral body or surrounding soft tissue (extradural) tumors may have an acute history of back pain and ambulatory or nonambulatory paraparesis. Careful historical examination may reveal previous (e.g., weeks to months) subtle back pain or pelvic limb lameness.



FIG. 42.10 Lateral radiograph of a dog with L7–S1 intervertebral foramen lysis *(arrow)* suggestive of nerve root tumor.

Physical Examination Findings

Physical and neurologic examination findings vary depending on anatomic location of the tumor (e.g., spine, paraspinal soft tissue, nerve root), degree of nerve root compression/ischemia, specific nerve roots involved, and associated secondary effects (e.g., paraneoplastic syndrome). Generally, patients present with any combination of LMN signs associated with cauda equina compression.

Diagnostic Imaging

Radiographic findings suggestive of vertebral neoplasia include vertebral body osteolysis and/or osteoproduction. These findings are generally associated with primary bone tumors (e.g., osteosarcoma, chondrosarcoma, fibrosarcoma). Lysis of an intervertebral foramen suggests a nerve root tumor (e.g., neurofibroma, meningioma) (Fig. 42.10). Diagnosis of neoplasia affecting the lumbosacral area and cauda equina is best accomplished using MRI (Fig. 42.11) or CT.

Laboratory Findings

Laboratory findings are generally normal or reflect paraneoplastic syndromes (e.g., hypercalcemia, monoclonal gammopathy). Patients with severe back pain and those treated with corticosteroids may have a stress leukogram.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for cauda equina dysfunction include DLSS; fracture/luxation; discospondylitis; vertebral osteomyelitis; fibrocartilaginous emboli; neoplasia of the spine, surrounding soft tissue, or nerve roots; chronic degenerative disc disease; herniated intervertebral disc extrusion; or congenital lumbosacral stenosis. Hip dysplasia can cause many signs similar to cauda equina disease, but this can be ruled out as a sole explanation for the patient's clinical signs with a neurologic examination.

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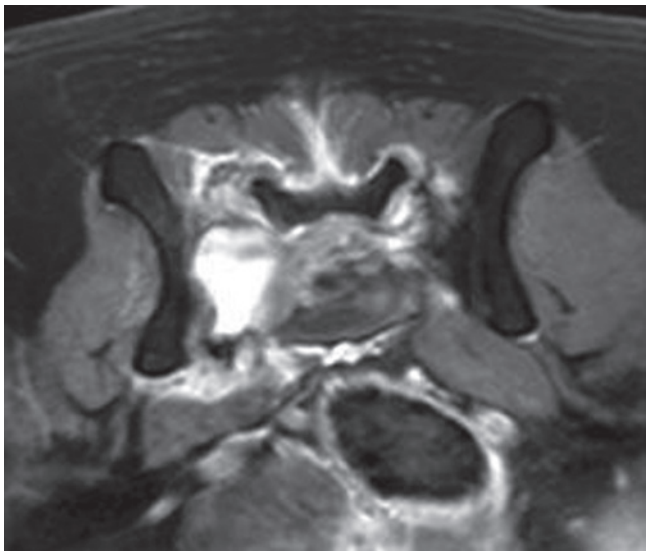


FIG. 42.11 Axial magnetic resonance image (T1 weighted with contrast) of the lumbosacral region of a dog. A large mass (chondrosarcoma) invading the vertebral canal is evident.

MEDICAL MANAGEMENT

Medical management is directed at both the primary lesion and secondary effects of the tumor. Glucocorticoid therapy is generally not recommended for primary treatment of nerve root injury. With most lumbosacral neoplasms, definitive medical treatment requires surgical exposure and incisional or excisional biopsy to determine tumor type and to plan appropriate adjunct chemotherapy, immunotherapy, irradiation, or combination therapy.

SURGICAL TREATMENT

Surgical treatment objectives include the definitive diagnosis of cauda equina compression (e.g., neoplasia, herniated disc, abscess); decompression and mass removal; incisional or excisional biopsy (wide surgical excision is preferred if possible); tumor staging, and, if necessary, spinal stabilization. If wide surgical excision creates spinal instability, stabilization is performed as described on p. 1438. Definitive histologic diagnosis is used to plan adjunct chemotherapy, radiation, or combination therapy.

Preoperative Management

Patients are given IV fluids before surgery. Preoperative management of patients with lumbosacral neoplasia is similar to that described for patients with any lumbosacral compressive lesion (see p. 1428). Preoperative steroids are not indicated because they do not protect nerve roots. If lumbosacral instability is suspected, care should be taken to support the pelvis during manipulations.

Anesthesia

Suggested anesthetic regimen for use in patients with spinal disorders are discussed on p. 1366.

Surgical Anatomy

The pertinent surgical anatomy of the lumbosacral area and cauda equina is discussed on pp. 1408 and 1428.

Positioning

Exposing neoplasms of the lumbosacral region generally requires a dorsal approach and wide dorsal laminectomy with unilateral or bilateral facetectomy and foraminotomy. If stabilization is needed, a dorsal approach is also recommended. Therefore position the patient in sternal recumbency with the hind legs tucked under the abdomen to gently flex the lumbosacral junction (see Fig. 42.3). This position facilitates exposure of the dorsal interarcuate space.

SURGICAL TECHNIQUE

Malignant Nerve Sheath Tumors

Tumors involving nerve roots of the cauda equina as they course through the lumbosacral vertebral canal are best approached using dorsal laminectomy, facetectomy, and foraminotomy as described on pp. 1428 to 1430. It is important to identify the specific nerve root or roots to be resected, and to have knowledge of the effect of resection on the patient's neurologic function. Neurologic deficits created by single or multiple nerve root resection may incapacitate the patient to an unacceptable degree. In such cases, biopsy for histopathologic diagnosis and adjunct medical management may be indicated.

NOTE If possible, excise the tumor so as to include 2 cm of adjacent, normal-appearing tissue.

Enter the vertebral canal by carefully elevating the inner periosteal layer with a dental or iris spatula and ophthalmic forceps. Remove any remaining epidural fat. Identify cauda equina nerve roots on the floor of the vertebral canal. Use a probe (e.g., Gross ear hook and spoon) to carefully follow the S1 and L7 nerve roots as they course along the floor of the vertebral canal. Identify the L7 nerve root as it lies against the caudal aspect of the L7 pedicle, and follow it as it disappears through the L7–S1 intervertebral foramen. Carefully examine each nerve root for evidence of diffuse enlargement or localized bulging. If the lesion is resectable, identify the nerve root involved, carefully cauterize the root cranial to the lesion using bipolar cautery, transect the root with a No. 11 scalpel blade, and carefully elevate the transected portion of the root. Follow the affected nerve root caudal to the lesion and complete the resection as described previously. Reexamine the cauda equina to rule out multiple nerve root tumors. Lavage the surgical wound and close tissues routinely.

Tumors Involving the Vertebral Body or Surrounding Soft Tissues

Tumors involving the vertebral body or surrounding soft tissues are best approached via dorsal laminectomy as described on p. 1429. *Perform a dorsal laminectomy to remove affected bone and provide cauda equina decompression. If bony involvement is extensive and nonresectable, use a trephine or bone curette to biopsy affected bone and perform a dorsal laminectomy to provide cauda equina decompression. If surrounding soft tissue structures are involved, biopsy representative areas. If neoplastic tissue is cavitated or abscessed, obtain samples for anaerobic and aerobic culture and susceptibility testing. If spinal instability is diagnosed or occurs as a result of decompression, perform spinal stabilization as described on p. 1438.*

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POSTOPERATIVE CARE AND ASSESSMENT

Patients with neoplasia of the lumbosacral spine should be monitored postoperatively in a similar fashion as patients with other lumbosacral spinal disorders. In general, strict confinement, analgesics as needed, short walks using an abdominal sling, frequent urinary bladder evacuation, and daily neurologic examinations are indicated. Long-term monitoring and adjunct medical therapy are dependent on tumor type and surgical margins.

PROGNOSIS

Prognosis depends on tumor type (biologic activity), surgical margins (percent of tumor resection), and sensitivity to adjunct therapy (chemotherapy, radiation therapy, or combination therapy). Malignant extradural neoplasms tend to have an unfavorable prognosis, benign extradural neoplasms have a favorable prognosis, and extradural-intramedullary neoplasms (e.g., mixed compartment tumors such as malignant nerve sheath tumors) have a guarded to unfavorable prognosis.

TRAUMA

DEFINITIONS

Traumatic or pathologic disruption of osseous and supporting soft-tissue structures of the caudal lumbar, sacral, and first caudal vertebrae (e.g., L6–Cd1 through L6–Cd5) may result in vertebral fracture or luxation and subsequent nerve root compression (i.e., cauda equina). Fractures of the lumbosacral spine are also called a *broken back, caudal lumbar fractures,* and *vertebral fractures.*

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Fractures and luxations of the caudal lumbar vertebra, lumbosacral articulation, sacrum, and caudal vertebrae generally result from direct flexional trauma to the hindquarters. Fractures are usually oblique or short oblique involving the vertebral body of L6 or L7 and may be accompanied by luxation of articular facets. Cranioventral displacement of the caudal segment typically occurs because of muscular forces acting on the sacrum and pelvis, along with the weight of the pelvic mass (Fig. 42.12). Lumbosacral fractures are fairly common due to the static-kinetic relationship of the relatively fixed sacrum to the mobile caudal lumbar and caudal vertebral bodies. The most common cause of fracture/luxation is vehicular trauma. The spinal cord typically terminates in the body of L6 in dogs; therefore neurologic signs are usually associated with trauma to nerve roots of the cauda equina instead of the spinal cord. In cats, the spinal cord often terminates over the body of S1. Because of the ability of nerve roots to resist traumatic injury, substantial displacement of the fracture or luxation may still leave the patient neurologically intact. Conversely, fractures or luxations may occur in which severe displacement of the fracture/luxation segments causes tethering or avulsion of nerve roots and will occasionally produce trauma to the caudal spinal cord; neurologic deficits in these patients are often profound. As mentioned previously in this chapter, stabilization of the lumbosacral junction may be warranted in some cases of DLSS as an adjunct to dorsal laminectomy with or without discectomy. The procedures discussed in this section for trauma to the lumbosacral region can be adapted to cases of DLSS.

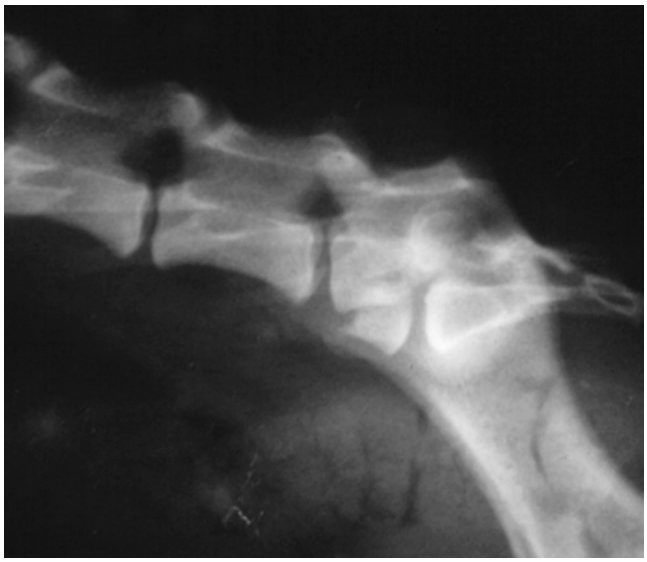


FIG. 42.12 Lateral radiograph of a dog with an L7 fracture. Notice the cranioventral displacement of the sacrum, pelvis, and L7 fracture fragment, which is typical with these fractures.

DIAGNOSIS

Clinical Presentation

Signalment

There is no specific age, sex, or breed predilection for canine or feline lumbosacral fracture/luxation. However, dogs are more likely to sustain this injury than cats, and there is a trend of dogs less than 3 years of age being affected more often.

History

The majority of dogs and cats with lumbosacral fracture/luxation have a history of recent vehicular trauma. Patients generally present with varying degrees of lumbar pain, ambulatory or nonambulatory paraparesis, and decreased anal and tail tone. Cats will occasionally present with fractures/luxations of the sacrococcygeal region resulting from suspected traction injury. It is theorized that car tires trap the tails of these cats as the cats are running to avoid being hit. The resultant traction applied to the cauda equina may result in temporary or permanent neurologic damage. These cats often display varying degrees of urinary and fecal incontinence, in addition to tail dysfunction. Some cats also exhibit pelvic limb dysfunction.

Physical Examination Findings

Severe traumatic injury often produces additional injuries (e.g., cardiopulmonary, urinary, diaphragmatic hernia, appendicular skeleton, a second vertebral fracture/luxation). A thorough physical examination of each system to identify concurrent injury is necessary. Patients that struggle during physical examination or those with profound neurologic deficits should be secured to a rigid platform to prevent further nerve root trauma until a definitive course of action can be taken.

Lumbosacral fracture/luxation may cause trauma and sustained compression to the cauda equina. The most commonly involved

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nerve roots are L6, L7, S1–S3, and Cd1–Cd5. Major nerves associated with these nerve roots include the sciatic nerve (L6, L7, S1, and/or S2), pelvic nerve (S1–S3), perineal and caudal rectal branches of the pudendal nerve (S1–S3), and caudal (coccygeal) nerves of the tail (Cd1–Cd5). Because of the regional anatomy innervated by the cauda equina, ischemia and/or compression may produce a wide variety of neurologic signs. Although a variety of neurologic deficits may occur, the most common presentation is lumbosacral hyperesthesia (pain on palpation of the lumbosacral junction), LMN ambulatory or nonambulatory paraparesis, varying degrees of anal sphincter hypotonia/atonia, and abnormalities in tail carriage and sensation.

Diagnostic Imaging

Neurologic examination localizes the lesion to the cauda equina. Survey radiographs are usually diagnostic and generally reveal an oblique or short oblique fracture through the vertebral body of L6 or L7, or a luxation or subluxation between L6 and L7 or L7 and S1. Although radiographs are a useful screening tool for trauma, we prefer CT imaging of lumbosacral trauma patients, as this modality is more sensitive than radiographs and allows for presurgical measurements for implant placement. MRI is also often used in lumbosacral trauma cases and has the advantage of delineating soft tissue structures more accurately than CT. The caudal fracture/luxation segment is characteristically displaced cranioventral to the cranial segment (see Fig. 42.12). Because of the relatively mild neurologic signs that may be seen with marked fracture or luxation displacement on survey radiographs, prognosis is based on neurologic examination findings, not radiologic findings. Moreover, as LMN signs predominate in patients with multiple spinal fractures, neurologic examination may not identify a second thoracolumbar fracture or luxation. A complete series of spinal radiographs or a whole body CT scan is always recommended in patients with spinal fracture or luxation.

NOTE Up to 20% of patients with spinal fracture or luxation have a second spinal fracture or luxation.

Laboratory Findings

Patients presenting with lumbosacral fracture/luxation secondary to severe trauma often have a stress leukogram and elevated hepatic enzymes. If patients sustain concurrent injuries resulting in uroabdomen, ruptured spleen, or pulmonary contusion, associated laboratory abnormalities may occur. Laboratory findings are usually nonspecific.

DIFFERENTIAL DIAGNOSIS

Fracture/luxation of the lumbosacral spine must be differentiated from other disorders causing cauda equina compression, including DLSS, discospondylitis, chronic degenerative disc disease, intervertebral disc extrusion, fibrocartilaginous embolism, and neoplasia of the spine, surrounding soft tissue, and nerve roots. Definitive diagnosis is made by analyzing history, neurologic localization, and imaging results.

MEDICAL MANAGEMENT

Objectives of medical management include immobilization of the spine and patient confinement until fracture/luxation stabilization and union occur. The following conditions are recommended: strict confinement for 4 to 6 weeks in a dry, elevated padded cage; analgesics/NSAIDs as needed to control back pain (do not use steroids and NSAIDs concurrently or in close temporal proximity to each other); urinary bladder expression or intermittent catheterization four times daily; passive physical therapy of the hind legs two to three times daily; and serial neurologic examinations twice daily to assess patient response to therapy.

SURGICAL TREATMENT

Objectives of surgical management are decompression of the cauda equina (fracture/luxation reduction) followed by adequate spinal stabilization. Infrequently, patients may require dorsal laminectomy for complete decompression (when bone fragments are present in the vertebral canal and/or intervertebral foramen) or for its prognostic value in evaluating the severity of cauda equina damage. The process of surgical reduction and stabilization provides immediate relief of back pain, allows freedom of movement, decreases patient morbidity, and protects spinal roots from further trauma. Surgical versus medical treatment is based on the patient's neurologic status at presentation, response to medical management, and serial neurologic examinations. There are numerous fixation methods for the lumbosacral region, some of which are described in this chapter. Adequate stabilization of the lumbosacral junction can be provided by a number of techniques, including (1) transilial pins with Kirschner clamps, bent pins, or bone cement; (2) transilial pins and plastic dorsal spinous process plates; (3) transilial pin, dorsal spinous process plates, and external skeletal fixation; (4) modified segmental spinal fixation; (5) vertebral body and/or pedicle Steinmann pins/screws embedded in polymethylmethacrylate (PMMA) bone cement; and (6) dorsal locking plates (string of pearls).[10,11] A pedicle screw-rod fixation method has been described for stabilizing the L7–S1 articulation in dogs with DLSS; this procedure could also be applied to trauma cases.[12] Transarticular cortical screw placement across the L7–S1 articular processes (facets) can also be used for stabilization but has been shown to have a fairly high incidence of implant failure in one study.[13] We recommend that these screws, if used, should not be the sole method of stabilization. Choice of technique is generally dictated by the surgeon's experience and equipment available.

Preoperative Management

Preoperative management of patients with cauda equina compression is similar to that described for any lumbosacral compressive lesion (see p. 1428). If concurrent spinal cord injury is not present, preoperative steroids are not recommended because they do not protect nerve roots. Lumbosacral instability predisposes the patient to further trauma, and care should be taken to support the patient's pelvis during preoperative manipulations.

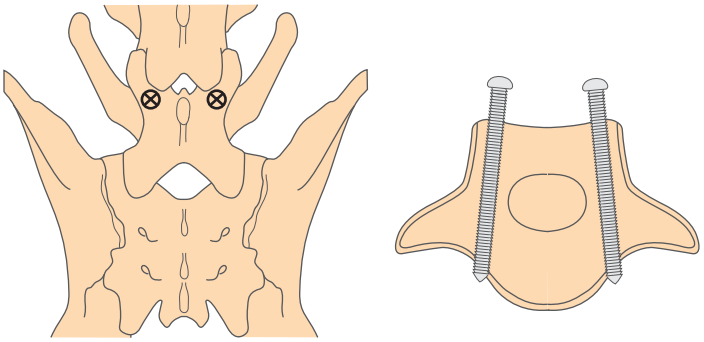
Anesthesia

Suggested anesthetic regimens for use in patients with spinal disorders are discussed on p. 1366.

Surgical Anatomy

Pertinent surgical anatomy of the lumbosacral area and cauda equina is discussed on pp. 1404 and 1428. For purposes of implant placement, the surgeon should also be familiar with implant entry points for pedicle pins/screws (Fig. 42.13).

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A B

FIG. 42.13 (A) Approximate dorsal implantation site and (B) cross-sectional view of the appropriate angle of implant insertion for caudal lumbar pedicle pins/screws.

Positioning

The surgical approach for each stabilization technique requires dorsal exposure. Positioning the patient in sternal recumbency with the hind legs tucked under the abdomen encourages flexion of the dorsal lumbosacral region and facilitates fracture reduction and cauda equina decompression (see Fig. 42.3).

SURGICAL TECHNIQUES

Transilial Pins

This procedure is limited to L7–S1 luxations and L7 fractures in which the dorsal lamina is intact. *Expose dorsal spinous processes and lamina of L6, L7, and the entire median sacral crest as described for dorsal laminectomy on p. 1429.* Because of the cranioventral displacement of the sacrum, visualization of the cranial aspect of the sacral crest is obscured by the lamina of L7. L7 laminectomy is not compatible with this technique. *Use a periosteal elevator or small osteotome to elevate epaxial muscles and expose the articular processes of L7. Use the tip of a Kelly or Carmalt forceps or a Senn retractor and carefully place it in the lumbosacral junction. Visually monitor the depth of placement of the forceps or retractor to avoid injuring nerve roots. Hook the tip of the forceps or retractor under the cranial lamina of the sacrum to act as a lever against the caudal lamina of L7 (Fig. 42.14).* Depending on patient size, a Senn retractor can also be used under the cranial lamina of S1 to lever the sacrum back into alignment with L7. Reducing the fracture or luxation facilitates adequate exposure of the lumbosacral junction. *Before attempting reduction, place a bone forceps (e.g., speed-lock reduction forceps) or towel clamp in the wing of each ilium or in the spine of L6 or L7 to provide countertraction.* These manipulations force the sacrum caudodorsally, to reduce the fracture/luxation. *Visualize the lumbosacral articular processes (facets); when the articular processes of L7 and S1 are reapposed, reduction is anatomic. Place a 0.062-inch (1.575 mm) Kirschner wire through each L7–S1 articular facet to maintain fracture or luxation reduction. Next, incise gluteal fascia on the dorsolateral crest of the wings of each ilium, elevate the middle gluteal musculature, and expose the dorsolateral aspect of each ilial crest. Place an appropriately sized Steinmann pin through the lateral aspect of the ilial wing, across the dorsal lamina of L7, and through the opposite ilial wing. Place a second pin in a similar fashion starting from the opposite side (Fig. 42.15A). Be sure both pins cross over the dorsal lamina of L7. Prevent pin migration by employing one of the following techniques: (1) bend the ends of each pin at a 90-degree angle (Fig. 42.13A); (2) connect the pins on each side with double Kirschner clamps of the appropriate size (Fig. 42.15B); or (3) notch the pins' ends with a pin cutter and incorporate them with PMMA bone cement (Fig. 42.15C). If indicated, remove the interarcuate ligament and perform sacral laminectomy to visualize nerve roots. Lavage the surgical wound with sterile physiologic saline solution, cover the ends of the pins by closing gluteal fascia, and close epaxial muscles by apposing dorsal midline fascia. Close subcutaneous tissue and skin routinely.* This procedure can be combined with dorsal spinous process plating (Fig. 42.16) for additional stability or if L6 is also fractured or luxated.

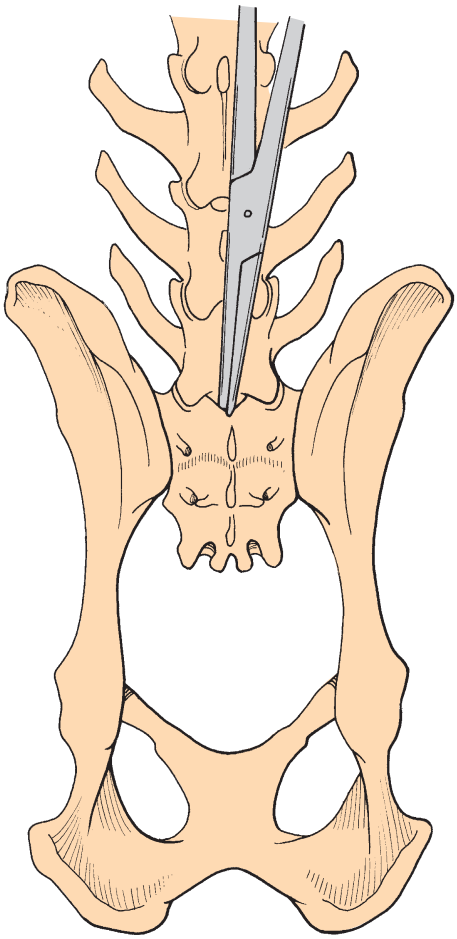


FIG. 42.14 Use of Kelly or Carmalt forceps to aid reduction of an L7 fracture. Hook the tip of the forceps under the cranial lamina of the sacrum and lower the jaws against the caudal lamina of L7.

Modified Segmental Spinal Fixation

Modified segmental spinal fixation can be used in patients of all sizes. It does not require deep exposure, and it is compatible with dorsal laminectomy, relatively straightforward to perform, versatile (it can be used in combination with other techniques), and results in a stable repair. Modified segmental spinal fixation is also described for thoracolumbar spinal injury on p. 1418. *Approach the dorsal aspect of the lumbar and lumbosacral spine as described earlier for transilial pins and dorsal spinous process plates. Expose three dorsal spinous processes cranial to the fracture or luxation.*

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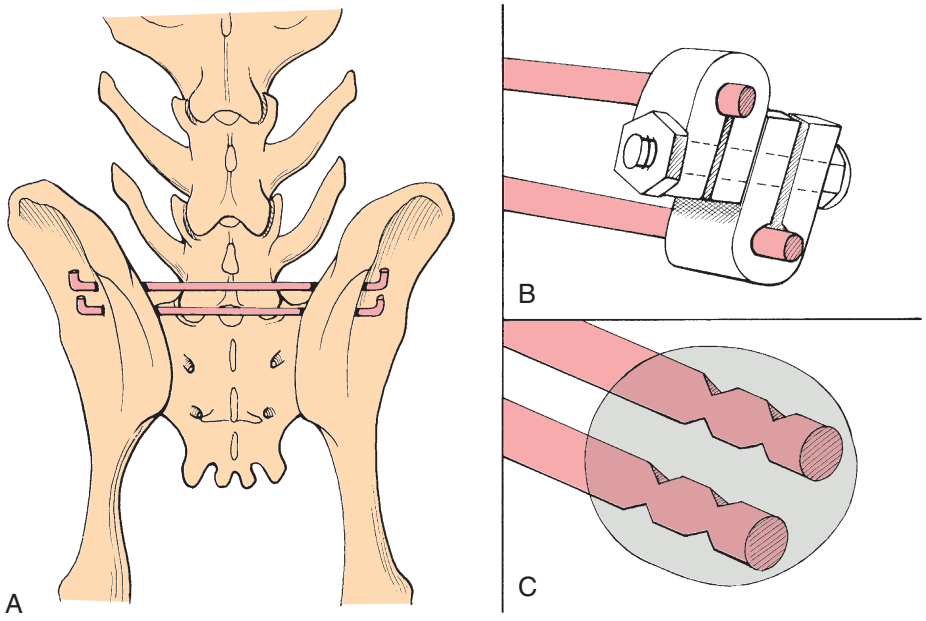


FIG. 42.15 Prevent migration of transilial pins by (A) bending the ends of each pin at a 90-degree angle, (B) connecting the pins on each side with a double Kirschner clamp, or (C) notching the pin ends with a pin cutter and incorporating them with bone cement.

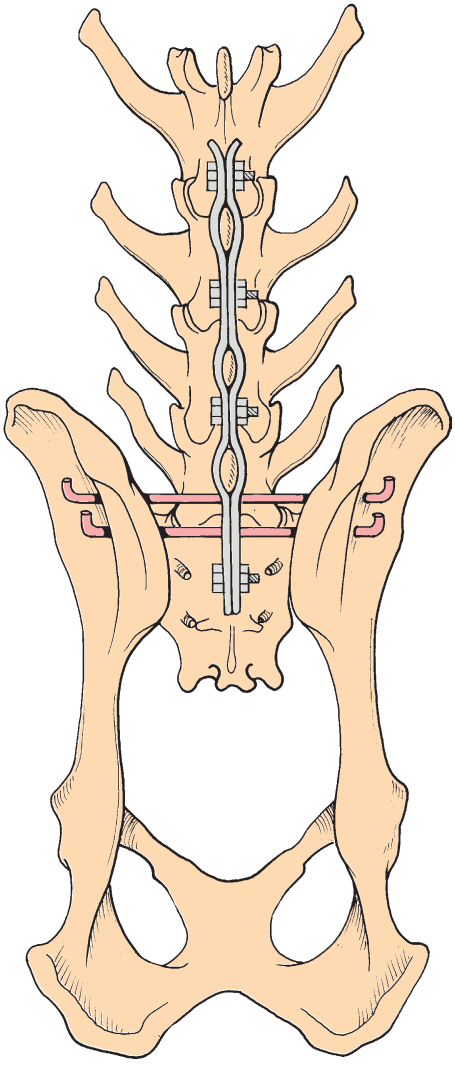


FIG. 42.16 Transilial pins and plastic dorsal spinous process plates to repair L7–S1 fractures or luxations.

Expose, carefully reduce, and maintain fracture or luxation reduction as described for transilial pinning on p. 1439. Perform a dorsal laminectomy at this time, if indicated (see p. 1429). Drill holes in the caudal articular processes and bases of dorsal spinous processes of at least two to three vertebrae cranial to the fracture or luxation. Drill holes in the cranial sacral articular facets to ensure a minimum of two points of fixation per pin in the sacroiliac segment. Preplace 3- to 4-inch (7–16 cm) lengths of 18- to 20-gauge stainless steel wire through each hole. Drill two holes transversely through each ilial wing at the level of the dorsal lamina of the sacrum. Bend four appropriate size and length Steinmann pins at a 90-degree angle, remove the points, and pass the pins through the holes drilled in the wings of the ilia. Place the pins alongside the lamina and attach them to the articular facets and dorsal spinous processes using the preplaced stainless steel wires (Fig. 42.17).

Steinmann Pins/Screws and Polymethylmethacrylate

Steinmann pins and/or screws and PMMA bone cement can be used in patients of all sizes. These constructs can also be modified by adding support rods (connecting the vertebral pins and/or screws) prior to PMMA placement (Fig. 42.18). This technique requires exposure of the dorsal surface of the transverse processes of L6 and L7 and is compatible with dorsal laminectomy, is versatile (it can be used in combination with other techniques), requires constant reference to an anatomic specimen (e.g., spine), and provides a stable repair. In the caudal lumbar vertebrae, pins/screws can be placed primarily in the vertebral bodies by directing the implants between the accessory process and transverse process (see p. 1423), or primarily in the vertebral pedicle, starting from a dorsal entry point just caudal to the cranial articular facet (roughly the middle of the facet) and directing the implant slightly laterally (see Fig. 42.13). This latter method of vertebral implant placement is often chosen for the caudal lumbar vertebrae (L6, L7) as the angle for more direct vertebral body implant placement can be difficult to achieve due to interference by the ilial wing. When placing implants primarily into the pedicle of caudal lumbar vertebrae, create a small entry point in the cortical bone with a small burr, prior to implant

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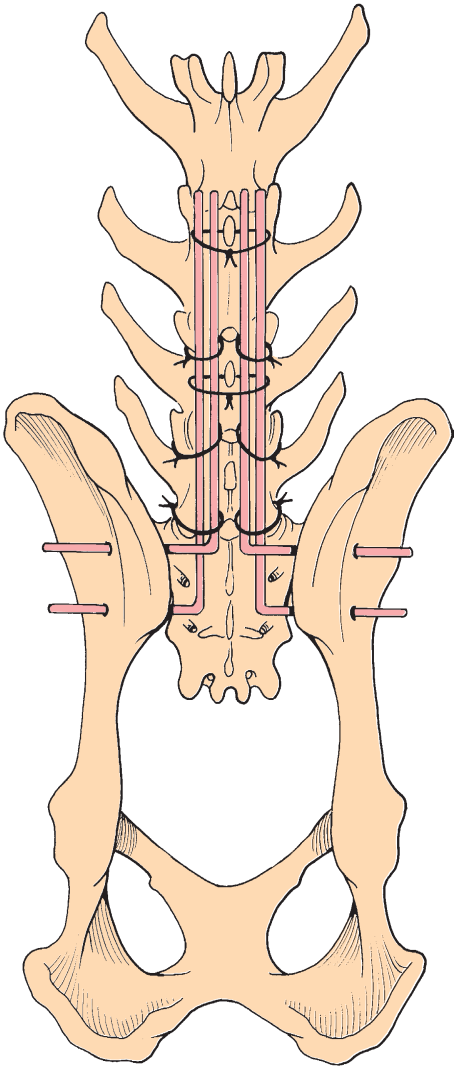


FIG. 42.17 Diagram showing proper pin placement for modified segmental spinal fixation of L6, L7, and S1 fractures or luxations.

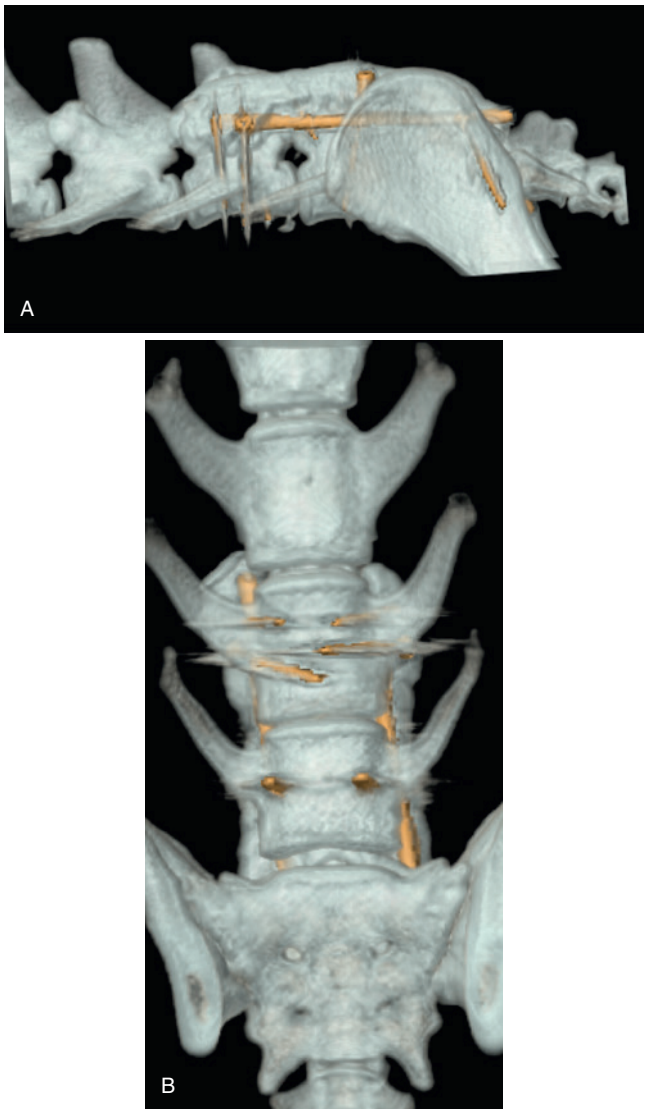


FIG. 42.18 (A) Lateral and (B) ventral views of postoperative three-dimensional computed tomography reconstructed images of a dog with a traumatic L6–L7 luxation repaired with screws, rods, and polymethylmethacrylate.

placement or predrilling (in case of screw placement). Otherwise, the implant or drill bit is likely to constantly slip off this curved area of bone. *Adequately expose the vertebrae cranial and caudal to the fracture or luxation. Incise muscle attachments to articular facets and periosteally elevate epaxial muscles until the dorsal aspect of the transverse processes of L6 and L7 and dorsal lamina of the sacrum are identified. Reduce the fracture/luxation and maintain reduction as described for transilial pinning on p. 1439. If the fracture involves the body of L6, place two pins/screws (one on each side) in the pedicle/body of L5 and two pins/screws in the pedicle/body of L7. If an L6–L7 luxation is being repaired, place two pins/screws in the pedicle/body of L6 and two pins/screws in the pedicle/body of L7. Place pins/screws and apply bone cement using the technique described on p. 1439. If the fracture involves the vertebral body of L7 or is an L7–S1 luxation, place two pins/screws in the pedicle/body of L6, two pins/screws in the pedicle/body of L7, and two pins/screws in the cranial articular process of S1 with penetration through the ilium. If the fracture involves the vertebral body of L7, pin/screw placement in L7 is dictated by the type of fracture present (e.g., avulsion fracture of the end-plate, place two pins/screws in L7; transverse fracture, place one or two smaller pins/screws, depending on the size of the fracture fragments; comminuted fracture, no pins should be placed in L7). Insert pins/screws directly into the pedicles/vertebral bodies of L6 or L7 using the accessory process and transverse process as landmarks. Insert pins/screws into the center of the cranial articular processes of S1. Drive the pin/screw until it penetrates the gluteal surface of the wing of the ilium. Direct the L6 and L7 pins/screws cranioventral and from lateral to medial, and the S1 pins/screws caudoventral and from lateral to medial. Drive pins/screws to exit 2 to 3 mm from the ventral aspect of the vertebral bodies and wings of the ilia. If pins are used (vs. screws), cut pins to leave 2 cm protruding and notch the exposed pinheads with a pin cutter. Thoroughly lavage and dry the surgical field (Fig. 42.19). Apply PMMA bone cement as described on p. 1439. If necessary, excise portions of epaxial muscles adjacent to the PMMA to facilitate closure. Close subcutaneous tissues and skin routinely.*

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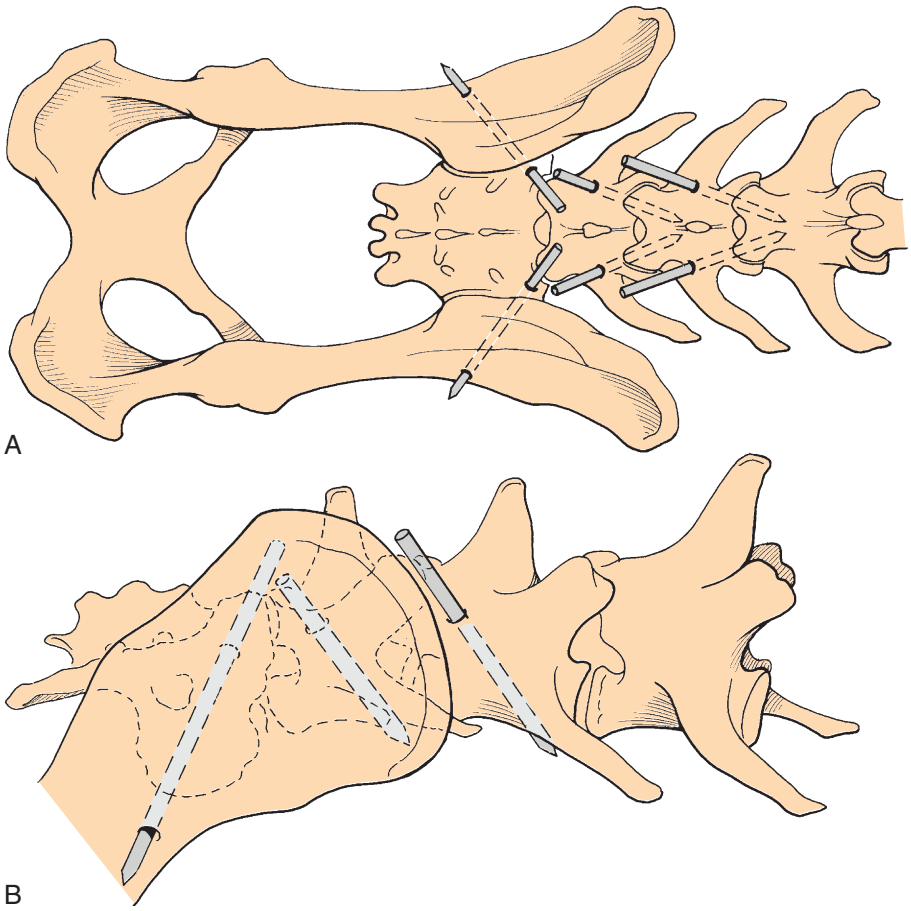


FIG. 42.19 (A) Dorsal and (B) lateral views showing proper pin placement in the iliac wings, articular facets of S1, and vertebral bodies of L6 and L7 when using Steinmann pins and polymethylmethacrylate to repair L6, L7, and S1 fractures or luxations.

SUTURE MATERIALS/SPECIAL INSTRUMENTS

Depending on the technique chosen, the following special instruments will be needed: plastic dorsal spinous process plates, bone cement, Steinmann pins, cortical screws, string-of-pearls plate set, skeleton, and Kirschner-Ehmer clamps and connecting bars.

POSTOPERATIVE CARE AND ASSESSMENT

Patients with lumbosacral fracture/luxation should be monitored postoperatively, as are patients with other lumbosacral disorders (i.e., strict confinement, analgesics as needed, short walks using an abdominal sling, frequent urinary bladder evacuation, and daily neurologic examinations). Long-term assessment consists of neurologic examinations and awake or sedated spinal radiographs at 1, 2, 3, 6, 9, and 12 months after surgery or when the fracture or luxation resolves.

PROGNOSIS

Prognosis depends on severity of cauda equina compression (i.e., on neurologic examination at presentation) and the treatment regimen chosen. Cauda equina nerve roots withstand considerably more trauma than the spinal cord. Patients with 100% lumbosacral vertebral canal compromise may retain neurologic function of the pelvic limbs, anus, urinary bladder, perineum, and tail; their prognosis is favorable. In general, the preservation of pain sensation (nociception) in the areas of innervation of the cauda equina nerve roots is a favorable prognostic indicator. However, patients with profound neurologic deficits (e.g., complete loss of motor function and deep pain perception in the sciatic nerve distribution, lack of anal tone and bladder tone with no perineal sensation) should be given a guarded prognosis, regardless of the percentage of vertebral canal compromise. As the severity of presenting neurologic deficits increases, early decompression and stabilization allow a more favorable prognosis. If neurologic deterioration during medical management is determined early, immediate surgical intervention produces a more favorable outcome. Cats with tail traction injuries often make full recoveries, especially if anal tone and perineal sensation are intact, and appropriate bladder management is instituted soon after injury. Failure to regain urinary continence within 1 month of trauma in these cats is a negative prognostic indicator. Lateralized sacral fractures in dogs tend to have a better prognosis for neurologic recovery than those located in a more central location.

NOTE The amount of vertebral canal compromise observed on lateral radiographs should not be used as a prognostic indicator.

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Nonsurgical Disorders of the Brain and Spine



There are a number of nonsurgical disorders of the brain and spine that neurosurgeons need to be familiar with because (1) these disorders can mimic surgical disorders and are often presented as such for diagnosis and possible surgery, and (2) some of these disorders require imminent diagnosis and treatment for a positive outcome. If presented with such patients, it is incumbent on the neurosurgeon to either be comfortable in diagnosing and treating these animals or to quickly recognize the nature of the disease process and arrange for care with the appropriate specialist. In some cases, a biopsy will be required for diagnosis. Although currently done infrequently, the use of stereotactic brain biopsy equipment that interfaces with both magnetic resonance imaging (MRI) and computed tomography (CT) is increasingly being used in veterinary neurosurgery. The distinction between surgical and nonsurgical disease of the brain and spine is not always clear; discospondylitis is usually considered to be solely a medical disorder; however, some patients will develop compressive lesions (e.g., infected epidural fat) and/or vertebral instability, either of which may require surgical intervention.

SPECIFIC DISEASES

CANINE COGNITIVE DYSFUNCTION/COGNITIVE DYSFUNCTION SYNDROME

DEFINITIONS

Canine cognitive dysfunction (CCD) is the canine analog of human Alzheimer disease (AD). This disorder is also referred to as **cognitive dysfunction syndrome (CDS). Beta amyloid (Aβ)** is a neurotoxic protein that accumulates in the brains of dogs and cats with cognitive dysfunction, and humans with AD, that forms plaques within the brain parenchyma. Accumulation of Aβ also leads to cerebrovascular disease, referred to as **cerebrovascular angiopathy. Tau protein** is another protein that accumulates in the brain of aging pets and humans with cognitive dysfunction.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

CCD is an age-related disorder, similar to AD in people, that occurs in elderly dogs; an analogous disorder also occurs in cats. Age-related cognitive dysfunction is best described for the dog, and this species appears to be the best animal model available for human AD. Similar to AD in people, the pathophysiology of CCD is uncertain. There are pathologic similarities between the brains of humans with AD and dogs and cats with age-related cognitive impairment. Cerebral vascular changes, meningeal thickening, gliosis, and ventricular dilatation occur in brains of both AD and CCD patients. More specifically, progressive accumulation of a neurotoxic protein called Aβ in the brain (in and around neurons) is a consistent feature in both AD and CCD. These accumulations coalesce to form plaques (neuritic plaques) and are most prominent in the frontal cerebral cortex and hippocampus in both human and veterinary disorders. The most common form of insoluble Aβ found in brain tissue is a 42 amino acid form, called $A\beta_{42}$. In addition to this insoluble form of the protein, smaller soluble oligomeric forms of Aβ have been identified; these are highly toxic and interfere with synaptic function.[1] In both human and veterinary disorders, the degree of Aβ accumulation has been correlated with the extent of cognitive impairment. In one histopathologic study of CCD dogs, however, cognitive impairment was associated with increased numbers of astrocytes and microglial cells, as well as elevated ubiquitin protein levels, rather than with levels of Aβ.[2] In addition to the accumulation of neurotoxic Aβ (protein in the aged canine and feline brain), intraneuronal accumulation of a hyperphosphorylated microtubular-associated protein (tau protein) has also been demonstrated. Tau protein is the precursor to neurofibrillary tangles (NFTs), another prominent histopathologic feature of human AD. The absence of mature NFTs in the brains of dogs and cats with age-related cognitive dysfunction has been argued as evidence against the canine and feline disorders as being analogous to human AD. However, the absence of NFTs in dogs and cats has a number of potential explanations. It is possible that dogs and cats do not live long enough for the tau proteins to develop into NFTs as they do in people. Although the amino acid sequence of Aβ protein is identical in humans and dogs, this is not the case for tau protein. The amino acid sequence of dogs and cats differs from that in people; this different sequence may affect the ability of tau protein to form NFTs. Other structural abnormalities found in the aging canine brain that are similar to those in humans include cerebral atrophy, ventricular enlargement, blood vessel wall fibrosis, amyloid deposition (meningeal and parenchymal), microhemorrhages and infarcts, axonal degeneration with myelin loss, astroglial hypertrophy and hyperplasia, and intraneuronal accumulation of several substances (lipofuscin, polyglucosan bodies, and ubiquitin).

The pathophysiology of CCD and AD is multifactorial and complex. There is evidence in both diseases that increased oxygen–free radical mediated cellular damage, decreased endogenous antioxidant defenses, inflammation (from various processes),

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ABSTRACT

Numerous nonsurgical conditions are likely to be regularly encountered by surgeons who perform brain and spinal surgery (e.g., cognitive dysfunction, ischemic brain and spinal cord problems, inflammatory brain disease, degenerative myelopathy, steroid-responsive meningitis-arteritis, and discospondylitis). Whether or not the attending surgeon will remain the primary clinician, it is important to have at least a working knowledge of the diagnostic and therapeutic options available. In some instances (e.g., inflammatory disorders), the surgical clinician may need to initiate treatment prior to seeking advice from another specialist if the patient is going to have a positive outcome.

KEYWORDS

Nonsurgical
cognitive dysfunction
dementia
degenerative myelopathy
fibrocartilaginous embolic myelopathy
infarct
inflammatory

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decreased mitochondrial function, DNA damage, vascular compromise, and neurotransmitter imbalance are all interrelated processes that are involved in progressive cognitive impairment. Neurochemical changes that occur in the aging brain are thought to contribute to progressive cognitive impairment. An age-associated decline in the brain neurotransmitter levels of acetylcholine, dopamine, norepinephrine, and *gamma-aminobutyric acid* (GABA) has been documented in CCD and AD. Of these abnormalities, cholinergic dysfunction appears to have the highest and most consistent correlation with age-related cognitive impairment. Other neurochemical abnormalities identified in brains of CCD and AD patients include increased acetylcholinesterase levels (associated with cholinergic decline), increased monoamine oxidase B (catalyzes the breakdown of dopamine, with subsequent formation of free radicals), and elevated cerebrospinal fluid (CSF) levels of lactate, pyruvate, and potassium.

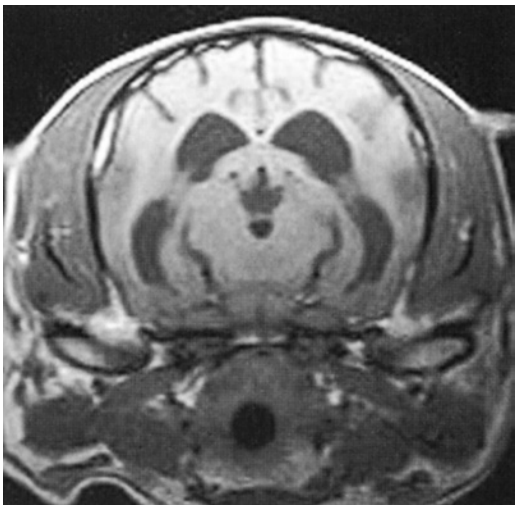


FIG. 43.1 Axial T1-weighted magnetic resonance image of a dog with cognitive dysfunction syndrome, demonstrating typical characteristics of brain aging.

DIAGNOSIS

Clinical Presentation

Signalment

CCD occurs primarily in older dogs, usually greater than 9 years of age. Cats are less commonly affected by CDS and are typically older than 12 years of age at presentation. There is no apparent breed or sex predilection for CDS.

History

Historical complaints regarding CDS are numerous and often nonspecific. They include inattentiveness, inactivity, aimless wandering (often pacing at night), demented behavior, disturbance of the sleep/wake cycle, urinary and/or fecal incontinence, difficulty navigating stairs, becoming lost in previously familiar environments, failure to recognize previously familiar people or animals, decreased interaction with family members, hearing loss, excessive vocalization (often at night), and anxiety. Cats with CDS occasionally exhibit overresponsive and aggressive behavioral patterns. Owners of CDS pets often describe their pets as acting "senile." In one study of dogs with CCD, four key clinical features were identified: (1) anxiety, (2) disturbance of the sleep/wake cycle (sleeping during the day, restless at night), (3) decreased interaction with owners, and (4) apparent confusion or disorientation.[3]

Physical Examination Findings

Dogs and cats with CDS typically display evidence of forebrain dysfunction. These patients have abnormal mentation and often respond inappropriately to their environment (dementia). Many dogs and cats with CDS will circle constantly in the examination room and either not respond or respond inappropriately to visual and auditory stimuli. In addition to the classic behavioral abnormalities indicative of CCD in dogs, suspected CCD dogs will occasionally have either transient central vestibular dysfunction or seizure activity of recent onset. Although not well-described as a clinical feature of CCD, vestibulocerebellar dysfunction and seizures are reported as a potential consequence of AD in people.

Diagnostic Imaging

The only imaging modality of use for the diagnosis of CCD is MRI. Brain imaging of AD patients can be normal or may reveal brain atrophy, ventricular enlargement, and lesions in the medial temporal lobes of the cerebral cortex (Fig. 43.1). Age-related changes appreciated on MRI of the brain in CCD patients are primarily reflective of brain atrophy and include ventricular enlargement, widened and well-demarcated cerebral sulci, and diffuse and scattered areas of T2-hyperintensity in periventricular white matter. Although these are consistent findings associated with the aging brain, they may be found in older patients without evidence of CCD. The thickness of the interthalamic adhesion as measured on transaxial T1- and T2-weighted magnetic resonance (MR) images was found to be significantly smaller in dogs with CCD compared with dogs without CCD (Fig. 43.2); an interthalamic adhesion thickness of 5 mm or less was found to be consistent with a diagnosis of CCD.[4]

Laboratory Findings

Laboratory findings are typically normal in dogs and cats with CDS, unless there is an age-related concurrent disorder (e.g., elevated blood urea nitrogen/creatinine from chronic renal disease). It is important to measure fasting/postprandial serum bile acids and/or blood ammonia to distinguish CCD from hepatic encephalopathy. Dogs with CCD should have normal serum bile acids and blood ammonia. Congenital portosystemic shunts are sometimes not diagnosed until the dog is older than 10 years old, and mild hepatic encephalopathy may mimic findings expected in CCD.[5] In one study, plasma concentrations of $A\beta_{42}$ were significantly increased in dogs with CCD, compared with older dogs with either mild cognitive impairment or no cognitive impairment.[6]

DIFFERENTIAL DIAGNOSIS

The main differential diagnoses for CDS are brain tumor and metabolic encephalopathy (e.g., hepatic encephalopathy).

MEDICAL MANAGEMENT

There are numerous proposed therapeutic approaches to CCD, with variable evidence of efficacy in improving cognitive function and/or delaying progression of cognitive decline. The use of oral

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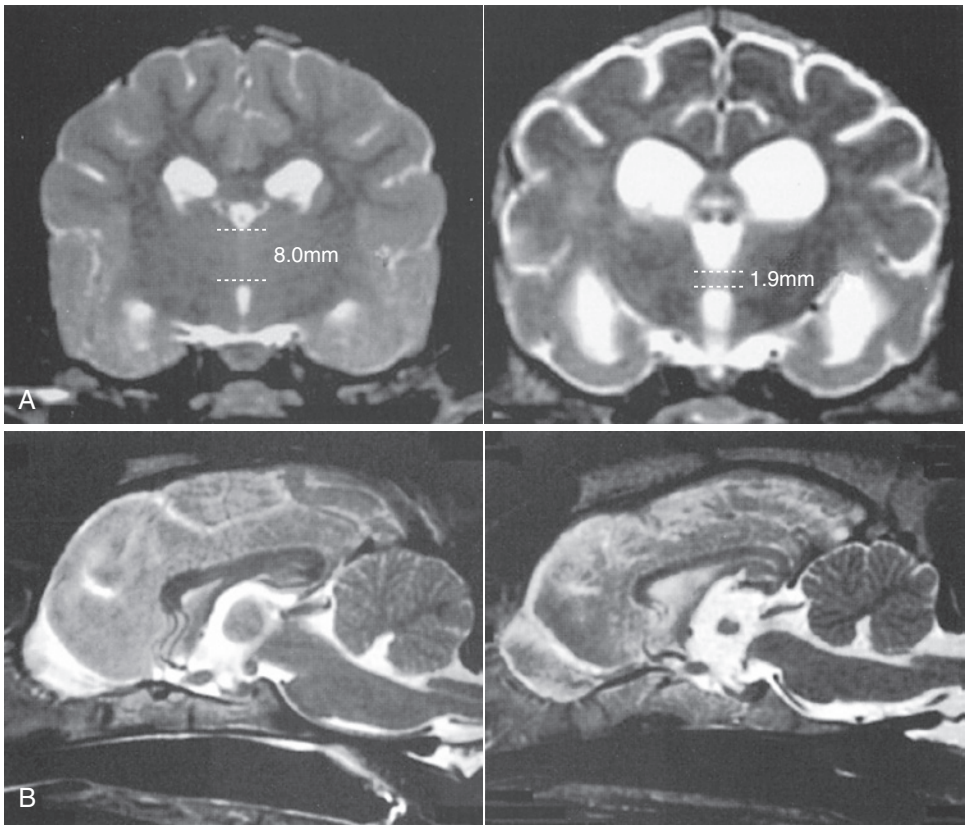


FIG. 43.2 (A) Axial and (B) midsagittal T2-weighted magnetic resonance images, demonstrating normal *(left)* and abnormal (*right;* cognitive dysfunction syndrome patient) interthalamic adhesion thicknesses in dogs. (Courtesy Dr. D. Hasegawa.)

L-deprenyl (selegiline; Anipryl, Atapryl, Carbex, Eldepryl, Zelapar), an irreversible inhibitor of monoamine oxidase B, has been purported to improve cognitive function and slow progression of the disease in the majority of dogs and cats with CDS. There is considerable variability in the degree of response achieved among patients, however. L-deprenyl is thought to exert its beneficial effects in the brain by restoring dopaminergic balance, as well as enhancing catecholamine levels and decreasing levels of damaging free radical species. The dosage for dogs is 0.5 to 1 mg/kg every 24 hours. Cats are administered 0.5 mg/kg every 24 hours. Most patients will exhibit a positive response within the first month of therapy. Despite apparent positive responses of both canine and feline CDS patients to selegiline, there is evidence that this drug does not have a significant effect on cognitive function in these patients or in people with AD. The clinical efficacy studies supporting selegiline use in CDS are based primarily on owner response to questionnaires rather than on standardized comparative cognitive testing procedures of treated and untreated patients. Because selegiline may produce nonspecific low-level hyperactivity by increasing brain catecholamine levels, the "response" observed by owners may not truly be representative of improved cognitive ability. Selegiline is not considered an effective drug for human AD because of variable responses and overall minimum improvement of cognitive function. The acetylcholinesterase inhibitor phenserine has exhibited efficacy in improving cognitive function in both dogs with CCD and humans with AD in clinical trials; to our knowledge, this drug is not yet commercially available for dogs. Behavioral changes in CCD patients may be alleviated with the use of GABA-ergic drugs, such as gabapentin or pregabalin. Because inflammatory changes have been identified in the brains of CCD dogs, the use of antiinflammatory drugs (e.g., carprofen) has also been proposed. Some naturally occurring phytochemicals (e.g., curcumin, resveratrol, green tea catechins) may hold some promise as treatment options for CCD. Oral S-adenosylmethionine has been shown to be effective in improving clinical signs of mental decline in CCD dogs in one study.[7] A large number of complementary therapies have been suggested for the treatment of CCD, with the primary goals of calming the patient, reducing anxiety, and normalizing the sleep-wake cycle. These include melatonin, valerian root, dog-appeasing pheromone, phosphatidylserine, ginkgo biloba, DHA (an omega-3 fatty acid), and various antioxidants and mitochondrial cofactors. The evidence for efficacy of these complimentary therapies is entirely anecdotal. There is convincing evidence that providing a diet fortified with antioxidants, mitochondrial cofactors, and essential fatty acids improves cognitive function and delays cognitive decline in dogs with CCD. This commercially available diet (Hills b/d) contains a mixture of fruits and vegetables, in addition to vitamins C and E, and mitochondrial cofactors (L-carnitine, DL-α-lipoic acid). Environmental enrichment, such as regular exercise and introduction of new toys, has also been demonstrated to improve cognitive function and delay cognitive decline in dogs with CDS. Progression of CDS appears to be more rapid in castrated versus intact male dogs, suggesting a potential role for hormone replacement therapy in this disease. Recent evidence suggests that the anticonvulsant drug levetiracetam (see Chapter 39) is effective in improving cognitive function and decreasing hyperexcitability in AD and animal models of AD; potential mechanisms of action include improved central nervous system (CNS) mitochondrial function

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(and associated improved synaptic transmission) and inhibition of oligomeric Aβ-induced astrocyte glutamate release.[8–10]

SURGICAL TREATMENT

There is no surgical treatment for CDS.

PROGNOSIS

The prognosis for CCD has long been considered guarded due to its progressive nature and lack of effective treatment options. However, two long-term CCD studies showed no effect of the diagnosis of CCD on survival, compared with older dogs without CCD.[3,6] This suggests that CCD, despite being a progressive neurologic disorder, can often be successfully managed medically.

DEGENERATIVE MYELOPATHY

DEFINITIONS

Degenerative myelopathy (DM) is an inherited degenerative disorder of the spinal cord, primarily affecting the thoracolumbar region (T3-L3 segments).

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

DM is a heritable, progressive degeneration of axons and myelin in all funiculi of the spinal cord, primarily in the thoracolumbar region. It is currently thought that DM is the canine analog of a form of human amyotrophic lateral sclerosis. A genetic mutation in the superoxide dismutase 1 *(SOD1)* gene has been identified in dogs with DM; this mutation leads to intraneuronal accumulation of cytotoxic cytoplasmic aggregates that stain with anti-SOD1 antibodies. Loss of axons with secondary demyelination has been demonstrated in both the CNS and peripheral nervous system of affected dogs. Dogs that are homozygous for the gene mutation are at risk for developing DM. As not all dogs homozygous for the SOD1 mutation develop clinical signs of DM and heterozygotes do not develop clinical features of the disorder, the mutation is suspected to be a recessive trait with incomplete penetrance.[11–13]

DIAGNOSIS

Clinical Presentation

Signalment

This is a disorder that primarily affects older (≥8 years), large-breed dogs. German shepherds are by far the most commonly affected, but other dog breeds and one cat have been reported to be affected. Additionally, a form of DM has been described in Pembroke Welsh corgis. There appears to be a female predominance for the disease in corgis. A list of dog breeds believed to be predisposed to DM is provided in Box 43.1.

History

The typical history is that of progressive (months), nonpainful pelvic limb weakness and ataxia. Clinical signs of dysfunction often start with dragging or scuffing the toes of the pelvic limbs, followed by ataxia and paraparesis. Dogs often have difficulty jumping.

BOX 43.1 Dog Breeds Thought To Be Predisposed to Degenerative Myelopathy

German shepherd
Boxer
Corgi (Pembroke and Welsh Cardigan)
Standard poodle
Rhodesian ridgeback
Collie
Irish setter
Bernese mountain dog
Chesapeake Bay retriever
Kerry blue terrier
Wire-haired fox terrier
Mixed-breed

Physical Examination Findings

Loss of pelvic limb proprioceptive ability (ataxia, toe dragging) is noticed initially, followed by gradual loss of voluntary motor function. Spinal reflexes in the pelvic limbs are typically normal to hyperreflexive. Decreased to absent patellar reflexes are found in approximately 10% to 15% of patients, however, and may reflect selective damage to dorsal lumbar nerve roots in these dogs. Pelvic limb tremors may be present during weight support. Late in the disease process, urinary and/or fecal incontinence may develop. Dogs often develop disuse atrophy of the pelvic limb musculature over time (see the discussion under Prognosis).

Diagnostic Imaging

Spinal imaging (myelogram, MRI, CT) is typically normal, but some dogs may have concurrent mild type II disc lesions that are probably clinically insignificant. In one study of CT/myelography performed in dogs with DM,[14] a number of abnormalities were identified when compared with normal dogs, including vertebral canal stenosis, deformed spinal cord, small spinal cord, focal attenuation of the subarachnoid space, and paraspinal muscle atrophy. A definitive diagnosis of DM is based on characteristic histopathologic lesions in the spinal cord at necropsy.

Laboratory Findings

Bloodwork (complete blood cell count [CBC], serum biochemistry) is generally normal. CSF results are typically normal or have increased protein concentration with a normal cell count. There is a genetic test available for DM to identify those dogs with the SOD1 mutation.

DIFFERENTIAL DIAGNOSIS

Major differential diagnoses for DM are chronic type II disc protrusion and spinal neoplasia.

MEDICAL MANAGEMENT

No proved effective therapies are available for DM. Treatment is supportive care. Intensive daily physiotherapy may improve survival time in DM dogs.

SURGICAL TREATMENT

There is no surgical treatment for DM.

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PROGNOSIS

The disease usually progresses over a 9- to 18-month period, eventually reaching a point at which the patient is nonambulatory in the pelvic limbs. The disease can progress to involve the thoracic limbs and eventually the brain stem in dogs kept alive after this degree of deterioration. This happens infrequently because most patients are euthanized once they reach the stage of nonambulatory paraparesis or paraplegia.

ISCHEMIC/VASCULAR DISEASE

DEFINITIONS

Infarct and **stroke** are synonymous terms used to denote a focal interruption of blood supply to a region of the CNS. In the brain, infarcts are often referred to as **lacunar** (small, from interruption of small vessel tributaries) or **territorial** (large, from interruption of a major vessel). They are also described as **hemorrhagic** or **nonhemorrhagic**, depending on whether or not there is an associated bleed around the region of vascular disruption. In the spinal cord, most infarcts are due to a disorder called **fibrocartilaginous embolic myelopathy (FCE),** where the material causing the infarct has been shown to be identical to nucleus pulposus fibrocartilage.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

There are multiple potential causes for brain infarcts, including systemic hypertension (primary/essential hypertension or secondary to underlying disease such as chronic renal failure, hyperadrenocorticism, or pheochromocytoma), cardiac disease, hypercoagulability, increased blood viscosity (e.g., polycythemia vera, multiple myeloma), intravascular neoplasia (e.g., lymphoma, hemangiosarcoma), infectious disease, and atherosclerosis (e.g., associated with hypothyroidism, diabetes mellitus, or hyperlipidemia). In people with strokes, an underlying cause is not identified in approximately 40% of cases; these infarcts are termed "cryptogenic." The percentage of cryptogenic strokes in dogs is believed to be similar to that in people. In contrast to humans, atherosclerosis appears to be rarely associated with canine brain infarcts; when it does occur in dogs, it is most likely to be associated with hypothyroidism. Brain infarcts in dogs are typically nonhemorrhagic and are most common in the cerebellum, cerebrum, and thalamic/midbrain regions. Multifocal brain infarcts have been reported but are comparatively uncommon. Cerebellar and cerebral infarcts tend to be territorial, involving the territories of large arteries, such as the rostral cerebellar artery and middle cerebral artery, respectively. These territorial infarcts tend to primarily involve the gray matter with variable levels of white matter involvement. Thalamic/midbrain infarcts tend to be smaller lacunar lesions involving the smaller perforating arteries of this brain region. In one report, over half of the dogs with brain infarcts had an underlying metabolic disorder that could potentially lead to thromboembolic disease, the most common being chronic renal disease and hyperadrenocorticism.[15] Systemic hypertension was documented in nearly 30% of those dogs whose blood pressure was documented. In another study of cerebellar infarcts in dogs, systemic hypertension was identified in over 40% of those patients in which blood pressure was measured.[16] In our experience there is usually an underlying disease present that could potentially explain the presence of hypertension in these dogs, with chronic renal disease and hyperadrenocorticism being the most common disorders.

FCE is a common syndrome caused by the embolization of arterial and/or venous supply to an area of the spinal cord. The embolizing material has been identified as fibrocartilage and is believed to originate from the nucleus pulposus of the intervertebral disc. The mechanism or mechanisms by which this material reaches the spinal cord vasculature from the disc are unknown. Theories center either around venous entry of disc material (e.g., extrusion either directly into a venous sinus or venous system of vertebral bone marrow—a Schmorl's node) with retrograde movement into the spinal arterial system (Fig. 43.3) or direct entry to the spinal cord arterial system (e.g., into normal surrounding vasculature or neovascularization over the annulus fibrosus associated with concomitant type II disc degeneration).

DIAGNOSIS

Clinical Presentation

Signalment

Dogs are much more likely to present with ischemic/vascular events to the brain and spinal cord than cats. There is a wide age distribution for both brain infarcts and FCE. Most dogs

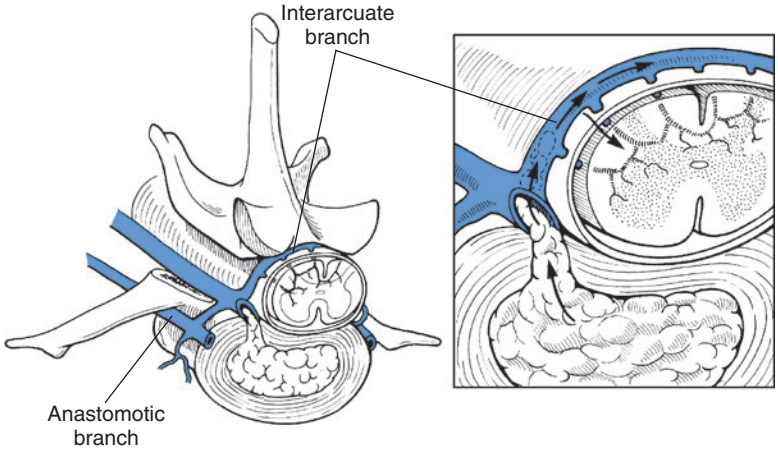


FIG. 43.3 Schematic illustration showing a proposed pathogenesis of a fibrocartilaginous embolism involving extrusion of nuclear material into the vertebral venous sinus or ventral spinal artery and then into small vessels supplying the spinal cord parenchyma.

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presented with brain infarcts are middle-aged to older with median and mean ages of 8 and 9 years, respectively. Cerebellar infarcts appear to be most common in small-breed dogs, most notably the Cavalier King Charles spaniel. It has been postulated that this predisposition may be related to this breed's propensity to develop heart disease, inherited platelet abnormalities, or to local aberrations in regional arterial (e.g., basilar artery) blood flow resulting from Chiari-like malformations (CLMs), which is common in the breed. In people with atlantoaxial instability, basilar artery compression has been associated with cerebellar infarction. In our experience, the combination of CLMs and cerebellar infarct is common in the Cavalier King Charles spaniel, whereas combinations involving cardiac disease and platelet abnormalities are not. Large-breed dogs appear to be predisposed to developing lacunar thalamic/midbrain infarcts. Greyhounds may be predisposed to brain infarcts; one proposed reason is the tendency for greyhounds to have higher resting mean arterial blood pressures than other breeds. FCE typically affects non-chondrodystrophic dogs, mainly of large and giant breeds, but smaller nonchondrodystrophic dogs (e.g., Shetland sheepdogs, miniature schnauzers) and a number of cats have been reported. FCE has also been described in a few chondrodystrophic small-breed dogs. Approximately 20% of FCE patients are dogs less than 20 kg. Miniature schnauzers are predisposed to FCE, which is the most common cause of myelopathy in this breed. Most dogs presenting with FCE are young to middle-aged (1–7 years of age) adults. In one large (n = 393 dogs) retrospective report of canine FCE cases, FCE was most common in middle-aged, large-breed dogs (30% of the total), the most common single breed reported was the miniature schnauzer, and small-breed dogs accounted for 24% of the cases.[17] Most of the reported cats with suspected FCE have been middle-aged to older at presentation (7–12 years of age), and the majority were domestic shorthaired cats. Older cats (14–15 years) have also been reported with cranial cervical spinal cord infarcts involving the ventral gray matter at the C1–C2 vertebral level. These infarcts occur in the territory of the ventral spinal artery. A large proportion of these cats had concurrent disorders that could potentially be the cause of the infarct (e.g., chronic renal failure, cardiomyopathy).[18,19] In one retrospective report of 19 cats with ischemic myelopathy, there was a preponderance of domestic shorthaired cats, a male predominance, and an average age at presentation of 10 years; these findings were similar to previous reports of feline spinal cord infarcts.[20] Based on the available literature and our experience, FCE in cats occurs over a wide age range (including juveniles) and in multiple locations along the spinal cord. Spinal cord infarcts in the ventral gray matter of the cranial cervical cord tend to occur in very old cats and are associated with an underlying disease condition that may be responsible for hypertension and/or hypercoagulability (e.g., chronic renal failure, cardiomyopathy).

History

For both brain infarcts and FCE, clinical signs of neurologic dysfunction are typically peracute or acute onset and nonprogressive after the initial 24 hours of onset. In rare cases of FCE the time course of neurologic deterioration progresses over several days. Specific owner complaints depend on the location of the ischemic event in the CNS. In many cases of FCE, the patient is observed by the owner to be doing something active (e.g., chasing a ball in the yard) at the time of the ischemic event. At the time of the event, if observed, it is also common for the dog to cry out as if in pain. Shortly following the vascular event, dogs with FCE typically do not appear to be in pain.

Physical Examination Findings

The neurologic findings for both brain infarct and FCE patients reflect the location of the lesion within the brain and spinal cord, respectively. Paradoxical vestibular syndrome appears to be a common phenomenon with cerebellar infarcts. Although most MR images of territorial cerebellar infarcts in dogs suggest pure cerebellar involvement of the infarct, many of these patients have signs of medullary dysfunction (e.g., paresis or nonambulatory status) as well. This apparent discrepancy is believed to be explained by secondary medullary compression from the edema around the infarct at the time of the ischemic event. By the time the patient is imaged, this swelling is typically not apparent on the MRI. In one recent retrospective report of 23 dogs with suspected cerebellar infarcts, neurologic deficits included head tilt (13), ataxia with (11) or without (4) hypermetria, nystagmus (8), decreased menace response (7), postural reaction deficits (7), and nonambulatory status (6).[21] Another unusual clinical presentation common in dogs with thalamic/midbrain infarcts is the presence of central vestibular dysfunction. Dogs with infarcts in this area of the brain have had clinical signs such as ipsilateral head tilt (often in addition to a head turn, which would be expected with a forebrain lesion), strabismus, and nystagmus. Seizure activity has been reported in dogs with cerebral infarcts, but it does not appear to be very common. Clinical signs of myelopathy in FCE patients vary, depending on both the location and severity of the spinal cord ischemic injury. Deficits are often asymmetric with FCE. In the large retrospective study of 393 FCE dogs mentioned earlier, the most common neuroanatomic localization was T3–L3 (33.1%), followed by L4–S3 (31.8%), C6–T2 (20.6%), C1–C5 (10.7%), and multifocal (3.8%); clinical signs of myelopathy were asymmetric in 69.5% of cases.[17] These patients are typically not in pain on presentation. However, a focal region of hyperesthesia that corresponds to the infarct location can often be found if these patients are examined within 12 hours of the onset of clinical signs. This mild hyperesthesia is probably due to transient meningeal swelling in the infarct region.

Diagnostic Imaging

Brain and spinal cord infarcts are unlikely to be visualized on any other modality than MRI. CT may be more sensitive in detecting acute hemorrhage in early hemorrhagic stroke, but it offers no other advantages over MRI. Most brain infarcts in dogs and cats are nonhemorrhagic. MRI allows for greater detail in imaging (including detailed multiplanar images) and fewer artifacts (like beam-hardening artifact with caudal fossa images) compared with CT. Some functional MRI techniques (diffusion weighting, perfusion imaging, and MR angiography) can be used to improve the accuracy of stroke diagnosis, especially in the peracute stage of infarction (and especially within the first few hours). Table 43.1 summarizes the characteristic MRI appearance of nonhemorrhagic brain infarcts in dogs over time from infarction, using standard MRI sequences. Overall, nonhemorrhagic brain infarcts tend to be hyperintense on T2-weighted and fluid attenuation inversion recovery (FLAIR) images (see Chapter 14), hypointense on T1-weighted images, and minimally to non–contrast enhancing (Fig. 43.4). Contrast enhancement, usually at the periphery of the infarcted region, is typically evident 1 to 8 weeks following the ischemic event, presumably associated

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TABLE 43.1 Characteristic MRI Appearance of Nonhemorrhagic Brain Infarcts Over Time

Stage	Time to Imaging	T2-Weighted MRI Findings	T1-Weighted MRI Findings	FLAIR Findings	Contrast Enhancement
Peracute	3–6 h	Hyperintense	Hypointense	Hyperintense	No
Acute	6–24 h	Hyperintense	Hypointense	Hyperintense	No
Early subacute	24 h–1 wk	Hyperintense	Hypointense	Hyperintense	Variable
Late subacute	1–6 wk	Hyperintense	Hypointense	Hyperintense	Yes
Chronic	>6 wk	Hyperintense	Hypointense	Hyperintense	Variable

MRI, Magnetic resonance imaging.

TABLE 43.2 MRI and CT Findings Associated With Different Stages of Hemorrhagic Brain Infarcts

Stage	Time to Imaging	T2-Weighted MRI Findings	T1-Weighted MRI Findings	CT Findings
Peracute	3–6 h	Slightly hyperintense	Isointense	Hyperdense
Acute	6–24 h	Hypointense	Isointense	Hyperdense
Early subacute	24 h–1 wk	Hypointense	Hyperintense	Hyperdense
Late subacute	1–6 wk	Hyperintense	Hyperintense	Variable
Chronic	>6 wk	Hypointense	Hypointense	Isodense

CT, Computed tomography; *MRI,* magnetic resonance imaging.

with disruption of the blood-brain barrier. The appearance of hemorrhagic infarcts on CT and MR images also varies with the time from the ischemic event and is dependent on the oxygenation state of hemoglobin. The "aging" stages of hemoglobin molecules progress sequentially as follows: oxyhemoglobin (peracute stage), deoxyhemoglobin (acute stage), methemoglobin (subacute stage), and finally hemosiderin and ferritin (chronic stage). Within the first several hours of a hemorrhagic infarct, the lesion is relatively isointense on T1- and T2-weighted MR images, but may show some hyperintensity on T2-weighted images due to edema surrounding the infarct. Deoxyhemoglobin subsequently forms over the next 24 hours, which is associated with low signal intensity on T1- and T2-weighted images. Intracellular methemoglobin predominates next, which is hyperintense on T1-weighted but hypointense on T2-weighted images. Subsequent accumulation of extracellular methemoglobin leads to a hyperintense signal on both T1- and T2-weighted images. Finally, hemosiderin and ferritin accumulate, which are low-signal intensity on both T1- and T2-weighted images. T2*-weighted (gradient recalled echo) sequences are very sensitive for identifying hemorrhagic lesions and should be routinely used in cases of suspected infarct.[22]

Both MRI and CT findings associated with different stages of hemorrhagic infarcts are summarized in Table 43.2. Hemorrhagic infarcts are also more likely to exhibit contrast enhancement compared with nonhemorrhagic infarcts (Fig. 43.5). The absence of an obvious spinal cord compressive lesion or mild focal thinning of the contrast column on myelography was the traditional method of tentatively diagnosing FCE. As with brain infarcts, MRI is also the preferred imaging modality for suspected FCE patients (Fig. 43.6); focal, sharply demarcated, hyperintense parenchymal lesions (suspected to be edematous, infarcted tissue) on T2-weighted and FLAIR images are characteristic. These lesions are isointense or hypointense to spinal cord on T1-weighted images and may display varying degrees of contrast enhancement (usually mild enhancement when present); the presence or absence of contrast enhancement may be related to the timing of MRI after the infarct occurs, often occurring approximately a week after injury in humans with FCE. The FCE lesions are often primarily within the gray matter of the spinal cord and unilateral in location. In one study of 52 dogs with suspected FCE, 11 dogs (21%) did not have any lesions apparent on MRI.[23] The appearance of lesions on MRI in this study was not associated with timing of imaging after infarction (in people, ischemic lesions may not be apparent on MRI within the first 48 hours of the infarct), but it was significantly associated with severity of neurologic dysfunction; ambulatory dogs were much more likely to have normal MRI results than nonambulatory dogs. This study also found a positive association between disease severity and lesion extent on MRI. In another large retrospective study for which 139 FCE dogs were imaged via MRI, 90.6% of dogs had T2 hyperintensity in the spinal cord parenchyma, 46% had subjective focal spinal cord swelling, and 22% had some level of contrast enhancement within or adjacent to the suspected infarct; 18% of cases had evidence of hemorrhage on T2* sequences.[17]

Laboratory Findings

Laboratory findings for patients with brain infarcts are variable, depending on whether or not there is an underlying disorder that may have led to the stroke event. Some patients may have laboratory evidence supporting chronic renal insufficiency or endocrine disease (e.g., hyperadrenocorticism, hypothyroidism), but many are within normal limits. Laboratory findings are usually normal for FCE patients. For both brain infarcts and FCE, CSF results may be normal or reveal mild, nonspecific abnormalities (mild increase in cell count and protein concentration, xanthochromia). In one large, retrospective study of canine

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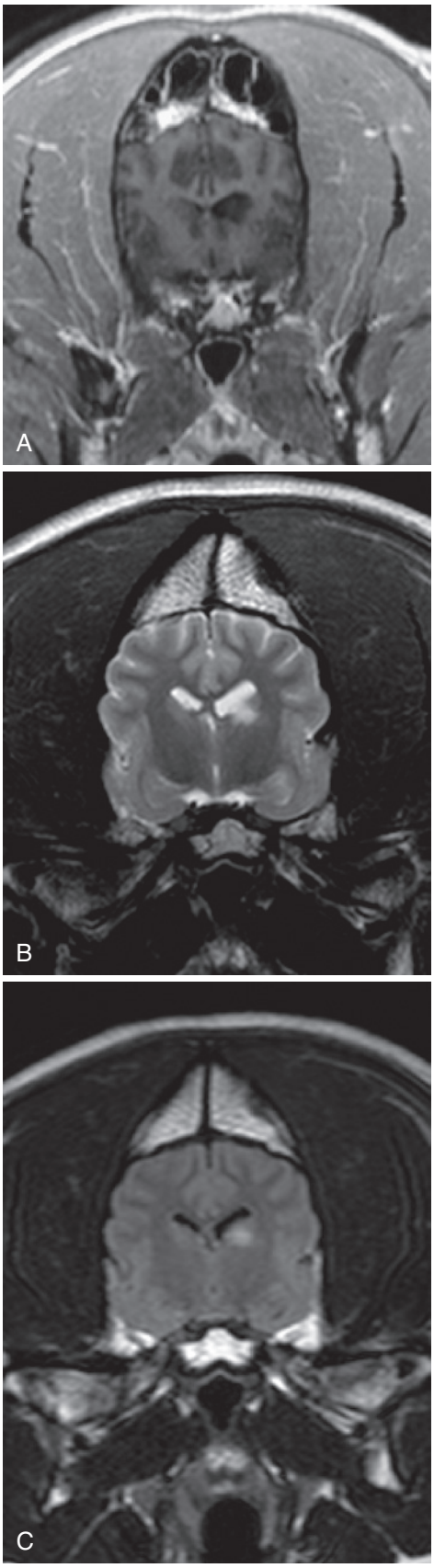


FIG. 43.4 (A) Axial T1-weighted with contrast, (B) T2-weighted, and (C) dorsal fluid attenuation inversion recovery images from a dog with a nonhemorrhagic cerebral infarct.

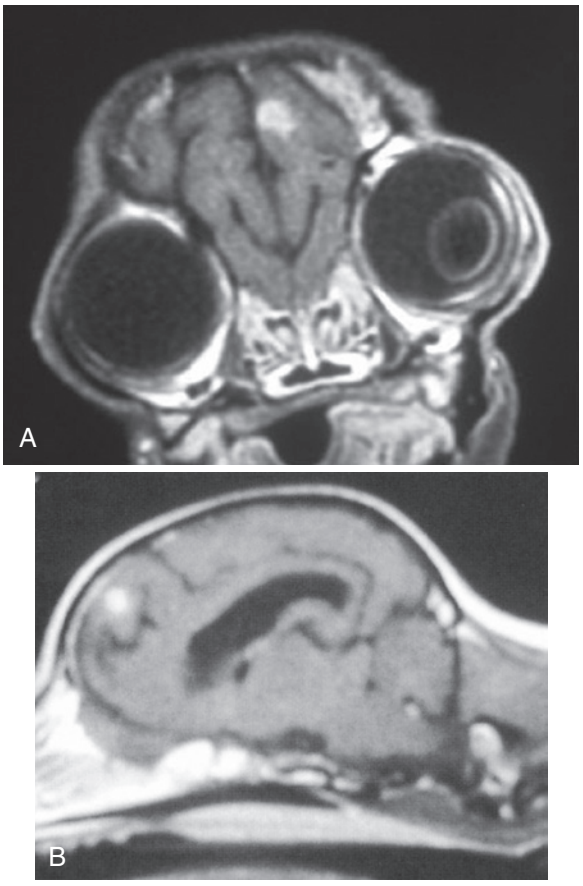


FIG. 43.5 (A) Axial and (B) sagittal magnetic resonance images (T1-weighted with contrast) of a dog with a focal hemorrhagic cerebral infarct.

FCE in which CSF results were available for 206 dogs, roughly half of the CSF results were within normal limits.[17]

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for brain infarcts include brain tumor, inflammatory/infectious disease (e.g., granulomatous meningoencephalomyelitis [GME]), and trauma. The most likely differential diagnosis for FCE is type I disc extrusion (particularly hydrated nucleus pulposus extrusion [HNPE]-type extrusions; see Chapters 40 and 41), followed by spinal neoplasia and inflammatory/infectious disease. In one study comparing dogs with presumptive FCE and dogs with HNPE, a number of distinguishing clinical features were identified (Box 43.2).[24]

MEDICAL MANAGEMENT

There is no specific medical management for either brain or spinal cord infarcts. For brain infarcts, medical management is directed at the underlying disease process if one is identified. For spinal cord infarcts, including FCE, medical management is focused on physical therapy and rehabilitation.

SURGICAL TREATMENT

Neither brain infarcts nor spinal infarcts are surgical disorders.

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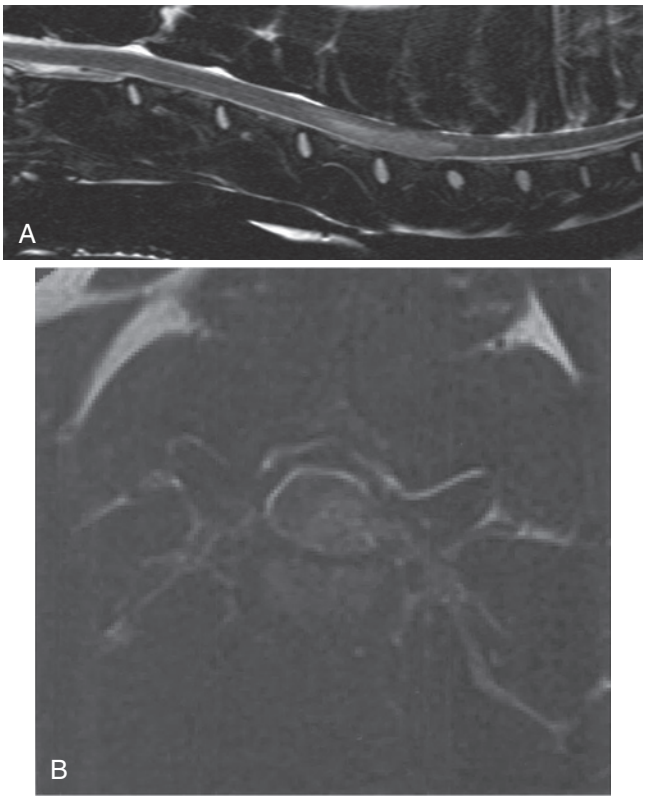


FIG. 43.6 (A) Midsagittal and (B) axial T2-weighted magnetic resonance images of the cervical region in a dog with fibrocartilaginous embolic myelopathy.

BOX 43.2 Clinical Features to Help Distinguish Between Fibrocartilaginous Embolic Myelopathy and Hydrated Nucleus Pulposus Extrusion

- English Staffordshire bull terriers are prone to FCE, whereas border collies are prone to HNPE.
- HNPE dogs are more likely to vocalize at onset of signs, compared with FCE dogs.
- HNPE dogs are more likely to have C1–C5 lesions than FCE dogs.
- HNPE dogs are more likely to exhibit spinal hyperesthesia on initial examination than FCE dogs.
- HNPE dogs are more likely to be ambulatory at time of hospital discharge than FCE dogs.
- FCE dogs are more likely to have L4–S3 lesions (and long-term fecal incontinence) than HNPE dogs.

FCE, Fibrocartilaginous embolic myelopathy; *HNPE,* hydrated nucleus pulposus extrusion.
Data from Fenn J, Drees R, Volk HA, et al. Comparison of clinical signs and outcomes between dogs with presumptive ischemic myelopathy and dogs with acute noncompressive nucleus pulposus extrusion. *J Am Vet Med Assoc.* 2016;249(7):767–775.

PROGNOSIS

The prognosis for dogs with focal brain infarcts is variable, but most have a guarded to fair prognosis for recovery of partial or full function. In one study of 33 dogs with brain infarcts, 10 dogs were euthanized; half of these were euthanized due to the severity of their underlying disease process rather than failure of their neurologic status to improve.[15] Another study of cerebellar infarcts also found a negative association between survival and presence of underlying systemic disease.[16] In a more recent retrospective study of 23 dogs with cerebellar infarcts, all dogs survived and were discharged from the hospital within 1 to 10 days of admission; in this case series, 12 dogs had concurrent underlying medical conditions. Although this study had only short-term follow-up, it does suggest that dogs with cerebellar infarcts tend to improve rapidly after initial infarction and most have a good prognosis for recovery.[21] The presence of an underlying or concurrent medical condition has also been associated with increased chance of repeat brain infarction within a 10-month period. The prognosis for functional recovery for dogs with FCE is somewhat variable, reflecting the range of lesion severity characteristic of this disease. In addition, recovery rates for these dogs vary considerably among published studies. Most of the recent literature suggests an overall favorable prognosis for dogs with FCE, whereas older literature is more pessimistic. For nonambulatory dogs, especially of the large and giant breeds, the prognosis for recovery is often guarded. Negative prognostic indicators reported include loss of deep pain perception (nociception), severe lower motor neuron damage, and owner reluctance to pursue prolonged physical therapy. There is some disagreement in the literature regarding the assertion that lower motor neuron damage portends a worse prognosis than upper motor neuron spinal cord damage in FCE cases. The degree of owner reluctance to pursue physical therapy is often associated with the size of the dog (e.g., prolonged physical therapy and bladder management for a paralyzed great Dane may not be feasible for many owners). In one report of 50 dogs,[25] 84% (42 dogs) had successful outcomes. In this study, the extent of the lesion as measured on MR images was predictive of outcome; dogs with a lesion length:vertebral length (C6 or L2, depending on whether the infarct was cervical or thoracolumbar) ratio (sagittal images) of 2 or less, or a cross-sectional (transaxial images) lesion area/spinal cord area percentage of less than 67%, were significantly more likely to recover, compared with dogs that had higher values for these parameters. Severity of neurologic signs at presentation was also significantly associated with a negative outcome in this study. The median time to maximal neurologic recovery for these dogs was 3.75 months. In a more recent retrospective study (2016) of 393 dogs with FCE, the overall prognosis was good to excellent, with 85% of dogs regaining the ability to walk unassisted; most patients reached this stage of neurologic improvement within 3 weeks, though persistent neurologic deficits were common in dogs that recovered the ability to walk unassisted (49.1%).[17] It appears that although FCE in dogs may be associated with a substantial level of morbidity and mortality, a large proportion of dogs may recover neurologic function following the infarct. Many of the cats diagnosed with FCE were euthanized shortly following presentation due to severity of neurologic deficits and presumption of a poor prognosis. Although the numbers of reported cats with ischemic myelopathy are much less than those described for dogs, the prognosis is generally favorable for recovery of neurologic function, whether the infarct is due to FCE or due to an underlying systemic condition (e.g., chronic renal failure, cardiomyopathy, hypertension). In one retrospective report of 19 cats with ischemic myelopathy due to a variety of suspected causes (FCE and underlying systemic disease), 79% of cases had a positive outcome.[20] In a similar retrospective case series in 11 cats, eight (73%) cats regained ambulatory function within 2 months of initial presentation.[18] In a retrospective report of eight older cats with cranial cervical cord infarcts associated with underlying disorders, all eight cats

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recovered normal to near-normal neurologic status within 3 months of onset of clinical signs; three of these cats had suspected recurrent ischemic episodes.[19]

NONINFECTIOUS INFLAMMATORY DISEASE

DEFINITIONS

GME is a common, suspected autoimmune, noninfectious inflammatory disorder of dogs. **Necrotizing encephalitis** (NE) is a less common suspected autoimmune noninfectious inflammatory disorder of dogs, distinct from GME clinically and histopathologically. The umbrella terms **meningoencephalitis of unknown etiology (MUE)** and **meningoencephalitis of unknown origin** are sometimes used to encompass this group of disorders.[26]

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

GME is characterized histologically by perivascular infiltrates of primarily mononuclear cells (lymphocytes, macrophages, and plasma cells) in the brain and/or spinal cord. The characteristic perivascular cellular infiltrates of GME both define the disease syndrome and account for the neurologic deficits. The underlying cause of this disease remains unknown; but there is evidence that GME is an autoimmune disorder, specifically a delayed-type (T cell–mediated) hypersensitivity reaction. Lesions predominate in the white matter with GME. There are three recognized clinical forms of GME: focal, multifocal (disseminated), and ocular. The ocular form is the least commonly encountered. In our experience, multifocal GME is the most common form of the disorder. NE includes two pathologically distinct disorders, referred to as necrotizing meningoencephalitis (NME) and necrotizing leukoencephalitis (NLE). These are also thought to be autoimmune disorders. Both NME and NLE are similar in that they are characterized by multiple cavitary necrotic nonsuppurative inflammatory brain lesions that involve both gray and white matter. The lesions in NME predominate in the cerebral cortex (gray matter). In NME, the lesions are typically found in the cerebrum, with consistent involvement of the leptomeninges. Extensive cerebral cavitations with a loss of demarcation between gray and white matter are typical for NME. NLE is characterized by similar lesions that often involve the brain stem in addition to cerebrum, with less consistent involvement of the leptomeninges and cerebral cortex (i.e., mainly white matter). Initial reports of these diseases in predisposed breeds led to the terms *pug encephalitis* or *pug/Maltese encephalitis* for NME and *Yorkshire terrier encephalitis* for NLE. It is believed that—considering the breed predilections of different types of meningoencephalitis of unknown origin—there is a genetic susceptibility component to these disorders in dogs.[27] Common genetic risk loci (for genes associated with regulation of immune system function) for the development of NME have been identified in several small dog breeds, including pugs, Maltese, and Chihuahuas.[28]

DIAGNOSIS

Clinical Presentation

Signalment

GME can affect any breed of dog of any age and either sex, but young to middle-aged (median age, 5 years) female dogs of small breeds (e.g., poodles, terriers) appear to be predisposed. NE also tends to occur in young, small-breed dogs, but a wide age range has been reported (approximately 7 months to 13 years). Pug and Maltese dogs are most commonly affected by NME, but other breeds reported with this disorder include Chihuahua, shih tzu, Pekingese, papillon, West Highland white terrier, Coton de Tulear, and Brussels griffon.[29] We also encountered a NME disorder in a Boston terrier. Yorkshire terriers appear to be the most common breed afflicted by NLE, but this disease may also affect other small-breed dogs. It is possible that NME and NLE represent variants of the same disease process. It is expected that more breeds will be reported with idiopathic NE. As with GME, the necrotic lesions observed on brain histopathology both account for the clinical signs of dysfunction and define the disease syndrome.

History

Multifocal GME is characterized by acute onset and rapid progression of CNS dysfunction, whereas dogs with focal GME tend to have a more insidious onset and slower progression of clinical signs. Common clinical complaints associated with GME include seizures, cerebellovestibular dysfunction, and cervical hyperesthesia. Isolated spinal cord dysfunction and visual deficits (optic neuritis) are uncommon clinical presentations of GME. Seizure activity is the most frequent clinical complaint with NE. The onset and progression of clinical signs of neurologic dysfunction with NE may be acute (disease course of ≤2 weeks) or chronic (disease course of 4–6 months). Clinical signs of forebrain dysfunction (e.g, seizures, circling, obtunded mental status, visual deficits with normal pupillary light reflexes, head-pressing) predominate. Neck pain is also common and may be due to the meningitis and/or the forebrain lesions. Yorkshire terriers with NLE often experience a chronic, progressive worsening of neurologic dysfunction over several months. In addition to clinical signs of forebrain dysfunction and neck pain, Yorkshire terriers with NLE often display clinical signs of brainstem dysfunction (e.g., central vestibular disease).

Physical Examination Findings

Physical examination findings are usually unremarkable, although some GME patients are febrile on presentation. Neurologic deficits are typically profound and reflect the location(s) of the lesions(s). Most GME patients have multifocal signs and often display central vestibular dysfunction. Most NE patients display primarily forebrain dysfunction.

Diagnostic Imaging

For both GME and NE, the preferred imaging modality is MRI, with CT being a poor second choice. The majority of GME patients have multifocal hyperintense (on T2-weighted and FLAIR sequences) lesions evident with variable contrast enhancement (T1-weighted with contrast). The contrast-enhancing lesions usually appear indistinct or "fuzzy" with blurred edges (Fig. 43.7). Some dogs with GME have secondary hydrocephalus evident on CT/MR images of the brain. MRI and CT findings have been described for NE as well. Lesions on MRI are usually iso- or hypointense on T1-weighted images, hyperintense on T2-weighted and FLAIR images, and usually non–contrast enhancing (Fig. 43.8). These cavitary lesions represent areas of brain necrosis.

Laboratory Findings

Bloodwork (CBC, serum biochemical panel) results are generally normal unless treatment with corticosteroids has begun prior to presentation, in which case one might see abnormalities such

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FIG. 43.7 (A) Axial and (B) dorsal T1-weighted with contrast magnetic resonance images of a dog with granulomatous meningoencephalomyelitis.

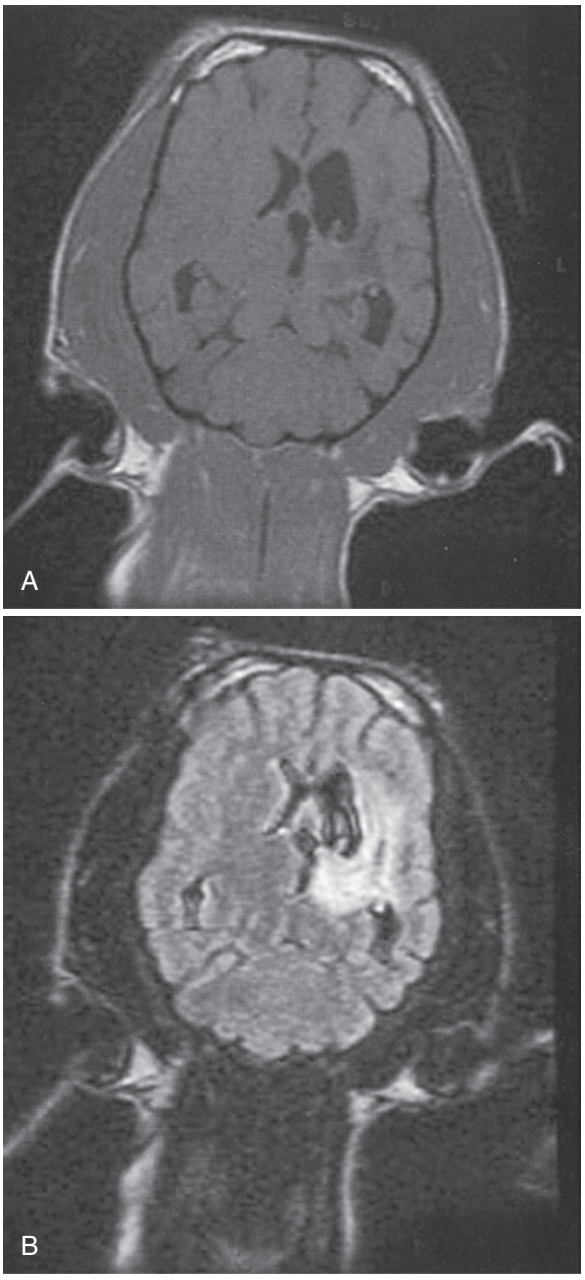


FIG. 43.8 (A) Dorsal T1-weighted and (B) fluid attenuation inversion recovery magnetic resonance images of a dog with necrotizing encephalitis.

as a stress leukogram or increased alkaline phosphatase concentration. CSF results for GME and NE are characteristically abnormal (see Box 37.4). A mainly mononuclear pleocytosis with a variable percentage of neutrophils (mean, approximately 20%) and elevated protein concentration is characteristic of GME. Uncommonly (≤10%), GME patients will have primarily neutrophilic or normal CSF results. In most cases of NE, a predominantly or exclusively mononuclear pleocytosis with elevated protein concentration is evident. The mononuclear cells in NME are usually primarily lymphocytic, whereas a mixture of lymphocytes and monocytes is usually seen in the CSF of NLE patients.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for GME and NE patients include brain tumor (especially if signs reflect a focal lesion), anomalous brain disorders (e.g., CLM, intracranial arachnoid cyst), brain infarct (if signs reflect a focal lesion), and infectious disease. The definitive diagnosis of GME and NE depends on histopathologic confirmation of the disease process. The tentative diagnosis is based on characteristic imaging and CSF results, in combination with supportive historical, signalment, and clinical presentation features.

MEDICAL MANAGEMENT

Although prednisolone remains a facet of the treatment of GME and NE, it should no longer be considered the mainstay of therapy for these disorders. In addition to prednisolone, therapies for

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BOX 43.3 Drugs for Treatment of Granulomatous Meningoencephalomyelitis and Necrotizing Encephalitis

- Prednisolone: 1–2 mg/kg PO q12h
- Procarbazine: 25 mg/m^2 PO q24h
- Cyclosporine: 3–5 mg/kg PO q12h
- Cytosine arabinoside: 50 mg/m^2 SC q12h for 2 days (typically 400 mg/m^2 over 24 h CRI)
- Azathioprine: 2.0 mg/kg PO q24h
- Leflunomide: 2.0–4.0 mg/kg PO q24h
- Mycophenolate mofetil: 20 mg/kg PO q12h, reduce to 10 mg/kg PO q12h after 3–4 wk (anecdotally seems to be more effective for NE vs. GME)

CRI, Constant-rate infusion; *GME,* granulomatous meningoencephalomyelitis; *NE,* necrotizing encephalitis; *PO,* orally; *SC,* subcutaneous.

GME and NE include cyclosporine, cytosine arabinoside, procarbazine, azathioprine (AZA), leflunomide, and mycophenolate mofetil (MMF; Box 43.3). Myelosuppression is the most likely adverse effect from procarbazine, although hemorrhagic gastroenteritis may also occur. Potential side effects from cyclosporine use in dogs include vomiting, diarrhea, hyporexia, weight loss, gingival hyperplasia, papillomatosis, hypertrichosis, and excessive shedding. Side effects from cyclosporine use are generally minimal if plasma levels are within target ranges. Myelosuppression is also a potential concern with the use of cytosine arabinoside. This almost never occurs with the dosage protocols used for GME and NE patients. In general, when using these drugs (especially procarbazine) CBC should be checked weekly for the first month, then monthly to every 2 months thereafter to monitor for myelosuppression. The most common side effect of AZA is bone-marrow suppression, necessitating regular CBC examinations. Other potential side effects include gastrointestinal upset, pancreatitis, hepatotoxicity, and poor hair growth. It may take approximately 2 weeks from initiation of AZA for a clinical response to be apparent. The most likely adverse effects of MMF use are gastrointestinal (vomiting, hemorrhagic diarrhea); these effects are usually avoidable if the initial dose is reduced within 3 to 4 weeks. In our experience, MMF tends to be more effective for NE than for GME. Antiseizure drugs should be administered to GME and NE patients with seizure activity, preferably drugs that do not cause sedation or polyuria and polydipsia (see pp. 1339–1340). Combination therapy with several of these drugs may be necessary for initial disease control, with the drugs slowly weaned over time, if possible. Drug weaning should not be initiated until there is an obvious response to therapy; one drug should be weaned at a time, and changes should not be done more quickly than every 4 weeks. Dogs with controlled GME and NE typically do not become drug free but are often maintained on a low enough level of therapy to avoid the obvious side effects of therapy.

SURGICAL TREATMENT

Neither GME nor NE is a surgical disorder, but surgical biopsy may be considered in some cases.

PROGNOSIS

In recent years, the prognosis for GME has improved dramatically, coincident with the development of improved treatment protocols. The prognosis for NE is still poor, but anecdotally it appears to have improved to some degree with new drug protocols. Historically, survival times for multifocal GME dogs treated with glucocorticoids alone have ranged from approximately 2 weeks to 3 months, although more recent evidence suggests considerably longer survival times.[30] Nonetheless, because of the variable and often poor response to glucocorticoid therapy, as well as frequent glucocorticoid-related side effects, a number of immunosuppressive drugs have been evaluated as adjunctive treatment options for GME patients. The three most promising drug options are procarbazine, cytosine arabinoside, and cyclosporine. Survival times exceeding 12 months have been reported for each of these drugs. Additionally, the use of these drugs appears to allow for successive decreases in glucocorticoid dosages, thereby minimizing adverse side effects associated with steroid use. Procarbazine (25 mg/m^2 orally, once daily) is an antineoplastic drug that crosses the blood-brain barrier and has some specificity for T cells. The cytotoxic effects of procarbazine are thought to be primarily via methylation of DNA bases. In one study of presumptive GME dogs, the use of procarbazine as an adjunct to prednisone was associated with a median survival time of 14 months,[31] regardless of clinical form of GME (the majority were multifocal). In one report of 10 dogs with noninfectious encephalitis of undetermined etiology,[32] cytosine arabinoside treatment was associated with a median survival time of approximately 1.5 years. In one study of 10 dogs with presumptive GME treated with cyclosporine,[33] the median survival time was approximately 2.5 years. In one study of dogs with MUE treated with AZA and prednisone, the median survival time was approximately 5 years. In that study, dogs that exhibited a complete response to therapy had significantly longer survival times than those with a partial response and dogs that did not relapse had significantly longer survival times than those that did relapse.[34] In our experience, most dogs with GME respond well to combination therapy and do well for at least 2 to 3 years. The response to therapy and prognosis for dogs with NE appears to be less than that for dogs with GME. The prognostic literature for MUE in dogs focuses on long-term outcome, but there is little information regarding short-term outcome for this group of disorders. In one retrospective study of 116 dogs with MUE, 30 (26%) patients died within the first week of diagnosis, despite treatment; significant negative prognostic indicators included decreased mental status, presence of seizures, and increased percentage of neutrophils on CSF analysis.[35] Despite improvements in prognosis for dogs with GME and NE, there appears to be a substantial early mortality for patients with these disorders.

STEROID-RESPONSIVE MENINGITIS-ARTERITIS

DEFINITIONS

Steroid-responsive meningitis-arteritis (SRMA) is a suspected autoimmune disorder characterized by inflammation of the meninges and meningeal arteries. This disorder is also often termed *corticosteroid-responsive meningitis/arteritis, aseptic meningitis,* and *sterile meningitis.*

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

This is a suspected autoimmune inflammatory disorder of the meninges and meningeal vasculature that tends to affect young

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adult large-breed dogs. In some cases, patients with this disorder have concurrent autoimmune polyarthritis. This is most typically an acute disorder, but a less common, chronic form of the disease has been described. The chronic form of the disorder is suspected to be due to relapse and/or inadequate treatment of the acute form of the disease.

DIAGNOSIS

Clinical Presentation

Signalment

This usually occurs in young (<2 years old), medium- to large-breed dogs, and may be the most common type of meningitis encountered in veterinary practice. There are several breeds predisposed to this condition, including boxers, beagles, Bernese mountain dogs, Weimaraners, and Nova Scotia duck tolling retrievers.[36]

History

In the most common ("classic") form of SRMA, the history is typically an acute onset of severe cervical hyperesthesia accompanied by lethargy and a stiff gait. The chronic form of the disorder is typified by cervical hyperesthesia as well but also by signs of spinal cord dysfunction (e.g., ataxia and paresis).

Physical Examination Findings

In the acute form of SRMA, the overwhelming clinical sign is profound cervical hyperesthesia. These dogs tend to adopt a hunched posture and resist any neck movement. They are often febrile on presentation and often have evidence of hyperesthesia in other spinal locations on palpation. Dogs with the chronic form of SRMA are more likely to have neurologic deficits such as ataxia and paresis, consistent with a compressive myelopathy.

Diagnostic Imaging

Survey spinal radiographs should be obtained to rule out disorders such as discospondylitis and vertebral physitis. In acute cases of SRMA, MRI of the cervical spine is often performed to rule out other causes of a cervical myelopathy. The MR results are often normal but may show evidence of meningeal enhancement on T1-weighted with contrast sequences. More obvious lesions on MRI may be apparent in chronic SRMA cases because of suspected meningeal fibrosis.

Laboratory Findings

Bloodwork results are often normal, but may show a leukocytosis with a left shift, especially in acute SRMA cases. CSF results are usually dramatic in acute SRMA cases, with a marked polymorphonuclear pleocytosis (predominantly or exclusively nondegenerate neutrophils) and elevated protein concentration (see Box 37.4). In chronic SRMA cases, the CSF is often less dramatic (may be normal) and may be mainly mononuclear or a mixed cell pleocytosis with or without elevated protein concentration. Elevated IgA concentrations in serum and CSF are characteristic of SRMA. Several acute-phase proteins (e.g., C-reactive protein and alpha-2 macroglobulin) have also been found to be consistently elevated in SRMA dogs.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for SRMA include infectious meningomyelitis (particularly bacterial), discospondylitis, type I disc extrusion, and spinal neoplasia.

MEDICAL MANAGEMENT

Treatment with immunosuppressive doses of prednisone (2–4 mg/kg per day) usually results in rapid improvement in acute cases. Additional treatment with oral analgesics (e.g., tramadol) can also be considered. If there is concern about an infectious etiology, concurrent antibiotics can be coadministered for 2 to 4 weeks. These patients should be slowly weaned off the prednisone over a number of months (e.g., 3–6 months), but approximately half of these dogs will require some level of long-term immunosuppressive therapy; most of the dogs requiring such extended therapy can be weaned to a very low dose of drug. There are anecdotal reports of successfully using other immunomodulatory drugs for this disorder (e.g., AZA, cyclosporine, mycophenolate), especially for cases of relapse.

SURGICAL TREATMENT

SRMA is not a surgical disorder.

PROGNOSIS

The prognosis for control of clinical signs with the acute form of this disease is typically excellent with appropriate treatment. The prognosis for the chronic form of the disease is fair to guarded.

DISCOSPONDYLITIS

DEFINITIONS

Discospondylitis is an infection and inflammation of the intervertebral disc and adjacent vertebral end-plates and vertebral bodies. The cause is most commonly bacterial (usually *Staphylococcus* spp.), but occasionally it is fungal (e.g., *Aspergillus*).

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Discospondylitis is an infection of the intervertebral disc and its contiguous vertebrae, usually by coagulase-positive staphylococci (e.g., *Staphylococcus aureus, Staphylococcus pseudintermedius*). Other bacteria have been reported (e.g., *Streptococcus, Brucella*), as well as fungal organisms (e.g., *Aspergillus terreus, Aspergillus deflectus*). There is evidence to suggest that some dogs that develop discospondylitis may have defective immunocompetence as a predisposing factor. Infectious organisms may gain access to the disc space and vertebrae via a number of proposed mechanisms. Hematogenous spread is thought to be the most common mechanism, even though the primary source of infection is not always found. The urinary tract is generally thought to be the most likely source of bacterial infection. Foreign body migration is another potential mechanism for development of discospondylitis, the best example being plant awn migration. The barbs of plant awns favor migration through tissue. These awns may carry bacteria with them to the disc space and/or serve as a nidus for bacterial localization once they arrive at the disc space. Iatrogenic infection may develop following spinal surgery or paravertebral injection. This is considered to be the least likely mechanism for bacterial localization to the disc space and vertebrae. In one retrospective study of 372 dogs that underwent decompressive surgery for intervertebral dis herniation,

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only eight (2.2%) dogs developed postoperative discospondylitis; risk of developing discospondylitis was significantly associated with body weights over 20 kg.[37] Although *Brucella canis* is an uncommon cause of canine discospondylitis, it has zoonotic potential; therefore the clinician should check for it (e.g., blood cultures, serology).

DIAGNOSIS

Clinical Presentation

Signalment

Discospondylitis is most commonly encountered in medium- to giant-breed male dogs of any age, but it has been reported in small-breed dogs as well as in cats. In one large retrospective study of dogs with discospondylitis,[38] older dogs were overrepresented, as were male dogs and purebred dogs (especially great Danes). The odds of having discospondylitis in this study were highest for dogs over 10 years of age.

History

Most dogs with discospondylitis display progressive clinical signs over at least several weeks, but some dogs develop signs acutely. Clinical signs are often nonspecific but usually include hyperesthesia associated with the spinal lesion(s). Dogs with lumbosacral discospondylitis tend to walk with a stilted pelvic limb gait. Decreased appetite, weight loss, depression, fever, and reluctance to move are other common nonspecific clinical features of this disease.

Physical Examination Findings

Most dogs with discospondylitis demonstrate evidence of hyperesthesia in the regions of the spinal lesions. Many dogs have no neurologic deficits or mild evidence of neurologic dysfunction (e.g., proprioceptive deficits with or without mild paresis). Some patients may be severely paretic or plegic. Dogs with discospondylitis may or may not exhibit systemic signs of disease such as lethargy, fever, and anorexia.

Diagnostic Imaging

Diagnosis of discospondylitis is usually based on characteristic radiographic findings with supportive historical and clinical features. Radiographs usually reveal collapse of the affected disc space(s), bony lysis in the end-plate regions of the affected disc space(s), and a variable component of bony proliferation and end-plate sclerosis (Fig. 43.9). These latter changes are often seen in chronic cases. A separate radiographic appearance, termed *vertebral physitis,* has also been reported, primarily in young dogs (<2 years of age). The majority of vertebral physitis cases have involved the lumbar vertebrae. In this condition, bony lysis seems to originate in the caudal physeal region of the vertebral body rather than the disc/end-plate region. Other clinical features of vertebral physitis appear to be indistinguishable from discospondylitis. Radiographic changes may lag behind clinical signs by as much as 2 to 4 weeks. A patient with normal radiographs and clinical features suggestive of discospondylitis may still have disease. Subtle lesions may also be missed if the radiographs are performed in the awake patient. In some cases, bone scintigraphy, CT, or MRI of suspected lesions may be valuable. Characteristic MRI features of discospondylitis have been described; these include hyperintense disc spaces and paravertebral soft tissues on T2-weighted and short tau inversion recovery images, heterogeneous contrast enhancement of affected disc spaces and end-plate regions, and hypointense affected vertebral bodies on T2-weighted images.[39]



FIG. 43.9 Lateral radiograph of a dog with discospondylitis at L7–S1.

Laboratory Findings

Bloodwork results are often normal, although leukocytosis is occasionally evident on a CBC. Some dogs have evidence of urinary tract infection on urinalysis. Bacteria may be cultured from the blood, urine, and/or affected disc spaces (fluoroscopically-guided needle or surgical aspirate); the reported success rate of such culture attempts varies but is approximately 50%. There is some evidence that culture of material obtained via needle aspiration of infected disc space(s) is more sensitive than culture of urine or blood.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for discospondylitis are primarily disc extrusion/protrusion and spinal neoplasia.

MEDICAL MANAGEMENT

Ideally, medical treatment of discospondylitis is guided by culture and antibiotic sensitivity testing of the offending organism. Because the organism is usually a *Staphylococcus,* first-generation cephalosporins or beta-lactamase–resistant penicillin drugs are often effective. In severely affected (e.g., paralyzed) patients, intravenous antibiotics should be administered for the first 5 to 7 days, after which oral antibiotics can be instituted. Concurrent treatment with antibiotics active against anaerobic bacteria should be considered, especially if there is minimal to no response to therapy within the first week of treatment. Antibiotic therapy has traditionally been recommended for at least several months. Discospondylitis patients usually require treatment well beyond several months. In one large retrospective study on canine discospondylitis,[38] the mean duration of antibiotic treatment was 53.7 weeks, suggesting that traditional recommendations regarding treatment duration may be too conservative. Analgesic drugs (e.g., oral codeine) may also be necessary. We have had success using oral Tylenol 4 with codeine in dogs with very

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painful discospondylitis. Dosing of this drug is based on the codeine (1–2 mg/kg, every 6–8 hours), not the acetaminophen. If alanine aminotransferase or serum bilirubin increases, acetaminophen should be stopped immediately until one can determine if acetaminophen toxicity is taking place. Each Tylenol 4 tablet contains 60 mg of codeine.

NOTE Tylenol 3 contains 30 mg codeine and 300 mg acetaminophen; Tylenol 4 contains 60 mg codeine and 300 mg acetaminophen. Be careful not to overdose the acetaminophen.

Surgical intervention may be warranted in patients with vertebral instability or with compressive lesions identified with CT, MRI, or contrast radiography. Treatment of patients with *Brucella* infections typically entails a combination of tetracyclines and aminoglycosides. *B. canis* infections can be difficult to cure; it is prudent to recheck the patient after medical management. Infections with *A. terreus* and *A. deflectus* can be difficult or impossible to resolve. Itraconazole has been used extensively, but posaconazole and voriconazole probably offer a better chance for success. Combination with terbinafine might increase efficacy of these azoles.

SURGICAL TREATMENT

Although discospondylitis is considered primarily a medical disorder, surgical intervention may be necessary in some cases. In patients with deteriorating neurologic status despite appropriate medical therapy, or in nonambulatory patients, the possibility of a compressive lesion exists. Possibilities include instability resulting from end-plate and disc compromise, extruded/protruded disc in the vertebral canal at the site of infection, and infected material within the vertebral canal causing spinal cord compression. Dogs with radiographic evidence and severe and/or progressive neurologic deficits often have compressive lesions at the radiographic site of discospondylitis, consisting of a combination of pus and infected epidural fat. The infected epidural fat assumes a brown discoloration and compresses the spinal cord. In patients that are deteriorating or that present with severe neurologic dysfunction, myelography, CT, or MRI (preferably) should be performed to investigate for compressive spinal cord lesions. In one MRI study of 23 dogs with discospondylitis, 17 (74%) had evidence of a static compressive spinal cord lesion at the abnormal site; a significant correlation between the percentage of spinal cord compression and degree of neurologic impairment was found in this study.[39]

Preoperative Management

See Preoperative Management for each specific location of the spine affected (i.e., cervical, thoracolumbar, and lumbosacral) on pp. 1366, 1404, and 1428, respectively.

Anesthesia

Suggested anesthetic protocols for animals undergoing spinal surgery are provided on p. 1366.

Surgical Anatomy

Anatomic descriptions for each location of the spine affected (i.e., cervical, thoracolumbar, and lumbosacral) are provided on pp. 1366, 1404, and 1428, respectively.

Positioning

Refer to the surgical procedure chosen for recommendations for appropriate surgical positioning.

SURGICAL TECHNIQUE

See surgical technique descriptions for each specific location of the spine affected (i.e., cervical, thoracolumbar, and lumbosacral) on pp. 1367, 1405, and 1428, respectively.

SUTURE MATERIALS/SPECIAL INSTRUMENTS

Avoid using nonabsorbable multifilament sutures (e.g., silk) in infected surgical sites.

POSTOPERATIVE CARE AND ASSESSMENT

After surgery, these patients should be assessed as described on pp. 1374, 1409, and 1430. Antibiotics should be continued for a minimum of 4 to 6 weeks.

PROGNOSIS

Prognosis is generally favorable with bacterial discospondylitis, particularly in cases with no or mild neurologic deficits. Prognosis is more guarded in dogs with resistant bacteria and in dogs with severe neurologic deficits. Obvious clinical improvement is generally expected within the first week of antibiotic therapy. Follow-up radiographs of affected disc spaces every 1 to 2 months are recommended to monitor the progress of disease. Fungal (e.g., *Aspergillus* spp.) discospondylitis is associated with a poor prognosis because the infection is usually disseminated at the time of diagnosis.

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44

Peripheral Nervous System Disorders and Diagnostic Techniques



An exhaustive discussion of the peripheral nervous system (PNS) abnormalities that affect dogs and cats is beyond the scope of this textbook and of little clinical use to the neurosurgeon. Much more important than distinguishing what specific disorder of the PNS is affecting a particular patient is the ability to distinguish that the patient has a PNS disorder rather than a myelopathy. Individually, most disorders of the PNS are not encountered frequently. However, as a group, surgeons deal with these diseases on a regular basis because they are often confused with either myelopathies or musculoskeletal disorders. The specific disorders covered in this chapter include acquired myasthenia gravis (MG), autoimmune polymyositis, polyradiculoneuritis, and tick paralysis.

MUSCLE AND NERVE BIOPSY

Position the patient in lateral recumbency and aseptically prepare the pelvic limb as you would for stifle surgery (see p. 37). Prior to making the skin incision, palpate for the peroneal nerve so that the nerve will be in the region of your surgical approach. Make a vertical skin incision caudal to the patellar ligament, from a few centimeters proximal to the level of the patella to a few centimeters distal to the tibial tuberosity. Make the incision approximately 2 cm caudal to the level used to access the stifle joint (see p. 1234). Use a blade or Metzenbaum scissors to expose the biceps femoris muscle and fascia. Pull up a portion of this muscle with DeBakey forceps and incise the muscle full thickness along its longitudinal striations using Metzenbaum scissors. Continue the incision until a digit can be placed beneath the muscle to palpate the underlying peroneal nerve. Using either a No. 11 blade or Metzenbaum scissors, remove a rectangular section of the biceps femoris muscle (at least 2 cm in length). Identify the underlying peroneal nerve and carefully free a segment of the nerve from surrounding fascia. Retract the edges of the incised biceps femoris muscle with Gelpi retractors to facilitate exposure of the common peroneal nerve (Fig. 44.1). Place a stay suture in the edge of the nerve at the proximal site of the planned biopsy, using 6-0 polydioxanone. While applying gentle tension to this suture, incise (with a new No. 11 blade) the nerve medial to the stay suture knot to begin the nerve biopsy. Do not remove more than one-third of the width of the nerve at the biopsy site. Using either the No. 11 blade or fine tenotomy scissors, remove a longitudinal strip of the peroneal nerve (1–3 cm, depending on patient size) for biopsy. If a distal neuropathy is suspected, procure a second biopsy specimen from the cranialis tibialis muscle. Close the site routinely.

SPECIFIC DISEASES

ACQUIRED MYASTHENIA GRAVIS

DEFINITIONS

Acquired MG is an autoimmune disorder of the neuromuscular junction in which autoantibodies are formed against nicotinic acetylcholine (ACh) receptors of skeletal muscle. The classic clinical manifestation of the autoantibody attack is skeletal muscle weakness, which worsens on repeated exertion.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

Acquired MG is a common autoimmune neuromuscular disorder of dogs that is rarely diagnosed in cats. Autoantibodies produced against the nicotinic ACh receptors of skeletal muscles impair neuromuscular transmission and produce weakness that worsens with exertion. Megaesophagus is a frequent feature of acquired MG in dogs because of the high proportion of skeletal muscle in the canine esophagus; but it is less common in affected cats. Several clinical forms of acquired MG have been identified, including focal, generalized, and acute fulminating. The thymus is believed to play an integral role in the initiation of the autoimmune response to ACh receptors, but the factors responsible for triggering the autoimmune response of acquired MG are still unknown.

DIAGNOSIS

Clinical Presentation

Signalment

There is a bimodal age distribution for acquired MG in both dogs and cats, with peak incidences at approximately 3 and 10 years of age. Both males and females are affected, although there may be a slight decreased tendency for sexually intact dogs to develop MG. Breeds most commonly afflicted with MG include German shepherds, golden retrievers, and Labrador retrievers. Other breeds with a relatively high risk for MG include Akitas, terriers, and German shorthaired pointers. Somali cats (related to Abyssinians) and Abyssinian cats are at risk for developing acquired MG.

History

Common historical complaints include difficulty swallowing, regurgitation (often described as vomiting by owners), ptyalism,

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ABSTRACT

Dogs and cats with selected disorders of the peripheral nervous system (PNS) are sometimes perplexing, appearing similar to patients with spinal cord dysfunction. It is important that surgeons have a thorough understanding of salient features of the more commonly encountered PNS disorders, as well as having an understanding of pertinent diagnostic tests and treatment options. Performing a muscle or nerve biopsy may be necessary in some animals to assist in diagnosing PNS disorders.

KEYWORDS

Peripheral nervous system
muscle/nerve biopsy
acquired myasthenia gravis
myositis
polyradiculoneuritis

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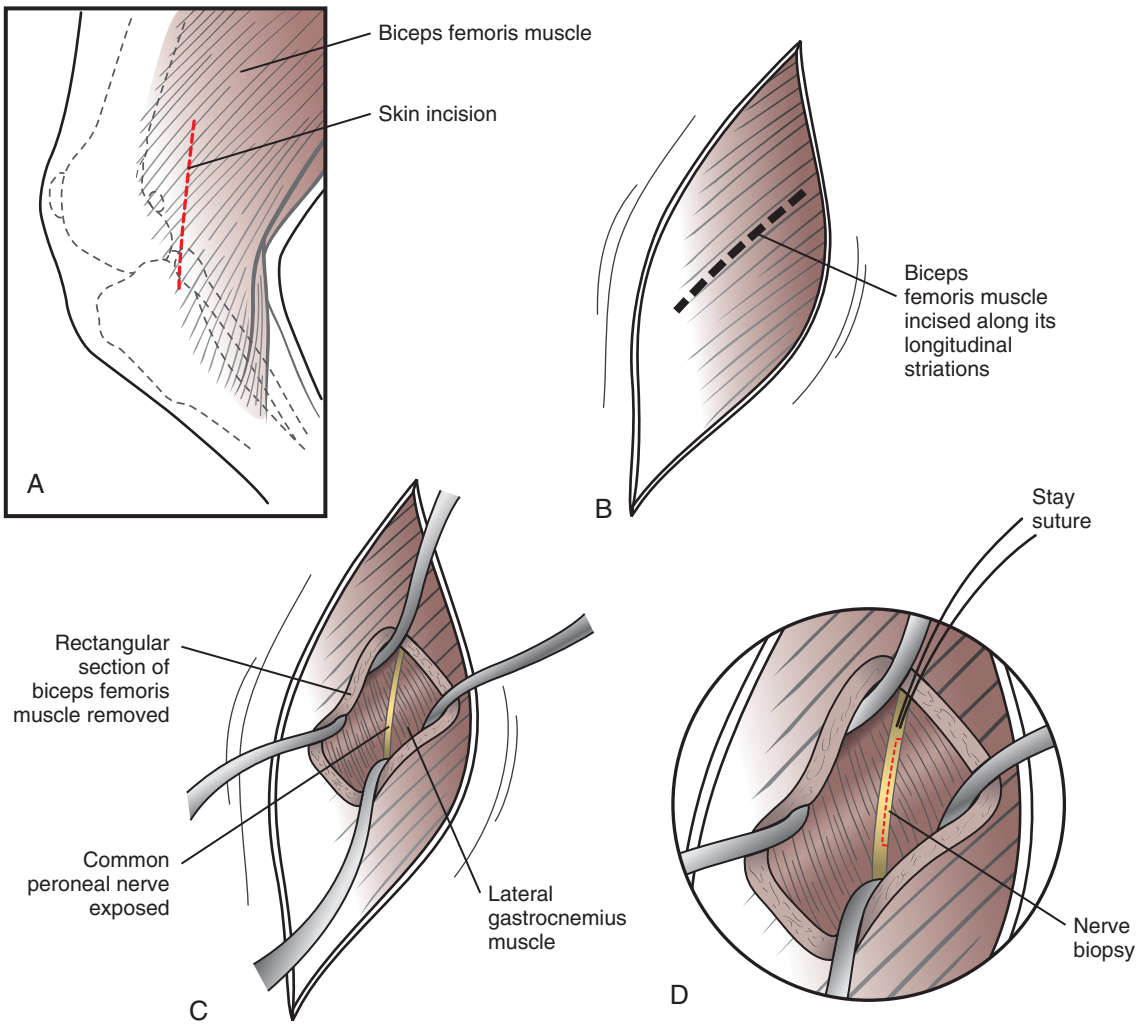


FIG. 44.1 Schematic illustration of the approach for a peroneal nerve biopsy. A section of biceps femoris muscle has already been removed for biopsy.

coughing, labored breathing, and limb muscle weakness (especially the rear legs). The specific constellation of clinical signs and time course of development reported by the owner varies with the clinical form of MG.

Physical Examination Findings

In addition to the historical complaints listed here, the nature of appendicular (limb) muscle weakness is variable in both distribution and severity. The clinical features of the different forms of acquired MG are provided in Box 44.1. Dogs and cats with MG typically do not have specific neurologic deficits but display varying levels of weakness. It is common for pelvic limb weakness to be the main (sometimes the only) clinical manifestation in dogs with MG; this feature often leads to misdiagnosis of MG as either a myelopathy or pelvic limb musculoskeletal disorder.

Diagnostic Imaging

Thoracic radiographs are recommended to look for megaesophagus, aspiration pneumonia, and mediastinal masses (i.e., thymoma). Mediastinal masses (typically thymoma) are much more common in cats with acquired MG than in myasthenic

BOX 44.1 Clinical Forms of Acquired Myasthenia Gravis in Dogs and Cats

Focal MG: No Clinical Signs of Appendicular (Limb) Muscle Weakness

Can present as megaesophagus, pharyngeal weakness, laryngeal weakness, and/or facial muscle weakness: each of these may be the sole presentation or may occur in any combination

Generalized MG: Clinical Signs of Appendicular Weakness

Typically see most (or all) of weakness in pelvic limbs, but thoracic limbs can be involved

Acute Fulminating MG: Rapid Onset and Progression of Severe Weakness

Profound focal and appendicular weakness
Acute onset, rapid progression to nonambulatory tetraparesis and respiratory distress
Usually fatal

MG, Myasthenia gravis.

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dogs; in one retrospective study of 235 cats with acquired MG, 52% had evidence of a mediastinal mass (vs. 3.4% in dogs).[1]

Laboratory Findings

Bloodwork (complete blood cell count [CBC], serum biochemistry) is usually normal or may reflect a septic process in the face of pneumonia. The most definitive blood test for the diagnosis of acquired MG is an immunoprecipitation radioimmunoassay that quantitates serum autoantibodies directed against the ACh receptor. Concentrations of ACh receptor antibody above 0.6 nM/L and 0.3 nM/L are diagnostic for MG in dogs and cats, respectively. The edrophonium (Tensilon) challenge test may provide a presumptive diagnosis of MG in patients while awaiting results of an radioimmunoassay if it is clearly positive (i.e., the patient displays a clear improvement in strength shortly after administration of IV edrophonium). However, some dogs with various myopathies will also display increased strength after receiving edrophonium. Although the association between MG and thyroid disease is tenuous, thyroid status should be evaluated in MG patients.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include other PNS disorders (e.g., myopathies, neuropathies), musculoskeletal disease, and metabolic disorders (e.g., hypoglycemia, hypoadrenocorticism).

MEDICAL MANAGEMENT

Medical management of acquired MG in dogs and cats is typically either oral pyridostigmine or oral pyridostigmine plus immunosuppressive therapy (see Box 30.14). Treatment options for canine and feline MG are summarized in Box 44.2. Prednisolone often causes an initial worsening of weakness in dogs with MG; this adverse effect is potentially severe enough to justify avoiding prednisolone as an immunosuppressive agent for canine MG. A similar adverse effect of prednisolone treatment has not been documented for cats with acquired MG.

BOX 44.2 Treatment Options for Canine and Feline Acquired Myasthenia Gravis

Anticholinesterase Drugs
Pyridostigmine (preferred anticholinesterase drug)
Neostigmine (only if pyridostigmine is not available)

Immunosuppressive Drugs
Azathioprine (dogs)
Cyclosporine
Mycophenolate mofetil (dogs)

Thymectomy
Thymic hyperplasia (not evaluated in dogs and cats)
Thymoma (not until MG signs controlled)

Hormonal Therapy
Thyroxine if dog is hypothyroid

Nutritional Support
Elevated feedings
Gastrostomy tube feedings

MG, Myasthenia gravis.

SURGICAL TREATMENT

Acquired MG is not a surgical disorder. Dogs and cats (less likely) that frequently regurgitate may require a stomach tube to be placed either surgically (large dogs) or endoscopically (small dogs and cats) (see pp. 97 and 98). In cases of acquired MG with associated thymoma, the thymoma may need to be removed if it encroaches on neighboring anatomic structures. It is best to have clinical signs of MG stabilized prior to attempting thymoma removal.

PROGNOSIS

The prognosis for acquired MG is guarded overall. Many dogs will experience a spontaneous remission. In our experience, these tend to be younger patients. Spontaneous remission appears to be uncommon in cats with acquired MG.[1] Patients with persistent or recurrent pharyngeal/esophageal dysfunction have a poor prognosis because of the constant risk of aspiration pneumonia. The acute fulminating form of MG in dogs and cats is usually fatal.

AUTOIMMUNE POLYMYOSITIS

DEFINITIONS

Autoimmune polymyositis is an autoimmune inflammatory disease of unknown pathogenesis that primarily affects appendicular musculature.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

This is an idiopathic autoimmune disorder. There is usually no identifiable cause for the autoimmune response, but systemic lupus erythematosus, use of trimethoprim-sulfa drugs in Doberman pinschers, and thymomas (usually in conjunction with acquired MG) (see p. 951) have all been associated with the development of this condition.

DIAGNOSIS

Clinical Presentation

Signalment

Although any age or breed dog can be affected, most are middle-aged, large-breed dogs of either sex. Newfoundlands and boxers are overrepresented. Newfoundlands tend to develop the disease at a younger age than other breeds. A substantial number of boxers with polymyositis may develop the disorder as a preneoplastic condition.

History

Clinical signs can be acute or chronic. Historical complaints include generalized weakness (often worsened by exercise), stiff gait, generalized muscle atrophy, dysphonia, myalgia, regurgitation (megaesophagus may be present), fever, and muscle swelling.

Physical Examination Findings

Physical examination findings concur with historical complaints and are generally consistent with a generalized myopathic disorder. Some dogs with this disorder will have concurrent masticatory

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myositis; this combination of autoimmune myopathies is referred to as overlap syndrome.

Diagnostic Imaging

Thoracic radiographs should be obtained to investigate for the presence/absence of megaesophagus. Abdominal ultrasonography may also be indicated if a paraneoplastic myopathy is suspected (especially in boxers).

Laboratory Findings

Basic bloodwork (CBC, serum biochemistry) is typically normal. Creatine kinase levels are typically elevated. Electromyographic examination typically reveals abnormalities, and muscle biopsy demonstrates myofiber necrosis, phagocytosis, and regeneration, with a nonsuppurative inflammatory infiltrate. Immunoglobulin localization to the sarcolemma may also be demonstrable immunohistochemically.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include infectious polymyositis (e.g., toxoplasmosis, neosporosis), preneoplastic/paraneoplastic myositis, and acquired MG.

MEDICAL MANAGEMENT

Treatment consists of oral prednisone therapy at immunosuppressive doses (e.g., 1–2 mg/kg, q12h) until clinical remission is achieved. If prednisone is ineffective or if it cannot be tapered because of disease recurrence, other immunosuppressive drugs (e.g., azathioprine, cyclosporine, mycophenolate mofetil) can be used.

SURGICAL TREATMENT

This is not a surgical disorder, but muscle biopsy is necessary for a definitive diagnosis.

PROGNOSIS

The prognosis is favorable in approximately 80% of cases. Relapses may occur with tapering or discontinuation of immunosuppressive drugs.

ACUTE IDIOPATHIC POLYRADICULONEURITIS

DEFINITIONS

Acute idiopathic polyradiculoneuritis is an idiopathic inflammatory disorder primarily involving both axons and myelin of ventral nerve roots; it occurs in dogs, and it is probably one of the most common polyneuropathies in this species. This disorder is similar to Guillain-Barré syndrome of people. It has been referred to also as **coonhound paralysis**.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

There is evidence that axonal loss is more prominent than demyelination in most dogs with polyradiculoneuritis. Demyelination is thought to be most severe in the ventral nerve roots in dogs with minimal myelin loss in the major nerve trunks. An autoimmune process is suspected, but the pathogenesis is uncertain. The term *coonhound paralysis* refers to those dogs with a history of being bitten or scratched by a raccoon shortly before developing clinical signs of disease. The term *idiopathic polyradiculoneuritis* refers to patients with an identical clinical disorder but with no possible exposure to raccoons. These two subcategories probably reflect the same disease syndrome, with the trigger for the inflammatory process being as yet unidentified in the latter subcategory. The typical clinical scenario for acute idiopathic polyradiculoneuritis describes a rapidly developing lower motor neuron (LMN) paresis/plegia, usually beginning in the pelvic limbs, and eventually involving the thoracic limbs.

DIAGNOSIS

Clinical Presentation

Signalment

There is a wide age range and no apparent sex predilection for this disorder. Certain breeds of coonhounds (e.g., Walker, black and tan, blue tick) are thought to be predisposed to this disease. However, one 2010 retrospective study found that the most common breed affected by this disorder is the Labrador retriever.[2]

History

The typical clinical scenario for acute idiopathic polyradiculoneuritis describes a rapidly developing LMN paresis/plegia, usually beginning in the pelvic limbs and eventually involving the thoracic limbs. Most affected animals will progress to being either nonambulatory tetraparetic or tetraplegic within 10 days of initial onset of clinical signs. It is fairly common for this stage of dysfunction to be reached within a 72-hour period of time. Development of life-threatening respiratory paralysis is a concern, especially in the more rapidly developing cases. Loss of voice (dysphonia, aphonia) is common, and some patients will also exhibit facial weakness.

Physical Examination Findings

In addition to the historical features described earlier, spinal reflexes are typically absent (with the exception of the perineal reflex, which is normal), muscles are hypotonic, and neurogenic atrophy develops quickly in recumbent patients. Proprioceptive placing reactions will be normal in those animals that still have enough motor ability to perform the efferent limb of these tests. These patients retain the ability to urinate and defecate, and they will readily eat and drink if the head is supported. Pain sensation also remains intact. In fact, these animals often seem hyperesthetic on limb manipulation, which may reflect the inflammatory nature of the disease.

Diagnostic Imaging

Thoracic radiographs are obtained to evaluate for atelectasis, aspiration pneumonia, the presence of megaesophagus, or metastatic lesions (i.e., paraneoplastic neuropathy).

Laboratory Findings

Bloodwork (CBC, serum biochemistry) is typically normal. Creatine kinase is usually normal but may be slightly elevated because of recumbency. Cerebrospinal fluid examination may demonstrate increased protein levels. Many, if not most, of these dogs will exhibit abnormal electromyographic activity with normal motor nerve conduction velocities. The patients often do not have enough functional axons to ambulate, but the remaining axons have essentially normal myelination.

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DIFFERENTIAL DIAGNOSIS

Differential diagnoses include paraneoplastic polyneuropathy, tick paralysis, acquired MG, protozoan polyradiculoneuritis/polymyositis, and botulism (rare).

MEDICAL MANAGEMENT

There is no known effective medical therapy for this disorder. In one study evaluating treatment of dogs with acute idiopathic polyradiculoneuritis using human intravenous immunoglobulin, the median time to unassisted ambulation of intravenous immunoglobulin–treated dogs was 27.5 days compared with 75.5 days for untreated historical controls; however, this difference was not found to be statistically significant.[3]

SURGICAL TREATMENT

This is not a surgical disorder; however, muscle/nerve biopsy may be part of the diagnostic approach.

PROGNOSIS

The prognosis for full recovery is often favorable but can be prolonged, usually taking several weeks to months. Some patients develop life-threatening respiratory paresis/paralysis in the acute phase of the disease (usually those dogs whose signs progress rapidly over 72 hours) and may need to be mechanically ventilated. Nursing care, physical therapy, and proper nutrition are essential for recovery. In one retrospective study of 58 dogs with acute idiopathic polyradiculoneuritis, 88% (51 of 58 dogs) survived the disease.[2]

TICK PARALYSIS

DEFINITIONS

Tick paralysis is an acute-onset, rapidly progressive LMN paralysis caused by a salivary neurotoxin produced by certain species of ticks. The neurotoxin interferes with neuromuscular transmission and has a neuropathic effect as well.

GENERAL CONSIDERATIONS AND CLINICALLY RELEVANT PATHOPHYSIOLOGY

In North America, tick paralysis is caused by *Dermacentor* species and affects only dogs. In Australia, a more severe form of tick paralysis affects dogs and cats; it is caused by a salivary neurotoxin carried by *Ixodes* ticks. The incidence of tick paralysis is highest in the summer months. After entering the circulation, the neurotoxin reaches the neuromuscular junctions and interferes with ACh release; the toxin is also believed to affect nerve transmission. A single tick can cause clinical signs, and the development of clinical dysfunction occurs approximately 5 to 9 days after tick infestation.

DIAGNOSIS

Clinical Presentation

Signalment

Any age, breed, and either sex of dog can be affected by tick paralysis.

History

The typical history is that of acute onset and rapidly progressive (24–72 hours) flaccid tetraplegia starting in the pelvic limbs.

Physical Examination Findings

Dogs with tick paralysis have a LMN paresis/paralysis of all four limbs. Spinal reflexes are depressed to absent, cranial nerves are usually normal (mild facial nerve paresis may occur), and pain sensation is normal; there is no evidence of hyperesthesia, and urinary/fecal continence is preserved. Dogs usually maintain a tail wag.

Diagnostic Imaging

Thoracic radiographs are typically normal. Megaesophagus may occur with the Australian version of tick paralysis caused by *Ixodes holocyclus*.

Laboratory Findings

Bloodwork (CBC, serum biochemistry) is typically normal.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include acute idiopathic polyradiculoneuritis, acute fulminating MG, and botulism (rare).

MEDICAL MANAGEMENT

Medical management of tick paralysis is removal of causative ticks. This may require shaving the dog to identify all the ticks and may also require the administration of an acaricide to ensure all ticks are dead. The tick sometimes "hides" in the ear canal, so it is important to check both ear canals thoroughly.

SURGICAL TREATMENT

This is not a surgical disorder.

PROGNOSIS

In North America, tick removal is generally curative, with recovery occurring within 1 to 3 days of removal. Tick paralysis caused by the Australian ixodid tick *I. holocyclus* is often fatal, even after tick removal.

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